SPORTS NUTRITION

Enhancing Athletic Performance

Edited by

BILL I. CAMPBELL

CRC Press
Taylor & Francis Group

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CRC Press
Taylor & Francis Group
Boca Raton London New York

CRC Press is an imprint of the
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CRC Press
Taylor & Francis Group
6000 Broken Sound Parkway NW, Suite 300
Boca Raton, FL 33487-2742

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Version Date: 20130531

International Standard Book Number-13: 978-1-4665-1359-4 (eBook - PDF)

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Dedication

I would like to dedicate this book to my wife, Joanne. You are the best blessing that God has given me. I love everything about you and love spending my life with you.

Contents

Foreword ix
Preface xi
About the Author xiii
Contributors xv

Chapter 1 Fat Metabolism 1
Sam Greeley and Bill Campbell

Chapter 2 Dietary Fat Intake Strategies to Enhance Performance 21
Mike Roberts, Joe Company, and Bill Campbell

Chapter 3 Carbohydrate Metabolism 45
Bill Campbell

Chapter 4 Dietary Carbohydrate Strategies for Performance Enhancement 75
Bill Campbell

Chapter 5 Protein Metabolism 125
Bill Campbell

Chapter 6 Dietary Protein Strategies for Performance Enhancement 155
Bill Campbell

Chapter 7 Nutrient Timing: Carbohydrate–Protein Combinations 181
Bill Campbell

Chapter 8 Energy Balance 199
Bill Campbell

Chapter 9 Enhancing Body Composition: Gaining Muscle and Losing Fat 239
Bill Campbell, Paul La Bounty, and Elfego Galvan

Chapter 10 Hydration and Performance 269
Jennifer Bunn

Appendix A: Overview of Bioenergetics 297
Bill Campbell

Foreword

The relationship between exercise training, athletic performance, fitness, and both acute and chronic nutrition has become one of the most important and fascinating areas of scientific study, research, and professional application. With so many types of training and diet philosophies and desired performance and physique, it remains difficult for many professionals to educate and provide effective guidance to maximize training benefits and optimize performance outcomes. Dr. Bill Campbell has proven uniquely qualified to effectively deliver both basic and advanced concepts in a user-friendly fashion for educators as well as translate complex research findings to practical application for sport nutritionists, personal trainers, strength and conditioning coaches, and other professionals. Each chapter of *Sports Nutrition: Enhancing Athletic Performance* is formatted masterfully, transitioning concept introductions to problem and solution overviews and practical applications.

Sports Nutrition: Enhancing Athletic Performance opens with a half dozen chapters addressing macronutrients and how they are processed in the body at rest during exercise. They examine how acute exercise and chronic training-related adaptations can influence the metabolism and destinations for carbohydrates, proteins, and fat. From there, the book overviews concepts related to nutrient timing—providing insight to more absolute applications vs. questionable, overstated, or underresearched concepts. Special attention is paid to the relationships between exercise on muscle protein synthesis (MPS), muscle protein breakdown (MPB), the influence of exercise and nutrient type, total and timing, and net protein balance.

The last three chapters of the book are dedicated to three critically important sport nutrition concepts that are often overshadowed or are treated as secondary considerations among athletes despite their having among the largest influences on acute performance, maximization of training adaptations, and long term performance and fitness improvement. Energy balance and understanding how to accurately assess both an individual's energy requirements for weight management as well as a sport and individualized macronutrient targets remain confusing and complex. The book again delivers with an easy to understand overview while highlighting both strengths and weaknesses in our current scientific understanding. Energy balance naturally transitions to the next chapter on body composition and how practitioners can understand and apply different tools in their practice. *Sports Nutrition: Enhancing Athletic Performance* closes with a chapter on hydration emphasizing the fundamental role of hydration in maximizing performance and easy to apply guidelines for successful hydration strategies.

It is an honor to be among the first to review this book. Dr. Campbell has clearly proven that he has the ability to translate his scholarship as a forefront university researcher and educator to a systematic, comprehensive, and practical resource for those who have not or will not be able to experience his skills and acumen in person. This book is an important resource and will remain a seminal work for years to come.

Robert Wildman
PhD, RD, FISSN

Preface

It is an exciting time in the field of sports nutrition. There is an ever-increasing flow of information emanating from scholarly journals pertaining to the many facets of sports nutrition. One of the problems that arises because of the extensive amount of scientific data available is that it is nearly impossible to stay on top of the information and to determine what is relevant. This book seeks to solve these problems. With over 1,000 references taken from the world's top academic journals, the reader will obtain the knowledge necessary to enhance exercise and sports performance. Many other sports nutrition books cover a lot of information ranging from improving performance and health to addressing the needs of various special populations (i.e., vegetarian athletes, athletes with diabetes, and athletes with eating disorders). This book is different. There is a laser-sharp focus on providing scientifically-based sports nutrition advice to maximize performance. In addition, content is devoted to exercise metabolism, which sets the stage for how certain nutrients exert their physiologic effects and ultimately lead to an improvement of athletic potential.

Starting in Chapter 1, the first nutrient that is discussed is fat. Numerous aspects of fat metabolism are discussed, including differences between fat metabolism at rest versus high-intensity exercise. In addition, evidence is reported not only that resistance exercise utilizes fat as an energy source during the exercise bout, but also that fat oxidation (the breakdown of fat) continues to be elevated for about 45 minutes in the postworkout period. In the chapter that follows, several dietary fat intake strategies are discussed that may enhance performance, including fat loading and the use of fatty acid-containing supplements. Carbohydrate metabolism is discussed next. Topics such as skeletal muscle glycogen depletion, lactate formation, and glycolysis are presented. Following this, several dietary carbohydrate strategies are discussed with the potential for improving both endurance- and resistance-exercise performance. Daily carbohydrate intake recommendations, low-carbohydrate diets, and methods for rapidly resynthesizing muscle glycogen are just a few of the topics presented in this chapter that are of interest to anyone looking for a performance advantage. The last macronutrient that is discussed is protein. Some of the questions that are posed in these sections include:

- Why are some types of protein considered high quality and others low quality?
- What are the best and worst sources of dietary protein?
- How much protein should be ingested at each meal?
- How many times per day should protein be ingested?
- What role does leucine have in relation to maximizing muscle protein synthesis?

Answers are provided to each of these questions and the conclusions that are made are based upon the latest scientific findings. In this sense, the reader can feel confident in his or her adoption of the recommendations made relative to protein

intake because the scientific foundation for the recommendations is clearly laid out and articulated.

Some of the most interesting information in the book is located in Chapter 9, "Enhancing Body Composition: Gaining Muscle and Losing Fat." This chapter lays out a scientifically-based strategy for losing body fat while making sure to maintain precious lean muscle mass. In addition, four principles are set forth that are integral for the athlete in the quest to optimize body composition. If these principles are not followed rigorously, body composition goals will be compromised.

This book addresses the needs of athletes who desire to improve their performance. The information presents not only performance nutrition principles but also the exercise biochemistry involved in the process of adaptation. After reading this book, the athlete and his or her support staff will no longer have any excuses for not knowing what to ingest, at what times, and in what amounts to improve performance and recover from training.

About the Author

Bill I. Campbell is an associate professor and director of the Exercise and Performance Nutrition Laboratory at the University of South Florida, a research laboratory dedicated to innovation in sports nutrition and metabolism research. As a researcher and author, Dr. Campbell has published over 100 scientific abstracts and papers related to sports nutrition and enhancement of sports performance. In addition, he has published more than 50 articles for health and fitness magazines (print and electronic media). He is a paid consultant to professional sports team organizations and sports entertainment corporations and has lectured on various topics related to sports nutrition and exercise performance to audiences all over the world.

Dr. Campbell is a member of the National Strength & Conditioning Association (NSCA) and the American College of Sports Medicine (ACSM), and he has been awarded "fellow" status with the International Society of Sports Nutrition (ISSN). He received his PhD in exercise, nutrition, and preventive health from Baylor University in 2007. During that same year, he also received the outstanding doctoral student award for research and teaching. In 2009, he received the outstanding undergraduate teaching award from the University of South Florida.

Contributors

Jennifer Bunn, PhD, is an assistant professor in the Department of Exercise Science at Campbell University in Buies Creek, North Carolina. At Campbell, she teaches courses in exercise physiology and sports nutrition and has worked with several of the athletic teams on campus to improve their nutrition. Her research interests include gender differences in aerobic and anaerobic training adaptations, behavior modification to improve healthy lifestyles, and ergogenic aids.

Joseph Company, PhD, obtained his BA in mathematics education from Goshen College, Goshen, Indiana. He taught mathematics for 6 years before returning to school to complete a master's degree in exercise physiology at the University of Missouri. His master's work involved validating body composition measurements in adult strength and endurance athletes. He continued his graduate work in the doctorate program of biomedical sciences at the University of Missouri, where he studied mechanisms of adipose tissue growth during and after a sudden decrease in physical activity. He obtained his PhD in 2013 and is affiliated with the University of Missouri.

Elfego Galvan is currently pursuing a doctoral degree in exercise physiology at Texas A&M University in the Exercise and Sport Nutrition Laboratory. He is a registered dietitian with bachelor's and master's degrees in nutrition sciences from Baylor University and the State University of New York at Buffalo, respectively. His research interests include nutrition and exercise interventions and the influence they have on sport performance and muscle metabolism in young, aging, and diseased populations. He is affiliated with the Department of Health and Kinesiology at Texas A&M University.

Sam Greeley obtained an MS in exercise science at the University of South Florida in 2012. While obtaining his degree, Sam also served as a teaching and research assistant. His master's work involved the investigation of the impact of continuous and discontinuous cycle exercise on affect. Currently, Mr. Greeley is the head soccer coach at Clearwater Christian College and is affiliated with the University of South Florida.

Paul La Bounty, PhD, MPT, CSCS, is an associate professor of human anatomy, physiology, and nutrition at Baylor University in Waco, Texas. Dr. La Bounty's research is focused primarily in the field of nutrition as it relates to exercise performance and body composition. He has authored and coauthored numerous peer-reviewed articles and abstracts in the areas of sport supplements, nutrition, and weight loss. Additionally, he has authored many consumer articles pertaining to performance nutrition and strength and conditioning for mixed martial artists and competitive grapplers.

Mike Roberts, PhD, obtained his BS degree from Baylor University in 2003 and continued to earn his master's degree there in the Exercise and Sports Nutrition Laboratory with Dr. Richard Kreider. Dr. Roberts then went on to the University of Oklahoma to obtain his PhD in 2010 and, afterward, began a 3-year postdoctoral fellowship under Dr. Frank Booth at the University of Missouri, where he performed basic cell and animal science research techniques. He is currently affiliated with the Department of Kinesiology at Auburn University. Dr. Roberts has published 45 peer-reviewed articles in journals such as the *Journal of Applied Physiology, American Journal of Physiology—Regulatory, Integrative, and Comparative Physiology, Sports Medicine, Experimental Physiology, Exercise and Sports Sciences Reviews, Medicine & Science in Sports & Exercise, Nutrition, Metabolism,* and *Journal of the International Society of Sports Nutrition*. His current research interests include (1) various physiological effects of different whey protein forms, (2) brain mechanisms that modulate exercise motivation, (3) mechanisms that regulate muscle hypertrophy, and (4) mechanisms that occur in response to physical inactivity.

1 Fat Metabolism

Sam Greeley and Bill Campbell

CONTENTS

1.1 Introduction 1
1.2 Triglycerides and Fatty Acids 2
1.2.1 Saturated Versus Unsaturated Fatty Acids 3
1.2.2 Location of Double Bonds in Fatty Acids 3
1.2.3 Fatty Acid Chain Length 5
1.3 Fat Digestion and Absorption 5
1.4 Fat Metabolism 6
1.4.1 Lipolysis 7
1.4.1.1 Transport of Fatty Acids from Adipose Tissue to Skeletal Muscle 9
1.4.2 Beta-Oxidation 9
1.5 Fat Metabolism at Rest and during Exercise 11
1.5.1 Rest 11
1.5.2 Low- and Moderate-Intensity Aerobic Exercise 12
1.5.3 High-Intensity Exercise 12
1.5.4 Exercise Duration 14
1.5.5 Endurance Training Adaptations 15
1.5.6 Resistance Exercise 15
1.6 Conclusion 16
Acknowledgment 16
References 16

1.1 INTRODUCTION

A number of different chemical compounds are found in food and within the body that are classified as fats (or lipids). Apart from water, fat is the most abundant substance in the body. From a physiological perspective, the most important fats fall into three main categories based on their chemical structure: triglycerides (also referred to as triacylglycerides), phospholipids, and sterols. Triglycerides make up the majority of fats found within the body and in foods. Phospholipids (consisting of a glycerol backbone, two fatty acids, and a phosphate group) are found in both plants and animals and constitute the cell membranes of various tissues found throughout the body. Sterols are very different when compared to triglycerides and phospholipids in their structure and function. Sterols possess carbon rings in their structure rather than carbon chains. Cholesterol is the most commonly known sterol. Because triglycerides

TABLE 1.1
Fat Storage Sites and Potential Energy in the Human Body

Fat Storage Location	Energy—kcal (kJ)*
Adipose tissue	122,850 (513,500)
Intramuscular triacylglycerides	3,070 (12,830)
Plasma triacylglycerides	45 (188)
Plasma fatty acids	4.5 (19)

make up the overwhelming majority of fats found in the diet and in the body, the terms triglycerides and fats will be used interchangeably throughout this chapter and text. Until recently, fat has been undervalued in its contributions to health and athletic performance. Most adults have over 100,000 kcal of energy in stored fat, which is approximately 50 times more than that available from carbohydrates stored in the liver and skeletal muscle. A person with a body mass of 200 pounds (91 kg) and 15% body fat has 30 pounds (13.6 kg) of fat. The vast majority of these energy reserves are stored as triglycerides in subcutaneous adipose tissue (~97.5%) while some exist in the form of intramuscular triacylglycerides (~2.4%). There is also a negligible amount of fat present as plasma triglycerides and plasma free fatty acids. Availability of substrate energy from these fat stores is summarized in Table 1.1.

Fats serve as the primary energy source at rest and during low- to moderate-intensity exercise. In addition to its role as a fuel source, fat also exerts pharmaceutical-like effects via its incorporation into cell membranes where it can affect biochemical processes and the physical nature of the cell (Lowery 2011). Dietary fat also provides essential fatty acids (EFAs) that the body is unable to synthesize. EFAs are needed to prevent deficiencies in many bodily systems. This chapter will discuss the various types of fat, how it is metabolized, and the effects that exercise training has on fat metabolism.

1.2 TRIGLYCERIDES AND FATTY ACIDS

Fat is made up of three elements: carbon, hydrogen, and oxygen. These atoms can be arranged in a myriad of ways to produce all types of fat. Triglycerides, also referred to as triacylglycerols or triacylglycerides, are the main component of human fat and provide energy in the form of fatty acids. Each triglyceride contains three fatty acid chains connected by ester bonds to a glycerol backbone (Figure 1.1). A fatty acid chain has a carboxyl group (COOH) on one end and a methyl group (CH_3) on the other end (Figure 1.2). Fatty acids can differ in several ways:

- Number of carbon–carbon double bonds
- Placement of the carbon–carbon double bonds
- Chain length

Each of these differences influences not only the effects that the fatty acids have on the body and its physiological systems, but also how they are metabolized. The following sections discuss these differences in more detail.

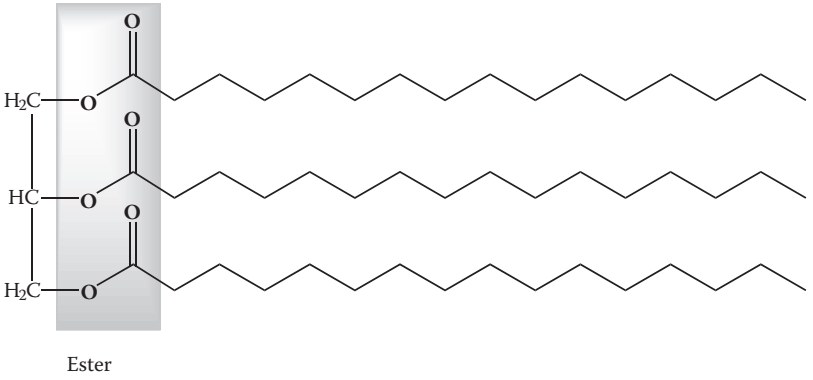

FIGURE 1.1 Triglyceride molecule.

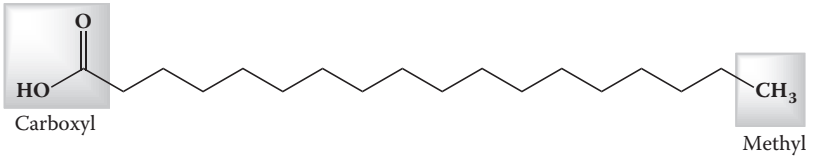

FIGURE 1.2 Fatty acid structure.

1.2.1 Saturated Versus Unsaturated Fatty Acids

There are two major categories of fat: saturated and unsaturated. The *degree of saturation* refers to the number of carbon–carbon double bonds that a fatty acid contains. If a fatty acid chain has no carbon–carbon double bonds, it is designated as saturated because it binds to the maximum number of hydrogen atoms possible (it is said to be saturated with hydrogen). Unsaturated fatty acids contain at least one double bond in their carbon chain. Unsaturated fats can be further categorized into monounsaturated (one double bond) and polyunsaturated (two or more double bonds). Figure 1.3 demonstrates the differences between saturated, monounsaturated, and polyunsaturated fatty acids.

1.2.2 Location of Double Bonds in Fatty Acids

A second difference in the makeup of fatty acids concerns the placements of the double bonds. Double bonds can be designated by counting from either end of the fatty acid chain. The carboxyl group is referred to as the alpha end and the methyl group makes up the omega end of the fatty acid. Paying attention to the different ends of a fatty acid chain is important because it provides for a consistent way of classifying fatty acids based on chain length and the number and location of single and double bonds. When naming omega fatty acids, the double bond that occurs closest to the omega end (methyl end) identifies the omega classification. There are omega-3, -6, and -9 classifications that signify the first double bond location from the omega end

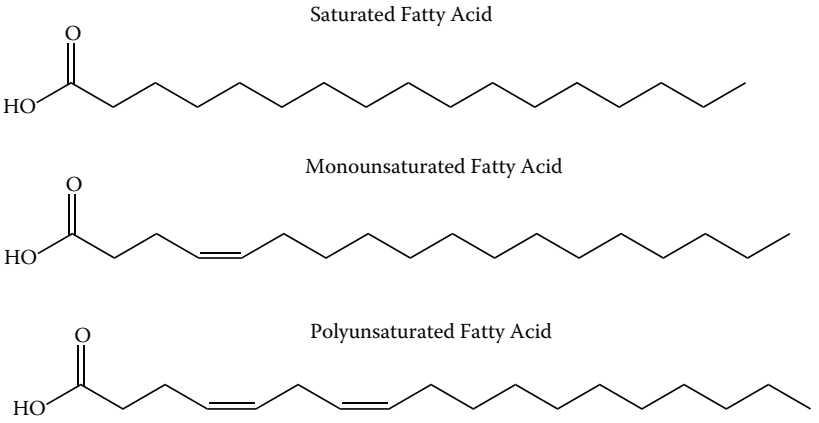

FIGURE 1.3 Fatty acids of various levels of saturation.

at the third, sixth, or ninth carbon in the chain. The structures of linolenic acid (an omega-3 fatty acid) and linoleic acid (an omega-6 fatty acid), which are considered essential fatty acids, are shown in Figure 1.4.

Human enzymes are able to insert double bonds into certain positions of fatty acid chains but cannot insert them into other locations. For example, human enzymes are unable to insert carbon–carbon double bonds closer than seven carbons from the methyl (omega) end. Thus, omega-3 and omega-6 fatty acids are termed EFAs because they must be provided by the diet since the body cannot synthesize them. Linoleic acid (omega-6) has two double bonds and linolenic acid (omega-3) has three double bonds (Figure 1.4). EFAs and their impact on health and exercise performance will be discussed further in Chapter 2.

In addition to the placement of the double bond within a fatty acid, another consideration is the arrangement of the hydrogen atoms at the carbon–carbon double bond. Unsaturated fatty acids can be in a *cis* or *trans* configuration. In the *cis* configuration, both hydrogen atoms attached to the carbons involved in the double bond are on the same side of the molecule. This causes the fatty acid to appear bent due to the

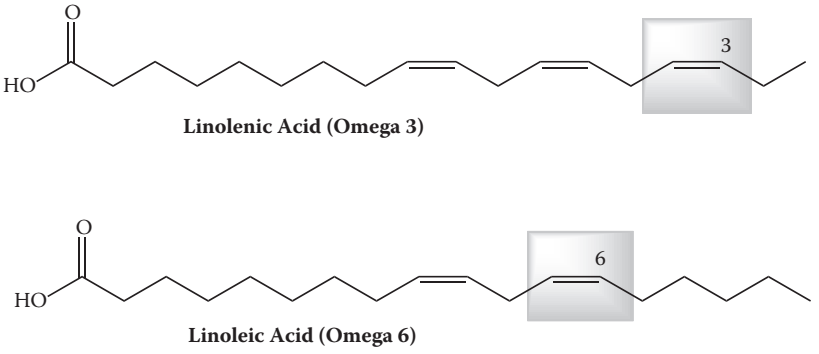

FIGURE 1.4 Essential fatty acids.

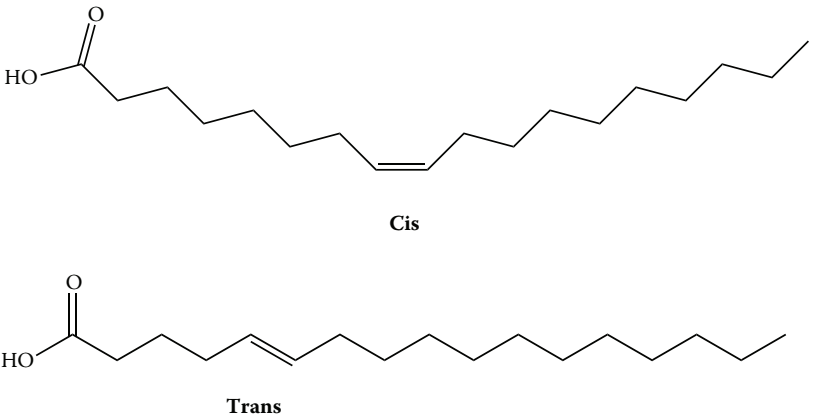

FIGURE 1.5 *Cis* versus *trans* configuration of a fatty acid.

hydrogen atoms repelling one another (Figure 1.5). Most naturally occurring fatty acids are in the *cis* category. In the *trans* configuration the hydrogen atoms linked to the carbons involved in the double bond are on opposite sides of the molecule, resulting in the chain taking on a straighter appearance much like saturated fatty acids (Figure 1.5). The shape of a *trans*-fatty acid (more straight and less curved), similar to a saturated fatty acid, allows fatty acids to pack in tightly next to each other in cell membranes. Other differences between *trans* and *cis* configurations include variations in melting points as well as chemical and enzymatic activities (Erasmus 1993). Naturally occurring *trans* fats can be found in small amounts in food, but most often are created by a food processing technique known as hydrogenation.

1.2.3 Fatty Acid Chain Length

The length of fatty acid chains can range between 4 and 24 carbons. Chain length is important in determining the characteristics and functions of fatty acids. Depending on the length of a fatty acid chain, it can be categorized as a short-, medium-, or long-chain fatty acid. For example, the typical length of fatty acid chains is between 16 and 22 carbons, and chains of this length are classified as long-chain fatty acids. How long the chain is will affect how the fatty acid is digested, absorbed, and used in the body (Fink, Burgoon, and Mikesky 2009). Table 1.2 identifies the common names of several saturated and unsaturated fatty acids of various chain lengths.

1.3 FAT DIGESTION AND ABSORPTION

Dietary fats are initially broken down into large fat globules in the stomach by gastric lipase enzymes. Large fat globules then pass into the small intestine, where they are then broken down into smaller fat globules through the actions of bile salts released from the gallbladder.

TABLE 1.2
Common Names of Fatty Acids of Various Saturation and Chain Lengths

Common Name of Fatty Acid	Fatty Acid Chain Length	Type of Fatty Acid	Number of Double Bonds
Butyric acid	4	Saturated	0
Caproic acid	6	Saturated	0
Octanoic acid	8	Saturated	0
Capric acid	10	Saturated	0
Lauric acid	12	Saturated	0
Palmitic acid	16	Saturated	0
Stearic acid	18	Saturated	0
Palmitoleic acid	16	Monounsaturated	1
Oleic acid	18	Monounsaturated	1
Linoleic acid	18	Polyunsaturated essential	2
Alpha-linolenic acid	18	Polyunsaturated essential	3
Gamma-linolenic Acid	18	Polyunsaturated essential (omega 6 derivative)	3
Arachidonic acid	20	Polyunsaturated essential (omega 6 derivative)	4
Eicosapentaenoic acid (EPA)	20	Polyunsaturated essential (omega 3 derivative)	5
Docosahexaenoic acid (DHA)	22	Polyunsaturated essential (omega 3 derivative)	6

Most fat digestion occurs in the proximal small intestine (i.e., duodenum). In the small intestine, pancreatic juices containing the pancreatic lipase act to liberate monoacylglycerols (MAGs), diacylglycerols (DAGs), and free fatty acids (FFAs) from the smaller fat globules. Long chain fatty acids are then absorbed in the small intestine and re-formed into triglycerides, which, along with cholesterol and proteins, are packaged as chylomicrons. Since fat is not soluble in water, the formation of chylomicrons is necessary for transport of these long chain fatty acids in the watery blood plasma. Short- and medium-chain fatty acids do not re-esterify in the epithelial cell. Rather, they diffuse into the blood and are bound to albumin in order to be transported to the liver through the hepatic portal vein. The recently formed chylomicrons are then released into the lymphatic system and eventually the bloodstream whereby muscle cells, fat cells, and/or other cell types can retrieve ingested fatty acids by "plucking" them off chylomicrons with an enzyme called lipoprotein lipase. Once free fatty acids enter a particular cell type, it can be oxidized as an energy fuel source, stored as a fuel source, or integrated into cellular structures (for instance, cell membranes).

1.4 FAT METABOLISM

The metabolism of fat for energy to be used by the body is essential for athletic performance. The ultimate goal of this process is to transport fatty acids to the

mitochondria of working muscles to be broken down to produce large amounts of ATP (adenosine triphosphate). This process includes the cleavage of fatty acids from their glycerol backbone (i.e., lipolysis), their subsequent transportation in the blood to the cytosol of the muscle, and then their transportation into the mitochondria for beta-oxidation.

1.4.1 Lipolysis

Lipolysis (hydrolysis of fat) is the process of breaking down triglycerides into glycerol and three fatty acid molecules. Physiologically, lipolysis occurs in three places:

- Cytosol of adipocytes (i.e., fat cells)
- Intramuscular triacylglycerol (IMTG) stores
- Plasma triacylglycerols

The fatty acids resulting from lipolysis are derived primarily from adipocytes and IMTGs. There are significantly more fatty acids available for oxidation from adipocytes than IMTGs. However, IMTG stores provide another source of fatty acids and are conveniently located in the muscle itself. Type I muscle fibers have a higher content of IMTG stores than type II muscle fibers. Estimates of the contribution of IMTG to total lipid oxidation have ranged from 20% at rest to 70% during exercise. There is evidence to suggest that IMTG utilization in muscle may be more important during recovery from exercise. Plasma triacylglycerols (catalyzed by lipoprotein lipase) also undergo lipolysis to liberate fatty acids as a fuel source, but contribute only minimally to energy production during exercise and are not well studied in humans. One study reported that the fatty acid uptake from plasma lipoprotein triacylglycerols occurs slowly and accounts for less than 3% of the energy expenditure during prolonged exercise (Jeukendrup and Gleeson 2010; Havel, Pernow, and Jones 1967). Plasma triacylglycerols are bound to lipoprotein complexes when transported between tissues. These lipoprotein complexes that transport plasma triacylglycerols include chylomicrons (produced in the absorptive cells of the small intestine) and very low-density lipoproteins (VLDLs), which are produced in the liver following the ingestion of dietary fat.

Catecholamines (epinephrine and norepinephrine) and insulin are the major plasma hormones regulating lipolysis in humans (Lafontan and Langin 2009; Jaworski et al. 2007). Catecholamines activate lipolysis by binding to beta-adrenergic receptors on the plasma membrane of adipocytes and inhibit the cascade by binding to alpha-adrenergic receptors, also located on the plasma membrane of adipocytes. Beta-adrenergic receptors are coupled with stimulatory proteins while alpha-adrenergic receptors are coupled with inhibitory proteins. Stimulating the beta-adrenergic receptors activates adenylate cyclase, which converts ATP to cyclic adenosine monophosphate (cAMP). cAMP activates cAMP-dependent protein kinase, which then phosphorylates hormone-sensitive lipase (HSL). Once activated, HSL catalyzes the removal of two of the three fatty acids from the triacylglycerol in the adipocyte. Monoacylglycerol lipase removes the final fatty acid from the glycerol backbone. This process of lipolysis via beta-adrenergic stimulation is summarized

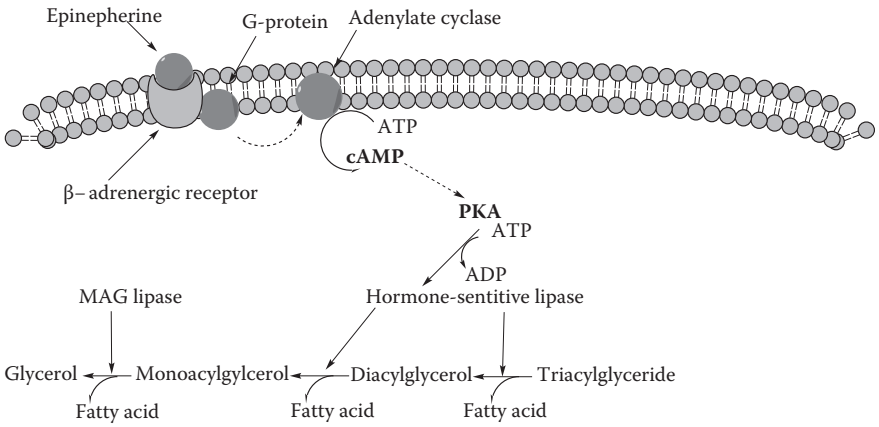

FIGURE 1.6 Lipolysis.

in Figure 1.6. The effect of catecholamines on the rate of lipolysis is dependent on their changes in plasma concentrations and binding affinities for the different adrenergic receptors. Other hormones that increase the lipolytic rate include glucagon and growth hormone.

Insulin has an inhibitory effect on lipolysis that is attributed to its stimulation of cellular phosphodiesterase-3, which degrades cAMP to AMP and reduces the signaling cascade responsible for activating HSL. Insulin activates phosphatidylinositol 3-kinase, which phosphorylates and subsequently activates phosphodiesterase-3 (Figure 1.7). Insulin's effect on lipolysis occurs primarily at rest, as insulin secretion decreases during exercise. Other factors in addition to catecholamines and insulin influence the lipolytic rate, but not as profoundly.

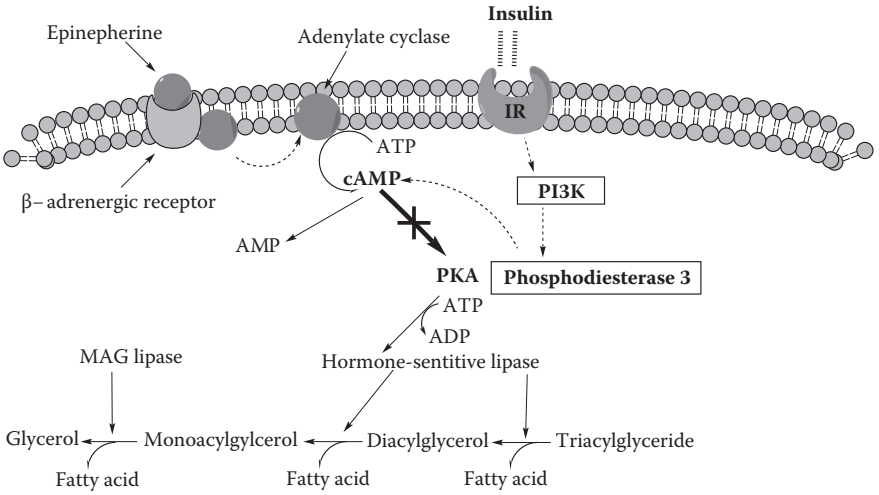

FIGURE 1.7 Inhibitory effect of insulin on lipolysis.

1.4.1.1 Transport of Fatty Acids from Adipose Tissue to Skeletal Muscle

The four products of adipocyte lipolysis are three fatty acids and a glycerol molecule. The glycerol molecule liberated during lipolysis is released to the blood. Therefore, the measurement of glycerol in the blood can be used as an index of the degree of lipolysis. Once in the blood, the plasma glycerol is taken up primarily by the liver and converted to glycerol 3-phosphate via glycerol kinase. Glycerol 3-phophate is then converted to dihydroxyacetone phosphate, which can enter either glycolysis or be converted to glucose via gluconeogenesis. The fatty acids produced during lipolysis can be released into circulation or remain in the adipose tissue and be used to form a new triacylglycerol, a process called re-esterification. At rest, about 70% of all fatty acids released during lipolysis are re-esterified (Jeukendrup and Gleeson 2010; Wolfe et al. 1990). During exercise, re-esterification is suppressed and the fatty acids that are released into circulation are taken up by skeletal muscle.

The fatty acids released to the circulation will bind with albumin for transport throughout the body (Wang et al. 2008; Ranallo and Rhodes 1998). Albumin is the most abundant protein in the blood, and one of its functions is as a carrier protein for transporting fatty acids. Albumin contains at least three high-affinity binding sites for fatty acids, which provide a large capacity to bind fatty acids. In order to be taken up by the muscle, fatty acids must first cross the endothelial lining of the blood vessel, then the interstitial space, and finally the plasma membrane/sarcolemma of the muscle cell. For many years, the transport of fatty acids into the muscle cell was believed to be a passive process. Recently, however, specific carrier proteins have been identified. At the endothelial lining, the albumin–fatty acid complex binds to specific albumin binding proteins (ABP), which aids the release of fatty acids from albumin and also facilitates their uptake into the active skeletal muscle. The entrance of a fatty acid into the muscle across the plasma membrane is facilitated by plasma membrane fatty acid-binding protein (FABPpm) and a fatty acid transporter (FAT/CD36) protein. Once in the cytosol, the fatty acid then binds to cytoplasmic fatty acid binding protein (FABPc) to be transported to the mitochondria for oxidation or will remain in the cytosol to be re-esterified into IMTGs.

1.4.2 Beta-Oxidation

The process of beta-oxidation (β-oxidation) transforms a fatty acid molecule into acetyl-CoA (coenzyme A) in the mitochondria. Prior to entering the β-oxidation pathway, fatty acids must be activated in the cytosol and then cross the two mitochondrial membranes. Once this occurs, the activated fatty acids are now able to enter the β-oxidation pathway. The following describes the activation of fatty acids in the cytosol and their transportation into the mitochondrial matrix and subsequent oxidation.

Long chain fatty acids in the sarcoplasm are activated by the enzyme acyl-CoA synthetase, resulting in the formation of fatty acyl-CoA (medium- and short-chain fatty acids undergo this reaction in the mitochondria). Now that the fatty acid is activated and is in the form of fatty acyl CoA (also referred to as acyl CoA), it must cross the outer mitochondrial membrane. Carnitine, a compound derived from the

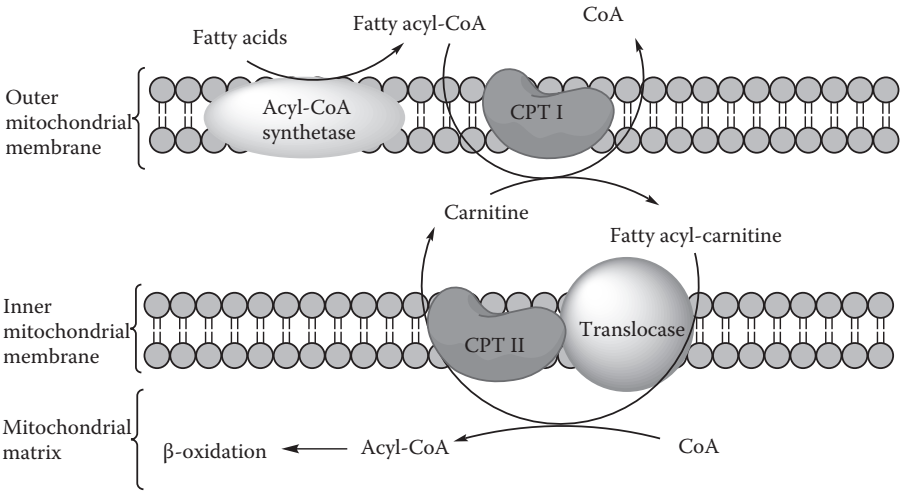

FIGURE 1.8 Long-chain fatty acid transfer from cytosol to mitochondria.

amino acid lysine, carries the fatty acyl CoA group into the mitochondria and then detaches the fatty acyl group from CoA, producing CoA and fatty acyl carnitine. This reaction is catalyzed by carnitine palmitoyltransferase I, located on the outer mitochondrial membrane.

The fatty acyl carnitine then crosses the inner mitochondrial membrane with the help of a transmembrane protein, translocase. Once in the mitochondrial matrix, the fatty acyl group is linked to CoA again. This reversal of the initial reaction (which occurred at the outer mitochondrial membrane) is catalyzed by carnitine palmitoyltransferase II, which is attached to the matrix side of the inner membrane. Carnitine then returns to the intermembrane space through the transmembrane protein translocase. Figure 1.8 demonstrates the process of transferring a long-chain fatty acid from the cytosol to the mitochondrial matrix. This process is often termed the carnitine shuttle.

Once in the mitochondrial matrix, fatty acyl CoA enters the pathway of beta-oxidation. Beta-oxidation is a recurring four-step process that involves the successive removal of 2-carbon acyl fragments from the carboxyl end of the fatty acid (Figure 1.9). The newly cleaved 2-carbon acyl fragment combines with coenzyme A to form acetyl-CoA. This process is repeated until the entire fatty acid molecule is oxidized. The final cycle produces two separate acetyl CoAs, instead of one acyl CoA and one acetyl CoA. Each cycle of β-oxidation produces one molecule of NADH, $FADH_2$, and acetyl CoA. NADH and $FADH_2$ are then oxidized in the electron transport chain to supply energy (i.e., resynthesize ATP), while acetyl CoA enters the Krebs cycle to produce three NADH and one $FADH_2$. The complete oxidation of fatty acids in the mitochondria depends on several factors, including the activity of enzymes of the β-oxidation pathway, the concentration of Krebs cycle intermediates, the activity of enzymes in the Krebs cycle, and the presence of oxygen.

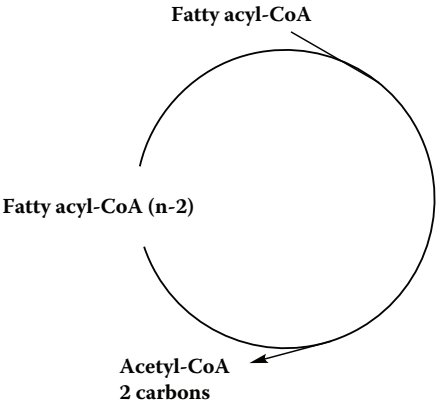

FIGURE 1.9 Acetyl-CoA production from β-oxidation.

1.5 FAT METABOLISM AT REST AND DURING EXERCISE

Carbohydrate and fat are always oxidized as a mixture, and whether carbohydrate or fat is the predominant fuel depends on a variety of factors, the most important being the intensity and duration of the exercise bout. There is a crossover from predominantly fat oxidation at rest and low intensities of exercise to chiefly carbohydrate usage at high intensities of exercise. This crossover effect is seen in both trained and untrained individuals. As a general rule, the relative contribution of fats to total oxidative metabolism decreases as exercise intensity increases (van Loon et al. 2001). Fat oxidation is the predominant fuel at low aerobic exercise intensities, and carbohydrate oxidation is the primary fuel during high exercise intensities. The changes in fat metabolism that occur during different aerobic exercise intensities, exercise durations, following endurance training, and during resistance exercise are discussed next.

1.5.1 Rest

After an overnight fast, most energy needs are provided by oxidizing fatty acids derived from adipose tissue triglycerides (Horowitz and Klein 2000; Klein et al. 1986). One study reported that, at rest, fat oxidation provided 66% of total energy expenditure, with free fatty acids and other fat sources (mainly intramuscular triglyceride stores) contributing about 50% and 16%, respectively (van Loon et al. 2003). In the resting condition, the amount of fatty acids released from adipose tissue typically exceeds the amount oxidized in the skeletal muscle. Because of this mismatch between available fatty acids and their oxidation rates, a large portion of the fatty acids are re-esterified back into triglycerides, primarily by the liver (Horowitz and Klein 2000). Resting plasma fatty acid concentrations are typically between 0.2 and 0.4 mmol/L (Jeukendrup and Gleeson 2010).

1.5.2 Low- and Moderate-Intensity Aerobic Exercise

The transition from rest to low-intensity exercise stimulates lipolysis, which increases the availability of fatty acids to the working skeletal muscle, contributing to an increase in the rate of their oxidation. During the first 15 min of aerobic exercise, plasma fatty acid concentrations usually decrease because the rate of fatty acid uptake by the skeletal muscle (and the subsequent oxidation) exceeds the rate of fatty acid appearance in plasma from lipolysis (Jeukendrup and Gleeson 2010). Thereafter, plasma fatty acids begin to increase as the lipolytic rate begins to exceed the oxidation of fatty acids in the skeletal muscle.

During mild- or moderate-intensity exercise (ranging from 25% to 65% VO_2max) fatty acid concentrations may reach 1 mmol/L, which is about two to five times greater than the resting concentrations of 0.2 to 0.4 mmol/L. As exercise intensity progresses from low intensity to moderate intensity, adipose tissue lipolysis increases approximately threefold, mainly due to an increase in β-adrenergic stimulation from circulating catecholamines. In addition, blood flow to adipose tissue increases (approximately doubles) and the rate of re-esterification is halved (Jeukendrup and Gleeson 2010). All of these factors serve to increase the delivery of fatty acids to the active skeletal muscle. This increase in plasma fatty acids is associated with a 5- to 10-fold increase in fat oxidation above resting amounts (Horowitz and Klein 2000; Phinney et al. 1983; Krogh and Lindhard 1920).

Total fat utilization increases until exercise intensity reaches approximately 60%–65% VO_2max (Achten and Jeukendrup 2003; Achten, Gleeson, and Jeukendrup 2002; Achten, Venables, and Jeukendrup 2003). Achten and colleagues conducted several well-designed studies and reported that the maximal rate of fat oxidation during running and cycling is approximately 60% VO_2max (Achten and Jeukendrup 2003; Achten et al. 2002, 2003). In relation to heart rate, the maximal rate of fat oxidation was observed at 74% of maximal heart rate (Achten et al. 2002). As a basis of comparison in terms of exercise intensity, prolonged self-paced exercise by young, fit men is typically performed at a low intensity of about 45% VO_2max (which is equivalent to 66% of maximal heart rate) (Swain et al. 1994; Evans et al. 1980).

The source of fatty acids undergoing oxidation also changes with a variation in exercise intensity. During exercise at 25% VO_2max, most of the fat oxidized is derived from plasma fatty acids and only small amounts are derived from IMTGs (Holloszy and Kohrt 1996; Klein et al. 1994; Romijn et al. 1993). During exercise intensity at 65% VO_2max, however, the contribution of plasma fatty acids declines, whereas the contribution of IMTGs increases and provides from one-third to about half of the fatty acids used for total fat oxidation (van Loon et al. 2003; Martin et al. 1993; Romijn et al. 1993). The estimated relative contribution of plasma fatty acid and IMTGs to total fat oxidation during exercise at three different intensities is shown in Figure 1.10.

1.5.3 High-Intensity Exercise

Shifts in energy substrate mobilization and utilization occur as exercise intensity increases. There is a progressive increase in the relative contribution of carbohydrate oxidation to energy expenditure and a corresponding decrease in the relative

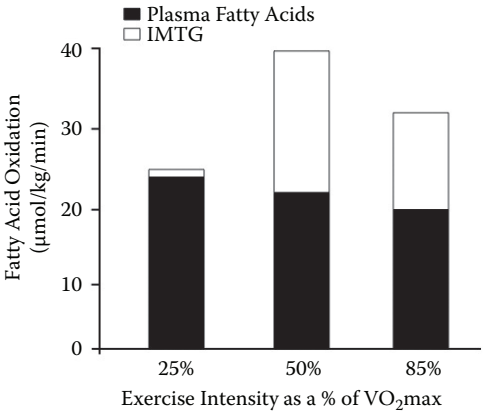

FIGURE 1.10 Contribution of plasma fatty acids and IMTGs to total fat oxidation. (Data adapted from Romijn, J. A. et al., 1993, *American Journal of Physiology* 265 (3 Pt 1): E380–E391.)

contribution of fat oxidation as exercise progresses from moderate intensity to high intensity (Brooks and Mercier 1994; Romijn et al. 1993). Achten and co-workers (2002) reported that, at exercise intensities above 75% VO_2max, fat oxidation rates decreased markedly. Further, the contribution of fat oxidation to energy expenditure became negligible above an intensity of about 90% VO_2max (Figure 1.11) (Achten et al. 2002).

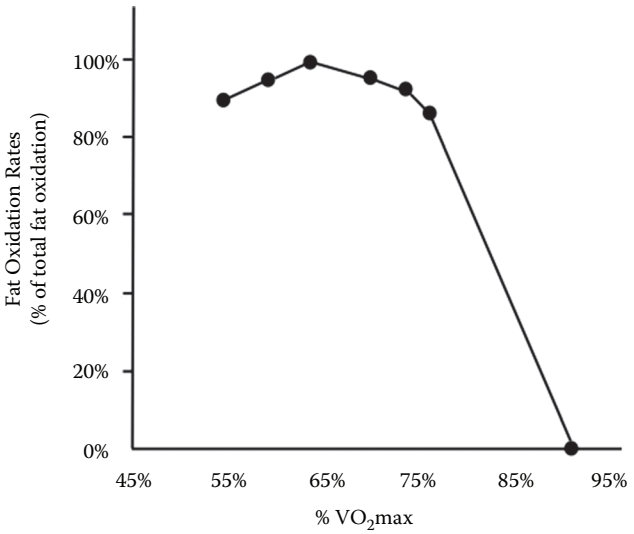

FIGURE 1.11 Contribution of fat oxidation during high-intensity exercise. (Data adapted from Achten, J. et al., 2002, *Medicine and Science in Sports and Exercise* 34 (1): 92–97.)

Despite a relatively high rate of energy expenditure during high-intensity exercise (>75% of VO_2max), fat oxidation is suppressed to values below those observed during moderate-intensity exercise (Horowitz and Klein 2000; Romijn et al. 1993; Jones et al. 1980). The limitation in fat use during high-intensity exercise is attributed, at least in part, to a decline in circulating fatty acids caused by a reduction in the release of fatty acids from adipose tissue (Horowitz and Klein 2000; Romijn et al. 1995). This decrease in the release in fatty acids is not caused by a reduction in lipolysis, but rather is likely due to a decreased adipose tissue blood flow and inadequate fatty acid removal by the bloodstream during high-intensity exercise (Horowitz and Klein 2000; Lambert et al. 1997; Hodgetts et al. 1991; Romijn et al. 1993, 1995; Jones et al. 1980; Rosell and Belfrage 1979).

Another reason for the suppression of fat oxidation during high-intensity exercise may be related to the increased glycogen metabolism in muscle (Jeppesen and Kiens 2012). The high rate of muscle glycogenolysis during high-intensity exercise increases the amount of acetyl-CoA derived from glycolysis, which increases malonyl-CoA concentrations in skeletal muscle (Horowitz and Klein 2000; Elayan and Winder 1991). Malonyl-CoA inhibits the enzyme carnitine palmitoyltransferase I (also known as CPT1), which is responsible for long-chain fatty acid entry into the mitochondria (Horowitz and Klein 2000; McGarry et al. 1977, 1983; Robinson and Zammit 1982). Because malonyl-CoA inhibits carnitine palmitoyltransferase I, lower levels of malonyl-CoA would be expected during exercise at low and moderate intensities, when long-chain fatty acid oxidation is high, whereas high concentrations of malonyl-CoA might be expected during high-intensity exercise, when long-chain fatty acid oxidation is low (Turcotte 2006). In summary, high rates of glycogenolysis (which occurs during high-intensity exercise) likely modify fat oxidation by impairing long-chain fatty acid transport into the mitochondria via carnitine palmitoyltransferase I inhibition (Horowitz and Klein 2000; Sidossis et al. 1997).

1.5.4 Exercise Duration

Exercise duration also plays an important role in fat metabolism. Lipolysis of adipose tissue triglycerides, plasma fatty acid uptake, and fatty acid oxidation increase progressively throughout a bout of constant, low- to moderate-intensity endurance exercise (Carey et al. 2001; Saltin and Astrand 1993). To illustrate this point, Ahlborg and colleagues (1974) observed the plasma free fatty acid uptake and fat contribution to total energy expenditure in subjects exercising for long periods of time. Specifically, six subjects were observed at rest and during 4 h of exercise at approximately 30% of maximal oxygen uptake. During the first hour of exercise, fat supplied approximately 40% of the energy, while in the fourth hour fat contributed approximately 65% of the total energy requirement. The increase in fat oxidation with increased exercise duration is associated with reduced glycogen stores. Reductions in blood glucose and insulin (a potent inhibitor of lipolysis) as well as increased glucagon output by the pancreas also contribute to the increase in fat metabolism that is associated with exercise duration.

1.5.5 Endurance Training Adaptations

One of the defining characteristics of endurance training is the metabolic shift to greater use of fat and a sparing of glycogen at a given submaximal intensity. Several factors contribute to this adaptive response:

- Increased density of the mitochondria in the skeletal muscles, which increases the capacity for fat oxidation (Horowitz and Klein 2000; Holloszy 1967)
- Increase in the number of capillaries within skeletal muscle, which enhances fatty acid delivery (Horowitz and Klein 2000; Saltin and Gollnick 1983)
- Increase in carnitine transferase, which facilitates fatty acid transport across the mitochondrial membrane (Horowitz and Klein 2000; Mole, Oscai, and Holloszy 1971)

The contribution of fat to total energy expenditure increases after endurance training (at both the relative and the absolute exercise intensities). This would seem to indicate that adipose tissue lipolysis and plasma fatty acid uptake are also increased with endurance training. However, this is not the case. Endurance training does not increase the whole-body lipolytic response during exercise performed at the same absolute exercise intensity (Horowitz and Klein 2000). Further, there is a decreased contribution of plasma fatty acids to fat oxidation following 12 weeks of endurance training (Martin et al. 1993). Therefore, since more fat is oxidized for energy after endurance training, but the rate of lipolysis is unchanged and there is a decrease in plasma fatty acid oxidation, it suggests an increased reliance on IMTGs as a source of fuel in the trained state (Horowitz and Klein 2000). Unfortunately, the studies investigating the changes in IMTG contributions to total energy expenditure following endurance training have reported conflicting results. Some (Horowitz and Klein 2000; Hurley et al. 1986; Phillips et al. 1996) but not all (Horowitz and Klein 2000; Kiens et al. 1993; Bergman et al. 1999) studies report a greater depletion of IMTG stores during exercise performed after endurance training as compared to before training.

1.5.6 Resistance Exercise

A majority of the scientific studies investigating substrate contribution during exercise has examined aerobic activities. In contrast, few studies have investigated the effects of acute resistance exercise and lipid metabolism. Resistance exercise has been shown to increase adipose tissue lipolysis, which indicates that fatty acids are being mobilized and are a possible fuel source (Ormsbee et al. 2007, 2009; Chatzinikolaou et al. 2008). One study showed that this activity peaked a few minutes into the exercise session and then declined steadily but remained above baseline (Chatzinikolaou et al. 2008). Intramuscular triglycerides may also provide a source of energy during heavy-resistance exercise. A study by Essen-Gustavsson and Tesch (1990) demonstrated a significant reduction in intramuscular triglycerides from biopsied muscle following an acute bout of resistance exercise. Similarly, it was reported that intramuscular triglycerides were significantly reduced by 27% following a single

bout of resistance exercise in type I muscle fibers, with no net changes in type IIa or IIx fibers (Koopman et al. 2006). Other investigations have also reported that type I muscle fibers undergo significantly more intramuscular triglyceride depletion during exercise as compared to type II muscle fibers (De Bock et al. 2005; van Loon et al. 2003). This is not a surprising observation considering that type I muscle fibers contain about two times more intramuscular triglyceride stores as compared to type II muscle fibers (De Bock et al. 2005; van Loon et al. 2003).

Resistance exercise upregulates lipolysis, but it is not clear to what extent this aids in the supply of energy during a resistance exercise session. Lipolysis continues to be elevated above baseline following resistance exercise. In a series of studies, Ormsbee and co-workers reported that lipolysis was elevated up to 20 min (Ormsbee et al. 2009) and 45 min (Ormsbee et al. 2007) following an acute resistance exercise bout. Fat oxidation was also measured in these studies and was significantly elevated for at least 40 min following exercise (Ormsbee et al. 2007, 2009). Other studies have reported elevations in fat oxidation lasting up to 2 h following resistance exercise (Binzen, Swan, and Manore 2001; Haddock and Wilkin 2006). In summary, it appears that an acute bout of resistance exercise increases adipose tissue lipolysis and fat oxidation. Elevations in lipolysis and oxidation have been observed for up to approximately 45 min following the resistance exercise bout. The elevated fatty acid oxidation rates are likely due to increases in lipolytic activity stemming from both adipose tissue and intramuscular triglyceride stores.

1.6 CONCLUSION

Fat is the most abundant energy source in the body and serves as the primary energy source at rest and during low- to moderate-intensity exercise. The energy provision from fat provides about two-thirds of total energy expenditure at rest, and as aerobic exercise increases from moderate to very high intensities, there is a progressive decrease in percentage of energy derived from bodily fat stores (adipose tissue and intramuscular triglyceride stores). Resistance exercise also relies upon lipolysis and fat oxidation in an effort to fuel this mode of exercise. The utilization of fat as a fuel source not only occurs during the resistance exercise bout, but also continues to be elevated for about 45 min in the postworkout period.

ACKNOWLEDGMENT

A sincere appreciation to Marcelle Issa for providing the artwork for each of the figures in this chapter.

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2 Dietary Fat Intake Strategies to Enhance Performance

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CONTENTS

2.1 Introduction 21
2.2 Dietary Fat Intake Strategies to Enhance Performance 22
2.2.1 Dietary Fat Intakes 22
2.2.2 Pre-Exercise Dietary Fat Strategies 23
2.2.2.1 Pre-Exercise Fasting 24
2.2.2.2 Pre-Exercise High-Fat Meal 25
2.2.3 Fat Loading 29
2.2.3.1 Endurance Exercise Performance 30
2.2.3.2 Anaerobic Exercise Performance and Ratings of Perceived Exertion 34
2.2.3.3 Fat-Loading Summary 35
2.3 Fatty Acid-Containing Supplements 35
2.3.1 Essential Fatty Acids 35
2.3.2 Conjugated Linoleic Acid 37
2.3.3 Medium Chain Triglycerides 38
2.4 Conclusion 39
References 40

2.1 INTRODUCTION

In the previous chapter, aspects of fat metabolism were discussed. Fat is a major fuel source for endurance activities and is even used for fuel during resistance exercise. In this chapter, a look at the various dietary fat strategies that have been investigated for their performance-enhancing benefits will be presented. In addition, there are several dietary fat supplements that have been purported to improve either exercise performance or body composition. A summary of each of these dietary fat supplements and its effectiveness will also be presented.

2.2 DIETARY FAT INTAKE STRATEGIES TO ENHANCE PERFORMANCE

Carbohydrates and fat serve as the primary nutrients that fuel exercise and training bouts. For this reason, many scientific studies have investigated the effects of various dietary fat strategies to maximize exercise performance. The first of these strategies to be presented in this chapter is the amount of dietary fat that should be ingested on a daily basis. Unlike carbohydrates and protein, specific recommendations based on grams of dietary fat per kilogram of body mass per day have not reached a consensus at this time.

2.2.1 Dietary Fat Intakes

Even though fat is an essential nutrient in the diet, no firm standards exist for optimal fat intake. In the general population, the acceptable macronutrient distribution range for fat is 20% to 35% of energy intake (Institute of Medicine 2005). On a relative basis, this equates to a range of approximately 0.8 to 1.25 g dietary fat/kg body mass. When fat intake is at 30% of total calories, the *Dietary Guidelines for Americans* (US Department of Health and Human Services and US Department of Agriculture 2005) recommends that the proportion of energy from fatty acids be 10% saturated, 10% polyunsaturated, and 10% monounsaturated and that sources of essential fatty acids be included. Since there is no recommended dietary allowance (RDA) for fat intake, it is important for the athlete to avoid intakes that are extreme on either end of the continuum (either too little or too much dietary fat intake). Diets that are too low in dietary fat combined with low total energy can lead to a negative energy balance. When energy balance is negative for long periods of time, training may suffer. Even when weight loss (in the form of fat loss) is pursued, dietary fat should not be reduced to levels below 20% of total daily calories. In contrast, consuming too much dietary fat can lead to the overconsumption of total calories, resulting in weight gain in the form of body fat. Because fat tissue does not help produce movement, it acts as "dead weight." When excess body fat is present, relative power production is decreased and performance will likely be hindered in most sports and activities.

Based on the available literature, there is no justifiable reason why athletes and physically active individuals should not follow the generally recommended fat intake range of 20% to 35% of total calories. While the research is limited, it appears that fat intake within this range does not adversely affect exercise performance. Van Zant, Conway, and Seale (2002) reported that when fat intake was 20% of total calories as compared to 40% of total calories, there was no effect on exercise training or strength exercise performance in moderately trained males. Hence, ingesting dietary fat above (40%) the recommended rage (20%–35%) does not improve resistance exercise performance. In relation to aerobic exercise, male duathletes (a duathlon consists of running and cycling) followed a diet containing 53% fat and a diet containing only 17% fat. After following this diet for 5 weeks, there was no difference in the time it took to run a half-marathon or in the total work output during a 20-min all-out time trial on a cycle ergometer (Vogt et al. 2003). This study was unique in that it compared dietary fat intakes below and

above the generally recommended intake of 20%–35% and reported no differences in aerobic exercise performance.

There is cause for concern in regard to exercise performance when dietary fat intakes go below 20% of total calories. Horvath and co-workers (2000) assessed the aerobic endurance performance of male and female aerobic endurance athletes after they ingested isocaloric diets with varying fat contents. The athletes consumed isocaloric diets consisting of either 16% fat, 31% fat, or 44% fat for 4 weeks before running at 80% VO_2max until voluntary exhaustion. The authors reported that the 31% fat diet resulted in a significant improvement in aerobic endurance performance in comparison to the 16% diet. The difference in aerobic performance between these two dietary fat intake groups was quite high, with the 31% fat diet improving run time to exhaustion approximately 17% more than the 16% fat diet. There were no differences in aerobic endurance performance between the 31% fat and the 44% fat diet groups, however.

The recommendation is that athletes consume a habitual diet of approximately 30% fat. Of this 30%, 10% should be saturated, 10% polyunsaturated, and 10% monounsaturated. Intakes above this amount have not been shown to affect exercise performance adversely, but may put some athletes in a situation where they are ingesting too many total calories, resulting in weight gain in the form of body fat. However, if the diet of the athlete is monitored and adjusted accordingly with activity levels, these concerns are mitigated. Athletes ingesting fat lower than 20% of total calories may be adversely affecting their exercise performance (Horvath et al. 2000). In terms of the strength/power athlete, reductions in endogenous testosterone production were reported with dietary fat intakes below 20% of total calories (Dorgan et al. 1996). Whether or not these reductions in endogenous testosterone concentrations resulting from low dietary fat intakes reduce strength/power exercise performance needs further investigation. Athletes report an average fat intake of 35% of total calories (Hawley et al. 1995), which is at the upper limit of the recommended range. A benefit of ingesting dietary fat in the upper level of the recommended range is that an athlete needing to lose weight can reduce energy intake by targeting calories derived from dietary fat and still be within the recommended range. This topic will be discussed in more detail in Chapter 9, "Enhancing Body Composition: Gaining Muscle and Losing Fat."

2.2.2 Pre-Exercise Dietary Fat Strategies

One of the goals of an endurance athlete is to tap into fat stores as much as possible and not oxidize carbohydrate (CHO) stores (liver and skeletal muscle glycogen) so that they can be utilized later in a competition or race. Utilizing less carbohydrate and more fat for ATP (adenosine triphosphate) resynthesis is referred to as a *glycogen sparing effect*. This glycogen sparing effect is a natural adaptation to endurance training and results in an increased capacity to oxidize fat for energy during submaximal exercise (Ivy 1999; Kiens et al. 1993). In addition to endurance training, several other approaches have been applied to increase fat availability, including:

- Caffeine intake
- Pre-exercise fasting

- Pre-exercise high-fat meal
- Fat loading

With respect to caffeine, most (Jenkins et al. 2008; Cox et al. 2002; Kovacs, Stegen, and Brouns 1998; Berglund and Hemmingsson 1982) but not all (van Nieuwenhoven, Brouns, and Kovacs 2005; Hunter et al. 2002) investigations report a significant enhancement of endurance performance. The ergogenic properties of caffeine ingestion were initially ascribed to a metabolic substrate shift caused by an increase in fatty acid availability and a decrease in glycogen breakdown (Helge 2000). However, increased fatty acid availability during exercise is not always present after caffeine ingestion, and therefore it is likely that other mechanisms (other than increasing fatty acid availability) play more important roles in endurance performance enhancement (Helge 2000; Graham 1998; Graham, Rush, and van Soeren 1994). The following sections discuss the other approaches related to dietary fat manipulation strategies and their effectiveness on exercise performance.

2.2.2.1 Pre-Exercise Fasting

While most athletes ingest a pre-exercise meal in the hours prior to competition or training, some choose not to do so. Triathletes have reported several reasons for not ingesting a meal that is high in fat 1 to 4 h prior to an exercise session/competition. These reasons include gastrointestinal distress consistent with bloating, diarrhea, and stomach cramping (Rehrer et al. 1992). Another motivation for fasting is that it has been proposed as a way to increase fatty acids in the blood (which spares muscle glycogen) and possibly improve exercise performance. In terms of glycogen stores, short-term fasting (24 h) depletes liver glycogen (Hultman and Nillson 1977; Nilsson and Hultman 1973), but not muscle glycogen (when no strenuous exercise is performed) (Knapik et al. 1988; Loy et al. 1986). In most (if not all) studies investigating the effects of fasting (from 14 h to 3.5 days), it is reported that plasma free fatty acid levels are significantly elevated both at rest (prior to exercise) and during endurance exercise (Zinker, Britz, and Brooks 1990; Knapik et al. 1988; Gleeson, Greenhaff, and Maughan 1988; Maughan and Gleeson 1988; Loy et al. 1986; Dohm et al. 1986).

Loy and co-workers (1986) gave competitive cyclists a 355-calorie meal either 3 h prior to endurance exercise (fed group) or 24 h prior to the same endurance exercise bout (fasting group). Fasting resulted in significantly higher pre-exercise free fatty acid values (~1.5 mmol/L) compared with the values of the fed group (~0.65 mmol/L). The subsequent endurance exercise caused an increase in free fatty acids in both the fed and fasting groups to values greater than 2 mmol/L, with no difference between the groups (Loy et al. 1986). In a similar study, moderately trained cyclists fasted for 36 h (fasted group) or ate a normal dietary menu on the evening before (fed group) an endurance exercise bout (Maughan and Gleeson 1988). Fasting resulted in significantly higher pre-exercise free fatty acid values (~1.2 mmol/L) compared with the values of the fed group (~0.5 mmol/L). Regardless of the fed or fasting state, plasma free fatty acids increased during the endurance exercise bout.

In summary, fasting (at least 24 h prior to endurance exercise) elevates resting, pre-exercise free fatty acid concentrations. Despite this favorable metabolic environment, pre-exercise fasting does not improve endurance performance. Thus, in spite of increased fatty acid availability, fasting does not improve endurance exercise performance and should not be recommended. Table 2.1 summarizes the research designs and endurance performance outcomes in studies comparing fasting states to postabsorptive and normal fed states.

2.2.2.2 Pre-Exercise High-Fat Meal

Ingesting a high-fat meal prior to endurance exercise serves to increase fat oxidation (and suppress carbohydrate oxidation) during the subsequent endurance exercise bout (Vukovich et al. 1993; Costill et al. 1977). If accomplished, this would spare glycogen and theoretically improve endurance exercise performance. It has been reported that increasing plasma fatty acids prior to endurance exercise does result in sparing muscle glycogen, but the researchers infused triglyceride emulsions and a compound known as heparin to induce elevations in plasma free fatty acids (rather than simply ingesting a pre-exercise high-fat meal) (Costill et al. 1977). Utilization of lipid–heparin solutions produces a very marked increase in fatty acid availability (a two- to fivefold increase) (Helge 2000). However, performance was not measured in these studies (Dyck et al. 1993; Vukovich et al. 1993; Hargreaves, Kiens, and Richter 1991) and it is obvious that this procedure is not applicable for athletes, but merely a laboratory procedure to manipulate substrate availability (Helge 2000).

In contrast to the use of a triglyceride emulsion and heparin, several studies have investigated elevations in plasma free fatty acids following a high-fat meal prior to endurance exercise. In addition to investigating fuel oxidation, an assessment of endurance exercise performance was also performed. In each of the studies discussed later, the athletes under investigation did not have compromised carbohydrate stores at the beginning of exercise. In one of these studies, trained cyclists and triathletes ingested a high-fat or a high-carbohydrate meal 90 min before an endurance exercise test in a double blind, randomized crossover design (Rowlands and Hopkins 2002). The exercise test consisted of a 1-h preload cycling bout at 55% peak power followed by a 50-min cycling test that consisted of five incremental loads ranging from 55% to 82% peak power. Following the incremental exercise test, a final 50-km time trial was performed. The purpose of the preload and incremental tests was to evaluate the metabolic effects of the high-fat versus high-carbohydrate meals. The purpose of the 50-km time trial was to assess whether the pre-exercise meals were able to influence endurance performance.

The high-carbohydrate and high-fat meals were isoenergetic and provided 15 calories/kg body mass (or an average of about 1100 calories/cyclist). The high-fat meal contained 5% carbohydrate, 10% protein, and 85% fat; the high-carbohydrate meal contained 85% carbohydrate, 10% protein, and 5% fat. In addition to the pre-exercise meal, but after the initiation of the exercise bout, cyclists consumed approximately 57 g of a carbohydrate beverage supplement per hour.

Fat oxidation increased during the 1-h preload cycling bout following ingestion of both the high-fat and high-carbohydrate meals. Toward the end of this segment, fat oxidation was significantly lower in the high-carbohydrate meal as compared to the

TABLE 2.1
Effects of Fasting on Endurance Performance

Study (Year)	Population	Design	Measurements	Findings
Dohm et al. (1986)	Nine physically conditioned male runners	Crossover design separated by 2 weeks Two treatments: • Fed treatment = ingested ~500 kcal 2–4 h prior to exercise bout • Fasting treatment = fasted 23 h prior to exercise bout	Participants ran at ~71% VO_2max until exhaustion	Endurance performance decreased by ~8% after the fast as compared to the fed condition (significant difference not reported)
Loy et al. (1986)	Ten competitive male cyclists	Crossover design separated by 1 week Two treatments: • Fed treatment = ingested 355 kcal 3 h prior to exercise bout • Fasting treatment = fasted 24 h prior to exercise bout	Cycling to fatigue; cycling at an initial 86% VO_2max; fatigue was determined when pedaling frequency could not be maintained at 65% VO_2max	Endurance performance significantly decreased by ~15% after the fast as compared to the fed condition
Loy et al. (1986)	Ten competitive male cyclists	Crossover design separated by 1 week Two treatments: • Fed treatment = ingested 355 kcal 3 h prior to exercise bout • Fasting treatment = fasted 24 h prior to exercise bout	Cycling to fatigue; cycling at an initial 79% VO_2max; fatigue was determined when pedaling frequency could not be maintained at 65% VO_2max	Endurance performance significantly decreased by ~22% after the fast as compared to the fed condition
Gleeson et al. (1988)	Six physically active males	Crossover design separated by 1 week Two treatments: • Fed treatment = ingested ~750 kcal 4 h prior to exercise bout • Fasting treatment = fasted 24 h prior to exercise bout	Cycle ergometer exercise performed to exhaustion at 100% VO_2max; exhaustion was defined as the point at which exercise could not be continued	Endurance performance significantly decreased by ~13% after the fast as compared to the fed condition

Maughan and Gleeson (1988)	Five physically active males	Crossover design separated by 1 week Two treatments: • Fed treatment = ingested food the evening before the morning exercise bout (postabsorptive state) • Fasting treatment = fasted 36 h prior to exercise bout	Cycle ergometer exercise performed to exhaustion at 70% VO_2max; exhaustion was defined as local muscular fatigue and dyspnea	Endurance performance significantly decreased by ~35% after the fast as compared to the fed condition
Knapik et al. (1988)	Eight healthy, nontrained male soldiers	Crossover design separated by 2–5 weeks Two treatments: • Fed treatment = ingested ~500 kcal 2–4 h prior to exercise bout • Fasting treatment = fasted 23 h prior to exercise bout	Participants ran at ~71% VO_2max until exhaustion	Endurance performance decreased by 15% after the fast as compared to the fed condition
Zinker et al. (1990)	Seven healthy males	Crossover design separated by 2 weeks Two treatments: • Fed treatment = ingested food the evening before the morning exercise bout (postabsorptive state) • Fasting treatment = fasted 36 h prior to exercise bout	Cycle to exhaustion at ~50 VO_2max; exhaustion was defined to be the moment when the work load could no longer be maintained	Endurance performance significantly decreased by 38% after the fast as compared to the fed condition

high-fat meal. Similarly, fat oxidation was also significantly lower in the high-carbohydrate group (as compared to the high-fat group) during the incremental cycling test. The authors stated that the likely reason for the reduction in fat oxidation was the pre-exercise plasma–insulin concentration that was observed following the high-carbohydrate meal (about 2.5 times higher than the high-fat meal). After the exercise bout was initiated, insulin concentrations declined in both groups to similar levels. Despite the substantial effects on plasma hormone concentrations (i.e., insulin) and fuel utilization, the pre-exercise meals had no effect on the 50-km time trial performance. While reporting a clear elevation in fat oxidation following the high-fat meal, the authors stated it is possible that any potential differences in performance relative to the high-carbohydrate meal were minimized by the maintenance of blood-glucose concentration associated with the ingestion of the carbohydrate supplement that was permitted during the exercise bout (Rowlands and Hopkins 2002). While this may be true, any study design that does not allow for carbohydrate ingestion during endurance exercise would not be simulating what endurance athletes practice during competitive races.

Another study utilizing endurance athletes (cyclists and triathletes) reported similar findings (Paul et al. 2003). Approximately 3.5 h before exercise, endurance athletes ingested a carbohydrate-rich meal, a fat-rich meal, or a calorie-free placebo beverage. The investigation was carried out in a randomized, double blind, crossover format, with 5–10 days separating each trial. The high-carbohydrate meal provided 3 g carbohydrate/kg body weight (an average of nearly 225 g or 900 calories carbohydrate) and the high-fat meal provided 1.3 g fat/kg body mass (an average of nearly 100 g or 900 calories fat). Three and one-half hours after consuming one of the test meals, the athletes exercised on a cycle ergometer for 30 min at an intensity that was 25 W above their lactate thresholds. The endurance athletes then rested for 15 min prior to completing a 20-km time trial.

The pre-exercise meals did not exert a significant effect on carbohydrate oxidation rate at any time during exercise (although the carbohydrate oxidation rate for the carbohydrate-rich meal was approximately 13% and 17% greater than the fat-rich and placebo meals, respectively). Conversely, fat oxidation was significantly higher in the fat-rich and calorie-free placebo meals during the last 5 min of the 30-min lactate-threshold phase and during the 20-km time trial. Fat oxidation rates were significantly higher for the fat-rich and placebo meals when compared to the carbohydrate-rich meal (about 75% greater than the carbohydrate-rich meal). Not surprisingly, insulin concentrations were significantly higher after the carbohydrate-rich meal, but not following the fat-rich or placebo meals. As could be predicted from high insulin levels, serum free fatty acids and glycerol concentrations (markers of lipolysis) were significantly lower in the carbohydrate-rich meal as compared to the fat-rich and placebo meals. Insulin is one of the primary hormones regulating lipolysis (exerting an inhibitory effect on lipolysis). Despite the dietary-induced changes in carbohydrate and fat oxidation rates, there were no significant differences among treatments for the 20-km time trial. While this may be surprising considering that one of the treatments included a noncaloric placebo meal, it is important to remember that the endurance athletes did not have compromised carbohydrate stores at the beginning of exercise. Rather, they were adequately nourished and hydrated.

Taken together, these studies (Paul et al. 2003; Rowlands and Hopkins 2002) indicate that ingesting a high-fat meal (in comparison to a high-carbohydrate meal) ranging from 90 min to 3.5 h prior to endurance exercise significantly increases fat oxidation. However, these substrate oxidation changes are not associated with an improvement in endurance performance. High-fat meals ingested prior to exercise/competition should be carefully considered. In a survey examining the relationship between gastrointestinal symptoms and dietary intake in triathletes competing in a half Iron Man triathlon, it was reported that all who had eaten within 30 min of the start vomited while swimming (Rehrer et al. 1992). Due to both gastrointestinal issues and the observation that endurance performance is not improved, it is not recommended that endurance athletes eat a high-fat meal prior to exercise.

2.2.3 Fat Loading

Carbohydrate stores are very limited in the body and can be depleted by approximately 3 h of continuous exercise. A depletion of carbohydrate stores during endurance exercise results in fatigue and decreases endurance exercise performance. Fat stores, on the other hand, are very large and are able to fuel activity for several days. A nutritional strategy known as "fat loading" seeks to fuel endurance activity (i.e., resynthesize ATP) primarily from fat stores by enhancing fat metabolism and sparing glycogen stores (Zderic et al. 2004). Fat loading has repeatedly been shown to alter substrate utilization by increasing fat oxidation at rest and during endurance exercise (Yeo et al. 2008; Stellingwerff et al. 2005; Staudacher et al. 2001). One reason that may explain the changes in fat metabolism following a period of ingesting a high-fat diet is reduced pyruvate dehydrogenase activation after a fat adaptation period (Stellingwerff et al. 2005). This finding could indicate that the increase in fat oxidation is at least partly caused by a reduction in the ability to oxidize carbohydrate because pyruvate dehydrogenase is a key enzyme in carbohydrate metabolism catalyzing the conversion of pyruvate to acetyl-CoA in the mitochondria, and thus controlling the entry of substrate into the tricarboxylic acid (TCA) cycle (Jeukendrup and Gleeson 2010).

Fat-loading strategies can also increase intramuscular triglyceride stores and subsequently increase the activity of "fat-burning" enzymes. This adaptation would be particularly important for endurance athletes, who have an increased capacity to store intramuscular triglyceride stores as compared to nonexercisers (Lowery 2011; van Loon et al. 2004). Raising the roughly 350 g of intramuscular triglyceride stores would appear advantageous regarding simple fuel supply. To test the hypothesis of increasing intramuscular triglyceride stores via dietary changes, six endurance-trained male cyclists ingested two diets: one that was high in fat and another that was lower in fat. The high-fat diet was composed of 60% fat, 24% carbohydrate, and 16% protein. The control diet was composed of 22% fat, 65% carbohydrate, and 13% protein. Both diets were ingested for 2 days and intramuscular triglyceride concentrations were measured before and after the 2-day dietary period.

Intramuscular triglyceride concentration was increased by 36% after the high-fat diet as compared to the lower fat control diet (Zderic et al. 2004). This (Zderic et al. 2004) and other (Tamura et al. 2008; Yeo et al. 2008) studies demonstrate that a short period of a high-fat diet can increase intramuscular triglyceride stores. In

contrast, when a low-fat diet is consumed for a few days, intramuscular triglyceride stores decrease. For example, seven endurance-trained cyclists were studied over a 3-week period. During the first week, all cyclists ingested a diet that provided 32% of energy from dietary fat. During the second and third weeks, they were randomly assigned to consume a diet that provided either 2% or 22% of energy from dietary fat. Intramuscular triglyceride concentration associated with the 2% fat diet was significantly reduced (by 21%) as compared with the 22% fat diet (Coyle et al. 2001). These two aforementioned studies (Zderic et al. 2004; Coyle et al. 2001) illustrate the impact that high- and low-fat diets have on intramuscular triglyceride content. Proponents of high-fat diets point to the fact that such diets do more than simply increase intramuscular triglyceride stores: They also increase lipolysis and fat oxidation during endurance exercise (Zderic et al. 2004; Coyle et al. 2001).

Clearly, skeletal muscle is highly sensitive to the dietary fat content of the diet due to the altering amounts of triglyceride and glycogen stores within its fibers. Though high-fat diets elevate intramuscular triglycerides, lipolysis, and fat oxidation during endurance exercise, there is a metabolic drawback to such diets. Several studies have reported that high-fat diets result in a significant reduction of skeletal muscle glycogen levels. Research from the 1930s demonstrated that short-term exposure to a high-fat diet resulted in impaired fatigue resistance (Jeukendrup and Gleeson 2010; Christensen and Hanson 1939). Later, muscle biopsy studies demonstrated that high-fat, low-carbohydrate diets resulted in decreased muscle glycogen levels and that this was the main factor causing lack of fatigue resistance during prolonged exercise (Jeukendrup and Gleeson 2010; Bergstrom and Hultman 1967; Hultman 1967).

Recent research has also confirmed these earlier reports. Endurance-trained cyclists ingested one of two isocaloric diets that differed in their fat and carbohydrate content. Both diets were consumed for a 1-week period and consisted of 22% fat, 68% carbohydrate, and 10% protein, or 2% fat, 88% carbohydrate, and 10% protein (Coyle et al. 2001). Skeletal muscle glycogen was 18% greater in the diet with the very low fat as compared to the higher fat diet. Similarly, when endurance-trained male cyclists ingested a high-fat (60% of total energy) or a low-fat (22% of total calories) diet for 2 days, resting muscle glycogen concentration was significantly lowered by 50% with the high-fat diet (Zderic et al. 2004).

To offset the negative effects that ingesting high-fat diets has on skeletal muscle glycogen, a short period of specifically timed high carbohydrate ingestion has been proposed (Helge 2000). A period of adaptation to a high-fat diet, followed by acute carbohydrate feeding, might theoretically induce the enzymatic adaptations in the muscle (favoring the oxidation of fat at submaximal exercise intensities) while also allowing for the optimization of pre-exercise glycogen stores. This strategy is typically associated with eating high amounts of fat (~60%–70% of energy in the diet) and relatively low intakes of carbohydrate for several days or several weeks, followed by a short period of eating high carbohydrate amounts (~70%–90% of energy in the diet) immediately prior to a competition.

2.2.3.1 Endurance Exercise Performance

High-fat diets are sometimes categorized as short term and long term. Short term typically implies the ingestion of a high-fat diet for period of time less than 2 weeks.

In contrast, long-term fat loading includes patterns of ingesting high-fat diets for greater than 2 weeks. In one long-term fat-loading investigation, 11 competitive duathletes ingested either a high-fat (with no subsequent period of carbohydrate loading) or high-carbohydrate diet for 5-week periods (Vogt et al. 2003). Specifically, the duathletes ingested either a high-fat diet (53% of energy from fat) or a low-fat, high-carbohydrate diet (17% fat; 68% carbohydrate) separated by a 2-week washout period. After the 5-week dietary treatments were completed, each duathlete participated in a half marathon (21 km) race.

The mean running time for the half-marathon was not significantly different between the two dietary treatments. Those on the high-fat diet treatment completed the half marathon in 80.2 min and those on the high-carbohydrate diet treatment completed the race in 80.4 min. The investigators also measured intramuscular triglyceride and skeletal muscle glycogen content before and after the dietary treatments. There was a statistically significant 2.3-fold higher intramuscular triglyceride content after the 5-week high-fat diet. Surprisingly, there was no significant difference in skeletal muscle glycogen content between the two dietary treatments. Reasons given for the lack of skeletal muscle glycogen depletion were that the fat content in the high-fat diet (53% of calories) was less than the typical proportions used in other investigations (typically greater than 60%). Also, the exercise training intensities of the participants during the 5-week intervention periods were relatively low in intensity (60%–65% of VO_2max). Such intensities are not primarily fueled by carbohydrate oxidation. Most, but not all (Lambert et al. 1994) long-term fat-loading investigations have also reported no effect on endurance performance (Goedecke et al. 1999; Pogliaghi and Veicsteinas 1999; Helge, Wulff, and Kiens 1998), while others have reported reductions in endurance exercise performance (Fleming et al. 2003; Helge, Richter, and Kiens 1996; Pruett 1970).

One hypothesis explaining why longer term high-fat diets do not work (and in some instances suppress endurance performance) is due to suboptimal adaptations to the training program while following a high-fat diet. Endurance athletes ingesting a normal diet that is relatively high in carbohydrates are able to adapt to the stimulus that the endurance training imposes on the body. By continually replenishing liver and skeletal muscle glycogen stores after training, they are able to enter the next training session fully recovered and consequently are able to train at progressively higher exercise intensities. In contrast, consuming a chronic high-fat diet (with low levels of carbohydrate) may not replenish glycogen stores and theoretically will reduce the intensity of subsequent training sessions and ultimately suppress endurance-training adaptations.

Adaptation to a fat-rich diet and its effects on exercise performance is influenced by several factors, including the duration of the fat adaptation period and the relative contributions of fat and carbohydrate in the diet (Helge 2000). Nearly all of the fat-loading investigations conducted in the past 15 years have utilized methodologies that included a period of ingesting a high-fat diet (~5–14 days) followed by a shorter time period (1–3 days) of carbohydrate loading. Table 2.2 summarizes much of the scientific literature investigating short-term high-fat diets followed by carbohydrate loading and their cumulative effect on endurance exercise performance.

TABLE 2.2
Short-Term High-Fat Diets Followed by Carbohydrate Loading

Study (Year)	Population	Design	Measurements	Findings
Stellingwerff et al. (2006)	Seven endurance-trained cyclists and triathletes	Crossover with a 2-week washout period Two 6-day treatments: • High-fat diet for 5 days (67% fat) followed by 1 day of CHO[a] loading (70% CHO) • High-CHO diet for 6 days (70% CHO)	Cycling time trial (4 kJ/kg body mass) to be completed as fast as possible (time trial followed a 20-min steady-state cycling at 70% VO_2max and a 1-min sprint); tests conducted on day 7	No significant difference between treatments in time trial performance (~13.2 min for each treatment)
Havemann et al. (2006)	Eight endurance-trained male cyclists	Crossover with a 2-week washout period Two 7-day isocaloric treatments: • High-fat diet for 6 days (~68% fat) +1 day of CHO loading (~90% CHO) • High-CHO diet for 6 days (~68% CHO) + 1 day of CHO loading (~90%CHO)	100-km cycling time trial conducted on day 8 of study	No significant difference between treatments in time trial performance (mean performance time was ~2.5% slower on the high-fat compared with the high-CHO diet)
Rowlands and Hopkins (2002)	Seven nationally competitive male cyclists and triathletes	Crossover with a 2-week washout period Three 14-day isocaloric treatments • High-fat diet (66% fat) • High-CHO diet (70% CHO) • 11.5-day high-fat diet (65% fat) followed by 2.5-day CHO loading diet (63% CHO)	100-km time trial that followed a 15 min test (aim was to cycle as far as possible); a 45-min steady-state (50% peak power) cycling bout; and a 1-h incremental test (total exercise time = 5 h)	No significant difference between treatments for 100-km time trial (high-fat diet slightly improved performance; high-CHO diet slightly decreased performance)

Burke et al. (2002)	Eight trained male cyclists or triathletes	Crossover with a 2-week washout period Two 6-day isocaloric treatments: • High-fat diet for 5 days (70% fat) +1 day of CHO loading (10 g/kg). • High-CHO diet for 5 days (70% CHO) + 1 day of CHO loading (10 g/kg)	Cycling time trial (7 kJ/kg body mass) to be completed as fast as possible (time trial followed 20 min of steady-state cycling at 70% VO_2max and a 1-min sprint); tests conducted on day 7	No significant difference between treatments in time trial performance (~25 min for each treatment)
Lambert et al. (2001)	Five endurance-trained male cyclists	Crossover with a 2-week washout period Two 13-day isocaloric treatments: • High fat diet (>65% fat) for 10 days + 3 days of a high-CHO diet (>65% CHO) • Habitual diet for 10 days (~50% CHO; 30% fat) + 3 days of a high-CHO diet (>65% CHO)	20-km cycling time trial (time trial followed 150-min cycle ride at 70% VO_2peak); time trial conducted on day 14.	High-fat diet improved the 20-km time trial performance by ~4% (a significant difference)
Carey et al. (2001)	Seven trained male cyclists or triathletes	Crossover with an 18-day washout period Two 8-day isocaloric treatments: • High-fat diet (69% fat) for 7 days + 1 day of a high-CHO diet (70% CHO) • High-CHO diet for 8 days (70% CHO)	1-h cycling time trial (time trial followed 4 h of cycling at 65% VO_2peak). Performance measured by distance covered in 1 h	No significant difference between treatments in distance covered during the 1-h time trial (high-fat treatment improved performance by ~4%)
Burke et al. (2000)	Eight trained male cyclists or triathletes	Crossover with a 2-week washout period Two 6-day isocaloric treatments: • High-fat diet for 5 days (>65% fat) +1 day of CHO loading (10 g/kg) • High-CHO diet for 5 days (70%–75% CHO) + 1 day of CHO loading (10 g/kg)	Cycling time trial (7 kJ/kg body mass) to be completed as fast as possible (time trial followed 2 h of cycling at 70% VO_2max); time trial conducted on day 7	No significant difference between treatments in time trial performance (however, mean time trial time was 8% faster with the high-fat treatment)

[a] CHO = carbohydrate.

2.2.3.2 Anaerobic Exercise Performance and Ratings of Perceived Exertion

Few studies have investigated the effects of fat loading on high-intensity, anaerobic exercise performance. Given the reality that high-intensity exercise is primarily fueled by carbohydrate, it is unlikely that changes in dietary fat intakes, such as fat loading, would have any effect on anaerobic exercise performance. One study investigated the effects of a high-fat diet on two Wingate tests (Fleming et al. 2003). A Wingate test is a cycle test in which the subject pedals as fast as he or she can for 30 s against a resistance that is ~7.5% of the subject's body weight. The main variables assessed during a Wingate test are peak power (occurs within the first seconds of the test), mean power (average power produced throughout the 30-s test), and fatigue index (percent fatigue).

In this study, 20 recreationally active men were assigned to ingest a high-fat diet or their habitual moderate- to high-carbohydrate diet for 6 weeks. The high-fat diet provided ~61% fat, 8% carbohydrate, and 30% protein. The habitual diet provided ~25% fat, 59% carbohydrate, and 15% protein. After the 6-week high-fat diet, no changes were observed in relation to mean power and fatigue when adjusted for body mass. However, peak power output was significantly decreased in the high-fat diet group (~7% reduction in peak power), but was unchanged in the habitual diet group (Fleming et al. 2003). A 7% reduction in power output is substantial for strength-power athletes who depend upon explosive power output for performance in their respective sports.

In a similar study, five trained cyclists underwent a randomly assigned 14-day high-fat or high-carbohydrate diet (Lambert et al. 1994). The high-fat diet contained ~67% fat, 7% carbohydrate, and 25% protein; and the high-carbohydrate diet contained ~74% carbohydrate, 12% fat, and 14% protein. The diets were separated by a 2-week washout period in which each athlete ingested a normal diet. In addition to a Wingate test, the investigators also included a high-intensity cycling test to exhaustion at approximately 90% VO_2max. It was reported that there were no significant differences between the two dietary treatments in terms of peak power production during the Wingate test (high-fat treatment produced 3.8% more peak power than the high-carbohydrate treatment, when adjusted for body mass) and time to exhaustion during the high-intensity cycling test (the high-carbohydrate treatment was able to exercise 50% longer [12.5 min] as compared to the high-fat treatment [8.3 min]). In consideration of these reports, it is clear that high-fat diets are not recommended for athletes relying upon their ability to produce high-power output levels.

In addition to performance assessments, ratings of perceived exertion have also been investigated in conjunction with fat-loading diets. Seven trained cyclists and triathletes undertook two 3-day dietary treatments in a randomized, crossover design with an 18-day washout period separating each diet (Stepto et al. 2002). Each athlete was prescribed either a high-fat (65% of energy) or an isoenergetic high-carbohydrate (70%–75% of energy) diet. On the third and final day of each treatment, the athletes completed a standardized laboratory training session consisting of a 20-min warmup at 65% of VO_2peak immediately followed by eight sets of 5-min work bouts at 86% VO_2peak with 1 min of recovery between each set. This laboratory session was structured to mimic workouts performed by ultra-endurance athletes during a taper.

Ratings of perceived effort of the legs were significantly elevated by 16% in the high-fat treatment as compared to the high-carbohydrate treatment (Stepto et al. 2002). Other investigations have also reported similar findings. During a maximal oxygen consumption test that followed a 6-week high-fat (61% of calories) diet, it was reported that ratings of perceived exertion were significantly elevated as compared to baseline values (Fleming et al. 2003). No changes in ratings of perceived exertion were observed following a control diet. Further, 1 h of steady-state cycling at 70% VO_2peak nonsignificantly elevated ratings of perceived exertion by approximately 6% in endurance-trained male cyclists (Havemann et al. 2006).

2.2.3.3 Fat-Loading Summary

Although the hypothesis that high-fat diets/fat loading may increase the capacity to oxidize fat and improve endurance performance during competition is attractive, little evidence indicates that it is true (Sherman and Leenders 1995). Most of the studies investigating fat loading on endurance performance have reported no endurance exercise improvement. The available studies that indicate a positive effect on performance were conducted at intensities lower than typical intensities during competition (Lambert et al. 1994; Jeukendrup and Gleeson 2010). In such situations, there is little reliance on carbohydrate to fuel the activity. Therefore, if a period of ingesting a high-fat diet does compromise carbohydrate stores, it may be masked because of the relatively low exercise intensities used in the investigation to study the performance-enhancing effects of fat-loading diets.

While only a few studies have investigated anaerobic exercise performance, it has been reported that power output is reduced following a high-fat, reduced-carbohydrate diet (Fleming et al. 2003; Lambert et al. 1994). Clearly, for athletes needing a high level of power output, high-fat diets are not recommended. Also, there is an elevated rating of perceived exertion with fat-loading strategies in endurance athletes. For these reasons, fat-loading dietary strategies are not recommended to improve exercise performance.

2.3 FATTY ACID-CONTAINING SUPPLEMENTS

The remainder of this chapter will outline research that either supports or refutes the usage of different fatty acids or fatty acid-containing supplements with the intent of improving body composition and/or athletic performance. While numerous fatty acids exist, this chapter will discuss the efficacy of essential fatty acids, conjugated linoleic acid (CLA), and medium chain triglycerides (MCTs).

2.3.1 Essential Fatty Acids

Essential fatty acids are fatty acids that cannot be synthesized at appreciable levels by the body and thus must be supplemented in the diet. The two essential fatty acids (EFAs) for humans include α-linolenic acid (omega-3) and linoleic acid (omega-6) (Figure 2.1). Common physiological functions of these fatty acids include:

- Comprising a substantial amount of the lipid material in cell membranes in all tissue types

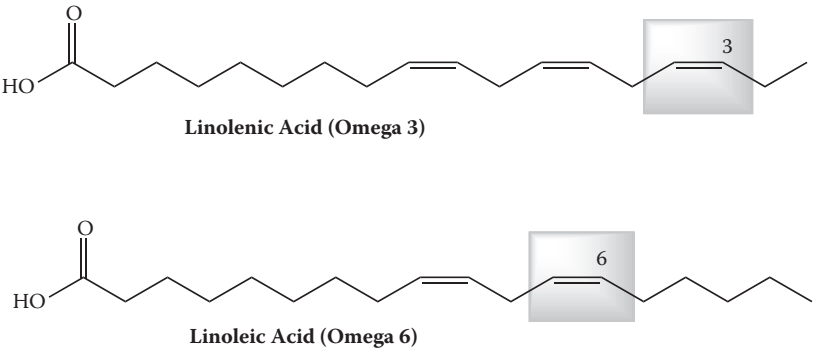

FIGURE 2.1 Omega-3 and omega-6 fatty acids.

- Forming signaling molecules such as eicosanoids, which regulate inflammation, blood clotting, and tissue repair following injury
- Serving as antioxidants (numerous human studies have determined that supplementing with omega-3 fatty acids reduces systemic markers of oxidative stress) (Visioli et al. 2012)

A plausible mechanism whereby omega-3 fatty acids exhibit their physiological effects may lie in their ability to replace more pro-inflammatory fatty acids (such as linoleic acid or arachidonic acid) within cell membrane structures (Nakamura, Flintoff-Dye, and Omaye 2008). Linoleic acid is found in vegetable oils (safflower oil, sunflower oil, and corn oil), almonds, and walnuts. Alpha-linolenic acid is found in leafy green vegetables, seafood, and walnuts. Fish oils contain two α-linolenic acid fatty acids: eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA).

Regarding athletic performance, the research on fish oil and EPA and DHA is in its infancy and concrete recommendations cannot be made at this point. The limited amount of research that has been conducted is promising, but much of the work has been conducted in nonathletic populations. For example, a recent study reported that 2 g/day fish oil supplementation increased strength gains and skeletal muscle activation patterns in older women who resistance trained when compared to a placebo group (Rodacki et al. 2012).

Supplementing older males with 14 g/day flax oil (containing α-linolenic acid) in conjunction with resistance training for 12 weeks also elicited significant increases in quadriceps muscle mass compared to a placebo group (Cornish and Chilibeck 2009). The increase in muscle mass observed in this study may have been due to an increased rate of muscle protein synthesis. Smith and colleagues (2011) reported that omega-3 fatty acids (providing 1.86 g of EPA and 1.5 g of DHA) significantly stimulated the rate of muscle protein synthesis as compared to a corn oil supplement in an older adult population.

Another study reported that healthy, active adults (aged 18–55 years) experienced favorable body composition changes following fish oil supplementation (Noreen et al. 2010). For a 6-week period, the subjects ingested either 4 g/day of a fish oil

supplement (containing 1.6 g of EPA and 800 mg of DHA) or 4 g/day safflower oil (acting as a placebo). At the end of the 6-week intervention, it was reported that the fish oil group significantly increased fat-free mass (1.1 lb. [0.5 kg]) and significantly reduced fat mass (–1.1 lb. [0.5 kg]) as compared to the safflower oil group (who lost 0.22 lb. [–0.1 kg] of fat-free mass and gained 0.4 lb. [0.2 kg] of fat mass). While all of these studies have reported favorable outcomes with omega-3 fatty acids/EPA-DHA supplementation, they were unfortunately conducted in nonathletic populations. So, while omega-3 fatty acid supplementation clearly has some benefits in healthy, older populations, there is still some uncertainty regarding its effects in athletic populations.

Tartibian et al. (2010) instructed amateur male wrestlers to ingest 1 g of omega-3 fatty acids (providing 180 mg EPA and 120 mg DHA) or a placebo for a 12-week period while they engaged in incremental wrestling training. At the end of the intervention, it was reported that omega-3 fatty acid supplementation significantly improved the pulmonary function of the athletes during exercise and after exercise. While pulmonary function was improved with omega-3 supplementation, no measures of exercise performance were conducted in this study. Several other studies have assessed changes in performance over time in conjunction with omega-3 fatty acids, but without success (Mickleborough 2013). When soccer players supplemented with omega-3 fatty acids (providing 1.6 g/day EPA and 1.04 g/day DHA), there was no effect on aerobic power or running performance (Raastad, Høstmark, and Strømme 1997). When Australian Rules football players supplemented with omega-3 fatty acids (providing 0.36 g/day EPA and 1.56 g/day DHA) for a 5-week period, there was no improvement in endurance exercise performance or recovery. Lastly, when trained cyclists ingested 2.4 g/day fish oil (providing 2 g/day EPA and 0.4 g/day DHA) over a 6-week period, there was no improvement in a 10-km time trial performance ride as compared to a placebo supplement (Nieman et al. 2009). Taking each of these studies into consideration, the data do not support the hypothesis that omega-3 fatty acid supplementation is effective in enhancing exercise performance (Mickleborough 2013).

2.3.2 Conjugated Linoleic Acid

Conjugated linoleic acid (CLA) is a family of roughly 30 isomers (compounds with similar chemical formulas but different molecular structure) of the omega-6 linoleic acid (pictured in Figure 2.1) family. Two common isomers of CLA include *trans*-10,*cis*-12 CLA and *cis*-9,*trans*-11 CLA. Beef, lamb, and dairy products are rich in CLA. The potential benefits that CLA may provide the athlete are not directly related to exercise performance, but rather to CLA's potential to improve body composition, particularly fat loss. Unfortunately, nearly all of the scientific investigations in which CLA has been studied have been in overweight/obese populations. Whigham, Watras, and Schoeller (2007) have combined data from numerous studies to contend that 3.2 g/day CLA reduces fat mass by approximately 90 g/week (or 1 lb. [0.45 kg] in 5 weeks). The authors also suggest that doses higher than 3.2 g/day do not appear to have additional effects on reducing body fat. The manner in which CLA reduces

body fat has been thoroughly researched and may include one or a combination of the following (Dugan, Aalhus, and Kramer 2004):

1. The potential ability of CLA to induce adipocyte, or fat cell, apoptosis
2. The ability of CLA to inhibit the adipocyte triglyceride uptake and storage
3. The ability of CLA to increase fatty acid oxidation
4. The ability of CLA to increase energy expenditure

CLA also appears to possess the remarkable ability of increasing muscle mass while reducing fat mass in those supplementing with this ingredient. Steck et al. (2007) examined the effects of supplementing obese humans with 3.2 g/day CLA, 6.4 g/day CLA, or 8 g of safflower oil (placebo condition) over 12 weeks. Authors reported a 1.4 lb. (0.64 kg) increase in lean body mass in the 6.4 g/day CLA group. Blankson et al. (2000) similarly determined that 6.8 g/day CLA supplementation elicited a nearly 2 lb. (0.9 kg) increase in lean body mass in obese individuals following 12 weeks of supplementation with resistance training while 1.7 g/day, 3.4 g/day, and 5.1 g/day did not statistically increase lean body mass following this intervention. Mechanisms whereby CLA supplementation increases muscle mass may be due to the fact that CLA isomers increase anabolic signaling pathways, which are known to increase muscle protein synthesis (i.e., phosphatidylinositol 3-kinase activation) (Mohankumar et al. 2013).

The limited data presented earlier suggests that supplementing with ~3–6 g/day CLA possesses potential health benefits, including: (1) increasing the reliance upon fat oxidation for fuel, which could lead to marginal weight loss (1 lb. [0.45 kg] in 5 weeks); and (2) increasing anabolic signaling in skeletal muscle, which could result in marginal weight gain (~1 lb. [0.45 kg] in 12 weeks). As stated previously, each of these studies was conducted in an overweight/obese population. Hence, extrapolating these findings to an athletic population and expecting similar results would be presumptuous.

2.3.3 Medium Chain Triglycerides

Medium chain triglycerides (MCTs) are triglycerides that contain two to three fatty acids that are 6–12 carbons in length. MCTs are found in abundance in both coconut oil and palm oil. Given their structural difference relative to conventional triglycerides (Figure 2.2), MCTs are easily absorbed into the bloodstream directly from the small intestine. In a sense, MCTs are fats that are metabolized like carbohydrates. Because of this attribute, researchers have investigated the efficacy of supplementing athletes with MCTs with the intent to improve quick energy production during prolonged endurance exercise.

Nosaka et al. (2009) fed recreationally trained athletes 6 g of MCT oil over a 2-week period and then had these individuals perform lower intensity cycling for 40 min followed by a higher intensity cycling bout until fatigue. The authors discovered that MCT oil supplementation achieved the following:

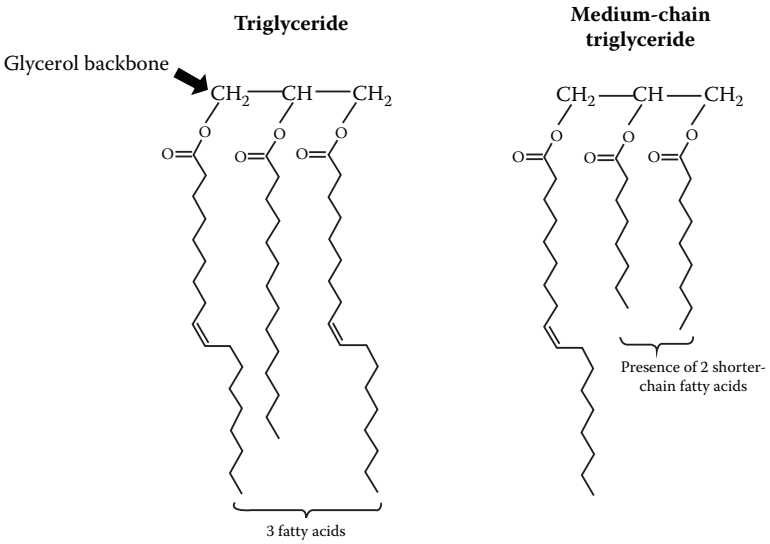

FIGURE 2.2 Difference between triglycerides and MCT.

a. Those that supplemented with 6 g/day MCT oil were able to cycle 4 min longer during the high-intensity bout than those that supplemented with longer chain triglycerides.
b. Blood lactate increases and perceived fatigue were lower in those that supplemented with MCT oil compared to those that supplemented with longer chain triglycerides.

However, prior evidence suggests that consuming 32 g of MCT oil prior to cycling exercise was less effective in aiding sprint performance compared to 75 g of carbohydrates and that MCT oil supplementation prior to exercise was associated with apparent increases in gastrointestinal distress during exercise (Goedecke et al. 2005). Likewise, similar evidence exists confirming the aforementioned study regarding the negative effects that 85 g of MCT oil exhibited on cycling performance (i.e., an 18% decrement) compared to the group that consumed 170 g of glucose (Jeukendrup et al. 1998). This study also reported that subjects consuming MCT oil experienced abdominal cramping. In summary, little evidence supports ingesting MCT immediately prior to or during exercise with the intent of increasing endurance performance.

2.4 CONCLUSION

Dietary fat intake recommendations for athletes and physically active individuals range from 20% to 35% of total calories. There is no research to support dietary fat intakes above or below this range in terms of improving endurance exercise performance. One strategy that some endurance athletes employ is to ingest a high-fat diet for several days to several weeks (approximately 70% fat) followed by a

very high-carbohydrate diet (approximately 70%–75% carbohydrate) for a few days leading up to an endurance event. Unfortunately, this type of short-term high-fat diet followed by carbohydrate loading does not result in improvements in endurance performance. On the other hand, employing this strategy does not negatively affect endurance performance either. In terms of fatty acid supplements, at present there does not appear to be any performance benefit from ingesting essential fatty acids, conjugated linoleic acid, or medium chain triglycerides.

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3 Carbohydrate Metabolism

Bill Campbell

CONTENTS

3.1 Introduction 45
3.2 Types of Carbohydrate 46
3.2.1 Monosaccharides 47
3.2.2 Disaccharides 48
3.2.3 Oligosaccharides 49
3.2.4 Polysaccharides 49
3.3 Carbohydrate Digestion and Absorption 49
3.3.1 Skeletal Muscle Glucose Uptake 50
3.3.2 Glycemic Index Versus Glycemic Load 50
3.4 Carbohydrate Metabolism 53
3.4.1 Blood Glucose Regulation 53
3.4.1.1 Phosphorylation of Glucose 54
3.4.2 Glycogen 54
3.4.2.1 Glycogen Synthesis 55
3.4.2.2 Glycogen Breakdown 56
3.4.3 Glycolysis 57
3.4.3.1 Carbohydrate Oxidation in Aerobic Metabolism 58
3.4.4 Lactate Formation and Metabolism 59
3.5 Carbohydrate Metabolism in Exercise 60
3.5.1 Crossover Concept 61
3.5.2 Skeletal Muscle Carbohydrate Metabolism during Exercise 62
3.5.3 Liver Glycogen Metabolism during Exercise 62
3.5.4 Control of Plasma Glucose during Exercise 63
3.5.5 Skeletal Muscle Glycogen Depletion 64
3.5.5.1 Aerobic/Endurance Exercise 64
3.5.5.2 Resistance Exercise 67
3.6 Conclusion 69
Acknowledgment 69
References 69

3.1 INTRODUCTION

Carbohydrates serve many important roles in the body and provide as much as 60% or more of the total energy in an endurance athlete's daily diet. Athletes are typically advised not only to eat a high-carbohydrate diet, but also to consume

carbohydrates before, during, and following exercise bouts in an attempt to replenish carbohydrate stores. The popularity of distance sports such as triathlons, marathons, and distance cycling has increased the attention that endurance athletes give to carbohydrates. As a result, extensive research on the importance of carbohydrates in the diet has been conducted since the 1970s. Such research has reported that during prolonged endurance events, severely reduced carbohydrate stores (muscle glycogen, liver glycogen, and blood glucose) are closely associated with fatigue and decreased endurance performance. Additionally, carbohydrate is important in the recovery phase (postexercise period) in terms of replenishing glycogen stores. Because of the importance of dietary carbohydrate, much research has focused on ways to optimize carbohydrate intake in an attempt to enhance carbohydrate oxidation and ultimately improve endurance performance in training and competition.

Carbohydrate availability may influence not only the performance of prolonged exercise, but also the performance of high-intensity exercise (such as resistance training). Carbohydrates are the only macronutrient whose stored energy generates adenosine triphosphate (ATP) anaerobically, which becomes important during maximal exercise that requires immediate energy release above levels supplied by aerobic metabolism. As an example, heavy-resistance exercise (an anaerobic activity) relies on carbohydrates as the primary nutrient to fuel the activity (Bloomer 2005; Binzen, Swan, and Manore 2001). Also, postexercise carbohydrate ingestion decreases skeletal muscle breakdown and amplifies the anabolic signaling pathways in skeletal muscle by virtue of carbohydrate's effects on secreting insulin. Both of these aspects of carbohydrate intake as it relates to optimizing resistance-training adaptations will be discussed in the following chapter.

In addition to fueling skeletal muscle, carbohydrates also serve as a fuel for the central nervous system (brain and spinal cord) and red blood cells. While nerve cells can use alternative fuel sources in limited quantities, red blood cells rely strictly on glucose. Glucose also represents the primary energy source for the brain (Pardridge 1983; Lund-Anderen 1979). It is often stated that nerve cells consume about 120 g glucose/day, which corresponds to a caloric expenditure of about 480 kcal (~2000 kJ). If this were to be true, this would serve as a lower level of daily carbohydrate intake for athletes to sustain optimal nervous system function.

This chapter discusses the various types of dietary carbohydrate and how these carbohydrates are digested and transported throughout the body. Next, an explanation of how ingested carbohydrate is stored in the body, as well as how it is liberated and utilized during periods of physical activity, is presented. Lastly, a discussion of carbohydrate metabolism is described to explain how various modes of exercise (such as endurance exercise and resistance exercise) influence its metabolism. Chapter 4 discusses how carbohydrate supplementation may be optimized to enhance exercise performance.

3.2 TYPES OF CARBOHYDRATE

As the name suggests, carbohydrates contain carbon and water with the general formula CH_2O. Dietary carbohydrates can be classified in a number of ways, including:

- The type of carbohydrate in the food
- The level of commercial processing that it has undergone
- The blood glucose or glycemic response to the carbohydrate within the body

One of the most common ways to classify carbohydrates is using the terms *simple* and *complex* carbohydrates. Sometimes simple carbohydrates are referred to as simple sugars and complex carbohydrates are referred to as starches. Simple carbohydrates are made up of only one or two carbohydrate molecules linked together, whereas complex carbohydrates are composed of longer and more complex chains of carbohydrates. Another classification scheme is to categorize carbohydrates into monosaccharides, disaccharides, oligosaccharides, and polysaccharides (Cummings and Englyst 1995). When combining the two aforementioned classification systems, simple sugars include both the monosaccharides and disaccharides, and complex sugars include oligosaccharides and polysaccharides. The following discussion outlines the primary carbohydrates found in the athlete's diet.

3.2.1 Monosaccharides

The monosaccharide represents the basic unit of carbohydrates. A monosaccharide is nothing more than a single molecule of sugar. The nutritionally important monosaccharides include glucose, fructose, and galactose (because they provide nutrients to the human body). Each of these monosaccharides has the same chemical formula ($C_6H_{12}O_6$), but possesses slightly different carbon-to-hydrogen-to-oxygen linkages (Figure 3.1). The bonding arrangements make fructose, galactose, and glucose unique in their function and different in their biochemical characteristics.

Glucose is the most typical sugar and contains 6 carbon, 12 hydrogen, and 6 oxygen atoms ($C_6H_{12}O_6$). It is the most important simple sugar for the human body and is the primary source of energy for the body's cells. Another name for dietary glucose is dextrose. Interestingly, glucose rarely exists as a monosaccharide in food but is joined with other sugars to form disaccharides and other complex carbohydrates. Glucose provides an immediate energy source as it can pass through the liver unchanged and enters the muscle for utilization or glycogen synthesis.

Fructose (also referred to as fruit sugar) occurs naturally in fruits and honey. Of the three monosaccharides, it has the sweetest taste. For this reason, it is incorporated as a potent sweetener in many commercially prepared breakfast and dessert products. The small intestine absorbs some fructose directly into the blood and the liver slowly converts it to glucose.

Unlike glucose, which can pass through the liver unchanged, galactose is converted to glucose by the liver and then is either stored as liver glycogen or released into the blood. Galactose does not occur freely in nature. Rather, it is combined with glucose to form the milk sugar lactose. In the body, galactose converts to glucose for energy metabolism. Figure 3.1 summarizes the individual monosaccharides that further bind together to form the disaccharides.

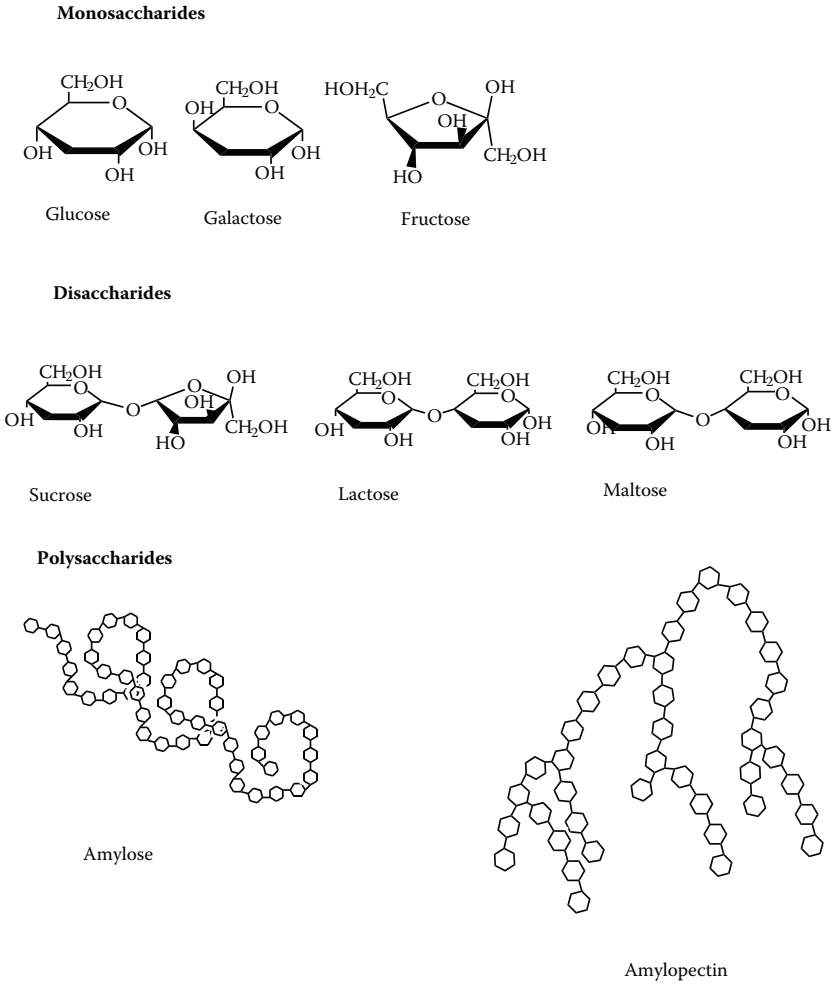

FIGURE 3.1 Saccharides.

3.2.2 Disaccharides

Combining two monosaccharides forms a disaccharide. Each disaccharide includes glucose as a principal component. The three disaccharides of nutritional significance include sucrose, lactose, and maltose. Sucrose is the most common dietary disaccharide, consisting of glucose and fructose. Foods that contain sucrose include table sugar, maple syrup, honey, and brown sugar. Lactose is found in natural form only in milk and comprises glucose and galactose. The other disaccharide, maltose, is composed of two glucose molecules. Maltose is present only in small amounts in an athlete's diet.

3.2.3 Oligosaccharides

Carbohydrates that are composed of short chains of three to nine linked sugars are known as oligosaccharides. Dietary sources of oligosaccharides are found in many vegetables. Manufactured oligosaccharides include maltodextrins and corn syrup. In particular, maltodextrin is a glucose polymer that is manufactured by breaking long starch units into smaller groups (some maltodextrins/glucose polymers are longer than nine linked monosaccharides and therefore are sometimes categorized as polysaccharides; see the following section). Maltodextrins are very common in sports drinks marketed for athletes.

3.2.4 Polysaccharides

Polysaccharides contain tens to thousands of monosaccharides combined in one molecule and are essentially the storage form of carbohydrate. Starch and fiber represent the two common forms of plant polysaccharides. Starch is the storage form of carbohydrate in plants. Plant starch is found in beans, potatoes, seeds, corn, and various grains that make bread, spaghetti, and pastries. Starch exists in two different forms: amylopectin and amylose (Figure 3.1). Amylopectin is a highly branched molecule consisting of a large number of glucose molecules, whereas amylose is a long chain of glucose molecules twisted into a helical coil. The predominance of one form or the other determines the "digestibility" of any food containing starch. Starches with a relatively large amount of amylopectin are rapidly digested and absorbed, whereas those with high amylose content are digested more slowly. Table 3.1 summarizes the classes of dietary carbohydrates.

3.3 CARBOHYDRATE DIGESTION AND ABSORPTION

Carbohydrates are ingested in the forms of polysaccharides (starches), disaccharides (sucrose, lactose, and maltose), and monosaccharides (glucose and fructose). Starch hydrolysis begins once food enters the mouth by the enzyme salivary amylase. Amylase is secreted by the salivary glands (and the pancreas) and its function is to break down polysaccharides into smaller linked glucose molecules and disaccharides. When the

TABLE 3.1
Carbohydrate Classifications

Simple Carbohydrates		Complex Carbohydrates	
Monosaccharides	Disaccharides	Oligosaccharides	Polysaccharides
Glucose	Sucrose	Maltodextrin[a]	Starch
Fructose	Lactose	Glucose polymers[a]	Fiber
Galactose	Maltose	Corn syrup	
		High fructose corn syrup	

[a] Some maltodextrins and glucose polymers are categorized as polysaccharides.

partially digested carbohydrate arrives in the acidic environment of the stomach, amylase activity decreases, leading to very little carbohydrate digestion in the stomach.

After reaching the stomach, partially digested carbohydrates are released into the small intestine. Most starch digestion occurs in the small intestine by pancreatic amylase. In the small intestine, pancreatic amylase, along with some other enzymes, completes the hydrolysis of starch into smaller branched chains of glucose molecules (oligosaccharides) and maltose. Enzymes on the surface of the small intestine complete the final stage of carbohydrate digestion to monosaccharide form. Specifically, sucrase breaks down sucrose to the simple sugars glucose and fructose, maltase degrades maltose to its glucose components, and lactase breaks down lactose to glucose and galactose.

The final products of carbohydrate digestion in the digestive tract are almost entirely glucose, fructose, and galactose—with glucose representing, on average, about 80% of these and galactose and fructose each representing no more than 10% of the products of carbohydrate digestion (Guyton and Hall 2000). Because glucose is the only carbohydrate that can be oxidized in muscle, much of the fructose and almost all of the galactose are transported to the liver, after absorption from the intestinal tract, and converted into glucose (Guyton and Hall 2000). The conversion of fructose and galactose occurs in the liver at relatively slow rates. Due to their conversion to glucose, little fructose and galactose are found in the circulating blood. Glucose thus becomes the final common pathway for transport of almost all carbohydrates to the tissue cells.

3.3.1 Skeletal Muscle Glucose Uptake

Before glucose can be used by skeletal muscle, it must be transported through the sarcolemma and into the cellular sarcoplasm. However, glucose cannot diffuse through the pores of the sarcolemma because the maximum molecular weight of particles that can diffuse readily is about 100, and glucose has a molecular weight of 180 (Guyton and Hall 2000). Hence, glucose is transported into the skeletal muscle via facilitated diffusion, which is made possible by the large numbers of protein carrier molecules that can bind with glucose. In bound form, glucose can be transported by the carrier protein from one side of the membrane to the other side (to the interior of the muscle fiber) and then released.

The protein carrier molecule that transports glucose into the sarcoplasm is GLUT-4. The GLUT-4 transporter migrates from vesicles within the muscle fiber to the plasma membrane (Wasserman et al. 2011; Koistinen and Zierath 2002). Insulin and physical activity (independent of insulin) stimulate the migration of GLUT-4 from intracellular vesicles to the cell membrane, where it is able to bind with glucose for its subsequent transport into the muscle fiber (Khayat, Patel, and Klip 2002; Richter et al. 2001; Kristiansen et al. 2000; Thorell et al. 1999).

3.3.2 Glycemic Index Versus Glycemic Load

The final product of carbohydrate digestion in the digestive tract is almost entirely glucose. Hence, blood glucose levels (and insulin levels) increase in the time period following

a meal containing carbohydrates. Different carbohydrate-containing foods have variable effects on blood glucose levels. Some foods increase blood glucose levels quickly and sharply, while other foods, even if high in carbohydrate content, do not elevate blood glucose levels to a great extent. In order to categorize foods based upon their effects on blood glucose elevations, the glycemic index was developed by researchers from the University of Toronto and was primarily used as a tool for individuals with diabetes looking to control their blood glucose (blood sugar) levels (Jenkins et al. 1981). Today, athletes also use this index as a way to choose foods to eat for health, weight loss, and performance.

In order to obtain the glycemic index of a particular food, researchers measure a portion of food that contains 50 g of carbohydrate. For instance, four slices of white bread, 1 ⅓ cups of rice, 1 ½ lb. of carrots, and two medium apples each contains about 50 g of available carbohydrate. After the food is measured, it is fed to a group of participants and their blood sugar responses are measured. The participants' blood sugar response to the food is then compared with the response to eating 50 g (about 3 tablespoons) of pure glucose. Following the test meal, if blood glucose levels rise to nearly the same levels as they did following the glucose ingestion, the test food is considered a high-glycemic carbohydrate. If blood glucose levels are not comparably elevated, the food is considered a low-glycemic carbohydrate. For example, oatmeal is approximately 49 on the glycemic index. When plain oatmeal that contains 50 g of carbohydrate is consumed, it will produce an increase in blood sugar approximately 49% of that obtained when the same amount (i.e., 50 g) of straight glucose is consumed.

In essence, the glycemic index is a numerical ranking of carbohydrate-containing foods based on their potential to raise blood glucose levels. Carbohydrates that are high on the glycemic index (>70) are quickly digested and absorbed. These carbohydrates tend to cause a rapid rise in blood glucose and in most cases a quick rise in insulin occurs. Conversely, carbohydrates that are low on the glycemic index (~55 and below) are more slowly absorbed and subsequently cause a relatively small increase in blood sugar and insulin. Specifically, the glycemic index of a carbohydrate source describes the rate at which glucose levels rise in the blood following consumption of the food source (Mondazzi and Arcelli 2009; Burke, Collier, and Hargreaves 1998).

The glycemic score for a food is largely determined by the rate at which ingested carbohydrate is available to enzymes in the small intestine for hydrolysis and subsequent absorption. In turn, gastric emptying and the physical availability of a sugar or starch to intestinal enzymes determine a food's intestinal digestion rate. Foods such as brown rice, whole-grain pasta, fruits, and vegetables have slow absorption rates and a low glycemic index (Foster-Powell, Holt, and Brand-Miller 2002). High glycemic index foods such as refined table sugar (sucrose) included in many sports drinks and soft drinks, white rice, and pasta promote a pronounced, albeit transient rise in both blood glucose and pancreatic insulin production (Foster-Powell et al. 2002). Table 3.2 lists some common foods and their glycemic index.

The glycemic index is not the only tool that can be used to determine the blood glucose response to a particular food item. The glycemic load uses the glycemic index as well as the actual amount of carbohydrate (i.e., the serving size of the food item) to determine the overall effect that a carbohydrate-containing food has on blood sugar

TABLE 3.2
Glycemic Index Versus Glycemic Load of Common Foods

	Glycemic Index	Glycemic Load	Serving Size	Carbohydrate Content
Beverages				
Apple juice	40	12	1 Cup	29 g
Orange juice	46	13	1 Cup	26 g
Gatorade	78	12	1 Cup	15 g
Breads and Muffins				
Bagel	72	22	1 Small	30 g
Whole wheat bread	69	9	1 Slice	13 g
White bread	70	7	1 Slice	10 g
Dairy Foods				
Skim milk	32	4	1 Cup	12 g
Low-fat yogurt	33	6	1 Cup	17 g
Ice cream	61	19	1 Cup	31 g
Fruits				
Apple	38	8	1 Medium	22 g
Banana	55	16	1 Medium	29 g
Orange	44	7	1 Medium	15 g
Vegetables				
Carrots (boiled)	49	8	1 Cup	16 g
Sweet corn	55	21	1 Cup	39 g
Baked potato	85	48	1 Cup	57 g

Source: Foster-Powell, J. et al., 2002, *American Journal of Clinical Nutrition* 76 (1): 5–56.

and subsequent insulin values. As stated before, the glycemic index compares different food sources that contain carbohydrates of the same quantity (i.e., 50 g of glucose is compared to 50 g of carbohydrate in oatmeal). However, this is not always realistic because many foods are not consumed in 50-g (1.76-oz.) portions. The glycemic load is calculated by multiplying the amount of carbohydrate in a given serving of food by the glycemic index of that same food and then dividing that number by 100.

For example, a medium baked potato has a glycemic index of ~85 and a typical candy bar has a glycemic index of ~55. However, the average serving size of a baked potato is about 173 g (6.2 oz.) and contains 37 g of carbohydrate. Conversely, a candy bar serving size is only 57 g (2 oz.) but contains 35 g of carbohydrate. The baked potato has a glycemic load of 28, while the candy bar has one of 35. Thus, even though the potato has a higher glycemic index, the candy bar has a greater effect on blood glucose than the potato even though the size of the candy bar is less than half that of the potato. Dr. Asker Jeukendrup, a respected sports nutrition researcher, reports that foods with a glycemic load of ≥20 are high, 11–19 are medium, and ≤10

TABLE 3.3
Classifications for Low-, Medium-, and High-Glycemic Index and Glycemic Load Scores

	Low	Medium	High
Glycemic index	≤55	56–69	≥70
Glycemic load	≤10	11–19	≥20

are low. Table 3.2 lists some common foods with their corresponding glycemic index and glycemic load values, and Table 3.3 lists the classifications of values for low-, medium-, and high-glycemic index and glycemic load scores.

3.4 CARBOHYDRATE METABOLISM

Carbohydrate serves as an essential, but limited, fuel source in the body. After carbohydrates are ingested, they are digested, absorbed, and released into the blood. The glucose present in the blood, referred to as blood glucose, is tightly regulated. In addition to the maintenance of blood glucose levels and the making of glucose from nonglucose sources (gluconeogenesis), several other processes comprise carbohydrate metabolism. These processes include:

- Glycogen synthesis and storage (occurring in the liver and skeletal muscle)
- Glycogenolysis (the breakdown of stored liver and skeletal muscle glycogen)
- Glycolysis (the breakdown of glucose to produce ATP)

Each of these processes involved in the overall process of carbohydrate metabolism is discussed next.

3.4.1 Blood Glucose Regulation

The liver and pancreas, as well as other organs, help keep blood glucose levels within a narrow range to match the carbohydrate energy needs of the various body tissues. Normal levels of blood glucose range from approximately 70 mg/dL (3.9 mmol/L) to 110 mg/dL (6.1 mmol/L). If blood glucose levels elevate above this range, certain steps must take place to decrease levels back to normal. For instance, after consuming a meal with large amounts of carbohydrates, the blood glucose level increases to about 140 mg/dL (7.8 mmol/L). Such a condition of high blood glucose levels stimulates the beta cells of the pancreas to secrete insulin. Insulin reduces blood glucose by increasing blood flow to insulin-sensitive tissues (such as skeletal muscle and adipose tissue), subsequently stimulating the diffusion of glucose molecules into these tissues.

In contrast, when blood glucose levels fall to low levels such as those found during fasting, the breakdown of liver glycogen (hepatic glycogenolysis) and gluconeogenesis facilitate an increase in blood glucose levels. This mechanism is attributed to the

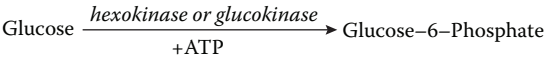

FIGURE 3.2 Phosphorylation of glucose.

hormone glucagon, which is secreted by the pancreatic alpha cells. Glucagon stimulates gluconeogenesis and glycogenolysis pathways in the liver to bring decreased blood glucose levels back to normal ranges. In summary, the major players keeping blood glucose levels within a narrow physiological range include the liver, pancreas, skeletal muscle, and adipose tissue, as well as the pancreatic hormones insulin and glucagon.

3.4.1.1 Phosphorylation of Glucose

Immediately upon entry into skeletal muscle or the liver, glucose is phosphorylated to form glucose-6-phosphate in accordance with the following reaction shown in Figure 3.2.

This phosphorylation is conducted by the enzyme glucokinase in the liver and by hexokinase in skeletal muscle. The phosphorylation of glucose is almost entirely irreversible, except in the liver cells. In the liver, another enzyme called glucose phosphatase is also available. When glucose phosphatase is activated, the reaction to convert glucose to glucose-6-phosphate is allowed to reverse, dephosphorylating glucose-6-phospate to yield glucose. Skeletal muscle cells do not contain glucose phosphatase; therefore, phosphorylation of glucose in a skeletal muscle cell serves to capture the glucose in the cell. This reaction occurs rather rapidly, to the extent that there is no accumulation of free glucose within contracting skeletal muscle (Katz et al. 1986). The intermediate glucose-6-phosphate plays a central role in the various conversions between glucose storage (in the form of glycogen) and glucose oxidation (via glycolysis) to yield ATP.

3.4.2 Glycogen

Glycogen is the carbohydrate source of energy most often used for exercise. It is needed for any intense bouts of exercise, sprinting, and resistance exercise. Similarly to starch being the storage form of carbohydrate in plants, glycogen is the storage form of carbohydrates in the human body. Glycogen is not simply a long string of repeated glucose compounds; it is a highly branched polymer (Figure 3.3). In fact, the glycogen molecule can be polymerized to almost any molecular weight, the average molecular weight being five million or greater (Guyton and Hall 2000). Glycogen is a convenient way to store glucose inside cells without having an impact on cell osmotic pressure since osmotic pressure depends on the number, not the size, of dissolved substances. For example, one molecule of glycogen may contain thousands of glucose molecules, yet produce a very small influence on osmotic pressure compared to thousands of individual glucose molecules (Houston 2001). The existence of many branches enables a faster breakdown for energy production when it is needed most: during high-intensity exercise. In addition, each gram of glycogen is stored with approximately 3 g of water.

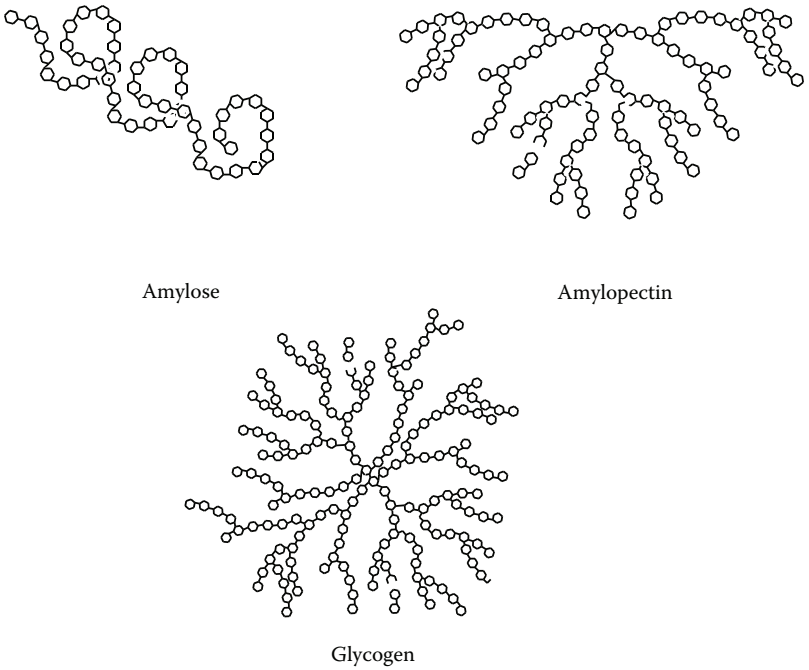

FIGURE 3.3 Highly branched structure of glycogen.

The two major sites of glycogen storage are the liver and skeletal muscles (there is no glycogen in the blood). The amount of glycogen that is stored in the skeletal muscle and liver is largely influenced by diet and physical activity. A 176-lb. (80-kg) male has approximately 400 g of muscle glycogen, 100 g of liver glycogen, and about 3 g of blood glucose (another carbohydrate source that, after being transported into the skeletal muscle, can be oxidized for quick ATP resynthesis). Hence, a well-fed 80-kg male possesses about 500 g of stored carbohydrate. Because each gram of either glucose or glycogen contains about 4 kcal, the typical male stores about 2000 kcal (~8371 kJ) of carbohydrate energy. This is enough total energy to fuel a relatively high-intensity 20-mile (~32 km) run (McArdle, Katch, and Katch 2009).

3.4.2.1 Glycogen Synthesis

The process of synthesizing glycogen from glucose is sometimes referred to as *glycogenesis*. This process occurs in both skeletal muscle and the liver. During the synthesis of glycogen in a skeletal muscle fiber, glucose that has entered the muscle fiber is converted to glycogen in a series of four enzymatically controlled steps. Upon entry into the skeletal muscle fiber, the first thing that occurs to glucose is that it is immediately phosphorylated by hexokinase to generate glucose-6-phosphate. Next, glucose-6-phosphate is converted to glucose-1-phosphate via phosphoglucomutase. Following this, glucose-1-phosphate is converted to uridine diphosphate (UDP)-glucose under the control of UDP-glucose pyrophosphorylase. Then, the UDP-glucose that was formed is added to the growing glycogen molecule (Figure 3.4). This reaction is

catalyzed by the enzyme glycogen synthase, which can add glucose residues only if the polysaccharide chain (i.e., glycogen) contains more than four residues.

3.4.2.2 Glycogen Breakdown

Glycogen can be broken down in both the liver and the skeletal muscle. With a normal liver glycogen content, liver glycogen stores will decrease by ~78% following a 12-h fast (Sherman and Wimer 1991). When glycogen is being degraded during exercise, it indicates that the body needs ATP to fuel skeletal muscle contraction. Broadly speaking, the goal of glycogen breakdown (also referred to as glycogenolysis) is to release glucose so that it can enter the glycolytic pathway to yield quick ATP synthesis. In the initial step of glycogen breakdown, individual glucose molecules are cleaved from glycogen to form glucose-1-phosphate, which is catalyzed by the enzyme glycogen phosphorylase. Two hormones, epinephrine and glucagon, can specifically activate glycogen phosphorylase, thereby causing rapid glycogenolysis (Chasiotis 1983, 1988). After it is formed, glucose-1-phosphate is converted to glucose-6-phosphate via the enzyme phosphoglucomutase. Glucose-6-phosphate is now able to enter glycolysis (explained later) to yield rapid ATP synthesis. The enzymatic reactions involved in glycogen degradation are summarized in Figure 3.4.

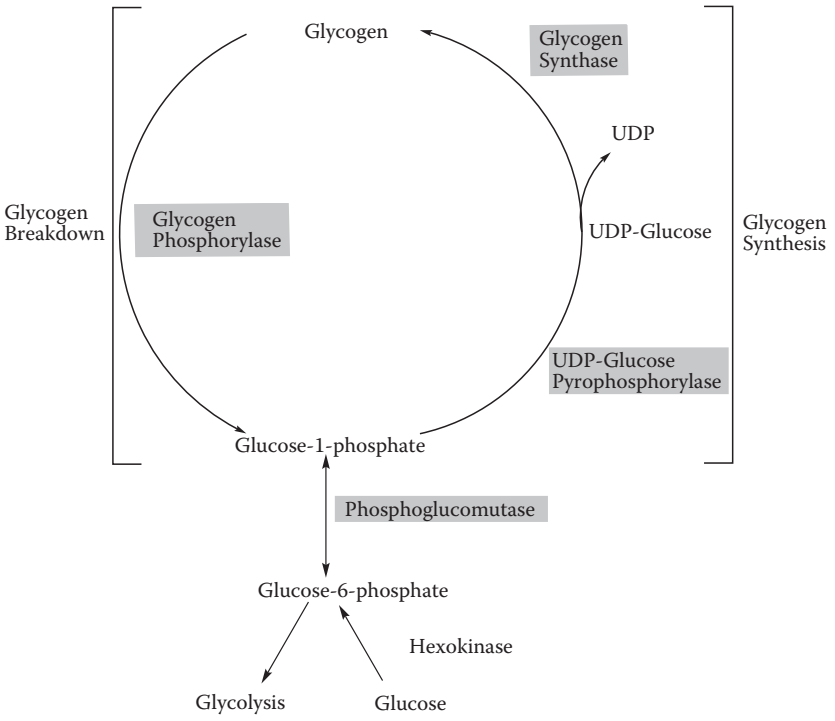

FIGURE 3.4 Glycogen synthesis and breakdown.

3.4.3 Glycolysis

The primary function of carbohydrate is to supply energy (i.e., ATP) for cellular work. In terms of glucose oxidation, this occurs in two stages. The first stage results in the anaerobic production of ATP and the second stage results in the aerobic production of ATP. The first stage of glucose degradation within skeletal muscle involves a series of chemical reactions, collectively termed glycolysis. Some activities that rely heavily upon the ATP generated during glycolysis include:

- Sprinting at the end of a 1-mile run
- 200-m sprint
- Grappling and repeated striking exchanges in mixed martial arts
- Prolonged games in a tennis match
- Resistance exercise aimed at inducing skeletal muscle hypertrophy

The skeletal muscle's capacity for glycolysis becomes crucial during physical activities that require maximal effort for up to 120 s. Glycolysis occurs in the sarcoplasm outside the mitochondria and does not require oxygen for ATP production. Specifically, glycolysis is a series of 10 chemical reactions that start with glucose and end with the production of pyruvate (Figure 3.5). In rested muscle, the activity of the glycolytic pathway is quite low. During moderate-intensity dynamic exercise, the rate of glycolysis may increase by 30- to 40-fold or more above resting levels (Houston 2001).

Assuming that the starting point of glycolysis is glucose (rather than glycogen), the first step of glycolysis in skeletal muscle is to phosphorylate glucose to produce glucose-6-phosphate (via hexokinase). Glucose-6-phosphate is then converted to fructose-6-phosphate, which is then phosphorylated to form fructose 1,6-diphosphate. The next step cleaves the six-carbon fructose-1,6-diphosphate into two three-carbon glyceraldehyde 3-phosphate molecules. The two molecules of glyceraldehyde 3-phosphate are oxidized, losing hydrogen atoms and gaining phosphate groups to form 1,3-diphosphoglycerate. Two molecules of NAD^+ are converted into two NADH in the process. The two 1,3-diphosphoglycerate molecules phosphorylate adenosine diphosphate (ADP) to yield two ATPs and two molecules of 3-phosphoglycerate. Next, the phosphate groups on 3-phosphoglycerate move to the second carbon, forming 2-phosphoglycerate. The two 2-phosphoglycerate molecules are dehydrated and form two high-energy phosphoenolpyruvate molecules. In the final step of glycolysis, the two phospoenolpyruvate molecules phosphorylate two ADPs and produce two more ATPs and two molecules of pyruvate.

In summary, glycolysis is simply the process of breaking down glucose to pyruvate to form ATP quickly. While glycolysis does generate ATP relatively quickly, there is not an abundance of ATP generated. In fact, glycolysis generates only about 5% of the total ATP formed during the glucose molecule's complete breakdown. The rest of the ATP that is formed from glucose oxidation occurs in the steps following glycolysis and involves the Krebs cycle and the electron transport chain—processes involved in the aerobic production of ATP.

Enzymes

1. Hexokinase
2. Phosphoglucose Isomerase
3. Phosphofructokinase
4. Aldolase
5. Triose Phosphate Isomerase
6. Glyceraldehyde 3-Phosphate Dehydrogenase
7. Phosphoglycerate Kinase
8. Phosphoglycerate Mutase
9. Enolase
10. Pyruvate Kinase

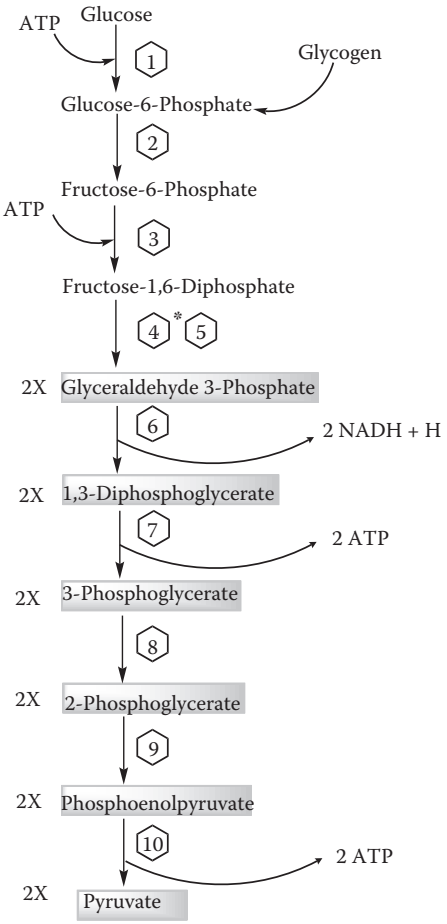

FIGURE 3.5 Glycolysis. *(An intermediate compound known as dihydroxyacetone phosphate is formed from the aldolase reaction, but it does not have a further role in glycolysis. Once formed, dihydroxyacetone phosphate is quickly converted into glyceraldehyde 3-phosphate via triose phosphate isomerase.)

3.4.3.1 Carbohydrate Oxidation in Aerobic Metabolism

The end product of glycolysis, pyruvate, is converted to acetyl-CoA in the outer mitochondrial membrane. The conversion of pyruvate to acetyl-CoA is catalyzed by the pyruvate dehydrogenase complex. Once acetyl-CoA is formed, it is ready to enter the Krebs cycle, which produces hydrogens and electrons that will be transported to the electron transport chain for eventual ATP resynthesis. Acetyl-CoA is also formed from the metabolism of fatty acids in a metabolic process referred to as β-oxidation. These processes are summarized in Appendix A, "Overview of Bioenergetics."

3.4.4 Lactate Formation and Metabolism

As stated previously, pyruvate is the end product of glycolysis. However, some of the pyruvate that is formed from glycolysis is converted to lactate under certain conditions, such as high-intensity exercise (Spriet, Howlett, and Heigenhauser 2000; Heigenhauser and Parolin 1999). During glycolysis, NAD^+ is converted to NADH via the oxidation (removal of a hydrogen and its associated electron) of the glucose molecule. During resting or low-intensity activities, a majority of the NADH formed during glycolysis releases its hydrogens (and associated electrons) to the mitochondria, and NAD^+ is re-formed. The hydrogens and their associated electrons released from NADH in the mitochondria ultimately join with oxygen in the electron transport chain to form water. As the intensity of exercise increases for more than a few seconds, the energy demands of the muscle fiber exceed either the oxygen supply or its rate of use, and the mitochondria cannot process all of the hydrogens (and their associated electrons) that were joined to NADH.

Continued release of anaerobic energy in glycolysis depends on NAD^+ availability. If NAD^+ is depleted, glycolysis will discontinue. In this prolonged high-intensity exercise state, NADH is unable to "donate" its hydrogens and electrons to the mitochondria, and must find another acceptor for the hydrogen molecules, so that NAD^+ can be reformed. Hence, NADH releases its hydrogens to combine temporarily with pyruvate to form lactate; this process is catalyzed by lactate dehydrogenase (LDH) and is a reversible reaction (Figure 3.6) (Spriet et al. 2000; Heigenhauser and Parolin 1999).

During recovery from the high-intense exercise, the additional hydrogens in lactate are removed and passed back to NAD^+ to re-form the pyruvate molecule, which is the reverse reaction and is also facilitated by LDH. It should be noted that another perspective exists in relation to the reason why lactate is formed during high-intensity activities. Some believe that lactate is produced in skeletal muscle when the rate of glycolysis exceeds the mitochondrial removal of pyruvate, rather than an inability of glycolysis derived NADH to donate its hydrogens directly to the mitochondria (Juel and Pilegaard 1999; Spriet et al. 2000).

Blood lactate exponentially increases with exercise of increasing intensity, closely following the increased glycolytic flux and reliance on muscle glycogenolysis. In the past, lactate was viewed as a metabolic waste product. However, recent research has confirmed that this is not the case. Once formed, lactate can act as a substrate for oxidative metabolism in contracting skeletal and cardiac muscle and a gluconeogenic precursor. In fact, contracting skeletal muscle is a major site of lactate oxidation,

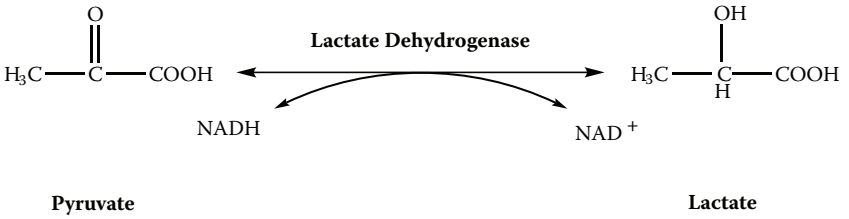

FIGURE 3.6 The conversion of pyruvate to lactate via LDH.

contributing as much as 30% of the total carbohydrate oxidation (Van Hall et al. 2003). During exercise, lactate can accumulate in the muscle fibers when its rate of production is higher than the rate of its release to the surroundings. Most (70%–80%) of the lactate formed during exercise is oxidized and used as a fuel energy source (Brooks 2002; Brooks et al. 1999). The remainder of the lactate (20%–30%) is carried by the blood and taken to the liver where it is converted to glucose. This metabolic pathway in which lactate is transported to the liver and subsequently converted to glucose is known as the Cori cycle.

Physiologic values of lactate range from 1 mmol/L at rest to over 20 mmol/L following maximal-intensity exercise lasting at least 30 s. Lower intensity exercise bouts result in lower lactate concentrations. When lactate is measured in the blood after maximal exercise, it is necessary to account for the fact that it takes a few minutes to peak. Thus, a blood sample taken right after the end of exercise will not produce a peak value. The best method to determine peak lactate levels following intense exercise is to take serial measurements spaced maximally 2 min apart for about 10 min or until lactate concentrations begin to decline. If only one blood sample can be taken, the preferred time is 5 min after the cessation of the exercise bout.

3.5 CARBOHYDRATE METABOLISM IN EXERCISE

A significant amount of stored fat is available to fuel certain types of exercise. However, due to the slowness of fat mobilization during exercise and the rate-limiting effects of the diffusion of fat from the blood to the muscle, fat oxidation cannot typically support muscular contraction that elicits >60% VO_2max (Sherman 1995; Koivisto 1986; Davies and Thompson 1979; Galbo et al. 1977). Thus, because athletes most often train and compete at exercise intensities requiring greater than 70% VO_2max, a source of fuel other than fat must be available (Sherman 1995), and that source of fuel is carbohydrate. The carbohydrates used during exercise can originate from any one of the following sources:

- Muscle and liver glycogen
- Blood glucose
- Carbohydrate ingested during exercise
- Gluconeogenic sources (glucose produced in the liver from amino acids, lactate, and glycerol)

The most important carbohydrate reserves are skeletal muscle and liver glycogen. Of these, muscle glycogen contains about 80% of the body's total carbohydrate reserves, and liver glycogen contains the other 20%. There is a very small amount of carbohydrate that exists as blood glucose, but this is relatively minor, accounting for ~1% of the total carbohydrate stored in the body.

In terms of priority of utilization, skeletal muscle glycogen is utilized first and serves as the "immediate" carbohydrate source of working muscle (Reichard et al. 1961). Subsequently, there is an increased uptake of blood glucose by working muscle that is supplied by an increased hepatic glucose output (Reichard et al. 1961).

Hence, skeletal muscle glycogen and blood glucose (originating either from ingested carbohydrates or from the liver) are crucial substrates for ATP resynthesis during endurance exercise. Differences between skeletal muscle and liver glycogen utilization and other concepts of carbohydrate metabolism are discussed next.

3.5.1 Crossover Concept

Fat and carbohydrate are the primary fuels metabolized during exercise. Protein is not metabolized during muscular contraction in substantial amounts unless the athlete is starved or bodily carbohydrate stores are maintained at low levels (Sherman and Lamb 1988). Carbohydrate and fat are oxidized as a mixture, and whether carbohydrate or fat is the predominant fuel depends on several factors, the most important being exercise intensity and duration. Fats are a primary fuel source for muscle during low-intensity (<30% VO_2max) exercise, whereas carbohydrates are the dominant fuel source during high-intensity (>70% VO_2max) exercise (Brooks and Mercier 1994). As illustrated in Figure 3.7, there is a progressive increase in carbohydrate metabolism and a decrease in fat oxidation with the increase of exercise intensity.

Also, as the exercise intensity increases from low to high, there is an exercise intensity at which the energy derived from carbohydrate exceeds the energy derived from fat; this point has been labeled as the "crossover" point by the highly respected exercise physiologist George Brooks (Brooks and Mercier 1994). Stated another way, as the exercise intensity increases above the crossover point, there is a progressive shift from fat to carbohydrate metabolism. As can be seen in Figure 3.7, an individual in the rested state is oxidizing nearly all fat for the majority of his or her fuel and, consequently, very little carbohydrate. At the other end of the spectrum, carbohydrate is burned exclusively during very high intensity exercise.

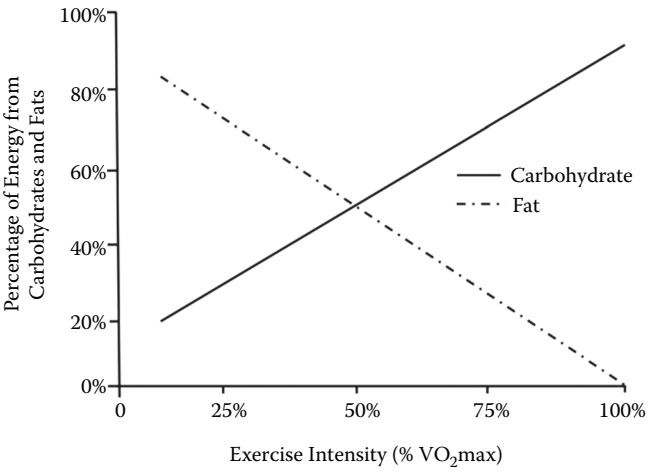

FIGURE 3.7 Crossover effect.

3.5.2 Skeletal Muscle Carbohydrate Metabolism during Exercise

The rates of glycogenolysis and glycolysis are low in resting muscle. During exercise, however, rates of both glycogenolysis and glycolysis are elevated. Skeletal muscle glycogen use during exercise depends primarily on the intensity of an exercise bout; more glycogen is used at higher intensities (Gollnick, Armstrong, Saubert, et al. 1973; Gollnick, Armstrong, Sembrowich, et al. 1973). Related to this increase is the fact that as the intensity of exercise increases from moderate to high, there is a greater reliance on fast twitch (type II) muscle fibers, which have a lower capacity for fat oxidation and a greater capacity for glycolysis.

The increase in muscle glycogen utilization during exercise results from the activation of glycogen phosphorylase, which catalyzes the first step in glycogen degradation (Figure 3.4). In the resting state, glycogen phosphorylase is inactive, but becomes activated during exercise. One of the main hormonal factors responsible for increasing glycogen phosphorylase activity is epinephrine (Chasiotis et al. 1983). Several studies have reported muscle glycogenolysis during moderate exercise is elevated because of increased plasma epinephrine (Febbraio et al. 1998; Jansson et al. 1986; Spriet, Ren, and Hultman 1988), which also increases with exercise intensity.

Skeletal muscle glycogen breakdown is not uniform throughout an exercise bout. Rather, the pattern of muscle glycogen metabolism during exercise is curvilinear (fastest during the initial stages of endurance exercise). Glycogenolysis is most rapid during the first 20–30 min. Afterward, glycogenolysis occurs at a slower rate until fatigue, which is likely related to muscle glycogen depletion (Watt et al. 2002; Sherman 1995; Coyle et al. 1986; Koivisto 1986).

In endurance activities, time to fatigue is directly proportional to the initial glycogen concentration (Sherman 1995; Bergstrom et al. 1967). When all other factors are equal, the concentration of glycogen stored in the skeletal muscle will play a direct role in how much is used. In other words, glycogen promotes its own use. For example, if skeletal muscle glycogen levels are high, more of that muscle glycogen will be used to fuel a given exercise bout than if the same exercise bout was undertaken with lower skeletal muscle glycogen levels. One of the explanations for increased muscle glycogen use when glycogen concentration is higher is related to the size of the glycogen molecule. When glycogen concentration is high, each glycogen molecule has a near maximal number of glucose units arranged in a highly branched fashion. In a larger glycogen molecule there is more branching, making the glucose units more accessible to glycogen phosphorylase, thus increasing the rate of glycogenolysis (Houston 2001). Overall, greater glycogen storage will increase its usage and the time until one becomes fatigued during endurance activities.

3.5.3 Liver Glycogen Metabolism during Exercise

Even though liver glycogen is an important carbohydrate reserve, it does not fuel exercise directly. Rather, the liver releases glucose to the blood, which is then subsequently taken up by the skeletal muscle to be oxidized for energy. The release of glucose from the liver helps maintain blood glucose homeostasis and prevent hypoglycemia. In a sense, the liver acts as a donor of glucose to the rest of the body in

times of increased glucose needs (such as during exercise). Glucose is produced in the liver via two mechanisms:

- Gluconeogenesis
- Glycogenolysis

Of these two sources, hepatic glycogenolysis (the breakdown of liver glycogen) provides the majority of glucose that is released from the liver. During moderate to heavy exercise lasting less than 30 min, almost the entire increase in liver glucose output is due to accelerated hepatic glycogenolysis (Wahren et al. 1971). Only a small amount (~15%) is accounted for by gluconeogenesis. As exercise duration continues (i.e., for several hours), gluconeogenesis increases to contribute about 50% of the total liver glucose output. The magnitude of glucose output by the liver depends on the liver glycogen content, which varies with the degree of fasting, carbohydrate intake in the days and hours before exercise, and the level of training of the athlete (Kjaer et al. 1987). Not surprisingly, liver glucose production increases linearly with exercise intensity. Hepatic glucose output rises linearly with exercise intensity until intensity reaches about 50%–60% VO_2max; as exercise intensity increases past this point, liver glucose output begins to increase exponentially.

There are two primary hormonal influences that affect hepatic glucose production during exercise: insulin and glucagon. A decrease in insulin levels (Redmon, Kubo, and Robertson 1995; Wasserman, Williams, et al. 1989), along with an increase in glucagon concentrations (Wasserman, Spalding, et al. 1989), has been shown to enhance hepatic glucose output during exercise. In addition to these pancreatic hormones, an increase in plasma epinephrine may also be responsible for increasing the rate of glycogenolysis in the liver during exercise (Sigal et al. 1996).

3.5.4 Control of Plasma Glucose during Exercise

Blood glucose concentrations during exercise are a function of the rate of liver glucose release and the rate of muscle glucose uptake (from the active skeletal muscles). During moderate-intensity exercise at ~60%–70% VO_2max, blood glucose concentrations remain constant at ~90 mg/dL (5 mmol/L) for up to 2 h because the rate of liver glucose release equals the rate of muscle glucose uptake. Thereafter, the rate of muscle glucose uptake remains constant and the rate of liver glucose release declines because of the gradual depletion of liver glycogen, provided exercise intensity remains constant. A major drop in blood glucose occurs only when exercise is prolonged for several hours. In contrast, if the intensity of exercise is increased (>60%–70% VO_2max), blood glucose normally increases, indicating that hepatic glucose output exceeds the skeletal muscle glucose uptake (Kjaer et al. 1991).

Gluconeogenic activity increases with increased exercise duration. Even though liver gluconeogenesis is accelerated as liver glycogen concentrations decline, the rate of gluconeogenesis cannot totally compensate for the declining liver glycogen concentration (Coyle and Coggan 1984; Ahlborg and Felig 1982). Thus, blood glucose concentrations can fall to ~45 mg/dL (2.5 mmol/L) after 2.5 h of cycling at 75% VO_2max

(Coyle et al. 1983, 1986; Ahlborg and Felig 1982). This decline in blood glucose is accompanied by a decline in total carbohydrate oxidation and eventual exhaustion.

Fatigue in some individuals is related to sensitivity to the decline of blood glucose (Coyle et al. 1986), whereas in other individuals fatigue occurs when blood glucose declines to hypoglycemic concentrations of <45 mg/dL (<2.5 mmol/L) (Felig et al. 1982). To offset the fatigue that is related to reductions in blood glucose, the endurance athlete can either elevate the pre-exercise concentrations of liver glycogen (by ingesting more dietary carbohydrate) or ingest a carbohydrate source during the endurance-exercise bouts.

3.5.5 Skeletal Muscle Glycogen Depletion

One of the defining characteristics of exercise's effects on carbohydrate metabolism is the reduction in glycogen stores following exercise. Skeletal muscle and liver glycogen are utilized during moderate-intensity/moderate-duration and high-intensity/long-duration activities. The major determinants of the extent to which glycogen stores are depleted are exercise intensity and duration. There is an exponential increase in glycogen breakdown with a linear increase in exercise intensity (Figure 3.8). The likely explanation for this observation is that with higher exercise intensities, more muscle fibers are recruited, particularly fast twitch type II muscle fibers. The greater the number of muscle fibers that are recruited during exercise, the greater is the potential for depleting the glycogen stored in these fibers.

3.5.5.1 Aerobic/Endurance Exercise

It has been well established that endurance exercise depletes muscle glycogen (Coyle et al. 1983; Hermansen, Hultman, and Saltin 1967; McConell et al. 1999). Exercise intensity, duration, mode (i.e., cycling vs. running), muscle group involvement,

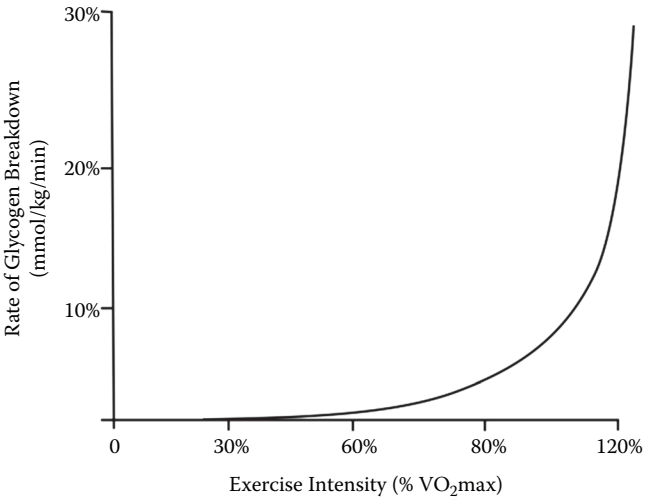

FIGURE 3.8 Glycogen breakdown rate with increasing exercise intensity.

individual biomechanical differences, and nutrition status are all factors that contribute to muscle glycogen depletion in endurance exercise. At exercise intensities less than 60% VO_2max, exercise can continue for hours without muscle glycogen depletion (Sherman and Wimer 1991; Saltin and Karlsson 1971). At exercise intensities of 65%–85% VO_2max, muscle glycogen decreases curvilinearly over time and exhaustion is related to depletion of muscle glycogen (Sherman and Wimer 1991; Hermansen et al. 1967).

However, at exercise intensities above 90% VO_2max, exhaustion occurs before muscle glycogen depletion. Therefore, there is a strong relationship between muscle glycogen depletion during exercise at 65%–85% VO_2max and exhaustion, but not at exercise intensities above or below this range (Sherman and Wimer 1991). One of the most cited studies used to demonstrate the impact of endurance exercise and skeletal muscle glycogen depletion was conducted by Costill and colleagues in 1971 (Costill, Sparks, et al. 1971). Five well-trained runners performed a 10-mile (16.1-km) treadmill run at ~80% VO_2max. Glycogen depletion rates were analyzed in three different muscles: the soleus, gastrocnemius, and vastus lateralis. Following the run, it was reported that the average amount of skeletal muscle glycogen across all three leg muscles was about 40% (Costill, Sparks, et al. 1971).

In a similar study (Costill, Bowers, et al. 1971), five endurance-trained men (who ran 5 miles [8 km]/day) exercised on a treadmill for 3 consecutive days. On each of the 3 days, the subjects ran 10 miles (16.1 km) on a treadmill at 80% VO_2max. The average amount of time for each exercise session was approximately 70 min. The diets of the runners contained only moderate amounts of carbohydrate (40%–50%), which was not enough to restore muscle glycogen after each successive daily run. Because of the suboptimal carbohydrate intake, prior to the run on the third day, skeletal muscle glycogen stores were about 50% of the pre-exercise levels on the first day of running exercise (Costill, Bowers, et al. 1971). This finding demonstrates not only that aerobic exercise depletes muscle glycogen, but also that successive days of endurance exercise have compounding effects on skeletal muscle glycogen levels if they are not adequately replenished (Costill and Miller 1980). Other examples of running and cross-country skiing also report significant glycogen depletion. For example, running a 30-km race in about 2.5 h depleted glycogen stores in the vastus lateralis by an average of 56% (Costill et al. 1973). During very long exercise durations, such as during an 85-km cross-country ski race that requires nearly 7.5 h to complete, skeletal muscle glycogen levels were depleted by 55% in the vastus lateralis (Fröberg and Mossfeldt 1971).

There is a specificity component to skeletal muscle glycogen depletion, meaning that skeletal muscle glycogen will be depleted in those muscles that are activated during the activity. During an 85-km cross-country ski race, which requires both lower and upper body contractions for long periods of time (6 to 10 h), four well-trained male athletes depleted their vastus lateralis and deltoid glycogen content by 60% and 87%, respectively (Bergstrom 1973). In two of the subjects, the skeletal muscle glycogen in the deltoids was nearly completely depleted. These glycogen depletion values were in consideration of the fact that each participant ingested approximately 300 g of carbohydrate in beverage form during the ski race.

Glycogen depletion patterns in skeletal muscle can also be delineated by muscle fiber type. When nine trained distance runners ran a 30-km race at an average intensity of 83% VO_2max, only a small amount of glycogen depletion occurred in the fast twitch muscle fibers, suggesting a primary reliance on slow twitch muscle fibers during prolonged running (Costill et al. 1973). Other investigations have reported similar findings in both trained and untrained endurance athletes engaging in running and cycling: that slow twitch fibers are the first to become depleted of their glycogen. However, as endurance exercise (at intensities ranging from 67% to 75% VO_2max) progresses, the fast twitch fibers also become depleted (Gollnick, Armstrong, Saubert, et al. 1973; Costill et al. 1974). These studies suggest a preferential utilization of slow twitch fibers during prolonged, intense exercise, with a secondary recruitment of fast twitch fibers occurring as the slow twitch fibers become depleted of their glycogen stores.

One of the adaptations that endurance training exerts on an individual is the ability to store more glycogen. To illustrate this training adaptation, six trained male cyclists and four males who were not engaged in cycling exercise (untrained) cycled at an intensity of approximately 67% VO_2max. The time spent cycling differed between the groups, with the trained group cycling for 180 min (3 h) and the untrained group for only 120 min (2 h) because they were not able to continue for a third hour. Muscle biopsies from the vastus lateralis muscle were taken at rest and after 20, 60, 120, and 180 min (for the trained group) of exercise. Table 3.4 displays the starting levels of glycogen as well as the extent to which each group depleted its glycogen stores.

Two things stand out in this table; first, the trained group started with nearly two times the amount of glycogen than the untrained group. Second, even after 60 min of exercise, the trained group had more muscle glycogen than the untrained group started out with. With consistent endurance exercise, there is an increase in the body's ability to store more glycogen as compared to the untrained state (Abernethy, Thayer, and Taylor 1990; Hurley et al. 1986; Matoba and Gollnick 1984).

TABLE 3.4
Muscle Glycogen Content at Rest and after Prolonged Cycling Exercise Performed at ~67% VO_2max

	Glycogen Levels in the Vastus Lateralis[a]								
Training Status	At Rest	20 Min	Decrease	60 Min	Decrease	120 Min	Decrease	180 Min	Decrease
Trained	182	139	24%	105	42%	63	65%	34	81%
Untrained	96	72	25%	30	69%	11	89%	Did not complete	Did not complete

[a] Millimoles per kilogram of wet muscle.

3.5.5.2 Resistance Exercise

Years ago, it was postulated that heavy resistance exercise was fueled exclusively by ATP and phosphocreatine (PCr) stores (Keul et al. 1978). Subsequent research has refuted this belief and it is now known that resistance exercise is also fueled by fat and carbohydrate sources (Ormsbee et al. 2007, 2009; Chatzinikolaou et al. 2008; Koopman et al. 2006; MacDougal et al. 1999; Tesch, Colliander, and Kaiser 1986; Tesch et al. 1998; Pascoe and Gladden 1996; Pascoe et al. 1993; Robergs et al. 1991; Essén-Gustavsson and Tesch 1990; Bell and Jacobs 1989; Dudley 1988; Lesmes et al. 1983; Gollnick et al. 1974). Skeletal muscle glycogen depletion (an indication of increased glycogenolysis) is moderately depleted following only one set of a typical resistance exercise, but significantly depleted following three sets. In one study, eight trained bodybuilders performed either one set or three sets of a biceps curl exercise performed at 80% 1 repetition maximum (RM) to failure. Skeletal muscle glycogen depletion was depleted by 12% and 24% in the biceps brachii after one and three sets, respectively (MacDougal et al. 1999) (Figure 3.9).

In another study using trained bodybuilders (Tesch et al. 1986), skeletal muscle glycogen (in the vastus lateralis) was depleted by 26% following five sets of 5–10 repetitions of four different lower body exercises: front squats, back squats, leg press, and leg extension. A very similar study (resistance exercise consisted of five sets of 12 RM of the front squat, back squat, leg press, and leg extension) using trained bodybuilders reported that skeletal muscle glycogen was depleted by 28% in the vastus lateralis (Essén-Gustavsson and Tesch 1990). Finally, when eight resistance-trained males performed unilateral leg extensions at 70% 1 RM for six sets of six repetitions, skeletal muscle glycogen was depleted 38% (Robergs et al. 1991). In each of these studies using resistance-trained bodybuilders, skeletal muscle glycogen was depleted between 24% and 38% following typical resistance-training routines.

Nearly the same amount of glycogen degradation is observed in non-resistance-trained individuals undergoing an acute bout of resistance exercise. Eight recreationally trained males performed about nine sets of six repetitions of unilateral

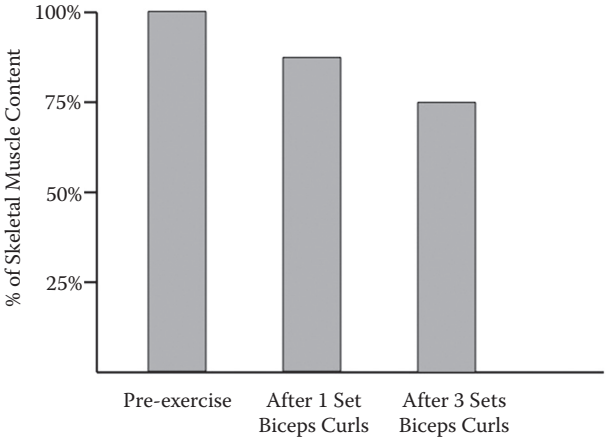

FIGURE 3.9 Skeletal muscle glycogen depletion following bicep curl exercise.

leg extensions at 70% 1 RM to failure, with 30 s of rest between each set. This workout resulted in a 29% depletion of skeletal muscle glycogen (Pascoe et al. 1993). Similarly, eight untrained males performing eight sets of 10 repetitions of leg press and eight sets of 10 repetitions of leg extension at an intensity of 75% 1 RM depleted their skeletal muscle glycogen stores by 33% (Koopman et al. 2006). In summary, it appears that a typical resistance-training workout (with multiple sets and exercises) depletes skeletal muscle glycogen by about 30%. Taken together, the studies suggest that the extent to which skeletal muscle glycogen is depleted does not differ very much between resistance-trained and -untrained individuals.

Some research has been more specific in its investigation of skeletal muscle glycogen depletion by examining the extent of depletion in the various muscle fiber types, including slow twitch muscle fibers (type I) and fast twitch muscle fibers (type IIA and type IIX). Eight untrained males performing eight sets of 10 repetitions of leg press and eight sets of 10 repetitions of leg extension at an intensity of 75% 1 RM depleted 23% of initial glycogen levels in the type I muscle fibers, 40% in type IIA muscle fibers, and 44% in type IIX muscle fibers (Koopman et al. 2006). Because the intensity was relatively high (75% 1 RM), there was a significant depletion in the fast twitch fibers.

In contrast, if the intensity is low, the fast twitch fibers are not recruited and subsequently no glycogen depletion occurs. To demonstrate this, six healthy males performed repeated 90° isometric leg extension contractions ranging from 10% to 50% of maximal voluntary contraction (MVC). At isometric tensions less than 20% MVC, there was preferential glycogen depletion in slow twitch fibers. At higher tensions, the fast twitch fibers became depleted (Gollnick et al. 1974). What was somewhat surprising in this investigation was the critical intensity at which glycogen was depleted from the slow twitch fibers to the fast twitch fibers: only 20% MVC. What this implies is that fast twitch fibers are recruited at intensities above 20% MVC, far below what most individuals believe to be the threshold for activating fast twitch muscle fibers. Unfortunately, this investigation did not specify the glycogen depletion in the specific subtypes of the fast twitch fibers (type IIA and type IIX muscle fibers).

Tesch and colleagues (1998) instructed nine males with 9 weeks of resistance-training experience to complete five sets of 10 concentric repetitions of the knee extension at 30%, 45%, and 60% 1 RM. Two minutes of rest between each set and a 45-min rest period between each varying intensity were included. In terms of glycogen depletion in the fast twitch fibers, type IIA muscle fibers showed glycogen depletion at all three intensities. In contrast, the type IIX muscle fibers only showed glycogen depletion at 60% 1 RM. In summary, glycogen was depleted in fast twitch fibers (type IIA) even at low intensities (30% and 45% 1 RM).

Combined, these studies (Tesch et al. 1998; Gollnick et al. 1974) demonstrate that the extent of skeletal muscle glycogen depletion among fiber types is dependent on the intensity of the workout. Also, training status (experience) does not affect glycogen depletion in either slow twitch or fast twitch fibers (Bell and Jacobs 1989).

One other area that has been studied within the context of resistance exercise and skeletal muscle glycogen depletion is contraction speed. This training variable elicits differential glycogen depletion in the various muscle fiber types. Four physically active (but not resistance trained) males completed 20 unilateral maximal 30 s

leg flexion and extension movements at 60°/s (slow speed) with one leg and at 300°/s (fast speed) with the other leg (Lesmes et al. 1983). At the slow contraction speed, skeletal muscle glycogen was depleted 35% in slow twitch muscle fibers and 39% in fast twitch muscle fibers. At the fast contraction speed, glycogen was depleted 48% in slow twitch muscle fibers and 56% in fast twitch muscle fibers. This and other reports demonstrate that the rate of glycogenolysis is greater in fast twitch fibers as compared to slow twitch fibers (Robergs et al. 1991; Lesmes et al. 1983). One final observation that can be made in relation to these resistance exercise studies discussed previously is that in light of the relatively high glycogen levels after resistance exercise, glycogen depletion does not seem to be a cause of fatigue and impaired muscle function during a resistance exercise session.

3.6 CONCLUSION

In relation to exercise and performance, the primary role of carbohydrates is to provide energy at relatively high rates. Among the different types of athletes, the endurance athlete pays particular attention to dietary carbohydrate intake and supplementation. This is likely due to the limited stores of carbohydrate in the body and the association between depleted glycogen stores and fatigue during endurance events. Once ingested, dietary carbohydrate can be stored as skeletal muscle glycogen, as liver glycogen, or be oxidized for energy via glycogenolysis or glycolysis. This chapter summarized all aspects of carbohydrate metabolism; the following chapter will discuss the effects of dietary and supplemental carbohydrate intake and its effects on improving exercise and sports performance.

ACKNOWLEDGMENT

A sincere appreciation to Marcelle Issa for providing the artwork for many of the figures in this chapter.

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4 Dietary Carbohydrate Strategies for Performance Enhancement

Bill Campbell

CONTENTS

4.1 Introduction 76
4.2 Aerobic Considerations 76
4.2.1 Daily Carbohydrate Intake 76
4.2.2 What about Low-Carb Diets and Endurance-Exercise Performance? 78
4.2.2.1 Train Low, Compete High? 83
4.2.3 Carbohydrate-Loading Strategies Prior to Competition 84
4.2.4 Monosaccharides and Endurance Performance 89
4.2.4.1 Glucose Supplementation and Endurance Performance 89
4.2.4.2 Galactose Supplementation and Endurance Performance 94
4.2.4.3 Fructose Supplementation and Endurance Performance 97
4.2.5 Carbohydrate Ingestion during Endurance Exercise (Multiple Transportable Carbohydrates) 99
4.2.6 Resynthesizing Glycogen in the Postexercise Period 102
4.2.6.1 Timing 103
4.2.6.2 Amount 104
4.2.6.3 Type 104
4.3 Resistance Exercise Considerations 105
4.3.1 Daily Carbohydrate Intake 105
4.3.2 Carbohydrate Ingestion Prior to and during Resistance Exercise 106
4.3.3 Postresistance Exercise Muscle Glycogen Resynthesis 111
4.3.4 Net Muscle Protein Balance and Insulinogenic Properties of Carbohydrate Intake 112
4.4 Conclusion 114
References 115

4.1 INTRODUCTION

The preceding chapter laid the foundation for how carbohydrates (CHOs) are used in the body by listing the various types of carbohydrates that are available and discussing how each of these is stored and metabolized during exercise. This chapter takes this information and builds upon it by discussing the role (if any) that carbohydrates have in terms of improving exercise performance. Aerobic and anaerobic athletes must make important decisions in regard to carbohydrate intakes. Questions concerning dietary carbohydrate intakes include:

- What amount of carbohydrate should be ingested on a daily basis?
- What is the importance of the timing of carbohydrate intake?
- What types of dietary carbohydrate should be ingested?

Each of these questions will be addressed, starting with aerobic exercise performance considerations and followed by anaerobic/resistance exercise considerations.

4.2 AEROBIC CONSIDERATIONS

Carbohydrate is the preferred fuel source for muscle during exercise at moderate intensities (~65%–85% VO_2max). Also, there is a direct relationship between the level of carbohydrate stores and performance in time to exhaustion tasks. Therefore, it is reasonable to suggest that a high-carbohydrate diet will elevate carbohydrate stores and enhance time-to-exhaustion endurance performance (Sherman and Wimer 1991). During prolonged endurance exercise, fatigue is often associated with skeletal muscle glycogen depletion and hypoglycemia (McConell et al. 1999; Coggan and Coyle 1987; Coyle et al. 1983, 1986). Supplementing carbohydrate, either by increasing the availability of glycogen in muscle before exercise or by ingesting carbohydrate during exercise, enhances endurance performance (Hawley, Shabort, et al. 1997; Coyle et al. 1983, 1986). Generally, the consumption of carbohydrates during endurance exercise does not spare muscle glycogen but rather reduces the reliance upon endogenous carbohydrate stored in the liver.

A contrasting point of view addressing the importance of dietary carbohydrates for endurance athletes is also presented in this chapter. In short, this view states that high-carbohydrate diets are not necessary for endurance athletes. While the majority of sports nutritionists and athletes do not hold this point of view, some compelling data will be presented in light of this controversial viewpoint.

4.2.1 Daily Carbohydrate Intake

Of the three macronutrients, carbohydrate has been the most intensely studied in terms of its application to fueling aerobic activities. The likely explanation for this is that while fat sources (plasma free fatty acids derived from adipose tissue and intramuscular triglyceride stores) are relatively plentiful, carbohydrate sources (skeletal muscle glycogen and plasma glucose derived from the liver and dietary carbohydrate) are limited (Burke et al. 2004). Because of the limitations in storing

carbohydrates, strategies have been proposed to enhance body carbohydrate availability. Scientific investigations have been very specific in terms of focusing on carbohydrate intake prior to, during, and following endurance exercise, but this has not replaced the need for more general recommended dietary carbohydrate intakes on a daily basis.

Earlier position papers on dietary guidelines for athletes have expressed carbohydrate requirements as a percentage of total energy (Brown 2002; American Dietetic Association and Canadian Dietetic Association 1993)—about 60%–70% of total energy intake. This strategy for recommended daily carbohydrate intake was not ideal, primarily because it does not address specific carbohydrate intakes, but rather assumes that the endurance athlete is ingesting adequate kilocalories on a daily basis. As an example, if an athlete were to undergo a severe hypocaloric diet and consume only 1000 kcal, but was consuming 150 g carbohydrate/day (which is a fairly low total daily carbohydrate intake), it could be ascertained that the athlete was ingesting adequate amounts of carbohydrate because, on a 1000-kcal diet, this equates to 60% of total kilocalories coming from carbohydrate. To address the specific carbohydrate needs of athletes it is important to express carbohydrate relative to body weight or fat-free mass. More recent position statements now recognize this fact (Rodriguez, DiMarco, and Langley 2009; Kerksick et al. 2008).

In this regard, the American College of Sports Medicine (American Dietetic Association et al. 2009) recommends a carbohydrate intake of 6–10 g carbohydrate/kg body weight/day. Specific carbohydrate intakes within this range will vary with an athlete's daily energy expenditure and type of exercise performed. Carbohydrate intakes in the upper range are especially important for athletes who engage in multiple workouts per day and whose fuel requirements for everyday training are likely to challenge or exceed normal body carbohydrate stores (Burke, Kiens, and Ivy 2004). The dietary carbohydrate recommendation of ~6–10 g/kg/day is cited by many sports nutrition textbooks and reviews (American Dietetic Association et al. 2009; McArdle, F. Katch, and V. Katch 2009; Reimers 2008; Dunford and Doyle 2012; Kreider and Leutholtz 2001; Sherman, Jacobs, and Leenders 1988; Kerksick et al. 2008).

The 2003 International Olympic Committee consensus on sports nutrition (Burke et al. 2004) also provided general daily carbohydrate intakes that were a little broader than the often cited 6–10 g/kg/day. The daily carbohydrate recommendations of the consensus were

- 5–7 g/kg/day for moderate duration/low-intensity endurance training
- 7–12 g/kg/day for moderate to heavy endurance training
- 10–12+ g/kg/day for extreme endurance-exercise programs (4–6 h or more per day)

An important factor to consider when determining recommendations for carbohydrate intake is the aerobic training program. Endurance athletes who train for long periods (90 min or more daily) should replenish glycogen levels by consuming carbohydrate intakes near the upper end of the recommended ranges, such as 8–10 g/kg/day (Reimers 2008; Jacobs and Sherman 1999; Sherman 1995; Sherman and Wimer

1991). In terms of grams of carbohydrate per day, this is equivalent to 600–750 g for an endurance athlete weighing 165 lb. (75 kg) (Reimers 2008). This level has been shown to restore skeletal glycogen adequately within 24 h (Reimers 2008; Costill et al. 1981; Kochan et al. 1979; Piehl, Adolfsson, and Nazar 1974). Athletes benefiting from this level of carbohydrate intake include those engaged in continuous aerobic activity for more than an hour on successive days, such as distance runners, triathletes, road cyclists, and cross-country skiers (Reimers 2008).

At times, even the upper ranges of the recommended 6–10 g/kg/day are not sufficient to replenish skeletal muscle glycogen, and the upper levels (10–12+ g/kg/day) of the 2003 International Olympic Committee's consensus on sports nutrition are necessary to replenish skeletal muscle glycogen. As an example, well-trained cyclists undertaking 2 h of training each day were found to have higher muscle glycogen stores after a week of a daily carbohydrate intake of 12 g/kg/day than when consuming 10 g/kg/day (Coyle et al. 2001). What are the actual daily intakes of endurance athletes? Dietary surveys of endurance athletes show that self-reported carbohydrate intakes are 7.6 and 5.7 g/kg/day for males and females, respectively (Burke et al. 2001, 2004). In summary, current carbohydrate intake recommendations ranging from 6 to 10 g/kg/day are sufficient to maintain skeletal muscle glycogen concentrations for most athletes training on successive days.

4.2.2 What about Low-Carb Diets and Endurance-Exercise Performance?

According to Burke and colleagues (2004), the recommendation of 6–10 g/kg/day of carbohydrate came from various studies that monitored muscle glycogen storage after 1 day of recovery from glycogen-depleting exercise (Burke, Collier, and Hargreaves 1993; Burke et al. 1995, 1996; Costill et al. 1981). In essence, the recommended range of dietary carbohydrate intake was not based on endurance performance, but rather on the skeletal muscle glycogen resynthesis levels following glycogen-depleting exercise. With adequate carbohydrate ingestion, skeletal muscle glycogen can be restored following glycogen-depleting exercise (Costill et al. 1981). To demonstrate this phenomenon, trained male runners depleted skeletal muscle glycogen levels and then ingested varying amounts of carbohydrate for the next 24 h. Carbohydrate intake was ingested at three different levels: 2.4, 4.7, and 6.6 g/kg/day. Skeletal muscle glycogen was resynthesized to a significantly greater extent in the 6.6 g/kg/day as compared to the lower levels of carbohydrate ingestion. However, even though skeletal muscle glycogen was significantly higher in this group, performance times in a 300-m sprint (taking about 48 s to complete) following the skeletal muscle glycogen resynthesis period were not different among the three levels of carbohydrate intake (Costill et al. 1981).

In another study, when well-trained triathletes ingested either 7 or 11.8 g of carbohydrate/kg/day after following muscle glycogen depleting exercise, it was reported that there were no differences in glycogen resynthesis values 24 h later (Burke et al. 1995). These studies reinforce the general recommendations of ingesting between approximately 6 and 10 g of carbohydrate/kg/day to maximize skeletal muscle glycogen (but not endurance-exercise performance) 1 day after glycogen-depleting exercise.

There is no question that particularly intense or voluminous training depletes muscle glycogen levels (Costill et al. 1971). In association with this muscle glycogen depletion, it has been inferred that suboptimal daily carbohydrate intake not only lowers muscle glycogen concentrations, but also impairs both training capabilities and maximal exercise performance ability. Sherman et al. (1993) stated the connection that optimal carbohydrate intakes, while effective for replenishing muscle glycogen levels, may not actually improve exercise performance. In support of their observation, rowers who consumed a moderate-carbohydrate diet while undertaking two daily intense training bouts reduced muscle glycogen concentrations, although rowing ability was maintained (Simonsen et al. 1991). In another study, cyclists and runners engaged in 7 days of intense cycling and running training. During the 7-day training period, the endurance athletes were randomly assigned to a moderate- (5 g/kg/day) or high- (10 g/kg/day) carbohydrate diet. To measure exercise performance, each athlete completed two maximal performance trials at 80% peak VO_2 until exhaustion. Muscle glycogen for cyclists and runners was maintained with 10 g/kg/day carbohydrate diet, but was significantly reduced by ~33% with the 5 g/kg/day carbohydrate diet. Despite these differences in glycogen levels after 7 days of intense exercise training, there were no differences between the two levels of carbohydrate intakes in terms of exercise performance (Sherman et al. 1993).

Tables 4.1 and 4.2 summarize much of the published literature in which several ranges of daily carbohydrate intakes—categorized into studies lasting less than 7 days (Table 4.1) and those lasting more than 7 days (Table 4.2)—were compared in terms of endurance performance and physiological responses.

By observing the results of the few studies that investigated low- versus high-carbohydrate intakes lasting less than 7 days, it appears as if not only the lower carbohydrate intakes resulted in significantly less skeletal muscle glycogen, but the higher carbohydrate diets also resulted in superior endurance performance in a time to exhaustion bout as well as a time trial (Walker et al. 2000). In these studies, the higher carbohydrate intakes were within the recommended 6–10 g/kg/day recommendation, and the lower carbohydrate intakes were less than the recommended amounts. At face value, this appears to present justification that consuming 6–10 g/kg/day carbohydrate improves endurance performance.

However, when the duration of ingesting a low-carbohydrate diet is increased to periods lasting from at least 1 week and up to and encompassing several weeks, the data favoring a higher carbohydrate intake are not as overwhelming. Out of the nine studies that were summarized in Table 4.2, three of them (Halson et al. 2004; Achten et al. 2004; Simonsen et al. 1991) reported a significant improvement in endurance performance in the higher carbohydrate intake group. In the other studies, there were either no differences in endurance performance or the lower moderate carbohydrate intakes (around 5 g/kg/day) resulted in significant (Lambert et al. 1994) or nonsignificant improvements in endurance exercise (Sherman et al. 1993; Phinney et al. 1983). What was consistent in each of these studies was that the lower carbohydrate diets always resulted in reductions in skeletal muscle glycogen as compared to the higher levels of carbohydrate intakes.

TABLE 4.1
Effects of Differing Levels of CHO Intakes (<7 Days) on Endurance Performance

Study	Population	Design and Diet	Performance Measurements	Glycogen Levels	Findings
Kavouras et al. (2004)	Twelve endurance-trained male cyclists	Randomized, crossover design; 3 days prior to cycling bout, subjects consumed: • High CHO (8.2 g/kg/day) • Low CHO (1.4 g/kg/day)	45-min cycling bout at 82% VO_2peak	44% Higher in the high-CHO diet than the low-CHO diet (~105 vs. 72 mmol/kg wet weight) prior to exercise bout	No difference between high and low CHO in physiological or perceptual responses (no performance measure)
Walker et al. (2000)	Six well-trained female athletes	Randomized, crossover design; 4 days prior to exercise bout, subjects consumed: • High CHO (8.2 g/kg/day) • Low CHO (4.7 g/kg/day)	Cycling at 80% VO_2max to voluntary exhaustion	13% Higher in the high-CHO diet than the low-CHO diet (709 vs. ~625 mmol/kg dry weight) prior to exercise bout	No significant differences reported; high-CHO diet cycled ~8% longer than low-CHO diet
Widrick et al. (1993)	Eight endurance-trained male cyclists	Randomized, crossover design; 2 days prior to the time trial, subjects consumed: • High CHO (6.5 g/kg/day) • Low CHO (2.3 g/kg/day)	70-km time trial	55% Higher in the high-CHO diet than the low-CHO diet (~170 vs. 110 mmol/kg wet weight) prior to exercise bout	High-CHO diet superior; low CHO diet took significantly longer (~3.6% more time) to complete the time

TABLE 4.2
Effects of Differing Levels of CHO Intakes (>7 Days) on Endurance Performance

Study	Population	Design and Diet	Performance Measurements	Glycogen Levels	Findings
Cox et al. (2010)	Sixteen male endurance-trained cyclists and triathletes	Parallel group design; athletes ingested one of the two diets for 28 days: • High CHO (8.5 g/kg/day) • Moderate CHO (5.2 g/kg/day)	100 Min of steady-state cycling at ~70% VO_2peak followed by a 7 kJ/kg body weight time trial	Maintained in both diets	No significant difference in time trial performance
Halson et al. (2004)	Six male endurance-trained cyclists	Randomized, crossover design; for 15 days (including 8 days of intensified training) athletes consumed: • High CHO (9.4 g/kg/day) • Moderate CHO (6.4 g/kg/day)	Cycle to volitional exhaustion at 74% VO_2max	Not measured	High-CHO diet was significantly better than the moderate-CHO diet in the cycle-to-exhaustion test during the intensified training period
Achten et al. (2004)	Seven trained runners	Randomized, crossover design; for 11 days (including 7 days of intensified training) athletes consumed: • High CHO (8.5 g/kg/day) • Moderate CHO (5.4 g/kg/day)	An 8-km all-out run on the treadmill followed by a 16-km all-out run; both tests conducted on day 11	Not measured	High CHO was significantly better in the 8- and 16-km performance compared to the low-CHO diet
Lambert et al. (1994)	Five endurance-trained male cyclists	2 Weeks prior to exercise bout athletes consumed: • Low-CHO diet (7% CHO) • High-CHO diet (74% CHO)	Power output via a 30-s Wingate test; cycle to exhaustion at ~90% and ~60% VO_2max (moderate intensity)	77% Higher in the high-CHO diet than the low-CHO diet (~121 vs. 68 mmol/kg wet weight) prior to exercise bout	No significant difference in power output or high-intensity exercise; low CHO = significantly better (88%) in moderate intensity

(continued)

TABLE 4.2 (CONTINUED)
Effects of Differing Levels of CHO Intakes (>7 Days) on Endurance Performance

Study	Population	Design and Diet	Performance Measurements	Glycogen Levels	Findings
Sherman et al. (1993)	Eighteen trained male runners	Athletes randomly assigned to one of two diets for a 7-day training period: • High CHO (10 g/kg/day) • Moderate CHO (5 g/kg/day)	Two maximal performance runs to exhaustion at 80% peak VO_2	55% Higher in high-CHO diet than the moderate-CHO diet (~125 vs. 80 mmol/kg wet weight) prior to exercise bout	No significant differences reported; moderate-CHO diet ran ~9% longer than low-CHO diet
Simonsen et al. (1991)	Twelve male and ten female collegiate rowers	Athletes randomly assigned to one of two diets for 4 weeks of intense, twice daily training: • High CHO (10 g/kg/day) • Moderate CHO (5 g/kg/day)	Mean power output for three maximal 2500-m rowing trials (separated by 8 min) over the 4-week study	25% Higher in the high-CHO diet than the moderate-CHO diet (~155 vs. 124 mmol/kg wet weight) prior to exercise bout	High-CHO diet significantly increased mean power output (10.7%) as compared to the moderate-CHO diet (1.6%)
Lamb et al. (1990)	Fourteen male collegiate swimmers	Randomized, crossover design; 9 days prior to swim bout, athletes consumed: • High CHO (12.1 g/kg/day) • Moderate CHO (6.5 g/kg/day)	Swim times of various distances (50–3000 m) recorded during last 5 days of diet phase	Not measured	No differences reported; high CHO offered no advantage over moderate CHO in swim velocities
Costill et al. (1988)	Twelve collegiate male swimmers	Increased training volume over 10 days, no CHO intervention: • High CHO (8.2 g/kg/day) • Low CHO (5.3 g/kg/day)	Swim times for two 25-yd. sprints and a 365-m endurance test	66% Higher in high-CHO diet than the moderate-CHO diet (~140 vs. 84 mmol/kg wet weight) after training	No difference in sprint and endurance performance; only low CHO had fatigue during training
Phinney et al. (1983)	Five competitive male cyclists	High-CHO diet for 1 week (>5 g/kg/day) followed by 4 weeks of a ketogenic diet providing ~0.27 g/kg/d CHO	Cycling to exhaustion at ~63% VO_2max	88% Higher in high-CHO diet than the ketogenic diet (~143 vs. 76 mmol/kg wet wt)	No differences reported; ketogenic diet exercised ~2.7% longer duration

4.2.2.1 Train Low, Compete High?

Some have advocated a low daily carbohydrate intake (even less than 5 g/kg/day) for much of the training period, followed by several days of very high levels of carbohydrate intake prior to an endurance event so that the body's carbohydrate stores can be maximized for competition. This strategy has become known as "train low, compete high" in relation to dietary carbohydrate intake. The main idea of this approach is to conduct the training sessions with relatively low glycogen stores. New molecular insights show that compared with high muscle glycogen content, an acute bout of endurance exercise commenced with low muscle glycogen results in a greater transcriptional activation of enzymes involved in fat metabolism, as well as an increase in adaptive responses favoring fat metabolism (Burke et al. 2011). What this means is that the endurance athlete will be able to utilize fat as an exercise fuel and have a reduced reliance on carbohydrate (Burke 2010). Specifically, exercising with low muscle glycogen stores amplifies two signaling proteins (adenosine monophosphate [AMP]-activated protein kinase and p38 mitogen-activated protein kinase), which have a role in mitochondrial biogenesis and other training adaptations (Burke 2010; Hawley, Tipton, and Millard-Stafford 2006).

Following a chronic low-carbohydrate diet in order to abide by the "train low" philosophy would be considered too risky for some endurance athletes accustomed to higher daily carbohydrate intakes. Because of this, more recent train-low protocols have utilized a different approach to reducing carbohydrate availability for training. One of these protocols incorporates the placement of training sessions (rather than dietary manipulation) to achieve low glycogen stores for specific workouts (Burke et al. 2011). In a comprehensive review on this topic of "train low, compete high," respected sports nutrition researcher Louise Burke (2010) noted that there are a number of potential ways to reduce carbohydrate availability for the training environment other than following a low-carbohydrate diet. Some of these exercise–diet strategies include (Burke 2010):

- Training two times in the same day (the second training session would be conducted with lower glycogen stores by limiting carbohydrate intake after the first training session)
- Training after an overnight fast
- Prolonged training while withholding carbohydrate intake during the session
- Withholding carbohydrate during the first hours of recovery

A few studies have produced support for augmenting these metabolic and training adaptations (Hulston et al. 2010; Morton et al. 2009; Yeo et al. 2008; Hansen et al. 2005). In the first of these studies, seven untrained male subjects completed a 10-week training program consisting of knee extensor exercise at 75% of maximal power output (Hansen et al. 2005). In this study, both of the subject's legs were trained with the same volume (5 h/week) over the 10-week period, but one of the legs was trained in a low-glycogen state and the other leg was trained in a high-glycogen state. To facilitate the low-glycogen training state, one of the legs was trained every other day in two separate workouts. After the first hour-long workout, glycogen was

depleted in the leg. After a 2-h rest period, another hour-long workout in the same leg was conducted, but this time it was completed in a glycogen-depleted state. The other leg was trained every day for an hour, but because there was no prior workout to deplete the glycogen in this leg, it was always completed with normal glycogen reserves. This 2-day training cycle was repeated for 10 weeks, and every week the subjects trained 5 days and then rested 2 days. Before and after the 10 weeks of training, a maximal workload test and an endurance test were performed for each leg separately so that the two legs could be compared. There were no differences in maximal workloads between the legs. However, the endurance test (measured as the time to exhaustion at 90% of post-training maximal power output) was nearly twice as great for the leg trained in the low-glycogen state as compared to the leg trained in the high-glycogen state (20 vs. 12 min).

These results demonstrate that endurance performance (although not traditional endurance performance such as running or cycling) was increased by training in a glycogen-depleted state—at least in previously untrained subjects undergoing a short-term training intervention (Hansen et al. 2005). Another interesting finding from this study was that resting glycogen stores were significantly higher in the low-glycogen state as compared to the high-glycogen state at the end of 10 weeks of the training (~11% greater). If maximizing resting glycogen stores is important for endurance performance, the findings from this study support training in a glycogen-depleted state (at times) in order to increase resting skeletal muscle glycogen store capacity, provided that adequate carbohydrates are ingested.

In another study using recreationally active men with a similar design (training once a day vs. twice a day), the low-glycogen training group induced greater oxidative enzyme adaptations than the high-glycogen group did, but there were no differences in endurance performance (Morton et al. 2009). There have also been two similar studies using trained endurance athletes (Hulston et al. 2010; Yeo et al. 2008). Both of these studies also incorporated twice-a-day training versus once-a-day training, and both reported significant adaptations in oxidative metabolism in the low-glycogen group. Also, training intensity during the training programs was compromised, but there were no differences in endurance-exercise performance between the low- and high-glycogen groups.

In summary, much of the literature on the "train low" concept is limited to twice-a day training (low glycogen for the second session). Despite increasing the muscle adaptive response and reducing the reliance on carbohydrate utilization during exercise, there is no clear evidence that these strategies enhance exercise performance in endurance athletes (Burke 2010), but there is evidence of improved endurance performance in untrained male subjects. Also, there is no evidence that this strategy decreases endurance performance in endurance-trained or -untrained populations.

4.2.3 Carbohydrate-Loading Strategies Prior to Competition

Carbohydrate is a limited fuel within the body and, during prolonged exercise, fatigue is often related to muscle glycogen depletion. Carbohydrate loading, commonly referred to as carbo loading, carb loading, or supercompensation, is a strategy used by endurance athletes, such as marathon runners, to maximize the storage

of glycogen in skeletal muscle. The degree to which muscle glycogen stores can be supercompensated varies, with increases ranging from less than 25% (Goforth et al. 2003; Walker et al. 2000; Burke et al. 2000) to a doubling or near doubling (Bussau et al. 2002; Sherman, Peden, and Wright 1991; Karlsson and Saltin 1971) of the preglycogen loaded value (Sedlock 2008). Carbohydrate loading is generally recommended for endurance events lasting longer than 90 min and is not of great importance to athletes involved in events that are short and explosive. The practice of carbo loading dates back to the late 1960s when a group of Scandinavian researchers (Karlsson and Saltin 1971; Bergstrom et al. 1967; Ahlborg et al. 1967) discovered a positive relationship between the amount of glycogen in the body and endurance performance. Different carbohydrate-loading protocols have been advocated since the late 1960s and generally are described as one of the following:

- Classical loading protocol
- Modified loading protocol
- One-day loading protocol

The classical loading protocol was based on a 7-day plan. The classic plan starts with a glycogen-depleting exercise bout 7 days prior to competition and is then followed by 3 days of extremely low carbohydrate intake (10% of total calories from carbohydrate and high intakes of protein and fat). Another exhausting exercise bout is conducted on day 4, after which an extremely high carbohydrate intake (90% of total calories) is followed for 3 more days leading up to the endurance competition. During the 3-day high-carbohydrate intake period, physical activity is limited. In a study in which subjects followed this protocol, glycogen stores were nearly doubled and the subjects exhibited significantly greater endurance in exercise lasting longer than 90 min (Karlsson and Saltin 1971).

Although the classical carbohydrate-loading protocol is effective in increasing muscle glycogen to very high concentrations, it also has some drawbacks that make certain athletes unwilling to integrate this approach into their training. A glycogen-depleting workout just a few days before as well as 3 consecutive days of inactivity immediately before a prolonged endurance event may be disruptive to the usual training program of an endurance athlete. Also, maintaining a 10% carbohydrate diet for 3 or 4 days (without sufficient time to become acclimated to the diet) causes gastrointestinal problems and mood disturbances in some athletes.

Subsequent research has demonstrated that high glycogen storage can be achieved without a depletion stage (a glycogen-depleting exercise bout followed with very low carbohydrate intakes) (Sherman et al. 1981). This “modified” loading protocol was based on a research study conducted by Sherman and colleagues in the early 1980s (Sherman et al. 1981). In this study, the newer, modified approach was compared against the classical approach. Trained runners slowly reduced their training over a 6-day period from 90 min of running at 75% VO_2max to complete rest on the day before a 20.9-km run. Those runners following the classic model ingested a low-carbohydrate diet (25% carbohydrate) for the first 3 days, followed by 3 days of a high-carbohydrate diet (70% carbohydrate). The runners following the modified approach ingested moderate amounts of carbohydrate (50% of calories) for 3 days

followed by 3 days of high carbohydrate (70%). Prior to the run, skeletal muscle glycogen levels were nearly identical in both groups. Also, there were no differences in the performance of the 20.9-kg run between the groups.

The modified loading protocol demonstrated that even without a glycogen-depleting period of exercise, trained athletes could store maximal amounts of muscle glycogen if fed a carbohydrate-rich diet for 3 days. Researchers from Australia investigated if maximal amounts of skeletal muscle glycogen could be stored after only 1 day of dietary manipulation (Bussau et al. 2002). In this investigation, eight endurance-trained male athletes were asked to eat a high-carbohydrate diet (10 g/kg/day) for 3 days while remaining physically inactive. Muscle biopsies were taken prior to carbohydrate loading and after 1 and 3 days of eating the carbohydrate-rich diet. Muscle glycogen content increased significantly (90% increase) from preloading levels after only 1 day and remained stable afterward despite another 2 days of the carbohydrate-rich diet. This 1-day carbohydrate-loading approach shows that combining physical inactivity with a high intake of carbohydrate enables trained athletes to increase muscle glycogen contents significantly within only 24 h.

Another version of a 1-day loading protocol utilized a short, intense bout of exercise on one day, which was followed with a day of rest and the ingestion of a high-carbohydrate diet (in endurance-trained males). The exercise bout consisted of high-intensity cycling exercise for 2.5 min at 130% VO_2max and then an all-out 30-s sprint. The next day the endurance athletes rested and ingested 10.3 g carbohydrate/kg body mass, and the result was an 82% increase in muscle glycogen (Fairchild et al. 2002). This particular version of a 1-day loading protocol shows that a combination of a short-term bout of high-intensity exercise followed by a high-carbohydrate intake enables endurance athletes to attain supranormal muscle glycogen levels within only 24 h (Fairchild et al. 2002).

Some researchers have combined the effects of fat adaptation and carbohydrate loading in an attempt to overcome the decrements in glycogen seen after fat adaptation (Burke et al. 2000, 2002; Brown 2002; Carey et al. 2001). This approach represents the "train low, compete high" concept in that the training period coincides with a low-carbohydrate/high-fat dietary period and that just prior to competition a carbohydrate-loading protocol is followed to maximize carbohydrate stores. Table 4.3 summarizes many of these studies and their effects on endurance performance, although the majority of them were less than 2 weeks in duration.

These studies show that there were generally no differences in endurance athletes' performance when a high-fat diet was followed by carbohydrate loading and a normal diet of high-carbohydrate intake. Regardless of the type and duration of the carbohydrate-loading protocols used, research examining the relationship between carbohydrate loading, muscle glycogen content, and exercise performance includes consumption of a high-carbohydrate diet up until the day of the endurance-performance event (Sedlock 2008). However, findings from some (Goforth et al. 2003; Roedde et al. 1986) but not all (Sherman et al. 1981) investigations suggest that a greater amount of glycogen storage occurs when performing glycogen-depleting exercise (such as in the classical model) rather than lighter exercise bouts (such as a taper utilized in the moderate model) before ingesting a high-carbohydrate diet (Sedlock 2008).

TABLE 4.3
Fat Adaptation Followed by Carbohydrate Loading

Study	Population	Design	Measurements	Findings
Stellingwerff et al. (2006)	Seven endurance-trained cyclists and triathletes	Crossover with a 2-week washout period; two 6-day treatments: • High-fat diet for 5 days (67% fat) followed by 1 day of CHO loading (~10.8 g/kg/day or 70% CHO) • High-CHO diet for 6 days (~10.5 to 10.8 g/kg/day 70% CHO)	Cycling time trial (4 kJ/kg body mass) to be done as fast as possible (time trial followed 20 min of steady-state cycling at 70% VO_2max and a 1-min sprint); tests conducted on day 7	No significant difference between treatments in time trial performance (~13.2 min for each treatment)
Havemann et al. (2006)	Eight endurance-trained male cyclists	Crossover with a 2-week washout period; two 7-day isocaloric treatments: • High-fat diet for 6 days (~68% fat) +1 day of CHO loading (~8–10 g/kg/day or 90% CHO) • High-CHO diet for 6 days (~68% CHO) + 1 day of CHO loading (~8–10 g/kg/day or 90% CHO)	100-km cycling time trial conducted on day 8 of study	No significant difference between treatments in time trial performance (mean performance time was ~2.5% slower on the high-fat compared with the high-CHO diet)
Rowlands and Hopkins (2002)	Seven nationally competitive male cyclists and triathletes	Crossover with a 2-week washout period; three 14-day isocaloric treatments: • High-fat diet (66% fat) • High-CHO diet (8.7 g/kg; 70% CHO) • 11.5-day high-fat diet (65% fat) followed by 2.5-day CHO loading diet (6.9 g/kg/day or 63% CHO)	100-km time trial that followed a 15-min test (aim was to cycle as far as possible); a 45-min steady-state (50% peak power) cycling bout and a 1-h incremental test (total exercise time = 5 h)	No significant difference between treatments for 100-km time trial (high-fat diet slightly improved performance; high-CHO diet slightly decreased performance)

(continued)

TABLE 4.3 (CONTINUED)
Fat Adaptation Followed by Carbohydrate Loading

Study	Population	Design	Measurements	Findings
Burke et al. (2002)	Eight trained male cyclists or triathletes	Crossover with a 2-week washout period; two 6-day isocaloric treatments: • High-fat diet for 5 days (70% fat) +1 day of CHO loading (10 g/kg). • High-CHO diet for 5 days (70% CHO) + 1 day of CHO loading (10 g/kg)	Cycling time trial (7 kJ/kg body mass) to be completed as fast as possible (time trial followed 20 min of steady-state cycling at 70% VO_2max and a 1-min sprint); tests conducted on day 7	No significant difference between treatments in time trial performance (~25 min for each treatment)
Lambert et al. (2001)	Five endurance-trained male cyclists	Crossover with a 2-week washout period; two 13-day isocaloric treatments: • High-fat diet (>65% fat) for 10 days + 3 days of a high-CHO diet (~7 g/kg/day) • Habitual diet for 10 days (~50% CHO; 30% fat) + 3 days of a high-CHO diet (~7 g/kg/day or 65% CHO)	20-km cycling time trial (time trial followed 150-min cycle ride at 70% VO_2peak); time trial conducted on day 14	High-fat diet improved the 20-km time trial performance by ~4% (a significant difference)
Carey et al. (2001)	Seven trained male cyclists or triathletes	Crossover with an 18-day washout period; two 8-day isocaloric treatments: • High-fat diet (69% fat) for 7 days + 1 day of a high-CHO diet (11 g/kg/day or 70% CHO) • High-CHO diet for 8 days (11 g/kg or 70% CHO)	1-h cycling time trial (time trial followed 4 h of cycling at 65% VO_2peak); performance measured by distance covered in 1 h	No significant difference between treatments in distance covered during the 1-h time trial (high-fat treatment improved performance by ~4%)
Burke et al. (2000)	Eight trained male cyclists or triathletes	Crossover with a 2-week washout period; two 6-day isocaloric treatments: • High-fat diet for 5 days (>65% fat) +1 day of CHO loading (10 g/kg) • High-CHO diet for 5 days (70%–75% CHO) + 1 day of CHO loading (10 g/kg)	Cycling time trial (7 kJ/kg body mass) to be completed as fast as possible (time trial followed 2 h of cycling at 70% VO_2max); time trial conducted on day 7	No significant difference between treatments in time trial performance (however, mean time trial time was 8% faster with the high-fat treatment)

Although the modified loading protocol of Sherman et al. (1981) produced muscle glycogen concentrations similar to the classic protocol, other studies yielded equivocal findings regarding the ability to maximize muscle glycogen concentration without initially depleting those stores (Sedlock 2008). The larger increases in muscle glycogen observed after glycogen-depleting exercise are suggested to result from factors such as a faster rate of glycogen resynthesis (Zachwieja et al. 1991), greater glycogen synthase activity (Bogardus et al. 1983), and enhanced glucose transport (Sedlock 2008; Price et al. 1994). Even though glycogen-depleting exercise may be necessary to achieve the highest levels of glycogen supercompensation, many athletes are not willing to engage in this type of protocol in the few days prior to an endurance competition.

Regardless of the protocol chosen, the relationship between carbohydrate loading, increases in muscle glycogen, and subsequent endurance performance is inconsistent (Sedlock 2008). For example, endurance performance was significantly improved with a 13% increase in muscle glycogen (Walker et al. 2000), but not with increases of ~18% (Burke et al. 2000) and 23% (Hawley, Palmer, and Noakes 1997). Likewise, large increases in skeletal muscle glycogen (~100%) were reported to increase (Karlsson and Saltin 1971) or have no effect (Sedlock 2008; Sherman et al. 1981) upon performance. Even though there are inconsistencies (improvement of endurance performance in some studies and no differences in endurance performance in others) with increased carbohydrate stores and endurance performance, there appear to be no decrements in endurance performance as a result of attempts to increase the body's glycogen stores.

4.2.4 Monosaccharides and Endurance Performance

As described in Chapter 3 ("Carbohydrate Metabolism"), the relevant monosaccharides are glucose, fructose, and galactose. Each of these monosaccharides is metabolized differently and therefore may have different effects on endurance performance. The following sections discuss the various monosaccharides and the investigations that have been conducted on them relative to endurance performance.

4.2.4.1 Glucose Supplementation and Endurance Performance

Due to the prominent status that glucose has in the body in terms of producing ATP (adenosine triphosphate), it is not surprising that there has been a lot of research investigating the effects of glucose supplementation on endurance-exercise performance. In fact, glucose supplementation has been studied in regard to its effects on performance when supplemented prior to and during endurance exercise, as well as the combination of both prior to and during the exercise.

4.2.4.1.1 Pre-Exercise Glucose Supplementation

Most but not all studies that have investigated the effects of glucose supplementation prior to endurance exercise have reported improvements in endurance exercise. Tables 4.4 and 4.5 summarize the scientific literature about those studies that have reported improvements in performance (Table 4.4) and those that have not reported an advantage to supplementing with glucose (Table 4.5) prior to endurance exercise.

TABLE 4.4
Summary of Studies Indicating That Pre-Exercise Glucose Supplementation Improves Endurance Performance

Study	Population	Treatment	Amount and Concentration	Timing of Ingestion	Exercise Bout	Results
Tokmakidis and Karamanolis (2008)	Ten male and one female recreational runners	Two treatments: • Glucose • Placebo	1 g/kg glucose; ~74 g in a ~19% solution	15 Min prior to exercise	Running at 70% VO_2max for 45 min followed by 80% VO_2max until exhaustion	Glucose treatment improved performance[a] by ~12.8% (83 vs. 73.6 min)
Spendiff and Campbell (2002)	Ten male upper-body-trained athletes (kayakers)	Two treatments: • Glucose • Placebo	0.58 g/kg glucose; 48 g in an 8% solution	20 Min prior to exercise	Arm cranking at 65% VO_2max for 60 min, followed by a 20-min performance trial to achieve max distance	Glucose treatment improved[a] distance covered by ~9% (12.55 vs. 11.5 km)
El-Sayed, Balmer, and Rattu (1997)	Eight male competitive cyclists	Two treatments: • Glucose • Placebo	0.36 g/kg glucose; ~25 g in an 8% solution	25 Min prior to time trial	1 h simulated cycling time trial	Glucose treatment improved[a] distance covered by 1.2% (equivalent to a 44 s improvement)
Goodpaster et al. (1996)	Ten male competitive cyclists	Two treatments: • Glucose • Placebo	1 g/kg glucose; ~75 g in a ~19% solution	30 Min prior to exercise	Cycling at 66% VO_2max for 90 min, followed by 30 min of "all out" cycling effort	Glucose improved work output by ~7.7% (~434 vs. 403 kJ)
Sherman et al. (1991)	Nine physically active males	Two treatments: • Glucose • Placebo	1.1 g/kg glucose; ~78 g in a 20% solution	60 Min prior to exercise	Cycling at 70% VO_2max for 90 min, followed by a performance time trial equivalent to 45 min of cycling at 70% VO_2max as quickly as possible	Glucose treatment improved performance[a] by ~12.8% (~41 vs. 47 min for the time trial)
Gleeson et al. (1986)	Six healthy, nonobese males	Two treatments: • Glucose • Placebo	1 g/kg glucose; ~71 g in an 18% solution	45 Min prior to exercise	Time to exhaustion on a cycle ergometer at 73% VO_2max	Glucose treatment improved performance[a] by ~13% (109 vs. 96 min)

[a] = significantly different at the $P \leq 0.05$ level.

TABLE 4.5
Summary of Studies Indicating That Pre-Exercise Glucose Supplementation Does Not Improve Endurance Performance

Study	Population	Treatment	Amount and Concentration	Timing of Ingestion	Exercise Bout	Results
Jentjens et al. (2003)	Nine endurance-trained cyclists or triathletes	Two treatments: • Glucose • Placebo	~0.34 g/kg, ~1 g/kg, and ~2.7 g/kg glucose; as 25, 75, and 200 g, respectively, in each respective ~4%, ~13%, and ~33% solution	45 Min prior to exercise	Cycling at 72% Vo_2max for 20 min followed by a time trial equal to approximately 40 min of cycling at 80% W_{max}	No difference in performance: 25 g glucose = 43.3 min; 75 g glucose = 43 min; 200 g glucose = 43 min; placebo = 42.5 min
Tokmakidis and Volaklis (2000)	Eight healthy males	Two treatments: • Glucose • Placebo	1 g/kg glucose; ~71 g in ~20% solution	30, 60, or 90 Min prior to exercise	Running at 65% Vo_2max for 60 min and then 75% Vo_2max until exhaustion	Glucose ingestion improved performance 3.4%, 5.6%, and 11.6% when ingested 30, 60, and 90 min before exercise as compared to the placebo, but differences were not significant
Chryssanthopoulos et al. (1994)	Five male and four female recreational runners	Two treatments: • Glucose • Placebo	~1 g/kg of glucose; 75 g in a 25% solution	30 Min prior to exercise	Running to exhaustion at 70% Vo_2max	Glucose treatment exercised ~10% longer than placebo (~133 vs. 121 min), but difference was not significant
Hargreaves et al. (1987)	Six male cyclists (four trained, two untrained)	Two treatments: • Glucose • Placebo	1 g/kg of glucose; 75 g in a ~21% solution	45 Min prior to exercise	Time to exhaustion on a cycle at 75% Vo_2max	No difference in performance (glucose = 92.8 min; placebo = 92.7 min)

Summarizing these findings, ingesting supplemental glucose 15 min to 1 h prior to exercise in amounts ranging from 0.36 to 1 g/kg body mass (in solutions ranging from 8% to 20%) significantly improves endurance exercise of both recreationally trained and competitively trained endurance athletes. Specific to trained, competitive endurance athletes, ingesting 1 g of carbohydrate (glucose)/kg body mass approximately 1 h prior to exercise is justified in terms of enhancing endurance performance.

Only two studies exist in which pre-exercise glucose supplementation actually decreased endurance performance. In the first of these studies, the trained cyclists ingested ~1.1 g/kg carbohydrate about 30 min prior to exercise. It was reported that performance was decreased by 19% at a workload requiring 80% VO_2max while cycling to exhaustion lasting about 50 min, but was not affected at an exercise intensity of 100% VO_2max lasting about 5 min (Foster, Costill, and Fink 1979). Another study that has supported the findings of this study reported that 100 g of glucose (~1.5 g/kg in ~28% solution) given to five trained male distance runners 1 h prior to an intermittent ride to exhaustion on a cycle ergometer at a workload requiring 85% VO_2max decreased performance by 25% compared to a control trial (Keller and Schwarzkopf 1984). It is important to note that in the past 30–40 years these studies have not been confirmed with other research supporting their findings. In contrast, a majority of the scientific evidence either supports the use of pre-exercise glucose supplementation or, at a minimum, suggests that pre-exercise glucose supplementation has no negative effect on endurance performance (refer to Tables 4.4 and 4.5).

4.2.4.1.2 Glucose Ingestion during Endurance Exercise

Research investigating the effects of glucose supplementation during endurance exercise closely mirrors the reports of glucose supplementation ingested prior to endurance exercise, with most studies reporting an enhancement of endurance performance (Smith et al. 2010; Hulston and Jeukendrup 2008; Green et al. 2007; Bjorkamn et al. 1984; Brooke, Davies, and Green 1975) and a few reporting no improvements in endurance performance (Carter et al. 2004). One of the best designed studies investigated three different levels of glucose ingestion during cycling exercise (Smith et al. 2010). In this study, 12 trained male cyclists and triathletes ingested glucose at 15 g/h (in a 1.5% solution), 30 g/h (in a 3% solution), and 60 g/h (in a 6% solution) during a 2-h cycling bout conducted at an intensity of ~77% VO_2peak. A 20-km time trial followed this and served as the endurance-performance measure (no fluid was ingested during the time trial). Ingesting 60 g glucose/h (0.77 g/kg/h in a 6% solution) improved performance by 4.7%, while the 30 and 15 g/h only improved performance by 3.8% and 3.3%, respectively (Smith et al. 2010).

Other studies supplying glucose supplementation during endurance exercise have reported similar performance-enhancing results, both in trained (Hulston and Jeukendrup 2008; Bjorkamn et al. 1984) and untrained (Green et al. 2007) populations. The amount of glucose ingested during endurance exercise ranged from 52 to 69 g/h (or, relative to body weight, ranged from 0.72 to 1.23 g/kg/h) and were provided in a 6% or 7% solution. The improvement in endurance performance ranged from 4% (Hulston and Jeukendrup 2008) to approximately 19% (Green et al. 2007; Bjorkamn et al. 1984). Assuming that glucose is the only carbohydrate source ingested during endurance exercise, the scientific literature supports an average ingestion of about

1 g/kg/h in a 6%–7% solution to improve endurance performance (Smith et al. 2010; Hulston and Jeukendrup 2008; Green et al. 2007; Bjorkamn et al. 1984).

4.2.4.1.3 The Issue of Rebound Hypoglycemia

Consuming a high glycemic index carbohydrate, such as glucose, in the hour before exercise results in hyperglycemia followed by a large increase in plasma insulin concentration (Costill et al. 1977; Koivisto, Karonen, and Nikkilä 1981). To demonstrate this, Costill and colleagues (1977) gave endurance-trained men 75 g of glucose 45 min prior to initiating treadmill running. The ingestion of glucose resulted in an increase of blood glucose from ~5 mmol/L (90 mg/dL) to 7.2 mmol/L (130 mg/dL), which was accompanied by a 3.3-fold increase in plasma insulin. In the first 20 min of running on the treadmill, plasma glucose dropped dramatically to a low of 3.5 mmol/L (63 mg/dL), which was significantly below baseline values. This hypoglycemic state persisted through the end of the 30-min treadmill run protocol. Other studies have reported similar findings: Ingesting glucose ~45 min prior to endurance exercise results in significant elevations in blood glucose and insulin (Koivisto et al. 1981; Foster et al. 1979). Then, after exercise begins, insulin and blood glucose levels decrease, with blood glucose levels decreasing to levels below baseline and reaching hypoglycemic levels. This phenomenon is referred to as rebound hypoglycemia.

TOPIC BOX 4.1 HYPOGLYCEMIA

Hypoglycemia is an abnormally low content of glucose in the blood. The term literally means "low blood sugar." Most healthy adults maintain fasting glucose levels above 4.0 mmol/L (72 mg/dL) and develop symptoms of hypoglycemia when the glucose falls below a critical threshold level (often 3 mmol/L or 54 mg/dL).

A potential issue with pre-exercise carbohydrate supplementation and the resulting rebound hypoglycemia is that the rise in insulin suppresses fat metabolism and accelerates carbohydrate oxidation. Suppression of fat metabolism is not ideal because optimal performance of endurance activities is associated with a greater ability to oxidize fat in the body. The other concern is that with an accelerated carbohydrate oxidation, the body's carbohydrate stores may be more rapidly depleted. This concern is largely unfounded, however, because the increase in carbohydrate availability associated with pre-exercise carbohydrate supplementation more than offsets the increase in exercise carbohydrate utilization (Hawley and Burke 1997).

The well-respected sports nutrition researchers John Hawley and Louise Burke addressed the issue of rebound hypoglycemia (Hawley and Burke 1997) very thoroughly. In their discussion, they cited the results of the Foster investigation (Foster et al. 1979), which reported that feeding 75 g of glucose 30 min before exercise impaired cycle time to exhaustion at 84% of VO_2max but did not alter the length of time subjects were able to ride during more intense (100% of VO_2max) exercise

(Hawley and Burke 1997; Foster et al. 1979). Foster and cohorts observed a rapid drop in blood glucose concentration during the first 10 min of exercise after subjects had been fed carbohydrate, but noted that this response was transient and was not associated with fatigue (Hawley and Burke 1997).

Hawley and Burke (1997) continued by stating:

> Unfortunately the results of this study have been so widely reported and publicized that warnings to avoid carbohydrate intake during the hour before endurance exercise have become part of sports nutrition dogma. However, a review of the literature reveals that this is the only study to find reductions in performance capacity after the ingestion of carbohydrate in the hour before exercise. Other investigations have found either no detrimental effect or improvements in performance ranging from 7% to 20%. (Chryssanthopoulos, Hennessy, and Williams 1994; Gleeson, Maughan, and Greenhaff 1986; McMurray, Wilson, and Kitchell 1983).

This assessment by these respected sports nutritionists indicates that concerns over pre-exercise carbohydrate supplementation leading to decrements in performance because of rebound hypoglycemia are exaggerated and not supported in the scientific literature. Despite this, many other studies have been conducted to offset the perceived harm resulting from rebound hypoglycemia. In particular, research investigating the effects of galactose and fructose supplementation has been conducted. For example, galactose ingestion produces a menial blood glucose response (Royle et al. 1978; Gannon, Khan, and Nuttall 2001) and virtually no insulinogenic activity (Ganda et al. 1979). Similarly, fructose ingestion does not appear to elevate blood glucose or insulin (Koivisto et al. 1981) and demonstrates little or no decline of blood glucose concentrations at the onset of exercise (Hargreaves et al. 1985; Koivisto et al. 1981). Another potential benefit of ingesting carbohydrate sources (such as fructose and galactose) that do not elevate insulin is that there would not be a corresponding inhibition of free fatty acid availability due to insulin's antilipolytic effects. The following sections consider the effects that fructose and galactose have on endurance-exercise performance.

4.2.4.2 Galactose Supplementation and Endurance Performance

A study investigating the effects of pre-endurance galactose supplementation on fuel utilization during the exercise bout may show some promise for potentially improving endurance performance (O'Hara et al. 2012). Following an overnight fast, nine trained male cyclists completed three bouts of cycling at 60% of their maximal power output for 120 min. Thirty minutes prior to exercise the cyclists ingested a 1-L solution of one of the following:

- Placebo beverage
- 75 g of galactose (7.5% solution)
- 75 g of glucose (7.5% solution)

Each of the carbohydrate supplements was given in a relative dosage of ~0.9 g carbohydrate/kg body mass. Also, each carbohydrate source contained stable isotope

carbon tracers so that their metabolism could be traced throughout the exercise bout. It was reported that glucose produced significantly higher exogenous carbohydrate oxidation rates during the first hour of exercise, while glucose rates derived from galactose (remember that a majority of ingested galactose is converted to glucose in the liver) were significantly higher during the last hour of exercise. Stated more simply, this means that the glucose supplementation was digested and absorbed rapidly, allowing the active skeletal muscles to take up the plasma glucose and fuel the exercise. In contrast, the galactose was digested more slowly and then was transported to the liver for its conversion to glucose and, over time, released to the blood to fuel the endurance exercise. Specifically, during the second hour of endurance exercise, the ingested galactose was the primary carbohydrate source oxidized (or catabolized) for energy.

To help explain this observation, it was also reported that the plasma glucose (~7.4 mmol/L or 133 mg/dL) and serum insulin concentrations were significantly higher (compared to the placebo and galactose supplementation) following the pre-exercise glucose supplementation directly prior to exercise. In contrast, at 1 h into the exercise bout, plasma glucose concentrations were significantly higher for the galactose and placebo treatments compared to the glucose supplementation. Regardless of the carbohydrate source, plasma glucose concentrations fell rapidly after the first 20 min of exercise—a common occurrence.

This study confirmed that the pre-exercise consumption of glucose provides a higher exogenous source of carbohydrate during the initial stages of exercise, but that galactose provides the predominant exogenous source of fuel during the latter stages of exercise (O'Hara et al. 2012). These results set the foundation for the potential ergogenic effects that galactose may have during prolonged endurance exercise. A few different studies have investigated the effects of galactose supplementation on endurance performance in well-trained male cyclists. In these studies, the galactose supplementation was ingested prior to exercise (Jentjens and Jeukendrup 2003; Stannard, Hawke, and Schnell 2009; Macdermid et al. 2012) and in some cases during exercise (Stannard et al. 2009; Macdermid et al. 2012).

In the first of these studies, eight well-trained male cyclists completed three exercise trials separated by 7 days (Jentjens and Jeukendrup 2003). The exercise bout consisted of 20 min of submaximal steady-state exercise at 65% of maximal power output immediately followed by a simulated time trial. The time to complete the simulated time trial determined if there were any differences in the various carbohydrate supplements ingested in terms of improving endurance-exercise performance.

Forty-five minutes prior to the start of exercise subjects consumed 500 mL of a beverage containing:

- 75 g of glucose in a 15% solution
- 75 g of galactose in a 15% solution
- 75 g of trehalose in a 15% solution (trehalose is a disaccharide containing two glucose molecules with a different bonding arrangement than maltose)

Each of the carbohydrate supplements was given in a relative dosage of ~1 g of carbohydrate/kg body mass. Plasma glucose concentration 15 min after ingestion

was significantly higher in the glucose treatment as compared to the galactose and trehalose treatments. Thirty minutes after ingestion, plasma glucose was still significantly higher following glucose ingestion when compared to galactose ingestion. This was accompanied by a more than twofold greater rise in plasma insulin concentration in glucose compared to galactose and trehalose. Despite these differences in plasma glucose and insulin prior to the initiation of the exercise bout, there were no differences observed in the time trial performance in the three trials (all finished the time trial in approximately 42 min or within 2% of each other) (Jentjens and Jeukendrup 2003).

Another study tested the hypothesis that supplementation with galactose before and during endurance exercise would improve endurance performance compared with a typical sports drink formulation (Stannard et al. 2009). Nine well-trained cyclists undertook three trials, each consisting of 120 min at 65% VO_2max followed immediately by a self-paced time trial. The time trial was set up in a way that required the completion of a set amount of work equivalent to 80% VO_2max for 30 min.

Prior to and during the cycling endurance-exercise bout, the cyclists consumed one of the following carbohydrate supplements:

- 100% Galactose in an 8% solution
- 50% Galactose/50% glucose in an 8% solution
- 80% Glucose/20% fructose in an 8% solution

The carbohydrate supplements were allocated in a randomized balanced design and administered in a double-blinded fashion. The supplements were consumed as 0.67 g/kg carbohydrate 45 min prior to exercise, increasing to 1 g/kg carbohydrate/h for the first 120 min of exercise and then decreasing to 0.33 g/kg carbohydrate during the time trial.

As expected, changes in plasma glucose and serum insulin were different following the galactose ingestion as compared to the other carbohydrate groups. Specifically, plasma glucose slightly decreased during the 45-min rest/pre-exercise period in the galactose group, while the other two treatments produced an increase in glucose concentration over this time period. Serum insulin concentration significantly increased during the 45-min rest/pre-exercise period in both the galactose/glucose and glucose/fructose groups, but did not change from baseline levels in the galactose group.

In relation to performance, mean time trial power was significantly less following galactose supplementation compared with the galactose/glucose supplementation trial. Further, the galactose group took significantly longer (~37.5 min) to complete the time trial as compared to the other carbohydrate treatments (~33 min). The authors concluded that ingestion of an 8% galactose-only solution is detrimental to endurance performance compared with equivalent volumes of iso-osmotic solutions containing 50% galactose/50% glucose or 80% glucose/20% fructose solutions (Stannard et al. 2009).

Another research group sought to determine the effects of galactose or a glucose-maltodextrin supplement (containing 50% glucose and 50% maltodextrin) on

self-paced endurance cycling performance (Macdermid et al. 2012). Both carbohydrate supplements were ingested prior to and during exercise. On two separate occasions, eight competitive male endurance-trained cyclists performed a 100-km time trial as fast as possible. In order to make time trial representative of competitive race cycling, four 1-km maximal effort sprints and four 4-km maximal effort sprints were inserted periodically throughout the 100-km distance. One hour prior to the time trial, approximately 38 g of galactose or glucose-maltodextrin was consumed in 600 mL of fluid in a 6% carbohydrate solution. During exercise, the galactose or glucose-maltodextrin was ingested at a rate of approximately 37 g/h. The cyclists ingesting the galactose supplement completed the time trial in ~138 min as compared to ~142 min for those ingesting the glucose-maltodextrin treatment. This was about 3% faster, but this time difference was not statistically different between the treatments (Macdermid et al. 2012). In summary, there does not appear to be any enhancement of endurance performance before and during exercise supplementation of galactose.

4.2.4.3 Fructose Supplementation and Endurance Performance

Several studies have investigated the effects that fructose ingestion has on endurance performance (Brundle, Thayer, and Taylor 2000; Hargreaves et al. 1987; Okano et al. 1988; Bjorkamn et al. 1984; McMurray et al. 1983). These studies have investigated fructose ingestion prior to endurance exercise (Okano et al. 1988; Hargreaves et al. 1987), during exercise (Bjorkamn et al. 1984), and both prior to and during endurance exercise (Brundle et al. 2000).

The first of these studies highlights the effectiveness of fructose supplementation in comparison with a placebo beverage on improving endurance performance (Okano et al. 1988). Twenty recreationally active males (and recently trained on a cycle ergometer) ingested two levels of fructose or a placebo (500 mL of flavored water) 4 h after eating a normal lunch. The lower level of fructose consisted of 60 g (in a 12% solution) and provided 0.94 g carbohydrate/kg body mass. The higher level of fructose consisted of 85 g (in a 17% solution) and provided 1.3 g/kg body mass. Each treatment was given 60 min prior to engaging in progressive cycling exercise starting at ~62% VO_2max and progressing to ~81% VO_2max at 120 min of exercise. Each subject cycled until exhaustion.

Time to exhaustion was significantly greater in the fructose treatments (145 min) as compared to the placebo treatment (132 min). Also, 85 g of fructose ingestion (1.3 g/kg) was significantly greater than the 60 g of fructose ingestion (0.94 g/kg) in terms of endurance performance. This study demonstrated that fructose supplementation, regardless of amount, is better than ingesting nothing in regard to improving endurance performance (Okano et al. 1988).

In contrast, Hargreaves and colleagues (1987) reported that fructose ingestion was no better than a placebo. In this study, six male cyclists ingested a normal diet for 2 days prior to each trial, which was separated by at least 1 week in a double-blinded manner. Six hours after the last meal and 45 min prior to exercise, each cyclist ingested the following:

- 75 g of fructose in 21% solution; 1 g CHO/kg
- 75 g of glucose in 21% solution; 1 g CHO/kg
- 350 mL of flavored water placebo

The cycling exercise was conducted at 75% VO_2max until exhaustion. The time to exhaustion was similar in all three trials: fructose = ~91 min; glucose = ~93 min; and placebo = ~93 min (Hargreaves et al. 1987). This finding is unique in that there is usually a performance benefit when carbohydrate is ingested prior to endurance exercise.

The two studies discussed next are similar in that fructose was given during endurance exercise. Eight trained physical education students (but not trained cyclists or runners) ingesting a normal diet engaged in a cycling exercise at 68% VO_2max until exhaustion (Bjorkamn et al. 1984). On three separate occasions, separated by 8 days, each subject ingested 52.5 g of fructose in 250 mL of water (21% solution providing 0.7 g CHO/kg body mass), 52.5 g glucose in 250 mL water (21% solution providing 0.7 g CHO/kg body mass), or a flavored water placebo every 20 min until exhaustion. The glucose treatment (137 min) resulted in a significant improvement in exercise to exhaustion as compared to both fructose (114 min) and the placebo ingestion (116 min). The fructose was no better than the placebo in this study (Bjorkamn et al. 1984).

Brundle and colleagues (2000) instructed 17 trained male endurance athletes ingesting a normal diet to cycle to exhaustion at an intensity of 75% VO_2max. One hour prior to exercise, each athlete ingested 1 g/kg body weight of fructose or glucose in a 14% solution (an average of ~70 g of carbohydrate). During exercise, 0.4 g/kg body weight of glucose or fructose in an 11% solution was ingested at 20, 50, and 90 min. Both the glucose (~160 min) and fructose (~160 min) groups had significantly longer exercise times to exhaustion as compared to the control group (~120 min). There were no significant differences between the glucose and fructose treatments, however (Brundle et al. 2000).

Taking each of these studies into consideration, fructose supplementation ingested prior to and during exercise will not decrease endurance performance. In some instances, it improves endurance exercise as compared to a placebo (Okano et al. 1988; Brundle et al. 2000). Other findings have reported that fructose ingestion is no better than a placebo (Hargreaves et al. 1987; Bjorkamn et al. 1984). When compared directly against glucose, fructose is either inferior (Bjorkamn et al. 1984) or offers no advantage (Hargreaves et al. 1987; Brundle et al. 2000).

The main reason that some favor ingesting fructose, rather than glucose or glucose polymers, prior to and during endurance exercise is because of the potentially adverse effects (but not substantiated) of rebound hypoglycemia. Another reason associated with rebound hypoglycemia is an attempt to maintain skeletal muscle glycogen stores during exercise. Several investigations have demonstrated that maintaining skeletal muscle glycogen is important and is one of the benefits of ingesting carbohydrates prior to endurance exercise (Bergstrom et al. 1967; Bergstrom and Hultman 1967; Hermansen, Hultman, and Saltin 1967).

In some studies, accelerated depletion of muscle glycogen has been reported when exercise is performed after glucose ingestion (Costill et al. 1977). However, given the

fact that fructose has a different metabolic effect on the body (lower blood glucose and insulin response), it has been suggested that fructose ingestion before exercise might provide an ideal carbohydrate source and improve glycogen utilization during endurance exercise (Koivisto et al. 1985). When fructose was directly compared to glucose, fructose was no more effective than glucose or a placebo in sparing muscle glycogen during long-term exercise (Koivisto et al. 1985).

4.2.5 Carbohydrate Ingestion during Endurance Exercise (Multiple Transportable Carbohydrates)

Carbohydrate ingestion during prolonged exercise (more than 1 to 2 h) can enhance performance. Carbohydrate ingestion during prolonged exercise most likely works by providing an alternative energy source to the working skeletal muscles, which therefore spares the body's small carbohydrate stores (liver and skeletal muscle glycogen). The quantity of carbohydrates consumed during exercise is determined by considering two factors (Fink, Burgoon, and Mikesky 2009):

1. The rate at which gastric emptying and intestinal absorption of carbohydrates occurs
2. The rate of carbohydrate oxidation (how fast the ingested carbohydrate is utilized for energy)

Carbohydrates ingested during endurance exercise help to maintain high rates of carbohydrate oxidation, which is necessary to maintain relatively high exercise intensities. In the past, it was suggested that the maximal oxidation rate of ingested carbohydrates was approximately 1 g/min (equating to an approximate maximal intake of 60 g carbohydrate/h during endurance exercise) (Jeukendrup and Jentgens 2000). Several strategies have attempted to influence the oxidation of ingested carbohydrate beverages, including the manipulation of the feeding schedule, exercise intensity, and the type and amount of the carbohydrate ingested. Some of these factors, such as the timing of the ingestion, have little to no effect on carbohydrate oxidation. In contrast, the amount and type of the carbohydrate have major effects on exogenous carbohydrate oxidation rates.

Some types of carbohydrates can be oxidized at much greater rates than other types (Adopo et al. 1994; Massicotte et al. 1989). Table 4.6 categorizes some of the different types of carbohydrates into those that are slowly oxidized, rapidly oxidized, and very rapidly oxidized. Traditionally, it was believed that the maximal amount of carbohydrate oxidation was between 60 and 70 g carbohydrate/h. A carbohydrate intake above this amount, it was believed, did not result in an increase in the oxidation of the carbohydrate, and therefore it was concluded that there was no point to ingesting more than 60–70 g carbohydrate/h during endurance exercise.

The likely reason that exogenous carbohydrate oxidation is limited to approximately 60 g/h is because of limitations in intestinal absorption. Using glucose as an example, when approximately 60 g of glucose is ingested per hour, it "saturates" its transporter SGLT1 (this transporter is responsible for taking glucose from the

TABLE 4.6
Oxidation Rates of Different Carbohydrates and Carbohydrate Combinations

Slowly Oxidized Carbohydrates (~30 g/h)	Rapidly Oxidized Carbohydrates (~60 g/h)	Very Rapidly Oxidized Carbohydrates (~90 g/h)
Amylose	Amylopectin	Glucose/fructose
Fructose	Glucose	Glucose/sucrose
Galactose	Maltose	Glucose/sucrose/fructose
	Maltodextrins	
	Sucrose	

inside of the intestines and delivering it to the blood vessels). If glucose is ingested above this amount, it will not result in more oxidation due to the fact that its intestinal transporter is saturated. Despite this proposed ceiling to carbohydrate oxidation, some research has reported carbohydrate oxidation rates above the 60–70 g/h rates. This research has demonstrated that in order to get more carbohydrate transported from the intestine to the blood stream, another type of carbohydrate must be ingested and that carbohydrate must use another type of transporter. This strategy for increasing carbohydrate availability and ultimately carbohydrate oxidation during exercise is referred to as "multiple transportable carbohydrates." Much of this work is attributed to the sports nutrition researcher Asker Jeukendrup and his research team (Jeukendrup 2010; Jeukendrup and Moseley 2010; Currell and Jeukendrup 2008).

Specifically, it has been demonstrated that fructose (which is a different type of carbohydrate than glucose) uses a different transporter called GLUT5. So, if enough glucose is given to saturate its transporter and enough fructose is ingested in the same beverage, more carbohydrate should be transported into the bloodstream due to the extra amount of carbohydrate that is ingested in the form of fructose. Several studies have confirmed that rates of carbohydrate oxidation have been significantly increased utilizing this multiple transportable carbohydrate strategy (Jentjens and Jeukendrup 2005; Wallis et al. 2005). A mixture of glucose + fructose (Jentjens, Moseley, et al. 2004), glucose + sucrose (Jentjens, Venables, and Jeukendrup 2004), or glucose + fructose + sucrose (Jentjens, Achten, and Jeukendrup 2004) results in approximately 20%–55% higher exogenous carbohydrate oxidation rates compared with the ingestion of an isocaloric amount of glucose and can lead to peak oxidation rates of approximately 1.7 g/min (Jentjens, Achten, et al. 2004). As stated before, this is significantly higher than the original theory that carbohydrate oxidation rates were maximized at about 1 g/min.

A common composition of the glucose/fructose or the maltodextrine/fructose is a 2:1 ratio. In order to achieve the high oxidation rates that these multiple transportable carbohydrates provide, large amounts of carbohydrate must be ingested. Unfortunately, ingestion of large amounts of carbohydrate is not possible during competitive events. For endurance events lasting for several hours (~3 h), a realistic goal would be to ingest 90 g carbohydrate/h with a mixture of glucose and

fructose, maltodextrin and fructose, or glucose, sucrose, and fructose. It is important to remember that if a single carbohydrate source were ingested at the rates designed for multiple transportable carbohydrates, the single carbohydrate source would accumulate in the intestine and lead to gastrointestinal distress (Jeukendrup 2010).

Multiple transportable carbohydrates have been shown repeatedly to increase carbohydrate oxidation rates. However, just because favorable metabolic adaptations occur in the body does not always mean that these changes equate to enhancements in exercise or sport performance. There have been a few studies that have investigated the ingestion of multiple transportable carbohydrates and their effects on endurance performance (Rowlands et al. 2012; McGawley, Shannon, and Betts 2012; Hottenrott et al. 2012). In one of these studies, amateur triathletes completed two simulated Olympic distance triathlons (McGawley et al. 2012). The swim and cycle sections of the main trials were of fixed intensities, while the running portion (10 km) was completed as a time trial. In one of the triathlons, the triathletes ingested 1.8 g/min (approximately 108 g/h) of a carbohydrate solution consisting of a 2:1 maltodextrin/fructose mixture in a 14.4% solution. In the other triathlon, the athletes ingested a placebo. Performance in the running segment was 4% faster when ingesting the carbohydrate solution (40.4 vs. 38.7 min) (McGawley et al. 2012).

TOPIC BOX 4.2 SPORTS DRINKS

Maltodextrins and glucose polymers are popular types of carbohydrates (categorized as both oligosaccharides and polysaccharides) found in sports drinks marketed to endurance athletes. They are absorbed rapidly from the digestive tract and are equally effective as other types of carbohydrates in terms of maintaining blood glucose levels during exercise (Murray et al. 1989; Flynn et al. 1987). When choosing between the monosaccharide or a glucose polymer/maltodextrin carbohydrate source, there appears to be no physiological or performance advantage of one type over the other. Most sports drinks available to athletes contain a combination of glucose, fructose, sucrose, and maltodextrin/glucose polymers. One of the reasons that maltodextrins and glucose polymers are frequently added to sports drinks is because they are less sweet than sucrose or glucose and the lower sweetness level permits a higher concentration of carbohydrate without making the product too sweet to consume (Manore and Thompson 2000).

Deciding the type and amount of carbohydrate to ingest during endurance exercise ultimately depends on the duration of the endurance event. When making decisions concerning the amount of carbohydrate to ingest during endurance exercise, it is important to understand that competitive endurance events lasting about 1 h are not limited by the availability of muscle glycogen stores, given adequate nutritional preparation. Therefore, ingesting supplemental carbohydrate when adequate carbohydrate stores exist in the body and when the endurance event is about an hour in length will not lead to enhancements of endurance performance. General

TABLE 4.7
Guidelines for Carbohydrate Intake during Endurance Events

Duration of Endurance Event	Recommended Intake	Carbohydrate Type
Less than 1 h	None	Not applicable
1–2 h	Up to 30 g/h	Most forms
2–3 h	Up to 60 g/h	Those that are rapidly oxidized[a]
Greater than 3 h	Up to 90 g/h	Those that are rapidly oxidized (multiple transportable carbohydrates)

[a] Refer to Table 4.6 for the oxidation rates of different types of carbohydrates.

recommendations for the types and amounts of carbohydrate intakes during endurance events are summarized in Table 4.7.

4.2.6 Resynthesizing Glycogen in the Postexercise Period

Following endurance exercise, muscle and liver glycogen stores are both depleted. The extent to which these stores are depleted depends primarily on the duration and intensity of the preceding endurance-exercise bout. Even though both storage sites are depleted, muscle glycogen is preferentially resynthesized first. The goal for most endurance athletes is to replenish stores of muscle and liver glycogen so that glycogen levels will not be a limiting factor during the next exercise session. Such would be the case for athletes who train two or three times per day. Another application for desiring rapid muscle glycogen synthesis would be for the athlete who trains intensely every day and must have an adequate recovery in the 1-day rest interval. Unless sufficient carbohydrates are consumed in the diet after training or competition, muscle glycogen will not normalize to pre-exercise levels. Interestingly, when no carbohydrate source (absence of food intake) is ingested after endurance exercise, glycogen levels are replenished, but not to pre-exercise levels. This observation is somewhat surprising because if no food or carbohydrate source were ingested following exercise, one would assume that glycogen levels would stay at the reduced postexercise levels until carbohydrate or other food sources are ingested.

Maehlum and Hermansen (1978) demonstrated this when they instructed five normal subjects to exercise to exhaustion at 70% VO_2max. Following this exercise bout, the subjects were not allowed to ingest food during the next 12 h. The exercise bout decreased muscle glycogen to ~30% of pre-exercise levels. After 4 h of recovery (with no carbohydrate or food ingestion), muscle glycogen had increased to ~40% of pre-exercise levels, but no further increases in muscle glycogen were observed during the next 8 h of recovery. This study demonstrated that, after endurance exercise, even in the fasted state, skeletal muscle glycogen levels are resynthesized to a small extent and the likely mechanism for this resynthesis is through an increased hepatic gluconeogenesis (Maehlum and Hermansen 1978).

This relatively slow rate of glycogen replenishment does not pose major problems for recreational athletes or for those who exercise three to four times per week and

ingest adequate amounts of carbohydrates in their diets. However, for endurance athletes or for those athletes who have multiple competitions or training sessions in a day or during consecutive days, the slow glycogen resynthesis rates (up to 24 h) may have an adverse effect on performance. For the athlete who wishes to maximize skeletal muscle glycogen as rapidly as possible following training or competition, a focus on the timing (Wallis et al. 2008; Parkin et al. 1997; Burke et al. 1996; Ivy, Katz, et al. 1988), amount (Ivy, Lee, et al. 1988), and type (Burke et al. 1993; Conlee, Lawler, and Ross 1987; Blom et al. 2007) of carbohydrate is important.

4.2.6.1 Timing

Following exercise in which skeletal muscle glycogen is significantly reduced, muscle glycogen synthesis increases rapidly at first, and then it gradually decreases over the next 24-h period. In the first hour after exercise, glycogen resynthesis can occur rather quickly in the absence of insulin. However, the presence of glucose and insulin will greatly facilitate the resynthesis rate. In this regard, the timing of carbohydrate ingestion following exercise is important for short-term muscle glycogen resynthesis. To demonstrate this, 12 male cyclists exercised on a cycle ergometer for over an hour to deplete skeletal muscle glycogen (Ivy, Katz, et al. 1988). On one occasion, the cyclists ingested a 25% carbohydrate solution (2 g/kg body weight) immediately after exercise and on another occasion they ingested the same carbohydrate beverage 2 h after exercise. During the first 2 h following exercise, the rate of muscle glycogen resynthesis was significantly greater when the cyclists ingested the carbohydrate beverage immediately after exercise as compared to waiting for 2 h. During the next 2 h of recovery, the rates of muscle glycogen resynthesis were similar in the two different treatments. However, the rate of muscle glycogen resynthesis was about 45% lower than that for the immediate postexercise carbohydrate ingestion observed during the first 2 h of recovery. The results of this investigation demonstrated that delaying the ingestion of a carbohydrate supplement after exercise results in a reduced rate of muscle glycogen storage.

Does the amount of carbohydrate ingested at each time point in the postexercise period affect the rate of glycogen resynthesis over a 24-h recovery period? The answer to this question was provided by Burke and colleagues (1996) when they fed eight triathletes either four large meals or 16 frequent, small meals following glycogen-depleting exercise over a 24-h period. Both feeding strategies provided 10 g carbohydrate/kg body mass over the 24-h period. There was no significant difference in muscle glycogen storage between the two groups 24 h after the glycogen-depleting exercise. The results of this study suggest that there is no difference in postexercise glycogen storage over 24 h when a high-carbohydrate diet is fed as small, frequent snacks or as large meals. Another practical application that this study provides is the finding that it is unnecessary for athletes who rest 1 or more days between intense training sessions to practice nutrient timing to stimulate glycogen replenishment, provided sufficient carbohydrates are consumed during the 24-h period after the exercise bout.

4.2.6.2 Amount

Cyclists ingested three different amounts of a glucose polymer solution immediately and 2 h following a glycogen-depleting cycling exercise that was 2 h in duration (Ivy, Lee, et al. 1988). The three different carbohydrate amounts were the following:

- 0 g of carbohydrate (placebo)
- 1.5 g/kg body weight (equivalent to 0.75 g/kg/h)
- 3.0 g/kg body weight (equivalent to 1.5 g/kg/h)

At 2 and 4 h after the exercise bout, it was observed that muscle glycogen storage was significantly increased above the placebo condition after ingestion of the medium and large carbohydrate intakes. However, muscle glycogen synthesis rates were not different between the 1.5 and the 3.0 g/kg body weight amounts. From this study, it can be concluded that 1.5 g/kg body weight is the maximal amount of carbohydrate intake above which no further benefits in skeletal muscle glycogen resynthesis occur. A general recommendation is to consume 1.0 to 1.5 g carbohydrate/kg body weight immediately after the training session and again every other hour for up to 4 h when there is less than 8 h of recovery available between two exercise or competition sessions (Burke et al. 2011; Rodriguez et al. 2009; Kerksick et al. 2008).

4.2.6.3 Type

In terms of the types of carbohydrates to ingest following exercise, it appears as if glucose, sucrose, and glucose polymers/maltodextrins are about equal in their abilities to resynthesize muscle glycogen. A landmark study demonstrated the effects that different types of carbohydrate ingestion after exercise can have on glycogen resynthesis rates (Blom et al. 1987). Two experiments were conducted in this study. In one experiment, three levels of glucose were tested: a high (1.4 g/kg body weight), medium (0.7 g/kg body weight), and low (0.5 g/kg body weight) concentration. In the other experiment, three types of carbohydrates (glucose, sucrose, and fructose) were tested at equal concentrations (0.7 g/kg/body weight). The high- and medium-glucose solutions replaced glycogen equally, as did glucose and sucrose fed at the same concentrations. Fructose did not replace muscle glycogen nearly as well as the other types of carbohydrates. (Fructose is better for resynthesizing liver glycogen than synthesizing muscle glycogen.)

Another difference in carbohydrate intake is the form of the carbohydrate in terms of being a liquid or solid. While a majority of the research literature has investigated liquid carbohydrate sources, they may not be more beneficial than solid carbohydrate sources in relation to the rates of glycogen synthesis (Reed et al. 1989; Keizer et al. 1987). When 1.5 g/kg/body weight was ingested in both liquid and solid form after exercise and 2 h after exercise, similar rates of muscle glycogen synthesis were observed (Reed et al. 1989).

In conclusion, the restoration of muscle glycogen stores after exercise depends on several factors, including the type of carbohydrate, the timing of the ingestion, and the amount of carbohydrate that is ingested. The addition of protein may also have

an impact on the rate of skeletal muscle glycogen resynthesis. This aspect will be discussed in Chapter 7 ("Nutrient Timing: Carbohydrate–Protein Combinations").

4.3 RESISTANCE EXERCISE CONSIDERATIONS

For the resistance-training athlete, the role that dietary carbohydrates have in the body is similar to the endurance athlete in some aspects and very different in other aspects. One area in which resistance exercise and endurance exercise are similar is in terms of their effects on skeletal muscle glycogen depletion. Both modes of exercise result in the depletion of skeletal muscle glycogen (MacDougall et al. 1999; McConell et al. 1999; Robergs et al. 1991; Sherman and Wimer 1991; Essén-Gustavsson and Tesch 1990; Coyle et al. 1983; Tesch, Colliander, and Kaiser 1986; Hermansen et al. 1967). An area in which resistance exercise is different from endurance exercise is that, unlike endurance exercise, dietary strategies such as carb loading and ingesting supplemental carbohydrate prior to and during resistance exercise does not appear to enhance resistance-exercise performance. This section will discuss the role that carbohydrates have in the overall daily diet of a resistance-training athlete as well as the effects that an acute ingestion of carbohydrate has prior to, during, and following a resistance-exercise session. Also, the insulinogenic/anabolic properties that carbohydrate intakes can elicit will be discussed.

4.3.1 Daily Carbohydrate Intake

Unlike endurance exercise (6–10 g/kg/day), there are no established guidelines for daily carbohydrate intake for athletes whose primary modes of training and competition are based on resistance training (strength athletes, bodybuilders, power lifters, etc.). This is likely due to the fact that there has been little to no scientific investigation into determining what optimal daily amounts of carbohydrate intakes should be for these types of athletes. Out of the few studies that have been conducted on daily carbohydrate intakes and resistance-training performance (but notably in nonathletic populations), it appears that relatively high carbohydrate diets are not needed to improve or maintain lean muscle mass gains. Four different studies utilizing overweight males and females (body mass index greater than 25 kg/m^2) reported that lower carbohydrate diets (ranging from 0.2 g/kg/day to 1.3 g/kg/day) combined with resistance training appear to be additive in the sense that they maximize fat loss while preserving/increasing lean body mass (Kreider et al. 2011; Jabekk et al. 2010; Quann 2008; Layman et al. 2005).

Another important factor is that, in low-carbohydrate diets, protein intakes are always higher as well. The interesting point that these studies make is that if resistance training is conducted while ingesting a hypocaloric, low-carbohydrate diet, fat loss is maximized and lean muscle mass is maintained. For an athlete, these findings provide the basis for the argument that fat loss is maximized with reduced caloric intakes and a significantly reduced carbohydrate intake. Unfortunately, as stated previously, these studies were not conducted in athletes, so until that is accomplished

it may be presumptive to assume that the results reported in overweight nonathletic individuals could be accurately extrapolated to strength athletes. The majority of studies have investigated the effects of acute carbohydrate supplementation in the hours before, during, and after resistance exercise. The following sections focus on the results published from these investigations.

4.3.2 Carbohydrate Ingestion Prior to and during Resistance Exercise

Because resistance training relies heavily upon carbohydrates as an energy source, it has been hypothesized that ingesting carbohydrates prior to a resistance-training bout will increase the total amount of work that may be performed during the workout (Kulik et al. 2008; Haff et al. 1999; Lambert et al. 1991). This hypothesis is based upon the fact that skeletal muscle glycogen is depleted during resistance exercise; as it is depleted, the intensity and subsequently the total work volume (measured by the amount of weight lifted × repetitions performed) is compromised. Stated another way, since glycogenolysis is an important source of energy during resistance exercise, the effects of carbohydrate supplementation may enhance acute resistance-exercise performance.

Research has demonstrated that resistance exercise does deplete skeletal muscle glycogen (Figure 4.1) (MacDougall et al. 1999; Tesch et al. 1998; Pascoe et al. 1993). Despite this depletion of skeletal muscle glycogen, the majority of studies investigating carbohydrate ingestion prior to and during an acute bout of resistance exercise do not report improvements in resistance-training performance. In the studies that have reported an enhancement (Haff et al. 2001) or near enhancement (Lambert et al. 1991) of performance, the resistance-exercise workout was not of a practical nature (i.e., 16 sets of lower body resistance exercise conducted on an isokinetic dynamometer or 17 sets of a leg extension exercise) and do not resemble workouts that are typically conducted by resistance-training athletes (Haff et al. 2001).

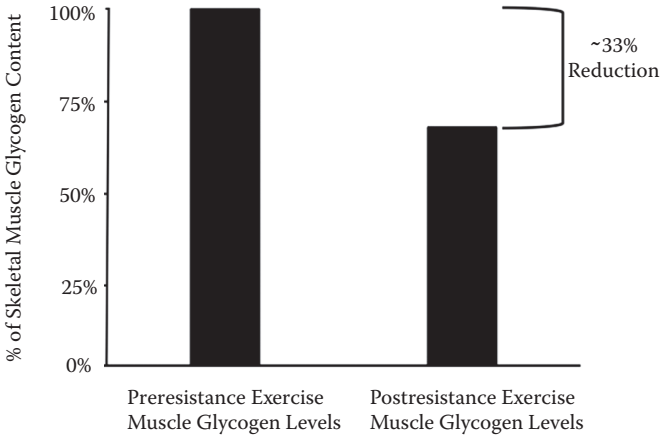

FIGURE 4.1 Skeletal muscle glycogen depletion.

Table 4.8 summarizes the investigations in which carbohydrate was ingested (in supplemental form) prior to or during a single resistance exercise bout and in which resistance-exercise performance was assessed. Based on these findings, it appears that pre-exercise carbohydrate ingestion does not lead to an enhancement of resistance-exercise performance, especially in workouts that would be typically performed by resistance-trained athletes. In addition to those published studies highlighted in Table 4.8, two additional studies that are only available in abstract form also reported the lack of an ergogenic effect with carbohydrate supplementation prior to (Vincent et al. 1993) and during (Conley et al. 1995) an acute bout of resistance exercise. Specifically, Conley and co-workers reported that carbohydrate supplementation (15 min before and after every set) did not significantly increase sets, repetitions, or total work as compared to an ingested placebo in a workout that lasted about 35 min. Vincent and colleagues observed that carbohydrate supplementation (100 g ingested immediately before training) did not improve total work, average power, peak torque, or work fatigue.

Though resistance-training performance does not appear to be enhanced with carbohydrate supplementation prior to and during a single exercise bout, there is evidence that such supplementation is effective for enhancing performance when two resistance-exercise sessions are conducted in the same day (Haff et al. 1999). In this study, six resistance-trained males (with the ability to squat 150% of their body mass) participated in two training sessions in the same day, and they performed this routine on two different occasions separated by at least 1 week. On one occasion, the athletes ingested a carbohydrate supplement and on another the athletes ingested a placebo beverage. The first/morning session was designed to deplete muscle glycogen stores and consisted of the following three exercises:

- Squats (five sets of 10 repetitions at 65% 1 RM [repetition maximum] with 3 min of rest between sets)
- Speed squats (five sets of 10 repetitions at 45% 1 RM with 3 min of rest between sets)
- One-legged squats (five sets of 10 repetitions at 10% 1 RM with 3 min of rest between sets)

The second/afternoon session was conducted 4 h after the morning session and consisted of sets of squats performed to exhaustion at 55% 1 RM with a 3-min rest period between each set. Sets were counted as the total number of 10 repetitions successfully completed. To determine the effectiveness of a carbohydrate supplement, each athlete ingested either a carbohydrate beverage or a placebo beverage before, during, and after the morning workout. During the morning session, the athletes ingested 0.3 g carbohydrate/kg body mass prior to and every 15 min during the workout, totaling 1.2 g carbohydrate/kg body mass during the hour-long morning session. Following the morning workout and before the afternoon workout, each athlete ingested 0.38 g of carbohydrate every hour for 4 h leading up to the second workout of the day. In comparison with the placebo, the carbohydrate beverage significantly increased the total number of sets (~19 vs. 11) and repetitions (~199 vs. 131) completed. As a percentage, the carbohydrate treatment

TABLE 4.8
Preresistance Exercise Supplemental Carbohydrate Ingestion

Study	Population	Treatment	Amount/Timing of Ingestion	Exercise Bout	Results
Kulik et al. (2008)	Eight resistance-trained males (1 RM squat ≥ 150% body mass)	Two treatments: • Carbohydrate • Placebo Randomized, double-blind treatments separated by 7 days	0.3 g/kg body mass ingested immediately before exercise and after every other successful set of squats; total amount of CHO ingested prior to and during bout was ~55 g	Sets of back squats (set = five repetitions) at 85% 1 RM to exhaustion	No significant difference between treatments for total sets (3.5 vs. 3.5) or repetitions (20.4 vs. 19.7) for the CHO and placebo groups, respectively
Haff et al. (2001)	Eight resistance-trained males (standard diet followed for 3 days prior to study)	Two treatments: • Carbohydrate • Placebo Randomized, double-blind treatments separated by 7 days	1 g/kg body mass ingested prior to (~95 g) and 0.51 g/kg body mass ingested after sets 1, 6, and 11 (~145 g); total amount of CHO ingested prior to and during bout was ~240 g	16 Sets of 10 repetitions at 120°/s on an isokinetic dynamometer	Carbohydrate treatment elicited significantly more work (~41 vs. ~38 kJ, or about 8% more work completed), but no differences existed for peak torque
Haff et al. (2000)	Eight resistance-trained males (1 RM squat ≥ 175% body mass)	Two treatments: • Carbohydrate • Placebo Randomized, double-blind treatments separated by 7 days	1 g/kg body mass ingested prior to (86 g) and 0.3 g/kg body mass ingested every 10 min during the workout (77 g); total amount of CHO ingested prior to and during bout was 163 g	Three sets of isokinetic leg flexion/extension prior to and following free weight protocol that involved three sets of 10 reps of the following: squats (65% 1 RM), speed squats (45% 1 RM), and one-legged squats (10% 1 RM)	No differences between the CHO and placebo treatments existed for any performance measure (total work, average work, peak torque, and average torque were measured before and after exercise

Dalton et al. (1999)	Twenty-two resistance-trained subjects undergoing 3-day energy restriction	Three groups: • Carbohydrate • Placebo • Control	1 g of carbohydrate/kg body mass 30 min prior to workout; total amount of CHO ingested was ~84 g	Five sets of 10 reps of squat, bench press, leg press, and leg extension from 80% 10 RM to 60% 10 RM; bench press and leg extension completed to failure at 80% of 10 RM on final set	No significant difference between treatments for bench press total reps and for leg extension total reps
Lambert et al. (1991)	Seven resistance-trained males	Two treatments: • Carbohydrate • Placebo Randomized, double-blind treatments separated by at least 7 days	1 g/kg body mass immediately before and 0.17 g/kg body mass after the 5th, 10th, and 15th sets; total amount of CHO ingested prior to and during bout was ~125 g	As many sets of 10 repetitions as possible at 80% of 10 RM on the leg extension machine; failure was defined as the point at which the subject could no longer perform seven repetitions for a set	Total number of sets (17 vs. 14) and repetitions (149 vs. 129) completed was ~19% and 15% greater for the CHO treatment as compared to the placebo, but results were not significant (results approached significance, however)

improved total sets by 65% and total repetitions by 51% in comparison to the placebo group.

Even though resistance-training performance was improved in this study, the workout (sets of between 10 and 20 squats performed at 55% 1 RM) lacks ecological validity. A proper interpretation of this study would be to indicate that carbohydrate supplementation ingested prior to and during (at a rate of 1.2 g/kg/h) a workout, as well as during a 4-h recovery period (at a rate of 0.38 g/kg/h) will significantly improve the volume of training during a subsequent high-volume, relatively low-intensity training bout conducted later in the same day.

Another consideration of the effects of carbohydrate supplementation prior to and during an acute bout of resistance exercise is the duration of the training bout itself. In those investigations in which the carbohydrate supplementation did not exert an ergogenic effect on resistance-training performance, it could be hypothesized that the exercise duration did not last long enough to cause carbohydrate availability to become a limiting factor. In the three studies that reported or at least suggested that carbohydrate supplementation was effective for improving resistance-training performance, the workouts were of a longer duration, lasting 56 (Lambert et al. 1991), 57 (Haff et al. 2001), and 77 (Haff et al. 1999) min. In the studies that reported no benefit of carbohydrate supplementation, the durations of the workouts were all less than 40 min (Conley et al. 1995; Haff et al. 2000; Kulik et al. 2008). Bringing this consideration into how these various studies are best interpreted, carbohydrate supplementation prior to and during an acute resistance-training bout will likely only be effective for improving resistance-training performance for those engaging in high-volume, low–moderate intensity exercise bouts lasting approximately 60 min or more and, potentially, when a second resistance-training session is undertaken in the same day.

Though performance may not be enhanced, carbohydrate ingestion prior to and during a resistance-exercise bout does decrease the rate and amount of glycogen loss. To illustrate this, Haff and colleagues (2000) instructed eight highly resistance-trained males to complete three sets of 10 repetitions of back squats at 65% 1 RM, speed squats at 45% 1 RM, and single-leg squats at 10% 1 RM on two separate occasions (the workout lasted about 39 min). During their two visits (separated by 1 week), the subjects ingested 1 g carbohydrate/kg body mass or a placebo 10 min prior to the resistance-exercise training session. Also, during the workout, the subjects ingested 0.3 g carbohydrate/kg body mass or the placebo beverage every 10 min. The total amount of carbohydrate that was ingested prior to and during the workout was about 160 g. Two measures were made prior to and after the lower body exercise bout: skeletal muscle glycogen levels and isokinetic leg exercise (to assess exercise performance).

The training bout resulted in ~27% decrease in muscle glycogen content in the placebo treatment, but only ~14% decrease when the carbohydrate beverage was implemented. There were no differences in isokinetic leg exercise performance. The results of this investigation indicate that the consumption of a carbohydrate beverage can attenuate the decrease in muscle glycogen associated with isotonic resistance exercise but does not enhance the performance of isokinetic leg exercise (Haff et al.

2000). In summary, it appears as if carbohydrate supplementation prior to and during resistance training can decrease the rate of muscle glycogen depletion and help to maintain daily glycogen stores, but does not lead to an enhancement of resistance-exercise performance.

4.3.3 Postresistance Exercise Muscle Glycogen Resynthesis

There have been numerous studies of muscle glycogen repletion following prolonged endurance exercise (Pedersen et al. 2008; Blom et al. 1987; Coyle et al. 1986; Wallis et al. 2008), but only a few have investigated the effects of muscle glycogen resynthesis after resistance exercise. Replenishing skeletal muscle glycogen is particularly important for an athlete (endurance athlete or intermittent sport athlete) who may have a team practice in the next few hours or the morning after a nighttime resistance-exercise workout. In comparison to other modes of exercise, such as endurance exercise and intermittent high-intensity exercise, rates of muscle glycogen resynthesis for resistance exercise are in the middle (faster than endurance exercise but slower than high-intensity exercise). Specifically, when no food/carbohydrate is ingested in the postworkout period, rates of muscle glycogen synthesis are ~1.5 to 2 mmol/kg wet weight/h following prolonged endurance exercise, ~1.3 to 11 mmol/kg wet weight/h following resistance exercise, and ~15 to 30 mmol/kg wet weight/h following short-term, high-intensity exercise (Pascoe and Gladden 1996).

To demonstrate the extent to which skeletal muscle glycogen can be resynthesized in the postworkout period with no food/carbohydrate ingestion, eight resistance-trained males performed six sets of six repetitions of leg extension exercise at 70% 1 RM (Robergs et al. 1991). Skeletal muscle glycogen levels were assessed prior to and immediately after exercise and 2 h after exercise. No carbohydrate (or food of any kind) was ingested in the 2-h postworkout time period. Muscle glycogen levels were 61% of pre-exercise levels immediately after exercise and then climbed to 79% of pre-exercise levels 2 h after exercise.

In one of the first studies to investigate the effects of carbohydrate ingestion on muscle glycogen resynthesis following a resistance-exercise bout, it was reported that carbohydrate ingestion following the workout resulted in significantly increased muscle glycogen resynthesis in comparison to a placebo (Pascoe et al. 1993). Recreationally fit college males engaged in a lower body workout protocol (sets of six repetitions of leg extensions performed at 70% 1 RM to fatigue), which induced a significant depletion of skeletal muscle glycogen content. In one of the two trials, the subjects ingested 1.5 g carbohydrate (glucose polymer)/kg body weight immediately after and again 1 h after the exercise bout. In the other trial (the two trials were randomized and separated by at least 7 days), the subjects ingested an equal amount of water at the same time as the carbohydrate feedings. For the water treatment, glycogen levels were 71%, 74%, and 75% of pre-exercise levels immediately after, 2 h after, and 6 h after exercise. For the carbohydrate treatment, glycogen levels were 67%, 86%, and 91% of pre-exercise levels immediately after, 2 h after, and 6 h after exercise. Without carbohydrate supplementation, the extent to which glycogen was resynthesized was minimal. In contrast, ingesting 1.5 g carbohydrate/kg body mass

immediately after and 1 h after the workout resulted in a significant resynthesis of skeletal muscle glycogen.

Another study also investigated the effects of postworkout carbohydrate ingestion and its effects on skeletal muscle glycogen resynthesis rates (Roy and Tarnopolsky 1998). Ten resistance-trained males performed a whole-body workout (three sets of nine exercises at 80% 1 RM) that included unilateral knee extension. Immediately after and 1 h following the exercise bout, a placebo or a carbohydrate supplement was given at a dose of 1 g/kg body weight. The carbohydrate consisted of 56% sucrose/44% glucose polymer from corn syrup solids. For the placebo treatment, glycogen levels were 73% and 76% of pre-exercise levels immediately after and 4 h after exercise. There was very little glycogen resynthesis occurring without any carbohydrate intake during the 4-h recovery period. For the carbohydrate treatment, glycogen levels were 74% and greater than 90% of pre-exercise levels immediately after and 4 h after exercise. Ingestion of carbohydrate led to a significantly greater mean rate of muscle glycogen resynthesis compared with the placebo condition.

Given that 1 g/kg/h (Roy and Tarnopolsky 1998) was as effective as 1.5 g/kg/h (Pascoe et al. 1993), it can be concluded that 1 g carbohydrate/kg/h is sufficient for resynthesizing skeletal muscle glycogen synthesis following resistance exercise to levels reaching above 90% of pre-exercise values 4 h after exercise. Following this dosing schedule, a 180-lb. (81.8-kg) individual would ingest about 82 g of carbohydrate immediately after and then again 1 h following a resistance-exercise workout (totaling ~165 g of carbohydrates within an hour after completing the resistance-exercise workout).

4.3.4 Net Muscle Protein Balance and Insulinogenic Properties of Carbohydrate Intake

In addition to restoring skeletal muscle glycogen, consuming carbohydrate after a resistance-training bout may also influence net muscle protein balance, affecting both muscle protein synthesis and muscle protein breakdown. (For a complete discussion of the concept of net muscle protein balance, refer to Chapter 5, "Protein Metabolism.") One of the first studies (Roy et al. 1997) to investigate this concept studied the effects of postworkout carbohydrate supplementation on muscle protein synthesis and muscle protein breakdown (via a marker of skeletal muscle breakdown, urinary 3-methylhistidine). In this original study, eight resistance-trained males participated in a randomized, double blind, placebo-controlled study in which they completed four sets each (eight sets total) of unilateral leg press and leg extensions for ~10 repetitions at 85% 1 RM. Immediately after and again 1 h after the lower body resistance-exercise bout, 1 g carbohydrate (glucose)/kg body mass or a placebo beverage was consumed. (Interestingly, this is the same dosing protocol recommended for restoring skeletal muscle glycogen to levels reaching ~90% of pre-exercise levels.) The total amount of carbohydrate ingested over the 2-h period was approximately 150 g.

Insulin levels peaked at 30 min (~125 μ IU/mL) after exercise and again 1.5 h (~160 μ IU/mL) after exercise in the carbohydrate group (both instances were 30

min after the ingestion of 1 g carbohydrate/kg body mass), but were unchanged in the placebo group. Muscle protein synthesis rates were increased by 36% following the carbohydrate supplement, but were only increased by 6% following the placebo ingestion. However, this difference did not reach the level of statistical significance. There was a significant difference in the marker of muscle protein breakdown. Urinary 3-methylhistidine excretion was significantly lower for the carbohydrate condition as compared to the placebo condition, exhibiting an 8% lower value over the 24-h collection period. The take-home message from this study is that carbohydrate supplementation (1 g/kg body mass) immediately and 1 h after resistance exercise can decrease skeletal muscle protein breakdown, resulting in a more positive net muscle protein balance.

Another study also investigated the effects of supplemental carbohydrate following a bout of resistance exercise and its impact on muscle protein synthesis and muscle protein breakdown (Borsheim et al. 2004). Half of the 16 recreationally active males and females in their late twenties who participated in this study were assigned to the carbohydrate group; the others were assigned to a placebo group. Each subject performed 10 sets of eight repetitions of leg extensions at 80% 1 RM, and the total workout time was about 20 min. Measures of protein synthesis and breakdown were measured for 4 h after the workout. The subjects ingested either 100 g (1.3 g/kg) of carbohydrate (maltodextrin) or a placebo beverage 1 h after the workout. Insulin levels peaked to ~105 μ IU/mL 1.5 h after the workout or 30 min after ingesting the 100 g of carbohydrate. Insulin levels were unchanged in the placebo group. Net muscle protein balance did not differ after the exercise bout and there were no differences between the two groups in the first hour after exercise. (Recall that the carbohydrate supplement was not ingested until 1 h after exercise.) However, during the second hour after ingesting the beverages, net muscle protein balance started to improve in the carbohydrate group and reached statistical significance by the third hour after carbohydrate ingestion.

Net muscle protein balance was significantly improved in the carbohydrate group, but not in the placebo group. Net muscle protein balance can be improved by increasing protein synthesis, decreasing protein breakdown, or a combination of both. In terms of protein synthesis, there were no differences between the groups either before or after the carbohydrate supplementation. Protein breakdown was significantly reduced following the ingestion of carbohydrate, but not following the placebo ingestion. Ingesting 1.3 g of carbohydrate in the form of maltodextrin 1 h after resistance exercise results in a significant reduction in the rate of skeletal muscle protein breakdown.

The effects that carbohydrate ingestion has on muscle protein synthesis and breakdown are attributed to the insulin that is secreted following carbohydrate ingestion. In the preceding studies, carbohydrate ingestion and the subsequent insulin response resulted in a suppression of skeletal muscle protein breakdown with no effects on muscle protein synthesis. Other studies also support the contention that insulin suppresses muscle protein breakdown (Heslin et al. 1992; Denne et al. 1991; Kettelhut, Wing, and Goldberg 1988; Gelfand and Barrett 1987). While elevations in insulin have been shown to increase rates of protein synthesis significantly, these were observed following the infusion of insulin rather than secreted in response to oral carbohydrate ingestion.

In one of these infusion studies, insulin was infused to levels well above normal physiological values, increasing 1,000-fold above basal levels (to levels of about 9000 μ IU/mL). Increasing insulin levels to this extent has been appropriately termed "extreme hyperinsulinemia" (Hillier et al. 1998). Another study that demonstrated an increase in protein synthesis infused insulin to physiologically high levels of nearly ~80 μ IU/mL (Biolo, Declan Fleming, and Wolfe 1995). Similarly, insulin was infused following a resistance-training bout to determine its effects on skeletal muscle protein synthesis and breakdown (Biolo et al. 1999). Following infusion, insulin levels went from ~8.6 to 52 μ IU/mL in the postexercise period. The increases in insulin suppressed protein breakdown but did not increase protein synthesis following the lower body resistance-exercise bout.

In relation to resistance exercise, carbohydrate intake recommendations should be based upon their ability to

- Improve muscular strength (and muscle mass if this is a goal of certain athletes)
- Replenish skeletal muscle glycogen levels if a sports practice or another resistance-exercise bout is planned in the next few hours
- Improve net muscle protein balance, particularly by improving (decreasing) rates of skeletal muscle protein breakdown

In relation to influencing skeletal muscle protein breakdown, ingesting 1 g carbohydrate/kg body mass immediately after and again 1 h following (or 2 g/kg body mass over a 1-h postexercise period) a resistance-exercise bout decreases the rate of muscle protein breakdown in resistance-trained athletes. In recreationally trained athletes, ingesting 1.3 g carbohydrate/kg body mass 1 h following resistance exercise also decreases muscle protein breakdown.

4.4 CONCLUSION

General recommended carbohydrate intakes for endurance athletes are 6 to 10 g/kg body mass/day. While this recommendation is likely sufficient to maintain skeletal muscle glycogen concentrations for most athletes training on successive days, specific carbohydrate intakes within this range will likely vary with an athlete's daily energy expenditure and type of exercise performed. Specific strategies that manipulate carbohydrate intakes include "train low, compete high" as well as different ways to load the body's glycogen levels prior to endurance events (carbohydrate-loading strategies). Each of these methods has demonstrated that pre-exercise glycogen levels can be significantly increased, but more research needs to be conducted in relation to performance before concrete recommendations can be made. About 60 min prior to endurance exercise, ingesting 1 g of glucose/kg body mass has been reported to enhance endurance performance or, at a minimum, not to affect exercise performance negatively. During endurance exercise, carbohydrate oxidation rates can be maximized if different types of carbohydrates are ingested, such as glucose/sucrose/maltodextrin plus fructose. In order to maximize rates of skeletal muscle glycogen resynthesis, ingesting 1.0 to 1.5 g carbohydrate/kg body weight immediately after the training session and again each hour for several hours is effective. Focusing on

a rapid rate of glycogen replenishment is important for those athletes that have multiple competitions or training sessions in the same day or during consecutive days.

Unlike endurance exercise (6–10 g/kg/day), there are no established guidelines for daily carbohydrate intake for athletes whose primary modes of training and competition are based on resistance training (strength athletes, bodybuilders, power lifters, etc.). Further, pre-exercise carbohydrate ingestion does not lead to an enhancement of acute resistance-exercise performance, especially in workouts that would be typically performed by resistance-trained athletes. The one benefit that carbohydrate ingestion does seem to elicit is its ability to decrease skeletal muscle protein breakdown, resulting in a more positive net muscle protein balance. Ingesting approximately 1 g carbohydrate/kg body mass immediately and 1 h after resistance exercise has been reported to improve (decrease) rates of skeletal muscle protein breakdown.

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5 Protein Metabolism

Bill Campbell

CONTENTS

5.1 Introduction 126
5.2 Essential Versus Nonessential Amino Acids 126
5.2.1 Complete Versus Incomplete Proteins 127
5.3 Protein Digestion and Absorption 127
5.4 Quantitative Measures of Protein Quality/Ranking of Proteins 128
5.4.1 Biological Value 129
5.4.2 Protein Efficiency Ratio 130
5.4.3 Protein Digestibility Corrected Amino Acid Score 130
5.5 Types of Protein 131
5.5.1 Milk Protein 132
5.5.1.1 Whey Proteins 132
5.5.1.2 Casein Protein 133
5.5.2 Egg Protein 134
5.5.3 Soy Protein 134
5.6 Methods of Assessing Protein Status 135
5.6.1 Urinary Urea Nitrogen 136
5.6.2 3-Methylhistidine Excretion 136
5.6.3 Nitrogen Balance 137
5.6.4 Isotopic Tracer Methodology 138
5.6.5 Arteriovenous Differences of Amino Acids across Skeletal Muscle 138
5.6.5.1 Limitations to the Arteriovenous Difference Method and the Superiority of Measures of Lean Body Mass Changes over Time 140
5.7 Protein Metabolism and Net Muscle Protein Balance 140
5.7.1 Skeletal Muscle Protein Synthesis 141
5.7.1.1 The Role of Muscle-Specific Genes in Skeletal Muscle Protein Synthesis 141
5.7.1.2 Translation Initiation 142
5.7.1.3 Akt-mTOR Pathway Regulation on Translation Initiation 143
5.7.1.4 Leucine and Translation Initiation 144
5.7.2 Muscle Protein Breakdown and the Ubiquitin Proteasome Pathway 146
5.8 Resistance and Endurance Exercise and Net Muscle Protein Balance 147
5.9 Conclusion 148
References 149

5.1 INTRODUCTION

In the human body, much of the dry weight (the body mass minus body water) consists of proteins. Proteins are involved in all aspects of life and are found in every cell of the body; they are needed to promote growth and to repair damaged cells and tissues. In comparison with carbohydrates and fats, protein plays a different role in an athlete's diet. The primary role for carbohydrates in relation to training is to provide energy for high-intensity activities (such as sprinting, high-intensity endurance exercise, and resistance exercise). The primary role for dietary fat in relation to training is also to provide energy. However, fat primarily fuels lower intensity exercise and is also the primary fuel utilized during recovery (in the hours after exercise and during the rest periods between sets in a traditional resistance-exercise workout) and at rest.

The primary role of protein is structural (collagen synthesis), regulatory (which includes peptide hormones such as growth hormone), and contractile (actin and myosin filaments). Unlike carbohydrates and fat, dietary protein should be viewed in a way that its ingestion emphasizes longer term adaptations. Every time an athlete engages in exercise, whether it is high-intensity interval training or endurance training, the activities provide a stimulus to the athlete's body to which he or she can adapt. Optimal adaptations to the training stimulus are related to dietary protein intake. If an athlete makes poor decisions with protein intake, he or she will have inferior adaptations to training programs. Conversely, if an athlete makes wise decisions regarding protein intake, he or she will maximize the adaptations resulting from the training stimulus.

Why are some protein sources considered high quality while other protein sources are regarded as low quality? How is dietary protein digested and absorbed? Related to this, are some proteins digested and absorbed faster than others? And, if so, what effects does this have in an athlete's body and how does it impact the adaptations to his or her training programs? These are the types of questions that will be answered in this chapter. In addition, the concept of net muscle protein balance will be presented.

5.2 ESSENTIAL VERSUS NONESSENTIAL AMINO ACIDS

Proteins are made up of approximately 20 amino acids (refer to Figure 5.1 for the common structure of amino acids). These amino acids include eight that are classified as *essential amino acids* that must be acquired from the diet because the body is unable to synthesize them. There are also approximately six conditionally essential

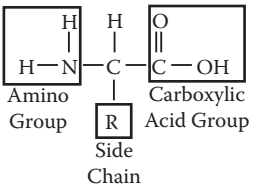

FIGURE 5.1 Common structure of an amino acid.

TABLE 5.1
Classification of Amino Acids

Essential Amino Acids	Conditionally Essential Amino Acids	Nonessential Amino Acids
Isoleucine	Arginine	Alanine
Leucine	Cysteine	Asparagine
Lysine	Glutamine	Aspartic acid
Methionine	Histidine	Glutamic acid
Phenylalanine	Proline	Glycine
Threonine	Tyrosine	Serine
Tryptophan		
Valine		

amino acids. They are called conditionally essential because, under certain conditions, the body has difficulty synthesizing them efficiently; thus, they typically must be obtained from the diet in order to provide sufficient amounts of these amino acids. Without dietary sources of essential amino acids, the body must catabolize its own protein stores (e.g., muscle) to provide the essential amino acids to meet essential protein needs. The body can synthesize the remaining amino acids fairly easily, so they are considered nonessential. Table 5.1 lists the 20 amino acids and summarizes them according to their designation as essential, nonessential, or conditionally essential.

5.2.1 Complete Versus Incomplete Proteins

Dietary protein is classified as either complete or incomplete depending on whether the protein contains adequate amounts of the eight essential amino acids. Animal sources of protein (meat, fish, poultry, milk, cheese, and eggs) contain all essential amino acids and are therefore considered complete sources of protein. On the other hand, vegetable (grains, legumes, nuts, seeds, and other vegetables) proteins are incomplete because they are missing or do not have enough of the essential amino acids. Some incomplete proteins are missing or are deficient in one or more essential amino acid, while another incomplete protein may be missing or is deficient in a different essential amino acid. If these two separate, incomplete proteins are combined and their amino acid assortments complement each other, the essential amino acids missing from one are supplied by the other. This practice of combining two incomplete protein sources to derive the needed essential amino acids is referred to as protein combining (and the proteins are said to be complementary proteins). The practice of ingesting complementary proteins is very important for the vegetarian athlete who chooses to restrict animal sources of protein.

5.3 PROTEIN DIGESTION AND ABSORPTION

The purpose of protein digestion is to liberate the amino acids from the consumed proteins (Berdanier 2000). During the digestive process, enzymes known as

proteases hydrolyze whole proteins into their component amino acids, dipeptides, and tripeptides. In contrast to carbohydrate digestion, which is initiated in the mouth via salivary amylase, protein digestion does not begin until it reaches the stomach and the food is acidified with gastric hydrochloric acid (Berdanier 2000). Once through the stomach, the amino acids are absorbed through the wall of the small intestine, pass into the blood, and then to the liver via the portal vein. There is a constant interchange of amino acids among the blood, the liver, and the body tissues, with the liver serving as the critical center in amino acid metabolism. The collection of amino acids in these bodily compartments is referred to as the free amino acid pool. The liver is continually synthesizing a balanced amino acid mixture for the diverse protein requirements of the body (Williams 2002). From the liver, the amino acids are secreted into the blood and carried as free amino acids or as plasma proteins (such as albumin and immunoglobulins). Metabolic fates of amino acids include the formation of the following:

- Structural proteins in the form of skeletal muscle
- Functional proteins such as enzymes
- Signaling proteins such as hormones

It is important to note that the various cells of the body will use only the amount of amino acids necessary to meet their protein needs. Those amino acids in the body's amino acid pool that are used neither to synthesize protein nor to synthesize metabolically important intermediates are deaminated, and the carbon skeletons are either oxidized or used for the synthesis of glucose or fatty acids (Berdanier 2000). In the process of deamination, the amino group (NH_2) containing the nitrogen is removed from the amino acid, leaving a carbon substrate known as an alpha-keto acid (some refer to the alpha-keto acid as the carbon skeleton of the amino acid). The carbon skeleton that is released may have several fates, including (Williams 2002):

- Oxidation for the release of energy
- Accepting another amino group and being reconstituted to an amino acid
- Being channeled into the metabolic pathways of carbohydrate and fat

The amino group that was formed in the process of deamination must be excreted from the body (Williams 2002). This process occurs in the liver, where the amino group (NH_2) is converted into ammonia (NH_3). Next, the ammonia is converted into urea, which passes into the blood and is eventually eliminated by the kidneys into the urine.

5.4 QUANTITATIVE MEASURES OF PROTEIN QUALITY/RANKING OF PROTEINS

As stated earlier, proteins are classified as complete and incomplete, with animal sources and vegetable sources of protein being high-quality and low-quality sources of protein, respectively. Other quantitative methods also exist for classifying the

quality of a protein in addition to determining whether it is complete or incomplete. Three such methods will be discussed in this chapter, including:

- Biological value
- Protein efficiency ratio
- Protein digestibility corrected amino acid score

Even though there are several methods for determining the quality of a particular protein, it is important to understand that protein quality ultimately depends on the amino acid profile of the protein; hence, complete protein sources that contain greater amounts of essential amino acids generally have higher protein quality (regardless of the classification system utilized to rank various proteins). For many years, bioassays, mainly with rats, were the methods of choice to assess the nutritional value of proteins (Schaafsma 2000). This value was expressed in parameters such as protein efficiency ratio, net protein utilization, and biological value (Schaafsma 2000).

5.4.1 Biological Value

The biological value method is a measure of the proportion of absorbed protein from a food, which becomes incorporated into the proteins of the organism's body. The higher the amount of ingested protein that is absorbed by the body (i.e., less of the protein has been excreted), the higher the biological value score is and the greater is the quality of the protein. In order to derive the biological value, it is necessary to measure the amount of nitrogen ingested (such as the amount of nitrogen in a whole egg) and the amount of nitrogen lost in the feces and the urine. Also, the amount of obligatory fecal and urinary nitrogen must be accounted for when measuring the amount of nitrogen that is excreted from the body. Obligatory fecal and urinary nitrogen is calculated when subjects are on a nitrogen-free diet. It is a measure of the loss of nitrogen from normal metabolism and not those losses from dietary sources. By subtracting obligatory nitrogen losses from total nitrogen loss in the feces and urine, the nitrogen losses from just the dietary nitrogen can be estimated (Campbell 2012). The formula for calculating the biological value is shown in Topic Box 5.1.

TOPIC BOX 5.1 BIOLOGICAL VALUE OF A TEST PROTEIN = I – (F – Fo) – (U – Uo) × 100

I = intake of nitrogen (from the test protein, such as a whole egg), F = fecal nitrogen, Fo = obligatory fecal nitrogen, U = urinary nitrogen, and Uo = obligatory urinary nitrogen. *Note:* Even when each of these variables is accounted for in relation to nitrogen intake and losses, additional losses from sweat, hair, and fingernails are not accounted for. However, the amount of nitrogen lost from these sources is considered negligible in the rested state (Campbell 2012).

Biological value is a measure of dietary nitrogen retained by the body for its needs and is expressed as a percentage of nitrogen utilized. The biological value can be expressed as biological value = nitrogen retained/nitrogen absorbed × 100. Theoretically, no nitrogen found in the feces and urine (i.e., above the obligatory values) after consuming the test protein implies that all of the nitrogen from the test protein was utilized by the body and yields a biological value of 100% (Campbell 2012). On the other hand, an amount of nitrogen found in the feces and urine (in addition to the obligatory values) equal to the amount of nitrogen contained in the test protein implies that none of the protein was absorbed and retained by the body and therefore the biological value would be 0%.

There are several drawbacks to using the biological value as a measure of protein quality. The measure does not provide information as to which tissues are affected. Rather, it only provides what is occurring on a whole-body level. The protein may be effective at promoting protein synthesis in one tissue or area of the body (such as the digestive system/gut) but not other tissues (such as skeletal muscle). In addition, the biological value test is time consuming, expensive, and sometimes impractical (Campbell 2012).

5.4.2 Protein Efficiency Ratio

The protein efficiency ratio (PER) method determines the ability of a protein to support growth in young, rapidly growing rats. This method has been applied for nearly 100 years for the routine assessment of protein quality of foods (Young and Pellett 1994). The PER is calculated by dividing the weight gain of the young rat (in grams) by the protein intake (also in grams) (Elango, Ball, and Pencharz 2009). The higher the PER value, the better the protein is.

Some have criticized the PER method by claiming that it overestimates the value of animal proteins and underestimates the value of vegetable proteins (Elango et al. 2009). This is primarily because the rapid growth rate of rats increases the proportion of the protein that needs to be essential amino acids, in comparison to the relatively slower growth rates in humans (Elango et al. 2009; FAO/WHO Expert Consultation 1990). Inherent with its methodology, the PER is actually a measure that best defines a protein's value for the growth of a healthy, young rat. Both the biological value and the protein efficiency ratio are largely considered the "old" methods of evaluating protein quality. The "new" method of assessing protein quality that is recognized by most food scientists is known as the protein digestibility corrected amino acid score.

5.4.3 Protein Digestibility Corrected Amino Acid Score

The modern method of evaluating protein quality is known as the protein digestibility corrected amino acid score (PDCAAS). The US Food and Drug Administration (FDA), the United Nations Food and Agricultural Organization (FAO), and the World Health Organization (WHO) have adopted the PDCAAS as the preferred method to determine protein quality. From a broad perspective, the PDCAAS is based on the combination of an age-related amino acid reference pattern that is representative of

human requirements plus estimates of the digestibility of the protein. Specifically, the method is based on the comparison of the concentration of the first limiting essential amino acid in the test protein with the concentration of that amino acid in a reference (scoring) pattern. This reference pattern is based on the essential amino acid requirements for humans 2 to 5 years old since this group matches or exceeds the amino acid requirement patterns of older children and adults. The digestibility of the food protein is also taken into account—that is, how much of the protein is absorbed by the body after digestion. The fecal method is used to evaluate digestibility.

PDCAAS scores range from 0.0 to 1.0, with 1.0 being the upper limit of protein quality and able to support growth and health. A protein with a PDCAAS of 1.0 indicates that the protein exceeds the essential amino acid requirements of the body and is therefore an excellent source of protein. Proteins with PDCAAS values exceeding a value of one are not considered to contribute additional benefit in humans; therefore, the upper range of the score is truncated at a value of one (Schaafsma 2000). Even though this method has been widely adopted, it is not without controversy. Several assumptions and principles that the PDCAAS uses are questioned by some. For further reading on these potentially problematic issues with the PDCAAS, Schaafsma (2000) and others (Darragh and Hodgkinson 2000) have authored concise summaries and also provide recommendations for improving the PDCAAS method (Campbell 2012).

5.5 TYPES OF PROTEIN

There are several different types/sources of protein. The characteristics of what makes one dietary protein a better choice than another type of dietary protein for an athlete is primarily based upon the quality of the different protein choices. Athletes should consistently ingest high-quality proteins—that is, animal sources of protein that are complete proteins and that have adequate amounts of the essential amino acids. Table 5.2 lists several types of protein sources and several quantitative protein scoring systems so that different types of proteins can be compared against one another in terms of their quality. The following sections introduce some of the more popular types of supplemental protein, including milk proteins (casein and whey), soy protein, and egg protein.

TABLE 5.2
Quantitative Scores for Different Types of Proteins

Protein Source	Biological Value	Protein Efficiency Ratio	PDCAAS
Beef	80	2.9	0.9
Casein	77	2.5	1.0
Egg (yolk + white)	100	3.8	1.0
Soy (concentrate)	74	2.2	1.0
Wheat gluten	64	0.8	0.3
Whey	104	3.2	1.0

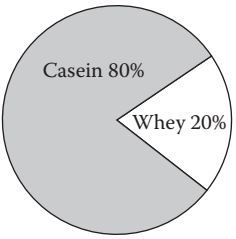

FIGURE 5.2 Composition of milk proteins.

5.5.1 Milk Protein

One cup of milk provides about 8 g of total protein. The protein component of milk is composed of numerous specific proteins. The primary group of milk proteins is referred to as caseins and all other proteins found in milk are grouped together under the name of whey proteins. Of these two milk proteins, approximately 80% of milk protein is casein and the remainder is whey protein (Figure 5.2). In contrast, human milk is about 70% whey protein and 30% casein protein. As cheese manufacturers transform milk into cheese, they separate milk's two protein types—whey and casein. The casein forms the curd, and the liquid whey is separated into different constituents, including whey protein. As will be discussed in the following chapter, a majority of the research that centers on protein supplementation and exercise has been conducted on these two milk proteins.

5.5.1.1 Whey Proteins

Whey protein is the most popular protein supplement marketed to athletes. In nearly every protein quality ranking score, whey protein is ranked the highest (one exception is egg protein, which ranks higher than whey protein in the protein efficiency ratio scoring method) (Table 5.2). Whey protein is extracted from whey, the liquid material created as a by-product of cheese production. There are actually several different subfractions of protein that are derived from whey. The protein fraction in whey comprises four major protein fractions and a few other minor protein fractions. The major protein fractions in whey are β-lactoglobulin, α-lactalbumin, bovine serum albumin, and immunoglobulins (Haug, Høstmark, and Harstad 2007). The minor protein fractions in whey are lactoferrin, lactoperoxidase, and glycomacropeptide. Each of these whey protein fractions and their percentage contribution to the total whey protein amount is summarized in Table 5.3.

When athletes or their support team purchase whey protein, they are purchasing one of several different finished whey protein products. Advances in processing technology, including ultrafiltration, microfiltration, reverse osmosis, and ion exchange, have resulted in the development of these different finished whey products (Marshall 2004). Whey proteins are commercially available as whey protein concentrates, isolates, and hydrolysates. The primary difference among these forms is the method of processing, plus differences in fat and lactose content, and amino acid profiles. Whey protein concentrate has anywhere between 25% and 89% protein, depending upon the quality of the protein product.

TABLE 5.3
Fractions of Whey Protein

Whey Protein Fraction	Percentage of Whey Protein
β-Lactoglobulin	50–55
α-Lactalbumin	20–25
Immunoglobulins	10–15
Glycomacropeptide	10–15
Bovine serum albumin	5–10
Lactoferrin	1–2
Lactoperoxidase	0.5

Source: Adapted from *Alternative Medicine Review* 13 (4): 341–347, 2008.

Whey protein concentrate is further extracted to become whey protein isolate. Whey protein isolate is the purest and most concentrated form of whey protein available. It contains 90% or more protein and very little fat and lactose. As the protein level in whey protein concentrate increases, the amounts of fat and lactose decrease. Whey protein hydrolysates are made from whey protein concentrate or whey protein isolate. Specifically, whey protein hydrolysates are enzymatically predigested, which allows the amino acids to be absorbed by the body more rapidly than intact proteins are. The amount of protein that is present in whey protein hydrolysate is variable.

5.5.1.2 Casein Protein

Casein makes up about 80% of the proteins in cow's milk and between 20% and 45% of the proteins in human milk (Kunz and Lönnerdal 1990). The caseins in milk form complexes referred to as micelles that are dispersed in the water phase of milk. The casein micelles consist of subunits of the different casein subtypes (alpha-1 casein [α-1 casein], alpha-2 casein [α-2 casein], beta-casein [β-casein], kappa casein [κ-casein), gamma casein [γ-casein]) and are held together by calcium phosphate bridges. All of the casein protein subtypes contain around 200 amino acids. The exact arrangement of the casein molecules in the micelle remains the subject of debate, although the subfraction κ-casein appears to surround the micelle due to its greater solubility. In addition to the different types of casein molecules, the casein micelle contains water and salts (mainly calcium and phosphorous).

Extracting the casein out of the milk and into a commercially available supplemental form of casein involves several processing steps. The first step is to defat the milk (producing skim milk) by a processing technique that involves separating the casein from whey (i.e., resolubilizing) and then drying it. Next, an acid is added to the skim milk to lower the pH, which causes the casein to precipitate out of the skim milk as a curd. Acid casein (as the curd is known) is insoluble in water, behaving much like sand. In order to make the casein curd more useful in food products (and to produce it as a supplemental form of protein), the acid casein curd is reacted with a strong alkali to result in an almost neutral protein product termed a caseinate.

Caseinates produced commercially consist of a mixture of the four casein subtypes (α, β, κ, γ).

Caseinates used in commercial supplements are available as sodium caseinates, potassium caseinates, calcium caseinates, and casein hydrolysates (depending on which type of alkali is used to neutralize the acid casein curd). The processing method affects the amino acid profile slightly, as well as the availability of α-, β-, γ-, and κ-casein subtypes. Sodium caseinate is the most water-soluble form of caseinate; calcium caseinate is usually the least water soluble of the caseinates and tends to sediment out of suspension within hours of being mixed into water.

5.5.2 Egg Protein

One large chicken egg contains approximately 70 cal and 6 g of protein. Many individuals believe that most of the protein in an egg is contained within the egg white (ovalbumin is the main protein found in egg whites), but this is not true. A little over half (about 3.5 g) of the protein content is found in the egg white, and the rest of the protein is contained in the egg yolk (about 2.5 g). Egg protein is a complete, high-quality protein that supplies all of the essential amino acids for humans (Table 5.2).

5.5.3 Soy Protein

Soy protein is derived from the soybean and is the most widely used vegetable protein source. Dehulling and defatting the soybean are the first steps that occur in the processing of the soybean. Dehulling is the process of removing the hulls (or chaff) from the soybean. Defatting the soybean is simply removing the oil by solvent extraction or by an expeller process in which the beans are heated and squeezed. Soybean meal is the term used to describe what is left of the soybean after dehulling and defatting.

Soybean meal (dehulled and defatted soybeans) is processed into three kinds of high-protein commercial products: soy flour, soy concentrates, and soy isolates. Each of these different types of soy products contains different amounts of protein (Figure 5.3). Of the three different categories of soy protein products, soy flour is the least refined form (Hoffman and Falvo 2004). Isolates are the most refined soy

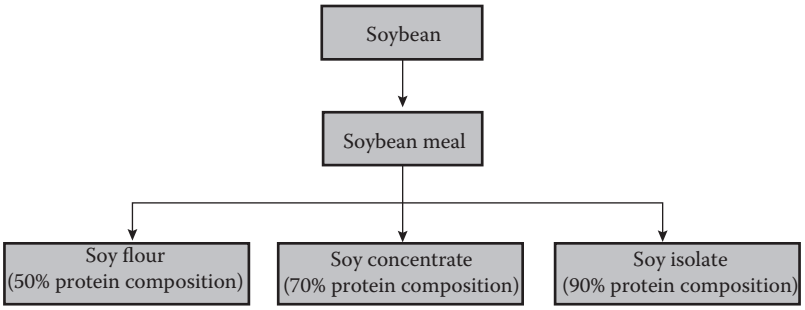

FIGURE 5.3 Types of soy protein.

protein product containing the greatest concentration of protein, but unlike flour and concentrates, contain no dietary fiber (Hoffman and Falvo 2004). Soy protein is classified as a high-quality protein source and its quality makes it a very attractive alternative for those seeking nonanimal sources of protein in their diet and those who are lactose intolerant. Soy is a complete protein with a high concentration of BCAAs (but contains fewer BCAAs than casein, whey, and egg proteins) (Hulmi, Lockwood, and Stout 2010).

5.6 METHODS OF ASSESSING PROTEIN STATUS

With the knowledge that carbohydrates and fat are the primary fuels for exercise, for many years protein was seen as not worthy of paying attention to or important for athletes. As different technologies developed, however, an appreciation of the central role that dietary protein and its component amino acids play in the overall metabolic response to exercise developed (Lemon 2012). Specifically, in the 1970s, interest in the potential role of protein as an exercise fuel was rekindled due to observations that nitrogen production tended to increase with prolonged endurance exercise of relatively high intensity (Lemon 2012; Décombaz et al. 1979; Haralambie and Berg 1976; Refsum and Strömme 1974).

Even though protein oxidation is increased during exercise, the total quantity of protein used as a fuel for skeletal muscle is likely no more than 10% of the total exercise energy expended (Lemon 1998, 2012; Rose and Richter 2009). This does not mean that protein intake for athletes is trivial, because protein and its component amino acids serve many other roles, such as building blocks for structural and functional proteins and as cell signaling compounds in skeletal muscle (Lemon 2012; Deldicque, Theisen, and Francaux 2005; Hawley et al. 2011; Koopman 2007). The primary role of protein is different from that of carbohydrates and fats and should be viewed in a way that its ingestion emphasizes longer term adaptations resulting from the athlete's training and conditioning programs. The primary determinant in making decisions regarding daily dietary protein intake is how the ingested protein is metabolized and ultimately partitioned in the body (Fürst and Stehle 2004; Pencharz and Ball 2003; Fuller and Garlick 1994).

The total amount of protein remains fairly constant throughout one's life span. Proteins are constantly degraded into their constituent amino acids—a process referred to as proteolysis. For protein mass to remain constant, new proteins must be synthesized to replace the degraded proteins. Of the 11 kg of protein in a typical 70-kg male, about 0.3 kg is degraded and replaced each day. The term "protein turnover" is often used to describe the simultaneous degradation and synthesis of bodily proteins. Protein turnover accounts for a significant portion of basal metabolic rate. It has been estimated that about 20% of energy expenditure at rest after overnight fasting is devoted to protein turnover (Welle and Nair 1990). Several methods are available for assessing protein status/protein turnover in the body with varying levels of relevance for the athlete. These methods include:

- Urinary urea nitrogen
- 3-Methylhistidine excretion

- Nitrogen balance
- Isotopic tracer methodology
- Arteriorvenous differences of amino acids across skeletal muscle
- Measures of lean body mass over time

Some of these methods are quite simple, while others are time consuming and invasive and require expensive and sophisticated laboratory equipment.

5.6.1 Urinary Urea Nitrogen

Urea serves an important role in the metabolism of nitrogen-containing compounds and is the main nitrogen-containing substance in the urine of humans. Most (approximately 90%) of the nitrogen excreted from the human body is incorporated in urea. The amount of urea excreted in the urine is an indication of whole-body protein breakdown. Urinary urea nitrogen is determined with a 24-h urine collection period in the following steps:

1. On day 1, the athlete urinates into the toilet.
2. Afterward, the athlete collects all urine in a special container for the next 24 h. The container should be refrigerated as much as possible.
3. On day 2, the athlete urinates into the container when he or she gets up in the morning.

Urea is made in the liver and then it diffuses into the blood. The blood then transports the urea from the liver to the kidneys, which then excrete it in the urine. Because there is some level of urea in the blood, it may be beneficial to measure the amount of urea in the blood to get a more complete picture of total urea produced in the body. The blood test that measures the urea concentration in the blood is known as "blood urea nitrogen" (BUN). While urinary urea nitrogen (and BUN) can be used as an estimate of whole-body protein breakdown, these measures have several drawbacks. For instance, the results are highly dependent on dietary protein intake. If dietary protein intake is increased, it will result in an increase in urea production. Conversely, if dietary protein is decreased, there will be a decrease in urea production. Also, urinary urea nitrogen does not provide detailed information, such as the amount of protein that has been catabolized from skeletal muscle.

5.6.2 3-Methylhistidine Excretion

Similarly to urinary urea nitrogen, urinary 3-methylhistidine is also a measure of protein breakdown. However, urinary 3-methylhistidine is used as an index of myofibrillar (actin and myosin) protein degradation (Bird, Tarpenning, and Marino 2006; Hickson and Hinkelmann 1985; Ballard and Tomas 1983; Dohm et al. 1982; Young and Munro 1978) and therefore is a highly valuable marker of protein breakdown for the athlete and his or her support staff. Estimates indicate that ~90% of total human 3-methylhistidine is located in skeletal muscle (Rooyackers and Nair 1997), validating its use as an index of myofibrillar protein degradation (Bird et al. 2006; Ballard and Tomas 1983;

Elia et al. 1981). 3-Methylhistidine is formed by the methylation of histidine after it is incorporated into the contractile proteins. Since the body cannot recycle 3-methylhistidine from degraded contractile proteins, it is excreted in the urine. Therefore, 3-methylhistidine is a urinary marker of skeletal muscle protein breakdown.

An elevated level of urinary 3-methylhistidine implies that the rate of actin and myosin breakdown has occurred. One of the limitations to utilizing this method is the fact that meat consumption increases 3-methylhistidine levels (Lukaski et al. 1981; Marliss, Wei, and Dietrich 1979). Meat and fish contain a relatively large amount of 3-methylhistidine and could cause erroneous results. However, with careful dietary controls, this method can be used for assessment of skeletal muscle protein breakdown (Ballard and Tomas 1983; Elia et al. 1981).

5.6.3 Nitrogen Balance

The nitrogen balance method is simply a measure of nitrogen intake and nitrogen excretion. The average nitrogen content of dietary proteins is about 16%. Therefore, nitrogen intake can be calculated by knowing how much dietary protein is ingested in the diet. Nitrogen excretion is not nearly as easy to calculate or measure. To measure nitrogen excretion, all routes of proteins exiting the body must be accounted for. The principal routes of protein (i.e., nitrogen) excretion from the body are urine, feces, sweat, and skin.

There are three basic states of nitrogen balance: positive, negative, and neutral. A positive nitrogen balance means that protein is being absorbed by the body and that nitrogen intake is greater than nitrogen output. A positive nitrogen balance is indicative of an anabolic state. A negative nitrogen balance means that the body is excreting protein and that nitrogen loss is greater than nitrogen intake. A negative nitrogen balance is associated with a catabolic state. A neutral nitrogen balance (equilibrium nitrogen balance) means that there is neither a net protein increase nor a net protein decrease by the body. Essentially, nitrogen intake and loss are equal and the body is not in an anabolic or catabolic state relative to protein metabolism.

While the nitrogen balance technique has been the method of choice for assessments of protein requirements (Lemon 2012; Institute of Medicine of the National Academies 2002; Rand, Pellett, and Young 2003), it is important to understand that the method has a number of limitations and drawbacks. These drawbacks include a delayed response to altered protein intake, confounding effects of energy intake (increased nitrogen loss when energy intake is inadequate), difficulties in quantifying all routes of nitrogen excretion, and the relatively shorter intervention periods in some investigations. Nitrogen balance experiments with controlled dietary periods lasting more than 2 to 3 weeks are desirable because they provide a longer time for metabolic adaptation to occur at a given protein intake and allow for quantitative assessment of changes in independent variables such as body composition and physiological or biochemical functions.

Taken together, these limitations generally result in an overestimate of intake and an underestimate of excretion. Essentially, the measured nitrogen balance tends to be more positive than it actually is, which produces an underestimate of the real protein requirement that should be ingested by the athlete (Lemon 2012). The most glaring

limitation of the nitrogen balance method is its inability to delineate tissue-specific changes in protein metabolism. Nitrogen balance is a measure of whole-body protein flux and does not provide specific information in regard to skeletal muscle protein synthesis and breakdown. Skeletal muscle has a lower rate of protein turnover relative to most nonmuscle tissues (Millward et al. 1997). Specifically, tissues such as the liver and gut have much higher turnover rates and deposit much more protein than others tissues, such as skeletal muscle (Wagenmakers 1999).

5.6.4 Isotopic Tracer Methodology

Another method of estimating protein status uses labeled isotopic tracers to model protein turnover. In this method, one or more of the atoms of an amino acid are substituted for an atom of the same chemical element, but of a different isotope. (An isotope is an atom with the same number of protons but a different number of neutrons.) Because the atom has the same number of protons, it will behave in almost exactly the same way chemically as other atoms in the compound and, with few exceptions, will not interfere with the reaction under investigation.

The difference in the number of neutrons, however, means that it can be detected separately from the other atoms of the same element. Atoms that are typically labeled include ^{15}N, ^{13}C, and ^{2}H. Relative to protein turnover studies, the stable isotopes that are most often used in whole-body studies are [^{15}N]glycine, L-[^{13}C]leucine, and L-[$^{2}H_5$]phenylalanine (Wagenmakers 1999; Thompson et al. 1989; Matthews et al. 1980; Waterlow, Golden, and Garlick 1978). The exact method of utilizing isotopic tracers will vary depending on what aspect of protein turnover is being modeled. For example, isotopic tracers can be used to estimate protein turnover at the whole-body level and also within individual tissues.

After ingestion or infusion, the amino acid tracer (i.e., the amino acid that is isotopically labeled) enters the free amino acid pool. Then, the labeled amino acid leaves the free amino acid pool via protein synthesis (i.e., incorporation into body proteins) or amino acid oxidation. If the amino acid is oxidized, it results in the production of urea, ammonia, and carbon dioxide. The primary advantage of the tracer method is that it reflects the metabolic fate of individual amino acids, thereby providing estimates of protein synthesis and protein breakdown. Often, the use of stable isotope tracer methodology, arterial-venous sampling (described in the following section), and muscle biopsies is used to quantify rates of muscle protein synthesis and breakdown. The idea of measuring rates of muscle protein synthesis and breakdown is described later in this chapter in Section 5.7, "Protein Metabolism and Net Muscle Protein Balance."

5.6.5 Arteriovenous Differences of Amino Acids across Skeletal Muscle

For the athlete, the focus of protein metabolism is often centered on skeletal muscle. As stated before, one of the primary drawbacks to the nitrogen balance method is that it is a whole-body protein turnover measure and does not provide specific information as to what is occurring in terms of protein synthesis and protein breakdown at the skeletal muscle level. Since athletes consistently train their skeletal muscles in

anticipation of strength, functionality, and, in certain cases, hypertrophy, any measure that is able to provide feedback at the skeletal muscle level is highly valued. Fortunately, protein turnover can be determined across a specific organ, such as skeletal muscle. When arterial and venous blood across a certain tissue (such as skeletal muscle) is sampled, the difference in the amino acid concentration between these two blood vessels gives information about the net exchange of specific amino acids. Often, isotopically labeled amino acids are used in conjunction with the measurement of arteriovenous differences to improve the value of the measurement.

The arteriovenous difference method can be explained by the fact that some amino acids are carried into a skeletal muscle by arteries and some are released by the skeletal muscle and are carried away by the veins. Some of the amino acids that are delivered to the skeletal muscle by the arteries are taken up by the muscle and incorporated into muscle proteins, indicating that protein synthesis is occurring. The amino acids that leave the muscle in the veins are a result of protein breakdown. The arteriovenous difference in the amino acid concentration provides a measure of net uptake and release by the skeletal muscle.

The most valuable information is obtained from amino acids that are not metabolized. Phenyalanine, tyrosine, and lysine are not metabolized in skeletal muscle and therefore these amino acids are typically used in this particular method to reflect the difference between net amino acid uptake from protein synthesis and the release of amino acids from muscle protein breakdown. The most often used amino acid is phenylalanine because it may give the best representation of overall amino acid metabolism (Borsheim et al. 2004).

In practice, what is usually done when using the arteriovenous method is to measure the amount of phenylalanine going into a muscle and the amount of phenylalanine coming out of a muscle. (Inserting catheters into the femoral artery and femoral vein is very common to accomplish this task of being able to sample and measure the phenylalanine at these two anatomical locations.) Because phenylalanine is neither produced nor metabolized in muscle, net phenylalanine balance reflects net muscle protein balance. Specifically, rates of muscle protein synthesis can be determined by the disappearance of phenylalanine from the arterial blood into the muscle, and the rates of muscle protein breakdown can be determined by the appearance of phenylalanine into the venous blood.

From a general perspective, if 10 arbitrary units of phenylalanine are going into the skeletal muscle (measured by its rate of disappearance from the femoral artery) and only 8 arbitrary units of phenylalanine are coming out of the skeletal muscle (measured by its rate of appearance into the femoral vein), it can be assumed that two units of phenylalanine have been incorporated into skeletal muscle protein and therefore reflect a state of increased protein synthesis (since the phenylalanine cannot be oxidized in the skeletal muscle). On the other hand, if eight arbitrary units of phenylalanine are going into the skeletal muscle (measured by its rate of disappearance from the femoral artery) and twelve arbitrary units of phenylalanine are coming out of the skeletal muscle (measured by its rate of appearance into the femoral vein), it can be assumed that four units of phenylalanine have been released from skeletal muscle protein stores and therefore reflect a state of increased protein breakdown (since the phenylalanine cannot be produced in the skeletal muscle). It is not

uncommon to use isotopically labeled amino acids (such as l-[$^{13}C_6$]phenylalanine) and skeletal muscle biopsies in conjunction with arteriovenous difference measures so that a more complete picture of muscle protein turnover can be determined.

5.6.5.1 Limitations to the Arteriovenous Difference Method and the Superiority of Measures of Lean Body Mass Changes over Time

It is important to note that the arteriovenous method, as valuable as it is in providing specific information in terms of protein turnover in skeletal muscle, is only used to provide protein turnover information over an acute period of time, such as several hours. For example, arteriovenous differences are generally used to determine the effects of exercise and nutritional interventions and their effects on muscle protein synthesis and muscle protein breakdown for several hours after the exercise bout is completed. If skeletal muscle protein synthesis is maximized and skeletal muscle protein breakdown is minimized for several hours following the exercise and nutritional intervention, the methods used for the exercise session and the amount and type of nutrients ingested are considered to be successful.

What often occurs next, however, is that the short-term changes in muscle protein synthesis and muscle protein breakdown are then *assumed* to be predictive of what would happen over several months of training and nutrient ingestion, which may or may not be the case. While short-term increases in muscle protein synthesis and decreases in muscle protein breakdown are ideal, these acute changes in protein turnover may not translate into longer term positive training adaptations. Therefore, decisions about program design and nutrient intake should not be solely based on acute protein turnover studies. Rather, these acute protein turnover studies should be interpreted as valuable but secondary to those investigations that have tracked changes in lean muscle mass and exercise performance over time, such as several weeks to several months, when a training program and dietary intakes were used as interventions.

In summary, measures of lean body mass changes over time (several weeks to several months) stand as the best method for determining the effectiveness of training programs and nutritional interventions when the variable of interest is skeletal muscle hypertrophy.

5.7 PROTEIN METABOLISM AND NET MUSCLE PROTEIN BALANCE

Proteins take part in an ongoing cycle of formation (referred to as protein synthesis) and degradation (referred to as protein breakdown or proteolysis). Interestingly, different modes of exercise have different effects on the rates of muscle protein synthesis and muscle protein breakdown in the time period following exercise. In Section 5.6.3 of this chapter, it was stated that there are three basic states of nitrogen balance: positive, negative, and neutral. What this represented was a measure of whole-body protein metabolism. As was stated in this section, the most glaring limitation of the nitrogen balance method is its inability to delineate tissue-specific changes in protein metabolism. Nitrogen balance is a measure of whole-body protein flux and does not provided specific information in regard to skeletal muscle protein synthesis and breakdown. A better measure of protein metabolism (especially for athletes) is

what is referred to as *net muscle protein balance*. This measure of protein metabolism provides insight into what is happening in regard to protein metabolism within skeletal muscle.

Net muscle protein balance is equal to muscle protein synthesis minus muscle protein breakdown. If skeletal muscle hypertrophy is to occur, the net muscle protein balance must be positive—meaning that rates of muscle protein synthesis exceed rates of muscle protein breakdown. When this occurs, net muscle protein balance is said to be positive. In contrast, during periods of skeletal muscle protein breakdown, rates of muscle protein breakdown exceed rates of muscle protein synthesis. When this occurs, net muscle protein balance is said to be negative. In the sections that follow, the primary cellular mechanisms of both muscle protein synthesis and muscle protein breakdown are presented.

5.7.1 Skeletal Muscle Protein Synthesis

As stated previously, net muscle protein balance is equal to muscle protein synthesis minus muscle protein breakdown. Muscle protein synthesis is associated with skeletal muscle hypertrophy. In order for skeletal muscle hypertrophy to occur, net muscle protein balance must be positive (synthesis must exceed breakdown). Within the context of skeletal muscle hypertrophy, myosin, along with the protein actin, comprises the functional component of skeletal muscle. Of these two primary functional skeletal muscle proteins, it is myosin that possesses the greatest ability to increase in size. Therefore, as muscle protein synthesis is discussed in the remainder of this section, the focus will be on myosin protein/myosin filaments and, in particular, the mechanisms that contribute to an increase in overall skeletal muscle hypertrophy. Before the mechanisms of skeletal muscle protein synthesis are discussed, it is important to look at the broader picture of skeletal muscle metabolism and recognize the role that genes have in skeletal muscle hypertrophy.

5.7.1.1 The Role of Muscle-Specific Genes in Skeletal Muscle Protein Synthesis

A gene is a segment of DNA that provides the information for the sequence of amino acids in a protein. From a general perspective, once genes are activated, they are copied into messenger RNA (mRNA) in a process known as transcription. Once mRNA is copied from the DNA, the mRNA serves as a template for which proteins are manufactured (translated).

Specific to the protein myosin, the first step in the process of hypertrophy is the activation of the myosin gene. Resistance training acts as a stimulus for activating many muscle-specific genes, including the myosin gene. Once the myosin gene is activated, it is copied into myosin mRNA via transcription. It is this myosin mRNA that then directs the process of changing amino acids into polypeptides (via a process known as translation) and, ultimately, a functional myosin protein that is added to the existing matrix of the sarcomere of the muscle fiber. Once the myosin protein has been synthesized, the myosin gene is said to be "expressed."

The aforementioned discussion of the pathway that muscle-specific genes take from activation to expression was a general overview of this highly intricate process.

5.7.1.2 Translation Initiation

There is a crucial point of regulation during the process of muscle protein synthesis that has a significant impact on the overall net muscle protein balance status. This point of regulation manifests itself just prior to the period in which myosin mRNA begins to add amino acids together to form the myosin protein (translation). This highly regulated step is known as translation initiation. By definition, translation initiation is a process in which myosin mRNA, initiator transfer RNA (tRNA), and small and large ribosomal subunits associate with each other to form a complex referred to as the initiation complex. This initiation complex must be formed before any amino acids can be bonded together and the process of muscle protein synthesis can begin. If the rate of translation initiation can be increased, then the rate of protein synthesis is also increased. In contrast, if translation initiation is suppressed, then the rate of protein synthesis is also decreased.

During translation initiation, all three of the major types of RNA are needed: mRNA, tRNA, and ribosomal RNA (rRNA). In addition to the presence of all three types of RNA, a number of protein factors must be present to control the initiation process. The protein factors that are essential to translation initiation are collectively called the eukaryotic initiation factors (eIFs). Specifically, there are three initiation factors: eukaryotic initiation factor 4E (eIF-4E), eukaryotic initiation factor 4G (eIF-4G), and eukaryotic initiation factor 4A (eIF-4A). In order for translation initiation to commence, these three eIFs must form a complex collectively known as eukaryotic initiation factor-4F (eIF-4F).

During the complex process of translation initiation, two ribosomal subunits, the 40S and the 60S ribosomal subunits, must combine to form the complete ribosome, described as the 80S ribosome. Before the 40S and the 60S ribosomal subunits combine with each other to form the 80S complete ribosome, the binding of initiator methionyl-transfer RNA (met-tRNA) binds to the 40S ribosomal subunit to form the 43S preinitiation complex. Next, the myosin mRNA binds to the 43S preinitiation complex to form what is called the 48S preinitiation complex. However, before myosin mRNA can bind to the 43S preinitiation complex, eIF-4F must be formed. eIF-4F is formed when eIF-4A, eIF-4G, and eIF4-E form a complex. A specific protein binds eIF-4E and subsequently does not allow it to bind with eIF-4A and eIF-4G, thereby not allowing the formation of the eIF-4F complex. This binding protein is called eIF-4E binding protein 1 (4E-BP1).

The binding of 4E-BP1 to eIF-4E prevents association of eIF-4E with eIF-4G and thus precludes formation of the active eIF-4F complex (Jefferson and Kimball 2001). The interaction between eIF-4E and 4E-BP1 is regulated by phosphorylation of 4E-BP1, whereby hypophosphorylated forms of 4E-BP1 bind to eIF-4E but hyperphosphorylated forms do not (Figure 5.4) (Bolster, Kimball, and Jefferson 2003). So, if 4E-BP1 is phosphorylated, it allows the eIF-4F complex to be formed, which subsequently allows myosin mRNA to bind with the 43S preinitiation complex.

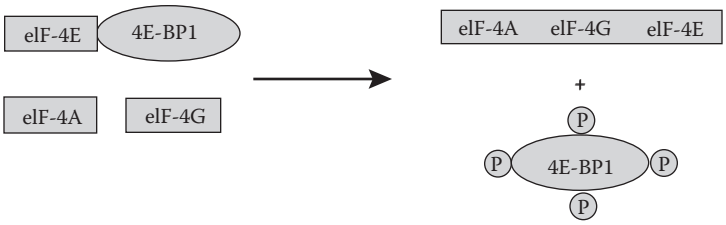

FIGURE 5.4 Formation of the eIF-4F complex via 4E-BP1 phosphorylation.

In addition to the formation of the eIF-4F complex, which allows the binding of myosin mRNA to the 43S preinitiation complex, there is yet another rate-limiting factor that may be associated with how the myosin mRNA binds with the 43S preinitiation complex. Ribosomal protein S6 (rpS6) is part of the small (40S) ribosomal subunit and, when phosphorylated, it has been correlated with increased protein synthesis (Dufner and Thomas 1999). Currently, the mechanism explaining how rpS6 enhances translation is still a mystery, but it is interesting to note that rpS6 is positioned near the mRNA binding site on the 40S ribosomal subunit and is thus located in a position that may permit a role in mRNA selection (Bolster et al. 2003). As stated before, when ribosomal protein S6 is phosphorylated, rates of protein synthesis are increased. The protein p70s6 kinase (p70S6k) phosphorylates ribosomal protein S6. Hence, when p70S6k is activated (via phosphorylation), it results in the subsequent phosphorylation and activation of rpS6 and enhances the translational capacity of the cell (An et al. 2003) and has been implicated in load-induced skeletal muscle hypertrophy (Xu et al. 2004).

Now that the 40S ribosomal subunit has been bound to both the tRNA and the myosin mRNA, it has formed the 48S preinitiation complex. The last step in the process of translation initiation is the 60S ribosomal subunit binds to the 48S preinitiation complex to form the 80S preinitiation complex.

To review, there were two compounds that needed to be phosphorylated in order for translation initiation to be optimized: p70S6k and 4E-BP1. Once these compounds are phosphorylated, the rate of translation initiation and, ultimately, protein synthesis increases.

5.7.1.3 Akt-mTOR Pathway Regulation on Translation Initiation

The Akt-mTOR pathway has been identified as a major regulatory pathway resulting in significant increases in the rates of protein synthesis. The Akt-mTOR pathway exerts its effects at the level of translation initiation. If the Akt-mTOR pathway is activated, it results in an increased rate of translation initiation and, ultimately, an increased rate of protein synthesis. Both of the compounds (4E-BP1 and p70S6k) that regulate translation initiation possess the same upstream regulator: the mammalian target of rapamycin (mTOR). Activation of mTOR has been shown to increase protein translation by activating p70S6K and inhibiting the activity of 4E-BP1 (Glass 2003). mTOR accomplishes this by phosphorylating both p70S6k (activating it) and 4E-BP1 (resulting in the releasing of eIF-4E), leading to increases in the rate of translation initiation (Figure 5.5).

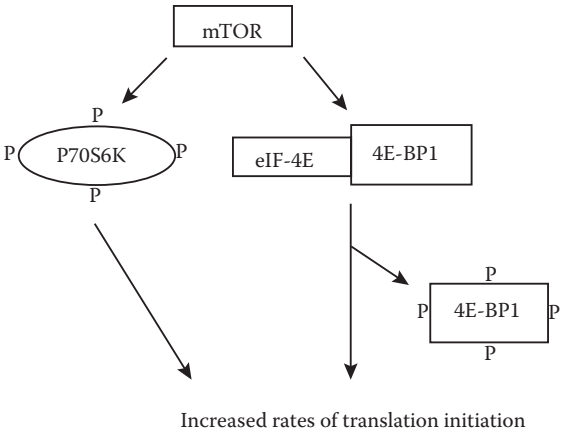

FIGURE 5.5 Increased rates of translation initiation via mTOR activation.

Directly upstream of mTOR is a compound known as Akt (also known as protein kinase B). Both of these compounds, Akt and mTOR, are part of the larger insulin signaling pathway (Figure 5.6). When insulin binds to its receptor, it promotes the phosphorylation of a family of substrates that includes insulin receptor substrate-1 (IRS-1). Once IRS-1 is phosphorylated/activated, it subsequently activates phosphatidylinositol 3-kinase (PI3-kinase). Following PI3-kinase activation, Akt is activated. Following Akt activation/phosphorylation, it has been shown that mTOR is subsequently activated (Kimball, Farrell, and Jefferson 2002). In summary, the proteins that constitute the insulin signaling pathway—the upstream protein kinase (PI3-kinase) as well as the downstream kinase Akt—work in concert through mTOR, thereby inducing the phosphorylation of 4E-BP1 and p70S6K and subsequently activating/amplifying protein translation (Gingras et al. 1998).

5.7.1.4 Leucine and Translation Initiation

Emerging data suggest that leucine supplementation ingested shortly following physical activity may enhance the anabolic status of the postexercise period. It appears that the effects that leucine exerts on the anabolic status are attributed to increases in protein synthesis. Multiple investigations have reported significant increases in protein synthesis following leucine ingestion. These findings have been reported in both animal models (Norton et al. 2012; Anthony et al. 2000) and human models (Dreyer et al. 2008; Katsanos et al. 2006).

What is the mechanism for leucine's effectiveness in elevating rates of protein synthesis? There is evidence to support that leucine is able to phosphorylate the regulatory compounds p70S6k and 4E-BP1, causing an increase in the rate of translation initiation (Norton et al. 2012; Dreyer et al. 2008; Anthony et al. 2000). One of the first studies to look at the effects of leucine on the Akt/mTOR pathway in humans was conducted by Greiwe et al. (2001). It was reported that phosphorylation of p70S6k increased fourfold in response to leucine alone, indicating that

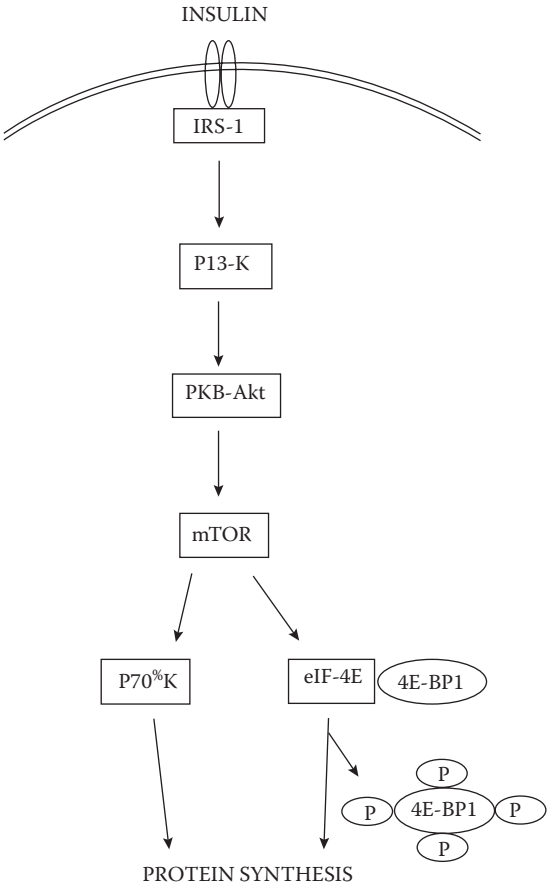

FIGURE 5.6 Insulin signaling transduction pathway.

physiological concentrations of leucine activate a key mediator of protein synthesis in human skeletal muscle.

To demonstrate just how important the amino acid leucine is to elevating protein synthesis via the phosphorylation/activation of p70S6k and 4E-BP1, Norton et al. (2012) conducted a series of studies and reported that low-quality protein sources that were lacking in leucine content were unable to increase rates of protein synthesis in rat models. In contrast, sources of protein that contained higher levels of leucine were able to elevate protein synthetic rates. In the first of these studies, rats were fed one of four meals (as well as a fasting group to serve as a control) containing 16% protein coming from the following:

- Wheat protein (6.8% leucine content providing 46 mg of leucine)
- Soy protein isolate (8% leucine content providing 54 mg of leucine)
- Egg white protein (8.8% leucine content providing 60 mg of leucine)
- Whey protein isolate (10.9% leucine content providing 74 mg of leucine)

Ninety minutes following ingestion, plasma leucine levels increased in the egg white and whey protein treatments, but not in the wheat or soy protein treatments. Rates of muscle protein synthesis were significantly elevated in the egg and whey protein treatments with the highest value observed in the whey protein group. No significant increase was observed in muscle protein synthesis in the wheat and soy protein groups. In terms of the markers of translation initiation, 4E-BP1 phosphorylation was also significantly higher in the whey and egg white protein groups as compared to the soy and wheat protein groups. The whey protein group increased the phosphorylation states of p70S6k significantly more than all other protein treatments.

In a second experiment, Norton and co-workers (2012) gave rats one of three meals with 16% protein coming from the following:

- Wheat gluten
- Wheat gluten plus leucine (to match the leucine content of the whey protein meal)
- Whey protein

In the hours following the ingestion of these meals, muscle protein synthesis increased in the whey and wheat-plus-leucine groups but not in the wheat-only group. Also, rates of protein synthesis were not different between the whey and the wheat-plus-leucine groups. Each of these experiments demonstrates that leucine content is a critical factor for evaluating the quantity and quality of proteins necessary for each meal in relation to stimulating muscle protein synthesis. Further, the data generated from these investigations establish a threshold requirement for a minimum leucine content within a meal in terms of stimulating muscle protein synthesis.

While there appears to be a minimum threshold of leucine ingestion to stimulate muscle protein synthesis, is it possible that more leucine is better? Several investigations have demonstrated that while leucine is an essential threshold signal for translation initiation, additional amounts of leucine above the threshold level do not produce additive effects on muscle protein synthesis (Debras et al. 2007)

5.7.2 Muscle Protein Breakdown and the Ubiquitin Proteasome Pathway

Net muscle protein balance contains two variables. While protein synthesis controls one aspect of protein balance, protein breakdown controls the other side of the equation. Muscle protein breakdown should not be looked at as a nefarious process, but rather as integral to proper maintenance of healthy muscle fiber function via its role in degrading old and damaged proteins (Mitch and Goldberg 1996). There are three main protein-degrading systems at work in skeletal muscle: the ubiquitin–proteasome pathway, the lysosomal system, and the calpain system.

The protein-degrading system that degrades the myofibrillar proteins (actin and myosin) is the ubiquitin–proteasome pathway (Mitch and Goldberg 1996). The process of degrading proteins begins with the "marking" of a protein by the covalent attachment of ubiquitin chains to the protein that is targeted for degradation. Ubiquitin is a small protein that makes a protein recognizable by the proteasome. The proteasome is a large, barrel-shaped protein complex made up of many subunits.

The degradation of proteins by the ubiquitin–proteasome pathway requires ATP (adenosine triphosphate) hydrolysis.

5.8 RESISTANCE AND ENDURANCE EXERCISE AND NET MUSCLE PROTEIN BALANCE

At rest, in the absence of an exercise stimulus and protein intake, net muscle protein balance is negative (Wagenmakers 1999; Phillips et al. 1997; Biolo et al. 1995). This means that the rate of muscle protein breakdown is greater than the rate of muscle protein synthesis. Resistance exercise and endurance exercise both have an effect on net muscle protein balance. Technical development and application of methods to measure muscle protein breakdown have lagged behind those of muscle protein synthesis. Therefore, there has been less scientific investigation into the effects of both resistance and endurance exercise and their effects on muscle protein breakdown. Also, since the methods used to measure rates of protein synthesis require constant isotopic tracer infusions, they are only suitable for measuring acute (meaning several hours) muscle protein synthesis following exercise.

Wilkinson and co-workers (2008) designed a study that investigated multiple aspects of resistance exercise and endurance exercise and their effects on muscle (myofibrillar) protein synthesis in both the trained and untrained states. Ten healthy, untrained males participated in the study. The subjects were not actively participating in any resistance-training activities or any programmed endurance activities. The subjects underwent two testing periods separated by 10 weeks of unilateral leg resistance or endurance-exercise training. During the first testing period (which was in the untrained state), the subjects performed a bout of resistance exercise (five sets of 8–10 repetitions at 80% 1 RM) with one leg and then a bout of endurance exercise (single leg cycling for 45 min at 75% VO_2peak) with the other leg. Prior to, during, and after the exercise bouts, the subjects ingested beverages that provided 1.1 g protein/kg body mass. Four hours after the exercise bouts, muscle protein synthesis was measured.

After the initial testing period, the subjects underwent a 10-week training program in which one leg carried out a resistance-training program (knee extension exercise) and the other leg carried out an endurance-training program (one-legged cycling). After the 10-week training period, the same procedures were followed, but this time the measures were conducted in the trained state, since the subjects trained for the previous 10 weeks.

Prior to training, resistance exercise resulted in a 68% increase in muscle protein synthesis in the hours following the workout. In the trained state, there was a 37% increase in muscle protein synthesis following the workout. Endurance exercise had no effect on muscle protein synthesis in the hours following the workout in both the untrained and trained states. In summary, this investigation reported that resistance exercise results in a significant elevation in muscle protein synthesis following resistance exercise, but that endurance exercise has no impact on rates of muscle protein synthesis (Wilkinson et al. 2008).

Kumar and colleagues (2009) completed a comprehensive review on the topic of the different modes of exercise and its effects on net muscle protein balance. The following are highlights of their impressive review as it pertains to net muscle protein balance:

- At rest, net muscle protein balance is negative (muscle protein breakdown is greater than muscle protein synthesis).
- After exercise, in the fasted state, there is a rise in the rate of muscle protein synthesis. Despite this rise, net muscle protein balance is still negative as the rate of muscle protein breakdown also increases. However, net muscle protein balance is less negative following exercise as compared to the rested stated because rates of muscle protein synthesis rise to a greater extent than do rates of muscle protein breakdown.
- When amino acids (containing leucine) or protein are ingested after exercise, net muscle protein balance becomes positive as the rate of muscle protein synthesis surpasses the rate of muscle protein breakdown.

In addition to an overview of net muscle protein balance, Kumar and colleagues (2009) also reviewed the effects that resistance exercise and endurance exercise have on muscle protein synthesis. Resistance exercise results in increased rates of muscle protein (myofibrillar) synthesis in the few hours following the exercise bout. Specifically, an acute bout of resistance exercise can increase the rate of muscle protein synthesis about two- to fivefold above pre-exercise levels. This increase occurs in both the trained and untrained states. However, in the trained state, the amount of the increase in the rate of muscle protein synthesis is reduced as compared to the untrained state (Wilkinson et al. 2008).

Endurance exercise imparts similar responses, at least in the untrained stated. Following endurance exercise, muscle protein synthesis is elevated in the hours following the exercise bout. However, this increased rate of protein synthesis is only observed in the untrained state. After a period of endurance training, rates of muscle protein (myofibrillar) synthesis are not elevated in the postexercise period.

Summarizing the differences between resistance and endurance exercise in the trained versus untrained states, following a period of training, it appears as if resistance exercise is still able to increase myofibrillar protein synthesis, but that endurance exercise is not able to impart the same effect. Rather, following a period of training, endurance exercise does result in an increase of mitochondrial protein synthesis in the postexercise period. Figure 5.7 summarizes the findings of this study and others as they relate to the rates of muscle protein synthesis and breakdown following a bout of resistance exercise.

5.9 CONCLUSION

The primary role of dietary protein is different from that of carbohydrates and fats and should be viewed in a way that its ingestion emphasizes longer term adaptations resulting from the athlete's training and conditioning programs. Several quantitative measures are used to rank the quality of dietary proteins. Regardless of the method used, if the dietary protein contains adequate amounts of the eight essential amino

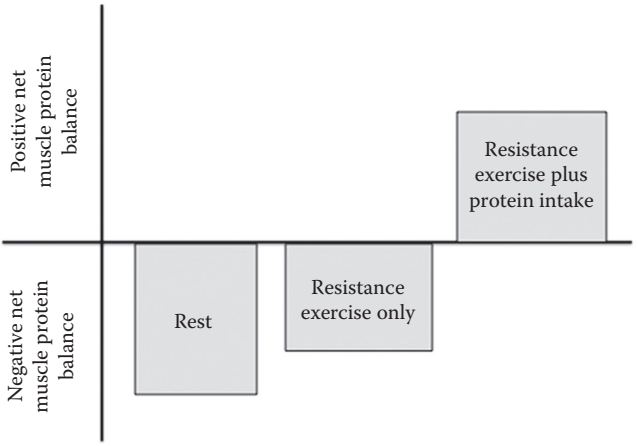

FIGURE 5.7 Net muscle protein balance.

acids, the protein is considered a complete, high-quality protein. In general, animal proteins are considered high-quality proteins and vegetable proteins are considered low-quality proteins.

Of all the different aspects of protein metabolism, net muscle protein balance plays an especially important role for the athlete. At rest, net muscle protein balance is negative. (Muscle protein breakdown is greater than muscle protein synthesis.) After exercise, in the fasted state, there is a rise in the rate of muscle protein synthesis. Despite this rise, net muscle protein balance is still negative as the rate of muscle protein breakdown also increases. However, net muscle protein balance is less negative following exercise as compared to the rested state because rates of muscle protein synthesis rise to a greater extent than do rates of muscle protein breakdown. When amino acids (containing leucine) or protein are ingested after exercise, net muscle protein balance becomes positive as the rate of muscle protein synthesis surpasses the rate of muscle protein breakdown.

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6 Dietary Protein Strategies for Performance Enhancement

Bill Campbell

CONTENTS

6.1 Introduction 155
6.2 Daily Protein Intake Recommendations 157
6.2.1 Factors Influencing Daily Protein Intakes 159
6.2.1.1 Protein Quality 159
6.2.1.2 Energy Intake 160
6.3 How Much Protein Should Be Ingested at Each Meal? 160
6.3.1 The Refractory Response of Skeletal Muscle to Elevated Plasma Amino Acid Concentrations 162
6.3.2 The Role of Leucine 163
6.4 Protein Timing—How Often Should Protein Be Ingested during the Course of the Day? 164
6.5 Safety Issues with Elevated Protein Intakes 165
6.6 Protein and Exercise Performance 167
6.6.1 Endurance Exercise 168
6.6.1.1 Central Fatigue Hypothesis 170
6.6.2 Resistance Exercise 171
6.6.2.1 Protein and Net Muscle Protein Balance 171
6.6.2.2 Comparison of High-Quality Proteins 172
6.7 Conclusion 176
Acknowledgment 177
References 177

6.1 INTRODUCTION

Dietary protein and specific amino acids have been investigated for their effects on improving exercise performance. Due to the fact that dietary protein is not a primary source of energy, as carbohydrates and fats are, the effects that dietary protein intake imparts on exercise performance are usually chronic in nature. What this means is that dietary protein is not simply ingested prior to or following a single training bout

and improvements in exercise performance/body composition are realized by the next day. In contrast, the effect that dietary protein has on enhancing performance or body composition is a process. In short, the process can be summarized as the following:

- The training session imparts a stimulus to the body.
- The body adapts to the stimulus over time, resulting in adaptations that are favorable for improving performance/body composition.

If protein is ingested in the proper amounts and at the right times, then the adaptations to the training stimulus are optimized. If dietary protein intake is inadequate, then the adaptations to the training stimulus are not optimized. This does not mean that gains in performance are not realized, but it does imply that the adaptations and, ultimately, exercise performance are not what they could be and the athlete is not reaching his or her full potential. The purpose of this chapter is to discuss how dietary protein can assist the athlete in optimizing training so that performance goals can be realized.

A discussion of optimal training programs is outside the scope of this chapter. However, the training program provides the stimulus to the athlete's body. After the stimulus has been imparted, it is the protein intake that allows the body to adapt optimally to the training stimulus. Therefore, the training program is of utmost importance when attempting to improve performance. While protein intake is very important, it is nonetheless secondary to a proper training program. Figure 6.1 summarizes these important concepts (from a resistance-training perspective) and highlights the role that dietary protein has in the adaptive process of skeletal muscle hypertrophy.

In summary, the process of training can be broken down into two simple processes: the process of stimulation and the process of adaptation. This chapter will focus on all of the aspects that dietary protein can contribute to the process of

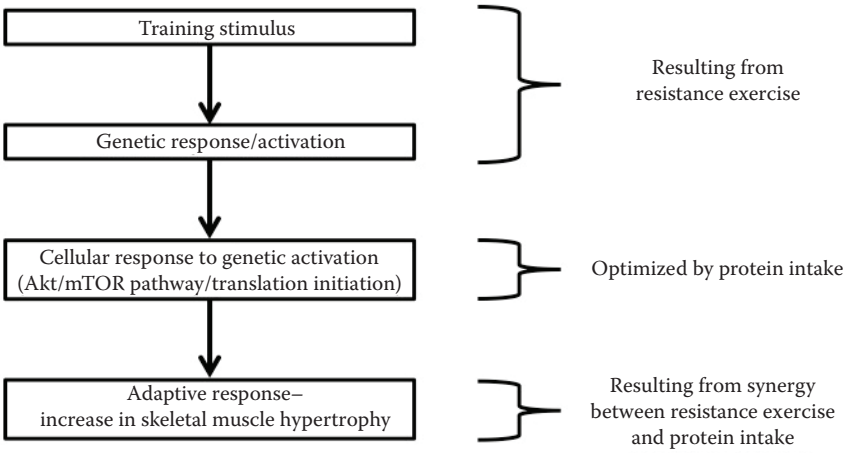

FIGURE 6.1 Synergy between resistance-exercise dietary protein intake relative to training adaptations.

adaptation; over time, this results in optimized outcomes in relation to exercise performance and body composition.

6.2 DAILY PROTEIN INTAKE RECOMMENDATIONS

There was a time when it was debated that athletes and physically active individuals needed more protein than the recommended dietary allowance (RDA) levels (0.8 g/kg body mass/day). Currently, this issue is no longer debated, as most researchers and professionals agree that athletes need to ingest dietary protein at levels higher than 0.8 g/kg body mass/day. The RDA for protein intake is estimated to be sufficient to meet the needs of nearly all (97.5%) healthy men and women age 19 years and older. This definition is important—*to meet the needs of nearly all healthy men and women*. When working with athletes, the goal is not simply to meet their dietary protein needs. Rather, the goal is both to meet their needs and to provide nutrients that will allow them to adapt optimally to their training programs. If an athlete ingested only the RDA for protein, the amount of protein would likely not be enough to (Campbell et al. 2007):

1. Offset the oxidation of protein/amino acids during exercise training (approximately 1%–5% of the total energy cost of exercise)
2. Provide substrate for lean tissue accretion
3. Repair exercise-induced muscle damage

What is the ideal amount of daily protein that will allow the athlete to optimize adaptive responses? Unfortunately, a specific answer to this question has not been adequately offered at this time. There are three academic societies that serve the athletic and physically active populations, and each of these organizations has stated that athletes should ingest relatively higher daily intakes of protein. Table 6.1 summarizes these recommended daily protein intakes for athletes.

The data on which these recommendations have been based is not in abundance. In fact, there have been only a handful of published research studies that have compared low versus high daily protein intakes and measured their effectiveness on

TABLE 6.1
Recommended Daily Protein Intakes from Academic Societies Serving Athletic Populations

Academic Organization	Recommended Daily Protein Intakes
American College of Sports Medicine (American Dietetic Association et al. 2009)	1.2–1.7 g/kg body mass/day
International Society of Sports Nutrition (Campbell et al. 2007)	1.4–2.0 g/kg body mass/day
National Strength and Conditioning Association (Reimers 2008)	1.5–2.0 g/kg body mass/day

net protein balance. Three of these studies were conducted with endurance athletes (Friedman and Lemon 1989; Meredith et al. 1989; Tarnopolsky et al. 1988) and three were conducted with strength/power athletes (Tarnopolsky et al. 1988, 1992; Lemon et al. 1992). In each study, it was reported that dietary protein intake needed to be above 0.8 g/kg body mass/day to remain in nitrogen balance for athletes and individuals participating in exercise training.

In relation to the endurance athletes, Friedman and Lemon (1989) gave well-trained endurance runners who were participating in their normal training (running 12–16 km/day) two different protein-containing diets for a 6-day period. The low-protein diet contained 0.86 g/kg body mass/day and the high-protein diet contained ~1.5 g/kg body mass/day. At the end of the 6-day intervention, it was reported that the higher protein intake resulted in a positive whole-body nitrogen balance while the lower protein intake resulted in a negative whole-body nitrogen balance. For strength athletes, Tarnopolsky et al. (1992) divided resistance-trained and sedentary subjects into three dietary protein intake groups: a low protein group (0.86 g/kg/day), a moderate protein group (1.4 g/kg/day), and a high protein group (2.4 g/kg/day). Following a 13-day intervention period, it was reported that whole-body nitrogen balance for the strength-trained participants was 1.41 g protein/kg body mass/day. The authors recommended a safety margin of one standard deviation, thus increasing the daily protein requirements to 1.76 g/kg body mass in order to provide enough protein substrate to adapt to the training stimulus.

In another study, Lemon and co-workers (1992) assessed protein requirements during the early stages of intensive bodybuilding training in previously non-resistance-trained males. During this randomized crossover study, each subject received an isoenergetic supplement. The higher protein supplement contained 2.6 g/kg body mass/day protein and the lower protein supplement contained 1.35 g/kg body mass/day. Each level of protein intake was studied for a 3.5-week period in which the subjects resistance trained for 1.5 h/day, 6 days/week. On the basis of 3-day whole-body nitrogen balance measurements, it was reported that the lower protein intake resulted in a negative net nitrogen balance and the higher protein intake resulted in a positive net nitrogen balance. The investigators calculated that a neutral net protein balance was about 1.4 to 1.5 g of protein/kg body mass/day, and that the recommended intake was approximately 1.6 to 1.7 g/kg body mass.

One investigation compared the dietary protein intakes of bodybuilders, endurance athletes, and sedentary individuals over a 20-day period (Tarnopolsky et al. 1988). For the first 10 days, each group ingested protein intakes at normal levels and then this was followed by 10 days of altered protein intakes. During the 10 days of altered protein intakes, the endurance athletes increased their protein intakes from 1.7 to 2.7 g/kg body mass and the sedentary individuals increased their protein intakes from 1.1 to 1.9 g/kg body mass. Since the bodybuilding group consumed habitually high levels of dietary protein, their protein intakes were reduced from 2.7 to 1.05 g/kg body mass during the 10 days of altered protein intake. The 3-day nitrogen balance data revealed that bodybuilders required 1.12 times and endurance athletes required 1.67 times more daily protein than sedentary controls to remain in nitrogen balance. For the bodybuilders, lean body mass was maintained during the 10-day altered protein intake period. While this study reported that bodybuilders do

not need excessive amounts of protein to maintain lean body mass for a short period of time, it did not address what ideal protein intakes should be for bodybuilders wishing to increase lean body mass.

Taking the data from each investigation into consideration, it is clear that both strength and endurance athletes need to ingest dietary protein intake levels that are higher than the RDA to remain in nitrogen balance. What is not clear from these investigations is a clearly defined optimal amount of dietary protein to maximize adaptations imparted by the training stimulus. It is also important to emphasize that the previous studies investigating various protein intakes were based on the whole-body nitrogen balance technique. This method is a measure of whole-body protein flux and is not an ideal measure of skeletal muscle protein metabolism. Future studies should investigate altered protein intakes and assess net muscle protein balance (measures of muscle protein synthesis and breakdown) in addition to whole-body nitrogen balance in athletes who are training. These data would provide more relevant outcomes for athletic populations.

Also, little research has been conducted on exercise activities that are intermittent in nature (e.g., soccer, basketball, mixed martial arts, etc.). In a review focusing on soccer players, a protein intake of 1.4–1.7 g/kg was recommended (Lemon 1994). Based on the available evidence, it appears that the recommendations of protein intakes for athletes posited by the academic societies (Table 6.1) are reasonable. The general recommendation for dietary protein intake for athletes is approximately 1.5 to 2.0 g/kg body mass/day. For a 200-lb. (91-kg) male athlete, this equates to a protein intake of 136 to 182 g/day. For a 135-lb. (61-kg) female athlete, this equates to a daily protein intake of 91 to 122 g.

The area of dietary protein intake needs further research. Also, other important factors need to be considered, such as the amount of protein that should be ingested with each meal and how many times per day dietary protein should be ingested. Each of these factors works in concert with total daily dietary protein intakes.

6.2.1 Factors Influencing Daily Protein Intakes

There are many factors that need to be considered when determining what constitutes an optimal amount of dietary protein for athletes. These factors include protein quality, energy intake, carbohydrate intake, mode and intensity of exercise, and the timing of the protein intake (Lemon 2000). Depending on the status of these factors, more or less daily protein may need to be consumed. Protein quality and daily energy intakes likely have the largest effect on overall protein requirements, so a brief discussion related to these two factors is presented.

6.2.1.1 Protein Quality

Protein quality is determined by the composition of its amino acids. Specifically, complete protein sources that contain greater amounts of essential amino acids are considered to be high-quality proteins. A low-quality protein is an incomplete protein and is lacking in one or several essential amino acids. The types of protein (high vs. low quality) that are ingested by the athlete on a daily basis will impact the protein requirements. For example, if the athlete is ingesting most of his or her protein

from high-quality sources, less protein will be needed on a daily basis. In contrast, if the athlete is relying on inferior sources of protein, then a greater daily amount of protein will need to be ingested to meet needs and for optimal adaptation to the training program. In essence, the higher the quality of the protein, the lower is the protein requirement. Young et al. (1975) compared the amount of beef protein and the amount of wheat protein that it would take to support body nitrogen balance in young males. It was determined that if the protein source ingested was whole wheat protein (a low-quality protein source), it would take 85% more protein as compared to the amount of protein that would need to be ingested if the source was beef protein (a high-quality protein source).

6.2.1.2 Energy Intake

When estimating the protein requirements for athletes, one must consider total daily energy intake. When energy intakes are below energy balance levels, the contribution of dietary protein needs to be increased due to the fact that protein can be metabolized as a source of energy in a state of negative energy balance. Hence, there is an inverse relationship between caloric intake and protein requirements. When caloric intake decreases, protein requirements increase. Walberg et al. (1988) investigated different protein intakes in weight lifters who were dieting. The male weight lifters were placed on an isoenergetic hypocaloric diet (18 kcal/kg body mass/day), which contained either a low-protein (0.8 g/kg) or a high-protein (1.6 g/kg) daily intake. The low-protein diet group achieved a negative net nitrogen balance while the high-protein diet group achieved a positive net nitrogen balance. The investigators concluded that a hypoenergy diet providing two times the RDA for protein was more effective in retaining body protein than a diet providing only the RDA for protein (Walberg et al. 1988).

Pikosky et al. (2008) reported similar observations. In their investigation, rather than decreasing energy intake, they created an energy deficit by increasing the amount of exercise in which the participants engaged. Following a 4-day baseline period of an energy balance diet and maintaining usual physical activity, there was a 7-day period of increased energy expenditure (1000 calories/day) via endurance exercise (bike, treadmill, elliptical) at 50%–65% VO_2peak. During the energy deficit period, one group consumed 0.9 g protein/kg/body mass/day and the other group consumed 1.8 g protein/kg/day. During the 7-day negative energy balance period, the lower protein group experienced a significant decrease in nitrogen balance while the high protein group experienced no differences in nitrogen balance. In Chapter 9, "Enhancing Body Composition: Gaining Muscle and Losing Fat," more data are presented that highlight the necessity of not reducing dietary protein intakes during a period of purposeful energy restriction.

6.3 HOW MUCH PROTEIN SHOULD BE INGESTED AT EACH MEAL?

When determining how much protein should be ingested at each meal, it is important to understand the perspective that an answer to this question often assumes: that

the nutritional goal of protein intake is the stimulation of muscle protein synthesis. While this goal is certainly important for most strength/power athletes, this particular physiologic response may not play a pivotal role for all athletes. Nonetheless, athletes of all types should seek to maximize the adaptations to their training stimulus, and one measure of dietary protein's effectiveness is its ability to stimulate muscle protein synthesis. With this assumption clearly stated (to ingest the optimal amount of dietary protein to maximize muscle protein synthesis), we seek to answer the question surrounding how much protein to ingest with each meal.

Several recent publications indicate that the maximum stimulation of muscle protein synthesis occurs with the intake of 20 to 30 g of high-quality protein. Whole protein by itself does not appear to be responsible for stimulating the metabolic pathways that initiate protein synthesis. Rather, it is the essential amino acid content and leucine in particular that have been shown to stimulate muscle protein synthesis via its activation of key regulatory factors in the Akt/mTOR cell signaling pathway that lead to an enhancement of translation initiation (Norton et al. 2012; Tipton, Gurkin, et al. 1999). While leucine appears to be the key amino acid that stimulates protein synthesis, the other amino acids, both essential and nonessential, that are provided by the ingested protein are important as all amino acids act as substrates for a given protein (such as myosin) undergoing synthesis.

When determining the amount of protein that should be ingested in one sitting to increase protein synthetic rates maximally, the best types of study designs would compare different amounts of protein (low vs. moderate vs. high) and then measure their effects on protein synthesis in the hours after ingestion. This type of study design has been conducted on a limited number of occasions. Symons et al. (2009) gave young (~35 years) and elderly (~68 years) males two different beef servings: a moderate serving and a large serving. The moderate serving (113 g) provided 30 g of protein (containing about 10 g of essential amino acids) and the large serving (340 g) provided 90 g of protein (containing about 30 g of essential amino acids). After ingestion, measures of muscle protein synthesis were made for about 5 h to determine if the larger serving of beef resulted in a larger rate of protein synthesis.

A moderate serving of lean beef containing about 30 g of protein increased muscle protein synthesis by approximately 50% in both young and older volunteers. Despite a threefold increase in protein and energy content, there was no further increase in protein synthesis after ingestion of larger servings of lean beef in either age group. The authors concluded that ingestion of more than 30 g of protein in a single meal does not further enhance the stimulation of muscle protein synthesis in young and elderly males. While the participants in this study were not athletes, there is no reason to believe that an athlete or physically active individual would respond differently to such an intervention. From this study alone, it can be concluded that 90 g of high-quality protein is no better than 30 g of the same high-quality protein in relation to stimulating muscle protein synthesis following a single meal.

Moore and colleagues (2009) took a similar approach in terms of giving male subjects different amounts of protein and subsequently measured rates of protein synthesis over a 4-h period. The protein synthesis measured in this study was mixed muscle, meaning that both myofibrillar and mitochondrial proteins were measured

for their rates of synthesis. In addition to the protein intake, there was also an exercise component that consisted of lower body resistance exercise. Specifically, the trained males completed four sets each of the leg press, leg extension, and leg curl, taking each set to failure at approximately 8–10 repetitions. After the resistance-exercise bout, the subjects ingested 0, 5, 10, 20, or 40 g of whole-egg protein in beverage form. While the 5- and 10-g doses significantly elevated levels of protein synthesis, protein synthesis was not maximized until 20 g of protein were ingested. The 40-g dose of whole-egg protein did increase protein synthesis levels above the 20-g dose (approximately 6% more), but the increase did not reach levels of statistical significance. In light of the nonsignificant finding, protein ingested above 20 g offers no further advantage in terms of stimulating muscle protein synthesis.

The excess protein that was ingested simply resulted in an increase in protein oxidation, meaning that the "extra" protein was metabolized rather than being incorporated into the protein synthetic processes. This finding makes sense, as overfeeding protein does not result in an increase of lean body mass. Specifically, amino acids supplied in excess of the requirements for protein synthesis are oxidized and their carbon skeletons are subsequently used for fuel or stored as body fat (Bohé et al. 2001; Motil et al. 1981).

Despite the increases in protein synthesis observed in the Moore et al. (2009) study, there was no statistically significant change in the phosphorylation of p70s6 kinase (p70S6k), which would have been expected given its role in translation initiation and protein synthesis. The essential amino acid content comprising the 20 g of whole-egg protein and observed to maximize rates of protein synthesis was 8.6 g. The dose of essential amino acids that maximally stimulates muscle protein synthesis after resistance exercise (~8.6 g) was very similar to that observed in the resting condition (10 g of essential amino acids) (Cuthbertson et al. 2005). In terms of leucine content, ~1.7 g (20 mg/kg body mass) resulted in maximal rates of protein synthesis (Moore et al. 2009).

6.3.1 The Refractory Response of Skeletal Muscle to Elevated Plasma Amino Acid Concentrations

It appears that there is an upper limit to the amount of protein that is ingested in one sitting and its ability to stimulate muscle protein synthesis maximally (Symons et al. 2009; Moore et al. 2009). Stated differently, ingesting excessive amounts of protein in a given meal will not result in increased rates of muscle protein synthesis. When this happens—when rates of muscle protein synthesis will not elevate despite elevations of amino acids—it is said that the skeletal muscle becomes refractory to stimulation. Bohé and co-workers (2001) conducted a study that clearly displayed the refractory response of skeletal muscle to elevated levels of infused amino acids. Amino acids were infused at a constant rate for a 6-h period. Overall, the amino acid infusion protocol resulted in a nearly threefold increase in muscle protein synthesis rates as compared to baseline. Following the amino acid infusion, the observed rates of muscle protein synthesis (MPS) were as follows:

- No increase in MPS for the first 30 min
- A significant rise of MPS between 30 and 60 min
- Peak MPS rates observed between 60 and 120 min (nearly a threefold increase)
- A robust fall in MPS from the peak value for the final 4 h of the amino acid infusion period (back to baseline levels)

Despite the fact that plasma amino acid levels were elevated for a 6-h period, muscle protein synthesis was only elevated for about a 2-h period following the amino acid infusion. This study demonstrates the refractory response of skeletal muscle and its apparent ceiling on rates of protein synthesis despite elevated concentrations of amino acids for up to 6 h. The concept of this refractory response of skeletal muscle has important consequences when making decisions as to the optimal meal/protein frequency on a daily basis. Practical recommendations related to this topic are discussed in Section 6.4, "Protein Timing—How Often Should Protein Be Ingested during the Course of the Day?"

6.3.2 The Role of Leucine

What if additional leucine is added to a protein/essential amino acid beverage? Does this result in an amplified muscle protein synthetic response? Based on the work of Norton et al. (2012) in rodent models, which clearly demonstrated a leucine threshold for stimulating muscle protein synthesis, it may be that the leucine content in any given protein intake is the limiting factor in terms of maximizing muscle protein synthesis, rather than the amount of the total protein content.

Pasiakos and co-workers (2011) reported that a leucine-enriched essential amino acid beverage resulted in a significant elevation of muscle protein synthesis as compared to a normal leucine-containing essential amino acid beverage. In this study, trained males ingested one of two different 10-g essential amino acid beverages during cycling exercise: one containing 3.5 g of leucine (46 mg/kg body mass) and the other containing 1.87 g of leucine (~25 mg/kg body mass). After the exercise bout (during a 3-h assessment period), muscle protein synthesis was 33% greater in the leucine-enriched essential amino acid beverage.

In the Moore et al. (2009) investigation in which 40 g of protein was observed to be no better than 20 g of protein after resistance exercise, the leucine content was ~1.7 g (20 mg/kg body mass) and 3.36 g (39 mg/kg body mass) for the 20- and 40 g egg protein supplements, respectively. In another study that incorporated a resistance-exercise component, Tipton, Ferrando, et al. (1999) gave healthy subjects either 40 g of mixed amino acids (4.4 g of leucine [69.8 mg/kg body mass]) or 40 g of essential amino acids only (8.3 g of leucine [131.7 mg/kg body mass]) a few hours after a lower body resistance-exercise bout. There were no differences in the rates of protein synthesis observed between the lower and higher leucine contents of the beverages.

Yet another study reported that leucine intakes of ~2.8 g (44.3 mg/kg body mass) were able to increase rates of muscle protein synthesis significantly (Paddon-Jones et al. 2004). However, this particular study did not investigate stepwise increases in leucine content, but rather provided the leucine (as part of an essential amino acid

TABLE 6.2
Amount of Protein Needed to Ingest Leucine at 45 mg/kg Body Mass for Individuals of Varying Body Weights

Body Mass (lb.)	Whey Protein Isolate (g)	Casein (g)	Soy Protein Isolate (g)	Milk Protein (g)	Chicken (g)
100 (~46 kg)	8–17	11–23	11–25	10–21	13–28
125 (~57 kg)	10–21	13–29	14–31	12–26	16–34
150 (~68 kg)	12–25	15–35	17–38	14–31	19–41
175 (~80 kg)	14–30	18–40	11–25	16–36	22–48
200 (~91 kg)	16–34	21–46	11–25	18–41	25–55
225 (~102 kg)	17–38	23–52	11–25	21–46	28–61
250 (~114 kg)	19–43	25–58	11–25	23–51	31–68

Note: Approximate ranges of protein intake were calculated from the Pasiakos et al. (2011) and Moore et al. (2009) studies in which maximal rates of muscle protein synthesis were measured.

beverage) as single doses and then monitored rates of protein synthesis for several hours after ingestion.

Based on the evidence that is available, it appears that when a leucine content of about 20 to 45 mg/kg body mass/day is ingested, rates of muscle protein synthesis are maximized. So, in order to answer the question about how much protein should be ingested at each meal to maximize rates of muscle protein synthesis, the leucine content of the protein must be considered. Beef, poultry, egg, casein, and fish are approximately 8%–9% leucine and whey protein isolate is about 12% leucine. So, when these different sources of protein are compared side by side, less whey protein isolate would need to be ingested to obtain the necessary amounts of leucine, while relatively more beef, poultry, and fish would need to be ingested. Table 6.2 summarizes the ranges of the different types of protein that would need to be ingested in order to maximize rates of muscle protein synthesis at one sitting.

6.4 PROTEIN TIMING—HOW OFTEN SHOULD PROTEIN BE INGESTED DURING THE COURSE OF THE DAY?

So far, recommendations have been made in relation to total daily dietary protein intakes as well as the amount of dietary protein that should be ingested at each meal to stimulate muscle protein synthesis maximally. The next logical step in providing practical information to the athlete and his or her support staff is to make a recommendation regarding the protein distribution throughout the day (or how many times protein should be ingested over the course of an entire day). Since changes in protein turnover in skeletal muscle are regulated on a meal-to-meal basis, a recommendation in terms of the frequency in which protein should be ingested is very important for the athlete.

Some of the factors that need to be accounted for when devising a dietary protein intake strategy are the refractory response of skeletal muscle and the time course for the protein synthetic response. In general, skeletal muscle protein synthesis is elevated for 2 to 3 h following ingestion of high-quality protein/essential amino acids that contain adequate amounts of leucine. If this fact were taken in isolation, one may be tempted to conclude that ingesting adequate amounts of protein every 2 to 3 h would be ideal to keep muscle protein synthesis rates at elevated rates consistently throughout the day. However, such an approach disregards the refractory response of skeletal muscle to aminoacidemia (prolonged elevations of plasma amino acids). One of the pioneering investigators in this area, Layne Norton, has suggested that it may be best to consume larger doses of protein that contain sufficient leucine to maximize muscle protein synthesis and then allow enough time for amino acid levels to fall between meals in order to resensitize the system (Norton and Wilson 2009).

Moore et al. (2009) speculated that no more than five or six times daily could one ingest adequate amounts of protein and expect muscle protein synthesis to be stimulated maximally. In a well-designed study, Paddon-Jones et al. (2005) sought to determine the impact of six protein/amino acid feedings on mixed muscle protein synthesis (mitochondrial and myofibrillar protein synthesis) as compared to only three protein feedings in a single day. The subjects in this study were recreationally active males. It was reported that mixed muscle protein synthesis was ~25% greater over a 16-h period when consuming three large meals (~850 calories; ~23 g protein, ~127 g carbohydrate, and ~30 g fat), supplemented with an additional three small 180-calorie meals containing 15 g of essential amino acids (containing ~ 3 g of leucine) and 30 g of carbohydrate, as compared to just three 850-calorie meals alone.

In summary, it appears as if as few as four and no more than six protein-containing meals could be ingested to stimulate muscle protein synthesis maximally. Using a guideline of four meals fits in nicely with the feeding patterns of Western nations: ingest adequate amounts of protein for breakfast, lunch, and dinner in addition to a high-quality protein postworkout beverage. If five or six protein feedings are adopted (as demonstrated in Paddon-Jones et al. 2005), the athlete would, by necessity, need to be very regimented in feeding schedules in consideration of the refractory response of skeletal muscle to elevated plasma amino acids from prior meals. In order to give some flexibility to the athlete in terms of feeding schedules, ingesting high-quality sources of protein is best achieved with a four or five meals per day strategy.

6.5 SAFETY ISSUES WITH ELEVATED PROTEIN INTAKES

The recommendations for dietary protein intake put forth in this chapter on a daily and per-meal basis are considered high by some, especially when compared to recommended dietary protein intakes (0.8 g/kg/day) of the sedentary population. Because of these relatively higher protein intake recommendations, safety concerns have been raised. Whenever dietary practices are outside the norm, it is appropriate to question potential safety concerns. In fact, if current dietary practices are not ideal—or worse, if they induce harm to the athlete's health—then such practices need to be challenged and alternative recommendations be made. In regard to

protein intake for the athletic population, two general safety issues are raised: One is renal stress and the other is calcium loss/bone catabolism leading to an increased risk for osteoporosis.

Unfortunately, when these safety concerns are raised, there is no substantiated evidence to support the relationship between higher protein intakes and the health-compromising outcomes related to renal stress and calcium loss. In a position stand on protein and exercise, the International Society of Sports Nutrition takes a rather bold stand in stating: "Both of these concerns (renal stress and calcium loss leading to an increased risk for osteoporosis) are unfounded as there is no substantive evidence that protein intakes in the ranges suggested (1.4 to 2.0 g/kg body mass/day) will have adverse effects in healthy, exercising individuals" (Campbell et al. 2007). While this statement appears to be true, it is also important to realize that the absence of evidence of harm does not necessarily mean evidence of absence of harm related to renal stress and calcium losses (Lowery and Devia 2009). Dr. Lonnie Lowery has uniquely framed this debate by stating that, at times, athletes (who seek out additional protein intake) and educators (who commonly advocate more conservative dietary protein intakes) are at odds with each other while there has been a lack of scientific consensus directly supporting the claims of either side (Lowery and Devia 2009). Lowery also highlights the fact that much of the existing data is epidemiological in nature—not cause and effect.

In relation to renal stress, population-based studies, professional observations, and laboratory findings suggest that that renal work is required when additional protein is ingested (Lowery 2012). However, does the additional work that is required of the kidney increase the risk for future renal disease? Many of the health concerns related to higher dietary protein intakes have come from observations on patients with pre-existing diseases, including kidney disease. Indeed, underlying disease may alter the recommendations and outcomes when it comes to ingesting relatively higher amounts of protein (Lowery 2012; Wrone et al. 2003). However, it is unsound scientific practice to take data obtained (via epidemiological methods) in diseased populations and extrapolate the findings to healthy, athletic populations. Similarly, it would be inappropriate to take the strength and conditioning programs of elite-level athletes and recommend them for those in the general population simply because the programs have been found to be effective for the hard training athlete. In reality, few prospective studies have included healthy, athletically trained populations in studies investigating protein intakes and renal health markers.

Two studies involving resistance-trained athletes have investigated the short-term effects of different levels of protein intake on markers of kidney health (Lowery and Devia 2009; Poortmans and Dellalieux 2000; Brändle, Sieberth, and Hautmann 1996). In the first of these studies, four different groups with varying levels of dietary protein intakes were investigated (Brändle et al. 1996). The four groups included bodybuilders supplementing their diets with protein (highest protein intake group), bodybuilders that did not supplement their diets with protein, vegetarians (lowest protein intake group), and other non-resistance-training subjects with no special diet. Each of these groups was on its respective diet for at least 4 months. The protein intakes were highly variable among the groups, ranging from ~0.29 g/kg body mass/day (g/kg/day) to 2.6 g/kg/day. The investigation found no correlation between

albumin excretion rate (elevated urinary albumin levels are a sign of chronic kidney disease) and dietary protein intake.

In the other study, Poortmans and Dellalieux (2000) investigated two types of athletes: body builders and other well-trained athletes (cyclists, judokas, and rowers). The body builders ingested ~1.95 g/kg/day and the other athletes ingested 1.35 g/kg/day for a 1-month period. At the end of the intervention, the authors reported that there were no major fundamental plasma differences for several variables usually associated with protein metabolism and renal impairment. In particular, the glomerular filtration rate assessed by creatinine clearance did not differ between the groups (Poortmans and Dellalieux 2000).

In addition to renal function, there are also some safety concerns that high-protein diets result in the leaching of calcium from bones, which may predispose some individuals to osteoporosis later in life. Indeed, some studies have reported an increase in urine acidity from increased dietary protein intakes (Schuette, Zemel, and Linkswiler 1980). The underlying mechanism was thought to be the acidifying effects of dietary protein on the human body, which required a buffer that came from the leached bone mineral (Lowery 2012). However, studies reporting this effect were limited by small sample sizes, methodological errors, and the use of high doses of purified forms of protein (Ginty 2003). It is now known that the phosphate content of protein foods (and supplements fortified with calcium and phosphorous) negates this effect. Also, researchers (Dawson-Hughes et al. 2007) have reported that only specific (aromatic) amino acids, such as histidine, phenylalanine, tryptophan, and tyrosine, bind with the calcium-sensing receptor to increase urinary calcium excretion (Lowery 2012).

As more data are presented, it appears that bone mineral density is not decreased but rather unchanged or increased in populations that ingest high amounts of dietary protein (Lowery 2012). One other consideration that needs to be made is the effect that physical activity, resistance exercise in particular, has on bone mineral density. Specifically, the mechanical stimulus induced by load-bearing exercise provides a stimulus that is able to increase and preserve bone mass.

There is little scientific evidence that ingesting higher than recommended (0.8 g/kg/day) amounts of dietary protein is detrimental to the health of healthy athletes. Safety concerns related to renal and bone health are not supported by the scientific literature. Of the limited data that are available, they tend to refute the detrimental effects. According to the limited scientific evidence, it appears that athletes ingesting protein at 1.5 to 2.0 g/kg/day are not causing harm to renal and bone health.

6.6 PROTEIN AND EXERCISE PERFORMANCE

Many individuals are conditioned to believe that endurance athletes should focus on carbohydrates and strength athletes should focus on protein in order for each type of athlete to perform well in his or her respective sports/activities. As discussed previously, protein intake reduces markers of muscle damage for an endurance athlete and also maximizes muscle protein synthesis when combined with resistance exercise. This section will focus on whether dietary protein intake is able to enhance

endurance- and resistance-exercise performance. In addition, a comparison of the most popular types of supplemental protein will also be discussed.

6.6.1 Endurance Exercise

Adding protein to a carbohydrate beverage during exhaustive endurance exercise results in the suppression of muscle damage in the days following the exercise bout. In addition, adding protein to a carbohydrate beverage also reduces the endurance athlete's feelings of muscular soreness. This aspect of protein supplementation will be more fully discussed in Chapter 7, "Nutrient Timing: Carbohydrate–Protein Combinations." In this chapter, the extent of the manipulation of dietary protein intakes on enhancing (or decreasing) endurance-related performance will be presented. The studies presented here investigated changes in protein ingestion over a period of time (several days to several weeks) rather than an acute elevation in protein intake in the hours before, during, or immediately after endurance exercise.

When dietary protein intakes are manipulated in an endurance athlete's diet, the changes should not be made without consideration of the carbohydrate content. For example, if carbohydrate intakes are reduced to below recommended levels, in spite of any changes that are made with dietary protein, performance may suffer. Macdermid and colleagues (2006) compared the influence of an isoenergetic, high-protein/moderate-carbohydrate diet with a diet that was based on guidelines for endurance athletes—meaning that the carbohydrate content was within recommended levels. The trained cyclists ingested the diets in a randomized, crossover fashion. The diets were followed for 7 days prior to a time trial and consisted of the following:

- A carbohydrate intake of 7.9 g/kg/day and a protein intake of 1.3 g/kg/day
- A carbohydrate intake of 5.9 g/kg/day and a protein intake of 3.3 g/kg/day

In both 7-day dietary treatments, fat intake and total energy intake were held constant. Prior to and at the end of the dietary intervention, a self-paced cycling endurance time trial was conducted as the primary measure of exercise performance. At the end of the 7-day dietary intervention, it took those on the higher protein diet 20% more time to complete the self-paced time trial, which was significantly longer than for those on the lower protein/higher carbohydrate diet. This study demonstrates that reducing carbohydrate intakes while simultaneously increasing protein results in an endurance-performance decrement. These findings are not surprising given that dietary protein is not a preferred energy source, and the dietary carbohydrate intakes in the higher protein treatment were below recommended intakes for endurance athletes (6–10 g of carbohydrate/kg body mass/day). Also, the length of the dietary period (7 days) may not have been sufficient to enable potential chronic adaptations to take place. A longer intervention period may have led to a greater adaptation to the diet and possibly a better performance. Regardless of these potential confounding factors, it is not advisable for endurance athletes to ingest suboptimal carbohydrate intakes and increase dietary protein intakes to levels that are substantially higher than recommended intakes. To the contrary, endurance athletes should ingest 6 to

10 g of carbohydrate/kg body mass/day and ingest dietary protein in a range of 1.5 to 2 g/kg body mass/day.

In a periodized training program, many endurance athletes will incorporate periods of high training volume, combined with limited recovery time, in an attempt to improve endurance performance. As the training volume increases, there is often a reduction in the time devoted to recovery, and this often results in an acute impairment in exercise capacity or performance (Halson et al. 2002; Jeukendrup et al. 1992). Sports scientists refer to this increase in training volume, which may lead to short-term reductions in performance, as "overreaching."

Researchers from England investigated the effects of additional protein intake during a short-term period of intensified training on subsequent endurance performance in highly trained male cyclists (Witard et al. 2011). Each cyclist completed two trials, both consisting of a 3-week training program divided into 1 week of normal training, 1 week of intensified training, and 1 week of recovery training. The primary measure of endurance performance was a simulated time trial in which the cyclists were required to complete a set amount of work as fast as possible. The time trial was conducted at the end of each of the 3 weeks and was placed after a 2-h submaximal exercise bout. The expectation was that performance would be compromised in the third week—the week after the intensified training period.

Energy intake was the same for both treatments (high protein and normal protein) throughout the 3-week mesocycle, with total energy intake increasing during the intensified training (week 2). Both protein and carbohydrate intakes were manipulated during the study, with the remainder of energy derived from fat. The diets for the high- and normal-protein treatments for the 3-week mesocycle were as follows:

- Week 1 (normal training): 6 g of carbohydrate/kg body mass; 1.5 g protein/kg body mass, remainder of energy came from fat; both groups ingested this diet during week one.
- Week 2 (intensified training): Protein intake was doubled to 3 g/kg body mass for the high-protein group and remained at 1.5 g/kg body mass in the normal-protein group. Carbohydrate was held constant at 6 g/kg body mass for both treatments.
- Week 3 (week of recovery): Protein intake remained doubled at 3 g/kg body mass for the high-protein group and remained at 1.5 g/kg body mass in the normal-protein group. Carbohydrate was held constant at 6 g/kg body mass for both treatments.

At the end of the intensified training and week of recovery, there were no significant differences in the time to complete the simulated time trial between the high-protein and normal-protein treatments (Witard et al. 2011). While no performance benefits were observed, the elevated protein intakes did not result in a decrease in endurance performance. It is important to note that carbohydrate intakes (6 g/kg/day) were kept within (but at the lower end of) the general recommended levels of 6 to 10 g/kg body mass/day. During periods of increased training volume, carbohydrate intakes should be increased to 8 to 10 g/kg body mass/day. The fact that carbohydrate intakes were kept at 6 g/kg/day could have been a limitation of this study. Also, it is

important to note that the lower protein intake treatment (1.5 g/kg body mass) was also within the general recommended levels of 1.5 to 2 g/kg body mass/day.

Very few studies have investigated the effects of prolonged periods of dietary protein manipulation and its effects on endurance performance. Based on the few studies reviewed here, it appears as if increasing protein intakes above recommended intakes does not enhance or decrease endurance performance.

6.6.1.1 Central Fatigue Hypothesis

To this point, we have looked at the effects of intact proteins and their effects on endurance performance. Some research has also investigated the effects of individual amino acids or the combination of several amino acids on exercise performance. One of the areas that have been investigated in relation to endurance performance has been the effects of branched-chain amino acids and their potential to delay fatigue. The branched-chain amino acids (BCAAs) consist of leucine, isoleucine, and valine. These three amino acids cannot be synthesized in the body and therefore are classified as essential amino acids and must be obtained from the diet.

One of the most interesting things about the BCAAs is their ability to be oxidized for energy in skeletal muscle—a characteristic that makes them unique among the other amino acids. The theoretical rationale for how the BCAAs may improve endurance performance stems from their potential to suppress central fatigue. Fatigue is defined as an inability to maintain a power output or force during repeated muscle contractions (Gibson and Edwards 1985). Fatigue is often described as having two contributing sources: central and peripheral fatigue. Peripheral fatigue is centralized to the contracting skeletal muscle and has several contributing factors, such as disturbances in excitation–contraction cycles and the availability of metabolic substrates (ATP [adenosine triphosphate], glycogen, etc.). Most often, peripheral fatigue is associated with decreased content of energy substrates, primarily glycogen, during exercise (Wildman 2004).

Unlike peripheral fatigue, central fatigue involves the central nervous system and encompasses neural impulses from the brain to the skeletal musculature involved in the transmission of the electrical impulse. In regard to neurotransmitters, the "central fatigue hypothesis" suggests that increased concentrations of brain serotonin can impair central nervous system function during prolonged exercise, resulting in fatigue and a subsequent decrement in performance. An increase in serotonin synthesis occurs when there are elevated levels of the amino acid tryptophan, which is an amino acid precursor to serotonin.

Studies in both rodent models and humans provide good evidence that brain serotonin activity increases during prolonged exercise and that this response is associated with fatigue (Davis, Alderson, and Welsh 2000). Therefore, if tryptophan levels are inhibited from crossing the blood–brain barrier, then theoretically serotonin production will be reduced and consequently fatigue will be delayed. Interestingly, tryptophan shares the blood–brain barrier transport mechanism with other large neutral amino acids, most notably the BCAAs (leucine, isoleucine, and valine). If BCAA blood concentrations are high, they compete with tryptophan for the blood–brain transport mechanism and essentially lower the transportation of tryptophan into the brain. In contrast, if BCAA blood concentrations are low, then tryptophan entry into

the brain increases (Kreider and Leutholtz 2001). Since BCAAs are oxidized for energy by the active skeletal musculature during exercise, blood levels of BCAAs decrease during endurance exercise, which allows tryptophan to cross the blood–brain barrier with limited competition.

Theoretically, if BCAAs were ingested before or during endurance exercise, it would result in an increase in plasma BCAAs and subsequently compete with tryptophan to cross the blood–brain barrier and result in a delaying of fatigue that occurs during endurance exercise. Specifically, oral ingestion of BCAA has been shown to elevate blood BCAA concentrations, which limits the amount of tryptophan that is able to cross the blood–brain barrier (Yamamoto and Newsholme 2000). While the idea of supplementing with BCAAs prior to and during endurance exercise seems good, the practice has not manifested in improved endurance performance.

To test the effects of BCAA ingestion and exercise performance, Blomstrand et al. (1991) gave experienced runners 16 g of BCAAs during a marathon (42.2 km) and monitored the effects on physical performance. When the runners were divided into two subsets—a "slow" group and a "fast" group—BCAA ingestion was found to result in a significant improvement in marathon time to completion for the "slow" group. However, overall marathon performance for the entire athlete sample was not affected by BCAA supplementation. Two other studies have also confirmed that BCAA ingestion did not improve endurance performance in endurance-trained males (Watson, Shirreffs, and Maughan 2004; van Hall et al. 1995). While theoretical rationale exists for BCAA supplementation to improve endurance performance, the available evidence consistently reports no endurance-performance enhancement.

6.6.2 Resistance Exercise

Resistance exercise is now a very common mode of activity for athletes of nearly all sports. Most athletes engage in resistance exercise for three main reasons:

1. To increase joint structure integrity (for the prevention of injury)
2. To increase maximal strength (for performance/power enhancement)
3. To increase lean muscle mass

In this section, as dietary protein is discussed in conjunction with resistance exercise, the focus will center on increasing lean muscle mass and strength development.

6.6.2.1 Protein and Net Muscle Protein Balance

As stated in the previous chapter, net muscle protein balance is equal to muscle protein synthesis minus muscle protein breakdown. For skeletal muscle hypertrophy to occur, net muscle protein balance must be positive: Synthesis must exceed breakdown. When net muscle protein balance is positive, it denotes an anabolic environment. To improve net muscle protein balance, an appropriate stimulus (such as resistance exercise) must be applied to the skeletal muscles. However, when resistance exercise is performed alone, in the absence of protein intake, net muscle protein balance is not improved to the point of becoming anabolic.

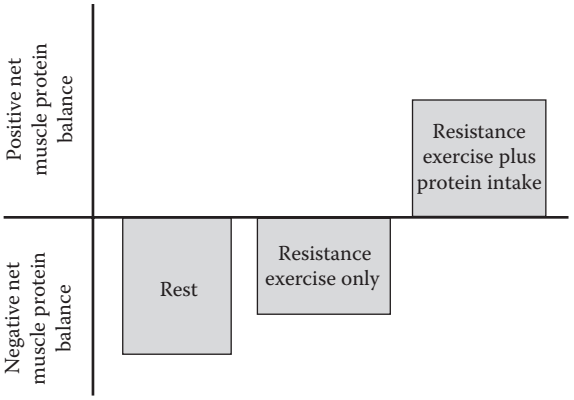

FIGURE 6.2 Net muscle protein balance.

As evidence for this observation, researchers assessed rates of protein synthesis and breakdown at rest and 3 h after a resistance-exercise bout in fasted participants (Biolo et al. 1995). At 3 h after exercise, protein synthesis had increased approximately 108% and protein breakdown increased 51%. Hence, resistance exercise improved net muscle protein balance by increasing protein synthesis at a greater rate than protein breakdown. However, even though net muscle protein balance was improved, it did not improve to the point of becoming positive. If protein is ingested along with a bout of resistance exercise, it results in a positive net muscle protein balance. Figure 6.2 demonstrates the effect that resistance exercise alone and resistance exercise plus protein intake have on net muscle protein balance.

6.6.2.2 Comparison of High-Quality Proteins

The past 15 years have witnessed a large amount of scientific investigation in relation to protein supplementation in conjunction with resistance exercise and their combined effects on muscle protein synthesis, skeletal muscle hypertrophy, and maximal strength. While there is a rather large difference between high- and low-quality proteins, even the various high-quality proteins are somewhat different in their digestive and metabolic characteristics. The following section will compare and contrast some of the high-quality proteins and their effects on muscle protein synthesis, lean muscle mass, and strength in conjunction with resistance exercise or a resistance-training program.

6.6.2.2.1 Skeletal Muscle Protein Synthesis

Several different types of protein are able to stimulate muscle protein synthesis, including whey, casein, soy, egg albumin, and beef proteins (Pennings et al. 2011; Moore et al. 2009; Symons et al. 2009; Wilkinson et al. 2007). Of these various protein sources, the three most popular commercially available protein supplements are whey, casein, and soy. It is no surprise, then, that these three proteins have been compared against one another in scientific literature in relation to their ability to stimulate muscle protein synthesis, lean body mass, and maximal strength. Based on leucine content alone, whey protein is superior as compared to both casein and

soy proteins in terms of stimulating muscle protein synthesis. In addition to the differences in leucine and essential amino acid content, one of the major differences in these three protein sources is their digestibility. Whey protein is associated with a very rapid, large, but transient increase in amino acid availability, while casein coagulates and precipitates when exposed to the stomach acid and, as a result, it is slowly released from the stomach during digestion (Churchward-Venne, Burd, and Phillips 2012). Soy protein is similar to whey protein in that it is considered a "fast protein" due to the relative speed with which its amino acids are found in the plasma following soy protein ingestion.

Tang and colleagues (2009) compared the effects of whey, casein, and soy protein on their ability to maximally stimulate mixed muscle protein synthesis. In this study, resistance-trained men conducted four sets of a unilateral leg press followed by four sets of unilateral leg extensions. Each set was conducted at a 10–12 RM (repetition maximum) intensity with a 2-min rest period between each set. Following the exercise bout, subjects ingested approximately 22 g of whey protein, casein protein, or soy protein. Mixed muscle protein synthesis was measured for 3 h after the exercise and protein ingestion. Since the exercise bout was conducted with only one leg, the other leg served as a nonexercise control, which was a very intuitive method that allowed for the determination of which protein stimulated mixed muscle protein synthesis to the greatest extent both in the rested and postexercise conditions.

In the rested condition, whey and soy protein elevated mixed muscle protein synthesis significantly more than did the casein treatment group. Whey was also superior to soy protein in the rested condition (in relation to stimulating muscle protein synthesis), but this difference did not reach the level of statistical significance. After resistance exercise, whey protein ingestion significantly increased mixed muscle protein synthesis to a greater extent than did either soy or casein ingestion. Soy protein, while inferior to whey protein, was significantly better as compared to casein protein. In summary, whey was superior to soy and casein, and soy was superior to casein, in relation to elevating rates of mixed muscle protein synthesis following resistance exercise. Other investigations have reported that milk proteins (a combination of whey [~20%] and casein [80%]) were superior in elevating muscle protein synthesis compared with soy protein when ingested after resistance exercise (Wilkinson et al. 2007).

In contrast to the Tang et al. (2009) study reporting that whey protein was superior to casein in relation to stimulating muscle protein synthesis in the 3 h following an acute bout of lower body resistance exercise, not all investigations have reported such observations (Tipton et al. 2004). Because the time period in which protein synthesis was measured in the Tang et al. study was only 3 h after exercise and because whey protein is a "fast" digesting protein and casein is a "slow" digesting protein (Dangin et al. 2001), the results indicating that whey protein was superior to casein may not be surprising since more of the amino acids (especially leucine) would have been made available to the skeletal muscle from the whey protein as compared to the casein protein. Given that the amino acids are released at a slower rate from the stomach after casein ingestion, what would be the effects on protein synthesis if a longer time period were studied after both whey and casein ingestion? Is it possible

that casein would have a similar effect on muscle protein synthesis to that of whey protein if the time period for measurement were increased?

Researchers from Denmark were able to answer these questions in a group of moderately active males (Reitelseder et al. 2011). The subjects completed 10 sets of eight unilateral repetitions of leg extensions at an intensity of 80% 1 RM. Immediately after the exercise bout, the subjects ingested approximately 20 g of whey protein, casein protein, or a placebo. Rates of myofibrillar protein synthesis were measured for a 6-h period after the exercise and protein ingestion occurred. Rates of muscle protein synthesis were reported in three different time periods: 1–3.5 h, 3.5–6 h, and the total 6 h time period. The rates of muscle protein synthesis resulting from whey and casein ingestion for each time period were the following:

- 1–3.5 h: Whey protein increased muscle protein synthesis rates ~25% more than casein (difference was not significant).
- 3.5–6 h: Casein protein increased muscle protein synthesis rates ~48% more than whey (difference was not significant).
- 6 h after exercise period: No difference in rates of muscle protein synthesis were observed between the whey and casein groups (although both groups were significantly better than a control group).

The findings from this study were interesting in that if only the first 3 h of protein metabolism are considered, the whey protein appears to be superior to the casein protein, which is in agreement with the findings reported by Tang and colleagues (2009). However, when the postexercise period was increased to 6 h, the slower digesting casein protein ingestion seemed to "catch up" to the whey protein such that there were no differences in overall muscle protein synthesis rates between them. While measures of muscle protein synthesis are important and can assist in helping the athlete make decisions on a meal-to-meal basis, the most valuable information comes from those studies that were conducted over several weeks or months and that measured actual changes in lean body mass and muscular strength.

6.6.2.2.2 Strength and Lean Muscle Mass

When comparing high-quality protein sources against one another, it is important to realize that these proteins are classified as high quality for a reason: They all have adequate amounts of the essential amino acids. However, as has been repeatedly stated throughout this and the prior chapter, the leucine content of whey protein (particularly the isolate form) is higher as compared to the other forms of high-quality proteins. This characteristic of whey protein arguably makes it the best choice for increasing muscle protein synthesis in the few hours (~3) following resistance exercise. In terms of chronic adaptations for which athletes are striving, such as increased strength and muscle mass, which high-quality protein source is best for the athlete? Fortunately, several studies have compared several high-quality sources of protein (whey, casein, and soy) ingestion in conjunction with a resistance-training program. In all but one finding (in a nonathletic population), whey protein was either better than or the same as casein or soy in terms of increasing muscular strength and lean body mass over a period of several weeks or months.

Recreational bodybuilders participated in a 10-week whole-body periodized and progressive resistance-training program (Cribb et al. 2006). Prior to and following the training program, maximal strength in the bench press and squat were conducted in addition to body composition measures (fat mass and lean muscle mass). Throughout the 10-week study, the participants supplemented their diets with either whey (hydrolyzed whey isolate) or casein at a dose of 1.5 g/kg body mass/day. In addition to this supplemental protein, the subjects also ingested protein from meals on a daily basis. Total daily protein intake was about 2.1 g/kg body mass/day for each of the two groups during the 10-week study.

While maximal strength significantly increased in both groups, the authors reported that maximal strength increases were significantly greater in the whey protein group for both exercises assessed compared to the casein group. Specifically, squat strength increased by ~94% in the whey group and 74% in the casein group. Bench press strength increased by 57% in the whey group and 21% in the casein group. In terms of body composition, there was a significant increase in lean body mass (~7%) and a significant decrease in body fat (10%) for the whey group. In contrast, there was no significant change in lean body mass or body fat over the training period for the casein group. This study clearly demonstrated the superiority of whey protein over casein protein.

In contrast, another study reported that casein was superior to whey protein in a group of police officers following a hypocaloric diet for 12 weeks (Demling and DeSanti 2000). In this study, the police officers were randomly placed into one of the following three diet groups (with each diet reducing total daily caloric intake 20% below energy balance levels):

- Diet alone without resistance exercise
- Diet and resistance exercise while supplementing with 1.5 g/kg/day using a casein protein hydrolysate
- Diet and resistance exercise while supplementing with 1.5 g/kg/day using a whey protein hydrolysate

Total weight loss was similar (about 5 lb.) in all three groups. With diet alone, body fat declined about 2.5%, while the whey protein group lost 4% body fat. The casein group lost significantly more (7%) body fat than the other two groups. Also, the casein group increased lean mass by 8.8 lb. as compared to a 4.4-lb. increase in the whey group. There were no changes in lean body mass for the diet-alone group. Lastly, the casein group increased muscle strength (in the chest, shoulders, and legs) to a significantly greater degree than the whey hydrolysate group did. Specifically, the maximum improvements in strength were 59% for the casein group versus 28% for the whey protein group.

This study (Demling and DeSanti 2000) was the only investigation that compared two high-quality proteins during a period of reduced energy intake. While the Cribb et al. (2006) study reported that whey protein was superior to casein in nondieting males, this study observed an advantage for casein when energy intake was below weight maintenance values. In situations in which catabolism is more likely to be elevated (during intense training or when following a hypocaloric diet), casein may be the preferred protein source to attenuate decreases in muscle protein breakdown

(Boirie et al. 1997) and lean body mass (Demling and DeSanti 2000). In this sense, perhaps a periodized approach should be taken in relation to the primary source of protein so that whey protein is prioritized during periods of noncaloric reductions and casein protein is prioritized during times of induced weight loss for the athlete.

It was stated previously that whey protein is superior to soy protein in its ability to maximize muscle protein synthesis in the hours following resistance exercise. This finding would lead one to believe that when whey and soy are compared against one another during a period of several weeks in which a resistance-training program was prescribed, the whey-supplemented groups would improve measures of lean body mass and strength to greater degrees than the soy-supplemented protein groups. Surprisingly, this has not been reported in three separate studies in which these two high-quality proteins have been compared (Kalman et al. 2007; Candow et al. 2006; Brown et al. 2004). In each of these studies, the subjects ranged from healthy, non-resistance-trained males and females to resistance-trained males with at least 1 year of resistance-training experience. The length of the studies ranged from 6 to 12 weeks in duration. In each investigation, soy protein was just as effective as whey protein in inducing favorable changes in lean body mass in conjunction with a resistance-training program.

One of the primary reasons that male athletes wish to avoid soy products is because of the isoflavones associated with soybeans. Isoflavones (i.e., phytoestrogens) are found abundantly in soybeans and soy products and therefore are often referred to as soy isoflavones. Soy isoflavones have the ability to bind to estrogen receptors, mimicking the effects of estrogen in some tissues and antagonizing (blocking) the effects of estrogen in others (Wang 2002). It is the potential agonist activity (exerting estrogen-like effects) of soy isoflavones that male athletes would want to avoid (Campbell et al. 2007). It should be stated that no research exists to document negative outcomes in males ingesting soy protein in relation to training adaptations. However, it makes sense for athletes who are not comfortable ingesting soy protein due to its isoflavone content to use whey and casein protein, because these are high-quality sources of protein with demonstrated effectiveness in improving body composition and strength when combined with a proper resistance-training program.

6.7 CONCLUSION

Athletes of all types engage in sport-specific and resistance training in order to maximize their performance. The process of training can be broken down into two simple processes: the process of stimulation and the process of adaptation. This chapter discussed the role that dietary protein has on the process of adaptation. In order to maximize the adaptive response to the stimulus provided by the training, it is recommended that athletes ingest approximately 1.5 to 2.0 g of protein/kg body mass/day. Ingesting this amount of protein in approximately four to five meals per day allows the athlete to adapt to the stimulus imparted by his or her training program. Also, by ingesting protein after training, the endurance athlete is able to suppress both muscle damage and muscle soreness and the resistance-training athlete is able to maximize rates of protein synthesis. Ingesting optimal amounts of dietary protein is not the

only consideration for the athlete. A consideration of the source of the dietary protein must also be made, with an emphasis on ingesting high-quality protein sources such as whey, casein, beef, poultry, and fish proteins.

ACKNOWLEDGMENT

A sincere appreciation to Nic Martinez for conducting research on the content related to this chapter.

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7 Nutrient Timing
Carbohydrate–Protein Combinations

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CONTENTS

7.1 Introduction 181
7.2 Does Nutrient Timing Work? 182
7.3 Endurance Performance Considerations 184
7.3.1 Suppression of Muscle Damage 184
7.3.2 Suppression of Muscle Soreness 189
7.3.3 Skeletal Muscle Glycogen Resynthesis 190
7.4 Resistance-Training Performance Considerations 192
7.4.1 Carbohydrate–Protein Ingestion Prior to a Single Bout of Resistance Exercise 192
7.4.2 Chronic Adaptations during and after Exercise Carbohydrate–Protein Ingestion 193
7.5 Conclusion 195
References 195

7.1 INTRODUCTION

Throughout this book, we have looked at how carbohydrates, protein, and fat can be individually consumed to provide improvements in performance. Over the past 15 years, there has been an emphasis in the scientific literature regarding how nutrients can be combined at specific times to maximize training adaptations leading to improvements in exercise and sports performance. The concept of combining nutrients and ingesting them at specific times has primarily concentrated on carbohydrate (CHO)–protein (PRO) combinations and is referred to as "nutrient timing." Simply stated, nutrient timing is focused on "when to eat" rather than solely on "what to eat." Nutrient timing has been studied under various modes of exercise, including running (Tsintzas and Williams 1998; Tsintzas et al. 1996), cycling (Jentjens et al. 2003; McConell et al. 1999), and resistance training (Esmarck et al. 2001; Tipton et al. 2001). A consistent pattern with nutrient timing is the ingestion of nutrients before, during, or immediately after a bout of exercise. Most of the data and knowledge that

we have concerning nutrient timing is related to what is ingested in the postworkout period.

Nearly all of the nutrient timing research is centered on carbohydrate–protein combinations, with the obvious exclusion of fat. There may be several reasons for this, including the fact that fat stores are not a limiting factor in terms of energy production (as carbohydrates are). Also, the primary role of protein is to elicit adaptations arising from the stimulus provided by the training. Dietary fat does not appear to have much of a role in this regard. It has often been suggested that adding fat to a postworkout recovery beverage should be avoided due to its potential to slow down the digestion and absorption of ingested carbohydrate, which would subsequently suppress the rate of skeletal muscle glycogen resynthesis.

Three different investigations have refuted this belief. The first of these studies reported that adding fat to the postworkout carbohydrate–protein beverage does not negatively alter the rate of skeletal muscle glycogen resynthesis following resistance exercise (Roy and Tarnopolsky 1998). Likewise, when subjects were given a postendurance workout beverage containing carbohydrate, protein, and fat (even up to 45% of the calories being derived from fat), neither muscle glycogen resynthesis rates nor glucose tolerance was altered (Fox, Kaufman, and Horowitz 2004). Finally, Burke and colleagues (1995) demonstrated that the addition of fat and protein to a postworkout carbohydrate beverage does not alter glycogen storage over 24 h, provided that carbohydrate intake is adequate. The following sections will investigate the validity of nutrient timing practices with a focus on carbohydrate–protein combinations. Also, the potential role that nutrient timing may have on improving endurance- and resistance-exercise performance and adaptations will be examined.

7.2 DOES NUTRIENT TIMING WORK?

The basis of nutrient timing involves the consumption of specific nutrients (primarily carbohydrate and protein) in and around an exercise session. Despite the fact that nutrient timing has been the focus of numerous research studies over the past 15 years, there is still some debate on the effectiveness of following nutrient timing principles. In one of the well-designed studies on this topic (Hoffman et al. 2009), it was reported that the timing of specific nutrients did not have an effect on training adaptations of resistance-trained males (American football players and power lifters). The athletes in this study were randomly assigned to one of three groups:

- The nutrient timing group received a protein supplement immediately before and immediately after workouts.
- The morning–evening group received a protein supplement in the morning and evening.
- The control group did not ingest a protein supplement.

All athletes participated in a 4-day/week supervised resistance-training program for a 10-week period. The protein supplement contained 42 g of protein (which contained 3.6 g of leucine, 3 g of isoleucine, and 1.4 g of valine) and 2 g of carbohydrate. Prior to and after 10 weeks of training, body composition, maximal strength (in

the bench press and squat), and upper and lower body power were assessed. At the end of the intervention, it was reported that there were no differences between the three groups in body composition (lean body mass, fat mass, and body fat percentage). In terms of muscular strength, maximal squat strength increased significantly for all three groups, but only the two protein supplement groups (nutrient timing group and the morning–evening group) realized significant increases in bench press strength. There were no differences between the three groups for any strength or power measures. These reports indicate that the timing of the protein supplement does not provide any added benefit to strength, power, or body composition changes in resistance-trained participants.

One possible explanation to the findings in this study was the fact that the morning–evening group significantly increased its dietary protein intake during the 10-week study intervention, and this was the only group to increase protein intake significantly above baseline levels. Such increases in dietary protein for this group may have partly explained the improvements in muscular strength, which could have made the morning–evening group more similar to the nutrient timing group and subsequently eliminated any differences that may have been observed if the protein intakes were kept constant between the groups. The increases in dietary protein intake notwithstanding, this study demonstrates that adhering to nutrient timing principles does not result in improvements in body composition and strength/power performance (Hoffman et al. 2009).

In contrast to this finding, an Australian study reported how important the actual timing of ingested nutrients is relative to strength and skeletal muscle hypertrophy (Cribb and Hayes 2006). The scientists who conducted this study sought to examine the effects of supplement timing on muscle-fiber hypertrophy, strength, and body composition during a 10-week resistance-exercise program. There were two groups of resistance-trained males in this study. Both groups conducted the same resistance-training workout for 10 weeks and both groups ingested the same supplement two times per day (a protein/creatine/glucose supplement). There was one major difference between these two groups, however: the timing in which the supplement was taken. One group took the protein/creatine/glucose supplement immediately before and after each resistance-training workout, while the other group took the exact same protein/creatine/glucose supplement in the morning and late evening. Before and after the 10-week study, the investigators assessed maximal strength (in the squat, bench press, and dead-lift), body composition, and muscle fiber type cross-sectional area (as a specific measure of skeletal muscle hypertrophy). The group that took the anabolic supplement immediately before and after working out demonstrated significantly greater increases in lean body mass and maximal strength (in the squat and bench press). Also, the changes in body composition were supported by a significantly greater increase in the cross-sectional area of the type II fibers (the fibers that have the greatest potential for hypertrophy). It is important to remember that both groups performed exactly the same workout and took the exact same supplements; the only difference was the timing of taking the supplement (Cribb and Hayes 2006). This study demonstrates the importance of nutrient timing and the effects that it can have on body composition, maximal strength, and skeletal muscle hypertrophy.

In the preceding paragraphs, two scientific publications give two different reports regarding the importance of nutrient timing. In the first study, nutrient timing appeared to have no effect on the training adaptations in resistance-trained males. In the second study, nutrient timing principles appeared to improve the training adaptations experienced by resistance-trained males significantly. Taking both of these publications into perspective and given the fact that nutrient timing may enhance training adaptations (Cribb and Hayes 2006) and does not appear to decrease the training adaptations (Hoffman et al. 2009), it is recommended that athletes ingest a carbohydrate–protein beverage/food source in the time period following their exercise and training bouts. This postworkout "meal" should count as one of the four to five meals that athletes should be ingesting on a daily basis. (For more information about meal frequency, refer to Chapter 9, "Enhancing Body Composition: Gaining Muscle and Losing Fat.")

7.3 ENDURANCE PERFORMANCE CONSIDERATIONS

Adding protein to a carbohydrate beverage prior to or during endurance exercise has been studied consistently over the past decade. While the majority of the studies conducted on this topic have reported no improvement in endurance-exercise performance, a few have reported that the addition of protein to a carbohydrate beverage that is ingested prior to and during an endurance event does result in a significant improvement in endurance performance. Tables 7.1 and 7.2 summarize the studies that have investigated the addition of protein to a carbohydrate beverage during endurance exercise.

There may be several reasons that explain why some investigations have reported that a carbohydrate–protein treatment improves endurance-exercise performance while others have not observed such an ergogenic effect. It is interesting to note that in the three investigations that demonstrated a performance-enhancing effect of carbohydrate–protein intakes, each utilized an inferior time-to-exhaustion test rather than a time trial. Exercise to exhaustion does not mimic the manner in which athletes typically compete (i.e., a race in which a fixed distance is performed as quickly as possible). When the research designs utilized time trials (Breen, Tipton, and Jeukendrup 2010; Saunders et al. 2009; van Essen and Gibala 2006), there was no performance benefit resulting from the carbohydrate–protein treatments.

7.3.1 Suppression of Muscle Damage

Even though carbohydrate–protein treatments may offer no advantage when time trials are used as the performance measure, there is no evidence to suggest that carbohydrate–protein combinations decrease endurance-exercise performance. In fact, there may be other benefits that these combinations provide that are valuable besides any potential performance improvements. Several studies have investigated the effects of carbohydrate–protein ingestion on muscle damage and muscle soreness following endurance exercise. Nearly every investigation has reported that carbohydrate–protein ingestion improves (reduces) both muscle damage and feelings of muscle soreness in the hours and days following endurance exercise.

TABLE 7.1
Summary of Studies in Which a Carbohydrate–Protein Supplement Does Not Improve Endurance Performance

Study	Population	Design and Diet	Performance Measurements	Findings
Breen et al. (2010)	12 Trained male cyclists	Randomized, double-blind crossover design; 2-h steady-state (55% VO_2max) cycling bout followed by a time trial lasting ~1 h; cyclists ingested one of the following treatments prior to and every 15 min during the steady-state bout: • CHO = ~60.25 min • CHP–PRO beverage = 65 g CHO/h + 19 g PRO/h	Time trial performance	No significant difference in the time to complete the time trial: • CHO = ~60.25 min • CHO–PRO = ~60.8 min
Saunders et al. (2009)	13 Recreationally competitive male cyclists	Randomized, double-blind crossover design; 2 sets of computer-simulated 60-km time trial separated by 7–10 days; cyclists ingested one of the following beverages (same CHO, but added PRO) every 5 km: • CHO beverage = ~60 g/h • CHO–PRO = ~60 g CHO/h + 14.3 g PRO/h	Time to complete 60-km time trial	No significant differences in time to complete 60-km time trial: • CHO = 135 min • CHP–PRO = 134.4 min • (CHO–PRO beverage was superior in the last 20 kg)
Valentine et al. (2008)	11 Recreationally trained male cyclists	Double-blind crossover design; four sets of rides to exhaustion on a cycle ergometer at 75% VO_2peak; in addition to a placebo, cyclists ingested three beverages separated by 5–10 days: • CHO = 77.5 g of CHO/h • CHO–PRO = 77.5 g CHO/h; 19.4 g PRO/h • CHO–CHO = 96.9 g CHO/h; isocaloric with CHO–PRO	Cycling time to exhaustion	No significant difference in time to exhaustion in the CHO–PRO (~126 min) and CHO–CHO group (~121 min); both CHO–PRO and CHO–CHO were superior to CHO and placebo treatments

(continued)

TABLE 7.1 (CONTINUED)
Summary of Studies in Which a Carbohydrate–Protein Supplement Does Not Improve Endurance Performance

Study	Population	Design and Diet	Performance Measurements	Findings
van Essen and Gibala (2006)	10 Trained male cyclists	Randomized, double-blind, placebo-controlled, crossover design; three sets of an 80-km cycling time trial performance separated by 7 days; cyclists ingested fluids every 15 min and total ingestion was approximately: • CHO = 60 g/h • CHO–PRO = 60 g of CHO/h + 20 g PRO/h	Time to complete 80-km time trial	No significant differences between CHO (135 min) and CHO–PRO (135 min) treatments; both treatments were significantly better than a placebo treatment (141 min)
Romano-Ely et al. (2006)	14 Male cyclists	Randomized, double-blind crossover design; two sets of rides to exhaustion on a cycle ergometer; the first and second rides were at 70% and 80% VO_2peak, respectively, and separated by about 22 h; cyclists ingested one of the following two nearly isocaloric beverages every hour during the exercise bout: • CHO beverage = ~60 g/h • CHO–PRO = ~45 g CHO/h + 11.25 g PRO/h	Cycling time to exhaustion	No significant difference in time to fatigue: • CHO = ~138 min • CHO–PRO = ~141 min

Notes: CHO = carbohydrate; PRO = protein.

TABLE 7.2
Carbohydrate–Protein Supplements Demonstrating Improvements in Endurance Performance

Study	Population	Design and Diet	Performance Measurement	Findings
Saunders et al. (2007)	13 Recreationally trained male and female cyclists	Randomized, double-blind crossover design; cyclists completed two timed cycle trials to volitional exhaustion at 75% VO_2peak; cyclists ingested one of the following treatments resulting in: • CHO gel = ~41 g CHO/h • CHO–PRO gel = ~41 g CHO/h + ~10 g PRO/h	Cycling time to exhaustion at 75% VO_2peak	CHO–PRO gel treatment rode significantly longer (13% longer vs. the CHO gel treatment: • CHO = ~103 min • CHO–PRO = ~117 min
Saunders et al. (2004)	15 Trained male cyclists	Randomized, double-blind crossover design; cyclists exercised at 75% VO_2peak to volitional exhaustion, followed 12–15 h later by a second ride to exhaustion at 85% VO_2peak; cyclists ingested 53 g CHO and 53 g CHO + 13 g PRO after the first exercise bout; the following was ingested during the cycling bouts: • CHO = ~39 g CHO/h • CHO–PRO = ~ 39 g CHO/h + ~10 g PRO/h	Cycling time to exhaustion at 75% VO_2peak followed 12–15 h later by a ride to exhaustion at 85% VO_2peak	CHO–PRO treatment rode significantly longer (29% and 40% longer) than the CHO treatment at 75% and 85% VO_2peak, respectively: • CHO = ~82 and 31 min • CHO–PRO = ~106 and 44 min
Ivy et al. (2003)	9 Trained male cyclists	Randomized, double-blind crossover design; 3-h cycling exercise that alternated between 45% and 75% VO_2max followed by cycling at 85% VO_2max until fatigue; cyclists ingested one of the following treatments prior to and every 20 min during the 3-h bout, which resulted in: • CHO beverage = ~55 g/h • CHP–PRO = ~55 g CHO/h + ~14 g PRO/h	Cycling time to exhaustion at 85% VO_2max	CHO–PRO significantly improved time to exhaustion as compared to CHO only (~36% longer): • CHO = ~20 min • CHO–PRO = ~27 min

Notes: CHO = carbohydrate; PRO = protein.

Muscle damage can be indirectly assessed using plasma creatine kinase and lactate dehydrogenase concentrations. Of these two markers, creatine kinase is the most common marker measured to estimate the extent to which skeletal muscle is damaged following exercise. Saunders, Kane, and Todd (2004) reported that plasma creatine kinase levels were significantly lower 12–15 h after a cycle time-to-exhaustion performance test at 75% VO_2peak when a carbohydrate–protein supplement was ingested as compared to carbohydrate alone. During the cycling time-to-exhaustion test, which took about 100 min to complete, the male cyclists consumed 39 g of carbohydrate and 10 g whey protein (0.14 g protein/kg body mass)/h. Immediately after the endurance exercise bout, the cyclists ingested an additional 53 g carbohydrate and 13 g whey protein (0.18 g protein/kg body mass). This carbohydrate–protein combination resulted in creatine kinase levels that were 83% lower as compared to carbohydrate ingestion alone (Saunders et al. 2004).

In a similar study (Saunders, Luden, and Herrick 2007), creatine kinase levels were also suppressed in the time period following endurance exercise when a carbohydrate–protein gel was ingested compared to a carbohydrate-only gel. Male and female cyclists cycled at 75% VO_2peak to exhaustion; the cycling lasted, on average, about 2 h. Every 15 min during the endurance exercise, the cyclists ingested a carbohydrate–protein gel or a carbohydrate-only gel. The amount of nutrients that were ingested during the cycling bout on a per-hour basis was 41 g of carbohydrate plus 10 g of protein (0.14 g of protein/kg body mass/h). The carbohydrate-only gel supplied the same amount of carbohydrate with no added protein. Immediately upon completion of the ride, the cyclists ingested a postexercise feeding of energy gel, which consisted of 50 g of carbohydrates plus 12.7 g of protein (0.18 g protein/kg body mass) or 50 g of carbohydrate alone. As a measure of indirect muscle damage, creatine kinase levels were measured prior to and 12–15 h following the exhaustive endurance exercise bout. Creatine kinase levels significantly increased (indicating more muscle damage) by 46% from pre-exercise to postexercise in the carbohydrate-only treatment, but did not significantly increase in the carbohydrate–protein treatment (an increase of only 23%). Stated another way, the addition of protein to a carbohydrate gel during and after exhaustive endurance exercise suppressed markers of muscle damage by 50%. Interestingly, endurance performance (as measured by cycling to exhaustion) was also significantly improved in each of the studies summarized previously (Saunders et al. 2004, 2007) as a result of adding protein to a carbohydrate supplement.

Other studies that have not reported benefits in endurance performance with the addition of protein to a carbohydrate supplement have nonetheless been consistent in their observation that markers of muscle damage are improved with the addition of protein. Romano-Ely and colleagues (2006) reported that a carbohydrate–whey protein supplement significantly decreased both creatine kinase and lactate dehydrogenase levels 24 h after endurance exercise as compared to a carbohydrate supplement alone. Valentine et al. (2008) also reported that postexercise creatine kinase levels were significantly suppressed when whey protein was added to a carbohydrate supplement during prolonged endurance exercise. Unlike other investigations that used whey protein, Saunders and co-workers (2009) added casein protein hydrolysate to a carbohydrate supplement during and after a 60-km time trial. Results from

this study were in agreement with others in reporting that creatine kinase levels were significantly lower in the carbohydrate–protein supplement as compared to the carbohydrate-only supplement.

The findings of Breen et al. (2010) have been in contrast with others in relation to the effectiveness of adding protein to a carbohydrate supplement and suppressing creatine kinase levels following endurance exercise. In this study, trained male cyclists ingested carbohydrate only or protein plus carbohydrate during a 2-h steady-state cycling ride at 55% VO_2max, followed by a time trial lasting about 1 h. Twenty-four hours later, it was reported that plasma creatine kinase levels increased 97% in the carbohydrate-only treatment and 33% in the protein-plus-carbohydrate treatment. Even though this was a rather large difference between the carbohydrate and carbohydrate–protein treatments in the trained male cyclists, the differences did not reach a level of statistical significance.

7.3.2 Suppression of Muscle Soreness

Another area related to indirect markers of muscle damage is the athlete's subjective feelings of muscular soreness. Most often, the athlete is asked to rate subjectively how sore his or her muscles feel prior to endurance exercise and then again about 24 h after exercise. Typically, a visual analog scale such as a 7-point Likert scale of muscle soreness is given to the athletes at baseline (prior to exercise) and again at one or more points following exercise (such as 24 and 72 h after exercise). A typical scale may range from 0 (no soreness present) to 6 (a severe pain that limits ability to move). Since adding protein to a carbohydrate treatment results in significant reductions in indirect markers of muscle damage, it would not be surprising if endurance athletes also experienced a reduction in muscle soreness resulting from the addition of protein to a carbohydrate treatment during endurance exercise.

Romano-Ely et al. (2006) reported that peak muscle soreness occurred 24 h after a 2.5-h cycling time to exhaustion test and was significantly higher when the athletes ingested only carbohydrate as compared to a carbohydrate–protein treatment. Saunders and colleagues (2009) reported that muscle-soreness ratings increased significantly 24 h after carbohydrate only was ingested during a 60-km time trial but did not increase when protein was added to the carbohydrate. Valentine et al. (2008) and Breen et al. (2010) both reported that postendurance exercise muscle soreness was lower in a carbohydrate–protein treatment as compared to carbohydrate-only treatment, but the difference did not reach the level of statistical significance.

It is clear that adding protein to a carbohydrate beverage or gel during exhaustive endurance exercise results in a significant suppression of muscle damage (as measured by plasma creatine kinase) at time periods ranging from 12 to 24 h after exercise. It also appears as if this strategy reduces the endurance athlete's feelings of muscular soreness as well. In order to realize these benefits, the amount of protein that was added to the carbohydrate treatments ranged from approximately 0.15 to 0.3 g of protein/kg body mass/h of exercise. In one investigation (Valentine et al. 2008), in which 0.26 g of whey protein/kg body mass/h was ingested along with carbohydrate during ~2 h of cycling to exhaustion, it was reported that 22–24 h after exercise, creatine kinase levels were about 8% lower than baseline values. This

was the only investigation to report such findings. Based on these findings, it is recommended that endurance athletes ingest approximately 0.25 g of protein/kg body mass/h of exercise in order to suppress markers of muscle damage and subjective feelings of muscular soreness.

Adding protein to a carbohydrate–fat supplement improves muscular soreness in nonendurance athletes as well (Flakoll et al. 2004). During a 54-day basic training period, US Marine recruits were given one of the following supplements immediately following exercise:

- Noncaloric placebo (0 g carbohydrate, 0 g protein, 0 g fat)
- Control (8 g carbohydrate, 0 g protein, 3 g fat)
- Protein (8 g carbohydrate, 10 g protein, 3 g fat)

The protein-containing supplement significantly improved muscle soreness scores in comparison with the control and placebo groups during the basic training period and on the last day of the training program. In addition, the added protein resulted in an average of 33% fewer total medical visits, 28% fewer medical visits due to bacterial/viral infections, and 37% fewer medical visits due to muscle/joint problems as compared to the non-protein-containing placebo and control groups. Results from this study indicate that adding small amounts of protein to a postexercise carbohydrate–fat supplement improves health and muscle soreness throughout prolonged, intense exercise training (Flakoll et al. 2004).

7.3.3 Skeletal Muscle Glycogen Resynthesis

For most athletes, resynthesizing depleted skeletal muscle glycogen stores does not need to be a priority. For example, recreational athletes who exercise 3 or 4 days per week do not need to devote specific carbohydrate intake strategies in the postworkout period to replenish skeletal muscle glycogen stores rapidly as long as they consume adequate amounts of carbohydrates on a daily basis. However, there are scenarios in which it is important for certain athletes to replenish depleted glycogen stores as rapidly as possible. For example, athletes who train two or three times per day or who train intensely for long periods of time on a daily basis likely benefit from postworkout carbohydrate ingestion for the purpose of quickly replenishing muscle glycogen stores. As was discussed in Chapter 4 (“Dietary Carbohydrate Strategies for Performance Enhancement”), the type of carbohydrate, the timing of the ingestion, and the amount of carbohydrate ingested all have an impact on the rate at which skeletal muscle glycogen is resynthesized following exercise.

In addition to carbohydrate intake, some research has discussed the potential for adding protein to postexercise carbohydrate to increase rates of muscle glycogen resynthesis. Researchers from the University of Texas, under the direction of Dr. John Ivy, were the first group to report the benefit of adding protein to a carbohydrate beverage after exercise (Zawadzki, Yaspelkis, and Ivy 1992). In this study, male cyclists cycled for 2 h on three separate occasions to deplete their muscle glycogen

stores. Immediately after and then again 2 h after the exercise bout, the cyclists ingested one of the following:

- 112 g carbohydrate (providing ~450 cal and 0.75 g CHO/kg/h)
- 41 g protein (providing ~160 cal and 0.3 g protein/kg/h)
- 112 of carbohydrate plus 41 g protein (providing ~600 cal and 0.75 g CHO/kg/h plus 0.3 g protein/kg/h)

Skeletal muscle glycogen levels were measured (via muscle biopsies) immediately after exercise (in the glycogen-depleted state) and 4 h after exercise (to determine the extent to which skeletal muscle glycogen levels were resynthesized). Both the carbohydrate and carbohydrate-plus-protein treatments produced significantly faster rates of glycogen storage compared with the protein-only treatment. While the total muscle glycogen concentrations did not differ between the carbohydrate and the carbohydrate-plus-protein treatments in the 4-h recovery period, there was a significant difference in the rates of glycogen storage that occurred. Specifically, the rate of glycogen storage was 38% (a significant amount) faster during the carbohydrate–protein treatment as compared to the carbohydrate-only treatment. The mechanism that was likely responsible for the observed increase was the insulin response. The plasma insulin response of the carbohydrate–protein treatment was significantly greater than that of the carbohydrate-only treatment. The authors concluded that postexercise muscle glycogen storage can be enhanced with a carbohydrate–protein supplement as a result of the interaction of carbohydrate and protein on insulin secretion.

One thing to keep in mind when critiquing this study was the total amount of kilocalories that were ingested during each of the treatments. The carbohydrate–protein treatment ingested nearly 150 kcal (33%) more than the carbohydrate-only treatment. Indeed, one of the criticisms of this investigation was the fact that it was impossible to determine if the greater rate of glycogen resynthesis was due to the added protein or if it was a result of greater energy availability because no isocaloric dose of carbohydrate was compared to the carbohydrate–protein group (Sunderland and Kerksick 2012). In order to address this, another study was conducted that used isocaloric carbohydrate and carbohydrate–protein treatments (Ivy et al. 2002). In this study, protocols similar to those of the Zawadzki et al. (1992) investigation were used, and again the carbohydrate–protein treatment was superior to an isocaloric carbohydrate beverage in terms of total glycogen storage and the rate of the muscle glycogen resynthesis during the first 40 min of recovery. The amounts of carbohydrate and protein ingested in this study (taken immediately after and then again 2 h later) were, respectively, 0.54 and 0.19 g/kg body mass/h.

Berardi and co-workers (2006) also reported that a carbohydrate–protein supplement (0.8 g CHO/kg/h and 0.4 g protein/kg/h) ingested immediately, 1 h, and 2 h after endurance exercise significantly increased muscle glycogen resynthesis rates as compared to an isocaloric carbohydrate-only beverage (1.2 g/kg/h).

Chapter 4 stated that "a general recommendation is to consume 1.0 to 1.5 g carbohydrate/kg/body weight immediately after the training session and again every other hour for up to 4 h when there is less than 8 h of recovery available between two exercise or competition sessions." In two of the three studies summarized previously

(Zawadzki et al. 1992; Ivy et al. 2002) in which a carbohydrate–protein supplement was ingested after glycogen depleting exercise, the amount of carbohydrates that were ingested was below recommended amounts for resynthesizing skeletal muscle glycogen synthesis (amounts ingested ranged from 0.54 to 0.75 g CHO/kg body mass/h). Other investigations (Tarnopolsky et al. 1997; van Hall, Shirreffs, and Calbet 2000) have reported that when postexercise carbohydrate intakes are ingested in high enough amounts (at least 1 g carbohydrate/kg body mass/h), additional protein does not appear to influence rates of glycogen synthesis positively.

Much more research in this area needs to be conducted before firm recommendations/doses can be made regarding optimal postexercise carbohydrate–protein ingestion. Also, it is important to remember that for most athletic and sporting events, rapid muscle glycogen replenishment is not needed. Accelerated muscle glycogen replenishment strategies are reserved for those athletes who train two or three times per day or who participate in tournament style competitions (soccer, basketball, swimming, etc.). While the data are somewhat equivocal at this time in terms of the benefits of carbohydrate–protein ingestion following exercise for the purposes of maximizing rates of skeletal muscle glycogen resynthesis, there are other benefits for including protein in the postworkout feeding. As stated in the prior section, adding protein to a carbohydrate source suppresses markers of muscle damage and subjective feelings of muscular soreness. For this reason and considering the fact that postexercise protein intake does not impede muscle glycogen resynthesis rates (and may improve them), ingesting protein with carbohydrate after glycogen-depleting exercise is a wise choice for the endurance athlete.

7.4 RESISTANCE-TRAINING PERFORMANCE CONSIDERATIONS

Individual resistance-exercise workouts are not enhanced with pre-exercise carbohydrate or protein ingestion alone. What if these two nutrients are combined? Does this result in an improvement of performance during a single bout of resistance exercise? Also, what if carbohydrate–protein ingestion occurs in the postworkout period? Does this result in cellular adaptations that over time will lead to improvements in strength and body composition? Very few studies have provided answers to these questions. In the few investigations that do exist, each used non-resistance-trained athletes rather than athletes in training.

7.4.1 Carbohydrate–Protein Ingestion Prior to a Single Bout of Resistance Exercise

Researchers from the University of Texas investigated the effects of a preworkout carbohydrate-plus-protein ingestion on resistance-exercise performance (Baty et al. 2007). Unfortunately, the subjects in this study were non-resistance-trained males rather than resistance-trained athletes. The subjects completed three sets of eight repetitions to fatigue in seven different upper and lower body exercises. They ingested a placebo beverage or a carbohydrate–protein beverage (in a 4:1 carbohydrate-to-protein ratio) at two time points prior to the exercise bout in the following manner:

- 30 min prior to exercise: 26 g carbohydrate plus 6.5 g whey protein or a placebo
- Immediately before exercise: 13 g carbohydrate plus 3.2 g whey protein or a placebo

In total, 39 g carbohydrate and about 10 g whey protein were consumed within 30 min prior to the workout in the carbohydrate–protein treatment group. Resistance-exercise performance was measured by assessing the total amount of weight lifted on the third and final set completed to fatigue on each of the seven exercises. There were no significant differences between the carbohydrate-plus-protein group and the placebo group in terms of total weight lifted during the third and final set of each exercise (Baty et al. 2007).

Another study was conducted to determine the effects on muscle damage and muscle soreness of a carbohydrate and protein supplement ingested prior to an eccentric resistance exercise (White et al. 2008). In this study, healthy, young, but sedentary males completed 50 maximal isokinetic eccentric quadriceps contractions with one leg in order to induce muscular damage and soreness. About 15 min prior to the damaging exercise bout, the subjects ingested 75 g carbohydrate and 23 g whey protein beverage or a placebo. Muscle damage and muscle soreness peaked 2 days after the exercise bout, but was unaffected by the carbohydrate–protein beverage.

Based on these studies, it does not appear that a carbohydrate–protein supplement ingested in the minutes prior to a single bout of resistance exercise will result in an enhancement of performance during the workout or reduce muscle damage and soreness in the days following a damaging workout. The finding relative to not improving performance is not surprising given that carbohydrate depletion is not a factor in an acute bout of resistance exercise and protein does not serve as a primary energy source. Any impact that a carbohydrate–protein supplement may have in enhancing performance for the strength minded athlete will likely be realized over a period of several weeks to several months and would result from the optimization of the cellular response that is cumulative over time.

7.4.2 Chronic Adaptations during and after Exercise Carbohydrate–Protein Ingestion

In prior chapters in this book, a great deal of attention was given to the benefits of ingesting protein alone and carbohydrate alone and their positive effects for enhancing adaptations that resistance-training athletes would find favorable. Carbohydrate ingestion following resistance exercise replenishes skeletal muscle glycogen and decreases skeletal muscle protein breakdown (via its impact on insulin secretion), resulting in a more positive net muscle protein balance. Protein ingestion following resistance is essential to maximize rates of muscle protein synthesis. Taken together, it would appear that carbohydrate-plus-protein ingestion would work synergistically in terms of improving net muscle protein balance when considering carbohydrate's suppression of muscle protein breakdown and protein's ability to maximize muscle protein synthesis. Very few studies have investigated the impact that a postworkout

carbohydrate–protein supplement would have on chronic strength and lean body mass adaptations when combined with a resistance-training workout.

Bird, Tarpenning, and Marino (2006) compared carbohydrate alone, essential amino acid (EAA) supplementation alone, and a carbohydrate–EAA treatment on body composition markers. Thirty-two non-resistance-trained male subjects completed two whole-body resistance-exercise sessions per week for 12 weeks. As soon as the workouts began, subjects began ingesting one of the following four beverages such that each treatment was consumed prior to the end of the workout:

- 40 g carbohydrate (approximately 0.5 g carbohydrate/kg body mass)
- 6 g EAAs
- 40 g carbohydrate + 6 g essential amino acids (CHO–EAA)
- Noncaloric placebo

Fat-free mass, muscle fiber cross-sectional area, and 3-methylhistidine excretion (a measure of muscle protein breakdown) were determined at the beginning and end of the 12-week training program. Fat-free mass significantly improved for all groups (including the placebo group), but only the carbohydrate–EAA group demonstrated significantly greater gains in fat-free mass (9 lb. [4.1 kg]) as compared to the placebo (4 lb. [1.8 kg]). All groups significantly improved type IIb muscle fiber cross-sectional area over the course of the 12-week training program. However, only the carbohydrate–EAA group (20% increase) and the EAA group (18% increase) resulted in significant improvements as compared to the placebo group (7% increase).

Muscle protein breakdown (as measured by urinary 3-methylhistidine) was measured prior to and 48 h after the last resistance-training session of the program. Remember, the higher the 3-methylhistidine values are following exercise, the greater is the skeletal muscle protein breakdown. The EAA (~5% decrease) and carbohydrate groups (13% decrease) showed no significant change in 3-methylhistidine excretion. Conversely, the carbohydrate–EAA group significantly decreased 3-methylhistidine (~26% decrease), suggesting an additive effect of carbohydrate and EAAs and their ability to suppress skeletal muscle protein breakdown (Bird et al. 2006).

One other study conducted in recreationally active women compared the effects of a carbohydrate–protein beverage (in the form of milk) versus carbohydrate alone during a period of resistance training (Josse et al. 2010). The recreationally active women resistance trained 5 days per week for 12 weeks and after each workout they ingested either fat-free milk or an isoenergetic maltodextrin beverage. The beverages were consumed immediately after and then again 1 h later and contained the following:

- Milk = 24 g carbohydrate and 18 g protein ingested at both time points
- Maltodextrin = 42 g carbohydrate ingested at both time points

Body composition and muscular strength (for nine different exercises) were recorded prior to and at the end of the study. Fat mass decreased significantly more in the milk group (3.5 lb. [1.6 kg]) as compared to the carbohydrate-only group (0.66 lb. [0.3 kg]). Lean muscle mass increased in both groups, but the milk group gained significantly more lean muscle mass (4 lb. [1.9 kg) as compared to the

carbohydrate-only group (2.5 lb. [1.1 kg]). In terms of strength gains, both the milk group and the carbohydrate only group significantly improved maximal strength in each of the nine different exercises. The only significant difference between the two treatments was bench press performance, in which the milk group improved significantly more (71%) as compared to the carbohydrate only group (47%).

It appears as if carbohydrate–protein/EAA intake during or after resistance exer- EAA essential amino acids alone. The benefits observed from the carbohydrate–protein/EAA intakes included:

- Superior gains in lean muscle mass
- Muscle fiber cross-sectional area
- Suppression of skeletal muscle protein breakdown

Unfortunately, the studies that have been conducted in this area have included non-resistance-trained male subjects and recreationally active female subjects. The adaptations in these populations is very favorable, but we do not know with certainty if these same improvements would be observed in a resistance-trained, athletic population.

7.5 CONCLUSION

The concept of combining nutrients and ingesting them at specific times has primarily concentrated on carbohydrate–protein combinations and is referred to as "nutrient timing." Simply stated, nutrient timing is focused on "when to eat" rather than solely on "what to eat." Does it matter when specific nutrients are ingested in terms of maximizing training adaptations? Some research has supported that the timing of the ingestion of nutrients is very important for maximizing adaptations (Cribb and Hayes 2006), while other research has not supported these findings (Hoffman et al. 2009). For the endurance athlete, ingesting a carbohydrate–protein beverage soon after exercise results in skeletal muscle glycogen resynthesis as well as the suppression of muscle damage and muscle soreness. There is some evidence to support the contention that rates of skeletal muscle glycogen resynthesis are maximized with the co-ingestion of carbohydrate and protein as compared to carbohydrate ingestion alone. To date, there is a lack of research that documents the benefits (or lack of benefits) of combined carbohydrate and protein ingestion in resistance-trained, athletic populations. In non-resistance-trained male subjects and recreationally active female subjects, it appears as if gains in lean muscle mass and reductions in skeletal muscle protein breakdown are experienced with carbohydrate–protein co-ingestion during or after resistance exercise.

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8 Energy Balance

Bill Campbell

CONTENTS

8.1 Introduction 200
8.2 Energy Balance Equation 200
8.3 Calorimetry, the Calorie, and Energy Balance 202
8.3.1 Energy Intake (i.e., Food) and the Calorie 202
8.3.2 Energy Expenditure and the Calorie 204
8.3.2.1 Direct and Indirect Calorimetry 205
8.4 Energy Intake 205
8.4.1 Measuring Energy Intake 206
8.4.1.1 Direct Observation 206
8.4.1.2 24-Hour Dietary Recall (Recall of Foods Eaten) 206
8.4.1.3 Food Frequency Questionnaires (Retrospective Questionnaires) 207
8.4.1.4 Food Records (Ranging from 1 to 7 Days) 207
8.4.2 Limitations of Measuring Energy Intakes 208
8.4.3 Energy Intakes and Macronutrient Composition of Athletes' Diets 210
8.5 Energy Expenditure 210
8.5.1 Resting Metabolism 217
8.5.1.1 Resting Metabolic Rates of Sedentary Versus Active Individuals 219
8.5.1.2 Resting Metabolic Rate Prediction Equations 219
8.5.2 Thermic Effect of Food 222
8.5.3 Thermic Effect of Activity 223
8.5.3.1 Methods for Determining Energy Expenditure of Athletes 223
8.6 Estimating Total Daily Energy Needs 225
8.7 Limitations of the Energy Balance Equation 227
8.8 Conclusion 230
References 230

8.1 INTRODUCTION

Except for periods of deliberate manipulation of energy intake to alter body composition, athletes should strive to achieve an energy intake that matches their total daily energy expenditure (Burke 2001). The idea of balancing energy intake with energy expenditure comprises the concept of the energy balance equation. An understanding of the factors that contribute to the energy balance equation is essential for athletes interested in improving their body composition. Energy balance occurs when energy intake (the sum of energy from foods, fluids, and dietary supplements) equals energy expenditure (the sum of energy expended as basal metabolic rate, the thermic effect of food, and the thermic effect of activity). Inadequate energy intake relative to energy expenditure may compromise athletic performance and minimize the benefits of training. On the other hand, excess energy intake often leads to gains in fat mass, which also compromises exercise and sport performance and decreases relative power production. In this chapter, the energy balance equation will be presented as well as a discussion of the variables contributing to the energy balance concept. Utilizing the principles set forth in the energy balance equation enables an athlete to meet energy needs and optimize body composition and, ultimately, athletic performance.

8.2 ENERGY BALANCE EQUATION

The human body obeys physical laws. The first law of thermodynamics (often called the law of conservation of energy) postulates that energy can be transferred from one system to another in many forms but cannot be created or destroyed. In relation to the human body, this means that the energy balance equation (Figure 8.1) dictates that body mass remains constant when caloric intake equals caloric expenditure. Conversely, if the energy balance equation is unbalanced, weight loss or weight gain will occur. One of the primary ways athletes achieve fat loss is by modifying nutritional intake, primarily the amounts and types of calories consumed. Body energy stores (particularly fat stores) can only increase when food intake exceeds energy expenditure. Conversely, energy stores can only be depleted when energy expenditure exceeds food intake. Thus, the balance between food intake and energy

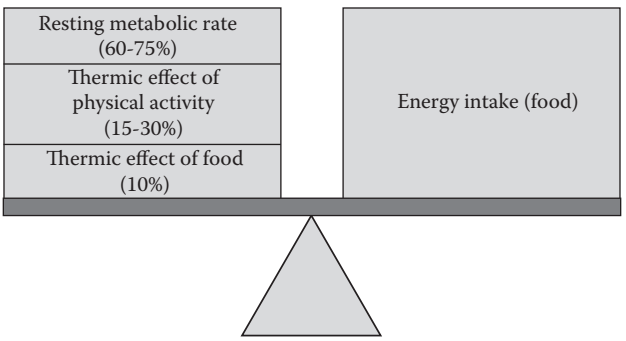

FIGURE 8.1 Energy balance equation.

expenditure determines the body's energy stores. These concepts are conveniently summarized in what is known as the energy balance equation. In terms of stimulating fat loss, the energy balance equation can be altered in the following three ways:

1. Reducing caloric intake below daily energy requirements
2. Maintaining caloric intake and increasing energy expenditure through additional physical activity above daily energy requirements
3. A combination of decreasing daily caloric intake and increasing daily energy expenditure

From a broad perspective, the energy balance equation is nothing more than a tool that conceptualizes energy intake (i.e., calories ingested from food) and energy expenditure (calories burned primarily through normal metabolic functions and physical activity). Hence, the energy balance equation can be stated as

$$\text{Change in body composition} = \text{energy intake} - \text{energy expenditure} \quad (8.1)$$

Equation (8.1) lists the conventional model for the energy balance equation. However, there are several other factors that contribute to the energy balance concept. For example, not all foods are digested with identical efficiency. As opposed to carbohydrates and fat, protein is the least efficient macronutrient in terms of extracting energy and processing it toward eventual energy storage; this concept of digestible efficiency is explained in more detail later in Section 8.5.2, "Thermic Effect of Food." Considering the differences in efficiency with which the macronutrients are digested, a correction factor can be applied to the energy intake aspect of the energy balance equation.

There are traditionally three components that contribute to energy expenditure: resting metabolic rate (RMR), thermic effect of food (TEF), and the thermic effect of physical activity (TEA). Research has also identified another aspect of energy expenditure that is often ignored: *nonexercise activity thermogenesis* (NEAT). Essentially, NEAT is associated with fidgeting, maintenance of posture, and other physical activities of daily life that are not classified as conscious voluntary exercise (Vanltallie 2001; Levine, Eberhardt, and Jensen 1999). Taking the digestibility correction factor and NEAT into consideration we get the following for the energy balance equation:

$$\text{Change in body composition} = \text{energy intake (corrected for digestion)} - \text{energy expenditure (RMR + TEF + TEA + NEAT)} \quad (8.2)$$

Regardless of which energy balance equation is used [Equation (8.1) or (8.2)], each provides a template on which program (both dietary and sport-specific training and conditioning) changes can be made in anticipation of reaching a desirable body composition. Before any dietary and training changes are implemented, an analysis of energy intake and energy expenditure should be performed. In addition, it is essential that planned, periodic body composition assessments be conducted in order to verify the following:

- Body mass reductions are resulting from reductions in body fat (not from lean muscle mass).
- Body mass gains are appearing primarily in the form of lean muscle mass (not fat mass).

The next sections discuss the variables of the energy balance equation in more detail. At the end of the chapter, an examination of the limitations associated with implementing the energy balance equation is presented.

8.3 CALORIMETRY, THE CALORIE, AND ENERGY BALANCE

Quantifying the variables of the energy balance equation—energy intake and energy expenditure—requires a knowledge of calorimetry. Calorimetry is the scientific process of measuring the heat of chemical reactions or physical changes. Whether one is measuring resting metabolic rate in humans or the number of calories in food, what is actually being measured is heat production (or an estimation of heat production). Following is a discussion on how calorimetry is used to measure the heat energy in food and in the resting metabolic rate.

The SI unit to measure heat energy is the joule. However, a common unit employed to measure heat energy is the calorie. A calorie (cal) is defined as the amount of heat required to raise the temperature of 1 g of water by 1°C. Because the calorie is very small, the term kilocalorie (kcal) is generally used to express energy expenditure and the energy value of foods. A kilocalorie is equal to 1000 calories and is defined as the amount of heat required to raise the temperature of 1 kg (or 1 L) of water by 1°C (specifically, from 14.5 to 15.5°C).

Many individuals confuse the terms "calorie" and "kilocalorie." What makes these terms confusing for many individuals is that they are often used interchangeably, despite their technical differences as just described. For instance, many individuals state that a given food contains "x" amount of calories, but what they mean is that the food contains "x" amount of kilocalories. Further, a kilocalorie is sometimes called the nutritionist's Calorie (note the capital letter C) because of its common use as a unit of food energy. Almost any time the word "calorie" is used with regard to food (such as the number of calories listed on a food label) or metabolism, it is synonymous with kilocalorie. In order to limit any confusion, the terms Calorie (with a capital C) and kilocalorie will be used interchangeably throughout this text and should be associated with the technical definition of a kilocalorie.

8.3.1 Energy Intake (i.e., Food) and the Calorie

Different foods contain different amounts of Calories (Table 8.1). What makes one food source possess more kilocalories than another food? The answer lies in the amount of heat that is liberated from the food source when it is burned (or oxidized). For example, if a particular food contains 300 kcal, then releasing the potential energy trapped within the chemical bonds of the food increases the temperature of 300 L (or kg) of water 1°C. The caloric value of food is measured

TABLE 8.1
Caloric Content of Various Foods

Food	Amount	Kilocalories
Lucky Charms, General Mills	1 Cup	114
Raisin Nut Bran, General Mills	1 Cup	209
Almond milk chocolate, Almond Breeze	8 Fluid oz.	110
Chocolate milk, whole	8 Fluid oz.	208
Peach pie	⅙ of 8-in. pie	261
Pecan pie	⅙ of 8-in. pie	452
French salad dressing	2 Tablespoons	21
Peanut butter spread	2 Tablespoons	210
McDonalds strawberry shake	16 oz.	560
PowerAde	16 oz.	144
Green beans	1 Cup	44
Coleslaw	1 Cup	82
Grouper, raw	3 oz.	78
Tuna, white, canned in oil	3 oz.	237
Prego traditional 100% natural sauces	1 Cup	263
Ragu light (two flavors)	1 Cup	96
Blue cheese, chunky, fat free, WishBone	2 Tablespoons	35
Blue cheese, chunky, WishBone	2 Tablespoons	170
Cappuccino cho chunk, Healthy Choice	½ Cup	120
Dulce de leche caramel, Haagen Dazs	½ Cup	290

Source: Pennington, J., and J. Douglass. 2005. *Bowes and Church's Food Values of Portions Commonly Used.* Baltimore, MD: Lippincott Publishing Company.

via a device known as a bomb calorimeter (Figure 8.2). The food sample is first placed in the bomb calorimeter, which is a stainless steel container surrounded by water. The heat generated as the food sample is ignited is then transferred into a known quantity of water within the water vessel, which results in a rise of water temperature that is measured to determine the energy value of the food accurately (Summerfield 2001).

In practice, computing the caloric value of foods is time consuming and labor intensive. However, each of the macronutrients (carbohydrates, proteins, and fats), when oxidized/burned in a bomb calorimeter, produces consistent and reliable values of heat energy (i.e., Calories). In terms of net energy values in the human body, 1 g of dietary fat provides about 9 Calories, while 1 g of carbohydrate or protein provides about 4 Calories. Hence, as long as the amount and composition of a food are known, the caloric values can be estimated accurately. For example, 100 g of cooked broccoli (about ⅔ cup) contains approximately 7 g of carbohydrate and 2 g of protein. Because 1 g of carbohydrate contains 4 Calories, the 7 g of carbohydrate found in 100 g of cooked broccoli is equivalent to 28 Calories. Likewise, 1 g of protein contains 4 Calories; therefore, the 2 g of protein contained in the broccoli is equivalent to 8 Calories. By adding the 28 Calories from the 7 g of carbohydrate

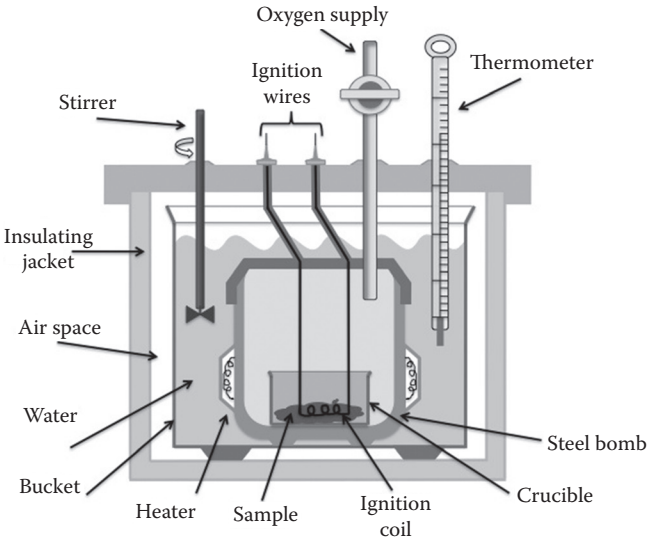

FIGURE 8.2 Bomb calorimeter.

and the 8 Calories from the 2 g of protein in 100 g of cooked broccoli, we get a total value of 36 Calories.

8.3.2 Energy Expenditure and the Calorie

Measurement of an individual's energy expenditure at rest or during a particular activity has many practical applications. An estimation of energy expenditure (both at rest and during activity) is important when establishing daily caloric intakes. If the goal is to lose body fat, it is essential to know how many Calories are expended on a daily basis so that caloric intake can be set at a level below this threshold.

When the body uses energy to do work, heat is liberated. The rate of heat production in humans is directly proportional to the metabolic rate. Therefore, measuring heat production (calorimetry) in humans yields a direct measure of metabolic rate. When this measurement of heat production is conducted in the rested condition, it is referred to as resting metabolic rate. Similarly to the energy value of food, the unit of measurement for resting metabolic rate is the joule or Calorie. In contrast to food, humans are not placed into a bomb calorimeter and burned/oxidized to determine the value of the heat energy that is released from the breaking of the chemical bonds of the body. Such a method would not only be unethical, but also would not be approved by many institutional review boards overseeing the research process. Rather than utilizing a bomb calorimeter, there are two techniques employed in the measurement of an individual's resting metabolic rate: direct calorimetry and indirect calorimetry.

8.3.2.1 Direct and Indirect Calorimetry

The process of measuring resting metabolic rate via the direct measurement of heat production is called direct calorimetry. Such a technique involves placing the human subject in a chamber insulated from the outside environment (usually by water surrounding the chamber) and having an inlet for oxygen to come into the chamber and an outlet for carbon dioxide to leave the chamber. By measuring the volume of water flowing through the chamber and the temperature change of the water, the amount of heat produced can be measured. Although direct calorimetry is considered to be a precise technique for the measurement of resting metabolic rate, there are considerable drawbacks to this method. These drawbacks include the construction of a chamber that is large enough for the athlete, in addition to the expense and engineering expertise that is needed.

Fortunately, there is another procedure that can be used to estimate resting metabolic rate. This technique is referred to as indirect calorimetry because it does not involve the direct measurement of heat production. Indirect calorimetry is relatively simple to operate and less expensive to maintain and staff than direct calorimetry. Indirect calorimetry requires the use of a metabolic cart, which analyzes the volume of ambient air that passes through the respiratory system and the amount of oxygen extracted from this ambient air (i.e., oxygen uptake).

A direct relationship exists between oxygen consumed and the amount of heat produced in the body (Anderegg et al. 2009; da Rocha, Alves, and da Fonseca et al. 2006; Jeukendrup and Wallis 2005; Ferrannini 1988). Hence, the term "indirect calorimetry" stems from the fact that the heat released by chemical processes within the body can be indirectly calculated from the rate of oxygen consumption. The caloric expenditure of resting metabolic rate (or energy expenditure in general) is often estimated to be approximately 5 kcal/L oxygen consumed. Therefore, if an athlete is undergoing an indirect calorimetry procedure and it is determined that his or her oxygen consumption be 0.3 L/min, this is equivalent to expending approximately 1.5 kcal/min (remember that every liter of oxygen consumed is equivalent to approximately 5 kcal). If an individual were to expend 1.5 Calories/min, this is equal to 2160 Calories expended over the course of an entire day. The use of indirect calorimetry to measure resting metabolic rate is very popular, but does require an investment of time for the athlete and the technician. Upkeep and maintenance of the metabolic cart must be conducted as well. To offset these challenges, several equations have been formulated to predict resting metabolic rate. These equations are discussed in Section 8.5.1, "Resting Metabolism."

8.4 ENERGY INTAKE

Estimations of energy intake can be used for everyday dietary planning and evaluation, and they are of interest for several reasons, including (Burke 2001):

1. Setting the potential for achieving the athlete's requirements for energy-containing macronutrients and the food needed to provide vitamins, minerals, and other non-energy-containing dietary compounds required for optimal performance and health

2. Assisting in the manipulation of muscle mass and body fat levels to achieve the specific physique that is ideal for athletic performance

8.4.1 Measuring Energy Intake

Energy intake represents the number of Calories that are ingested on a daily basis from the nutrients' protein, carbohydrate, and fat (other forms of dietary energy such as fiber and alcohol will not be discussed to keep the focus on the primary macronutrients). The recording of energy intake is usually conducted over a period of 1 to 7 days (Thompson and Byers 1994). Broadly speaking, energy intake can be measured via four methods (Hill and Davies 2001):

- Direct observation
- Recall of foods eaten (24-h dietary recall)
- Retrospective questionnaires (food frequency questionnaires)
- Food records (range from 1- to 7-day dietary/food records)

8.4.1.1 Direct Observation

Direct observation involves placing the individual or athlete in a research laboratory setting (often for several days at a time in a metabolic chamber) and physically observing all foods and beverages (with known caloric values) that are ingested. By observing all food and beverage intake, it is relatively simple to calculate the total amount of Calories that are ingested. The direct observation method yields more accurate and reliable results, but is cumbersome and places the athlete in an artificial environment. Hence, it is rarely utilized except in research studies. Therefore, other methods of recording dietary intake are commonly used because they are more practical and time efficient.

8.4.1.2 24-Hour Dietary Recall (Recall of Foods Eaten)

Often used as a quick nutrition assessment, many times 24-h dietary recalls can be used on a spontaneous basis to determine an athlete's daily food intake (Moffatt et al. 2011). In this method, the athlete is asked to list the foods and beverages that were ingested within the past 24 h. The 24-h dietary recall can be performed by two different methods. The first is when the athlete is asked to start from the beginning of the previous day and provide in detail all of the food and beverages consumed from the beginning of the day before. The second method starts with the current day and works backward. For example, the athlete would be questioned on what he or she ate prior to this visit and then work back over the past 24 h (Moffatt et al. 2011). An advantage of the 24-h dietary recall is that it involves minimal burden on the athlete, it can be scheduled around daily activities, and it can be done in person or over the telephone in a brief amount of time (about 15–30 min) (Magkos and Yannakoulia 2003; Moffatt et al. 2011).

One of the potential problems with a 24-h dietary recall is the misrepresentation of the usual diet (Sempos et al. 1985). It is important to ask the athlete if the diet consumed within the past 24 h was a normal diet or if it was a variation from the normal

diet. For example, if in the previous day the athlete returned home on a flight from an out-of-state competition and upon returning received treatment from the athletic training staff for an extended period of time, these events may have caused changes in diet during the past 24 h that are not typical.

8.4.1.3 Food Frequency Questionnaires (Retrospective Questionnaires)

Food frequency questionnaires are designed to assess habitual diet by asking about the frequency with which food items or specific food groups are consumed over a reference period (from several days to a year). Specifically, a food frequency questionnaire is a limited checklist of foods and beverages with a frequency response section for athletes to report how often each item was consumed over the specified period of time. The length of the list of foods can range from approximately 20 to 200 items. Food frequency questionnaires can be administered in several formats, including paper and pencil, web based, interviewer administered, or telephone interview. For obvious reasons, questionnaires that are self-administered (paper and pencil or web based) are much less burdensome than other assessment methods. In its simplest form, the food frequency questionnaire consists of a short or long list of foods, or categories of foods, with options to indicate how often each is consumed within a specified period of time, varying from 1 day to several months (Magkos and Yannakoulia 2003). Categories ranging from "never" or "less than once a month" to "greater than six servings per day" are used and athletes are required to choose one of these options.

One of the limitations of food frequency questionnaires is their specificity to certain populations (Moffatt et al. 2011; Magkos and Yannakoulia 2003). They are developed by reviewing a large number of diet records or recalls to determine the most common food items for the population as a whole (Magkos and Yannakoulia 2003; Block et al. 1986). Hence, their applicability for assessing the intakes of people whose eating patterns deviate considerably from those of the mainstream is largely limited (Magkos and Yannakoulia 2003; Coates and Monteilh 1997). Currently, there is one food frequency questionnaire that has been developed for use with physically active men (Magkos and Yannakoulia 2003; Fogelholm and Lahti-Koski 1991). This 122-item questionnaire showed good agreement at the group level but poor agreement at the individual level when validated against a 7-day diet record (Magkos and Yannakoulia 2003; Fogelholm and Lahti-Koski. 1991). In comparison to the other methods, the food frequency questionnaire has more variability in the energy intake values reported (Andersen et al. 2003; Buzzard et al. 2001).

8.4.1.4 Food Records (Ranging from 1 to 7 Days)

Diet records can be recorded for many different time periods, with the most popular being 1-, 3-, 4-, and 7-day diet records. For athletes, a 3- to 7-day diet-monitoring period is believed to provide reasonably accurate and precise estimations of habitual energy and macronutrient consumption (Magkos and Yannakoulia 2003; Deakin 2000; Black 2001). The 3-day diet record is advantageous because it is usually more representative of typical intake than a 1-day diet record, but not as cumbersome as a 7-day diet record. Generally, a food record consisting of three consecutive days is recommended, as studies have shown that incomplete records get more frequent as

the number of days increases. This is referred to as respondent fatigue (Biró et al. 2002; Thompson and Byers 1994). Also, the 3-day diet record has been reported to be valid (Lührmann et al. 1999) and often serves as a reference method for other measures of energy intakes (unfortunately, these findings are reported in populations other than athletes) (Eysteinsdottir et al. 2012; Tokudome et al. 2005; Ke et al. 2005; Schröder et al. 2001; Trumble-Waddell et al. 1998).

A 3-day diet record requires the athlete to record all foods and beverages consumed during a designated 3-day period, with one of the days comprising a weekend day. Completion of a 3-day diet record may include the weighing or quantifying (usually in household measures) of all food and drink for the 3-day period. The days chosen for examination should be typical and should include two weekdays and one weekend day. The 3-day diet record is essentially a form that prompts the athlete to provide as much detail as possible about dietary intake, such as the specific food/beverage consumed, the quantity of the food/beverage consumed, time of day, and the method of preparation (if applicable). A sample 3-day diet record is provided in Table 8.2. In comparison to the other methods of estimating food/energy intake, the 3-day diet record is the most commonly used approach (Burke 2001). While the 3-day diet record appears to be a better choice of estimating energy intake than the 24-h dietary recall and food frequency questionnaires (Crawford et al. 1994), the need to weigh and record intake over several days can be seen as tedious and time consuming and thus is often associated with poor compliance or an alteration of the diet during the recording period (Hill and Davies 2001; Barrett-Conner 1991; Black et al. 1993).

Food records can be analyzed using composition of food tables or computerized diet-analysis programs. Data that have been coded and put into a computerized nutrition analysis program can be easily analyzed for calories, carbohydrate, protein, and fat content. In addition, computerized diet-analysis programs also typically indicate the proportion of calories (percentage of calories) from the macronutrients.

8.4.2 Limitations of Measuring Energy Intakes

Regardless of the method used to measure energy intake, the goal is to attain the most accurate report on the total amount and composition of Calories ingested from normal dietary intakes of the athletes under investigation. In general, all methods of measuring dietary intake are hampered by errors of reliability and validity (Magkos and Yannakoulia 2003; Black 2001). The emergence of the doubly labeled water (DLW) technique (Schoeller and van Santen 1982) for measuring free-living energy expenditure in humans has enabled the accurate validation of dietary assessment methods (Magkos and Yannakoulia 2003). Nonetheless, the principle behind the validation of measures of energy intake using measures of energy expenditure is that of the energy balance equation. As stated above, energy balance occurs when energy intake equals energy expenditure under conditions of stable body weight. If energy intake is not matched by energy expenditure, energy balance no longer exists and a change in body weight will result. Thus, if energy intake is lower than energy expenditure, body weight will be reduced. What has been observed in many investigations in which energy intake and energy expenditure (via the doubly labeled

TABLE 8.2
Three-Day Food Record Table

Name: _____ Date: _____

Instructions:

Do not change your eating habits while keeping your 3-day food record.

Tell the truth. Record what you really eat so that an accurate analysis can be made.

Fill out the following food record form for 3 days, including one weekend day.

Time	Food/Beverage Ingested	Amount (cups, fluid ounces, tablespoons, slices, pieces, etc.)

Basic rules to remember:

- Write down EVERYTHING! Keep your food record form with you all day long and write down everything you eat and drink. A piece of candy, a handful of potato chips, a can of soda, or a small bagel may not seem like much, but calories in these foods can add up.
- Record what you eat right away! Don't depend on your memory at the end of the day. Record what you eat and drink as you go, meaning you should carry a pen and paper or use a list on your phone, etc.
- Be specific. Make sure you include "extras," such as cheese on your sandwich or vegetables, butter, and salad dressings.
- Estimate amounts. If you have a bowl of cereal or ice cream, measure out or estimate the actual amount (rather than writing "bowl" of cereal or "bowl" of ice cream).

water technique) were measured was that reported energy intake values were consistently less than energy expenditure values—without a concomitant reduction in body weight. Assuming the validity of the energy expenditure measures, the only explanation was that energy intake was underreported (the individuals were reporting far fewer Calories consumed than what they were actually ingesting).

Previous research clearly indicates a significant and systematic underreporting of food intake by virtually all populations investigated, including the overweight and obese (Goris, Westerterp-Plantenga, and Westerterp 2000; Buhl et al. 1995), adults (Bathalon et al. 2000; Tomoyasu, Toth, and Poehlman 1999), and children

(Bratteby et al. 1998; Champagne et al. 1998). Unfortunately, the underestimation of energy intake is also widespread in athletic populations (Drenowatz et al. 2012; Aerenhouts et al. 2011; Ebine et al. 2000; Hill and Davies 2002). In a comprehensive review on this topic, Magkos and Yannakoulia (2003) stated that in published studies using doubly labeled water to validate self-reported energy intake in athletes, underreporting accounts for approximately 10%–45% of total energy expenditure. The inaccurate reporting of dietary intake among athletic populations has important implications when attempting to make changes to their dietary programs. In light of this, results from the various methods to assess energy intake should be interpreted with caution. However, there are measures that can be taken to increase the accuracy of reporting the energy intakes of athletes (Magkos and Yannakoulia 2003). For example, when food intake was recorded by trained staff (Jones and Leitch 1993) or with the assistance of dieticians (Sjödin et al. 1994), the estimated energy intake closely matched the doubly labeled determined energy expenditure, regardless of the dietary assessment method used or the types of athletes involved.

8.4.3 Energy Intakes and Macronutrient Composition of Athletes' Diets

Despite the known limitations of assessing energy intakes from indirect measures, there is still value in obtaining indirect measures of energy intakes. Tables 8.3 to 8.8 summarize some of these published data while classifying male and female athletes as intermittent sport athletes, endurance athletes, and strength/power athletes. By knowing the macronutrient compositions of the athlete's diet, extreme dietary habits can be avoided, and this allows the athlete to make more informed decisions about which macronutrients can be reduced during a period of caloric restriction in an effort to reduce body fat. When analyzing the self-reported energy intakes of male and female athletes, an interesting trend manifests itself. When kilocalories are compared as a total daily amount (an absolute measurement), males appear to ingest significantly more kilocalories than their female counterparts. However, when the energy intakes are controlled for by body mass, the self-reported energy intakes of male and female athletes are much closer. Further, when energy intakes are reported in terms of fat-free mass, the differences between male and female athletes become even less.

8.5 ENERGY EXPENDITURE

The other half of the energy balance equation (in addition to energy intake) is energy expenditure. The total daily energy expenditure of each athlete is unique, arising from the contributions of three principal components: basal metabolic rate/resting metabolism, thermic effect of food, and the energy expenditure of physical activity. In terms of their relative contributions, basal metabolic rate is the largest contributor, as it represents about 60%–75% of total daily energy expenditure. The thermic effect of food comprises approximately 10% of total daily energy expenditure and the energy expenditure of physical activity contributes approximately 15%–30% of total daily energy expenditure.

Direct calorimetry and indirect calorimetry are two of the methods used to measure energy expenditure in humans. Another method is the doubly labeled water

TABLE 8.3
Self-Reported Energy Intakes of Male Intermittent Sport Athletes

Study	Population	Energy Intake Method	Energy Intake per Day (kcal)	Energy Intake (relative to body weight) (kcal/kg)	Macronutrient Composition
Russell and Pennock (2011)	10 Professional soccer players	7-Day diet record	2831	42.3	56% CHO 16% PRO 31% FAT
Lundy et al. (2006)	34 Professional rugby players	4-Day diet records	4230	46	51% CHO 18% PRO 25% FAT
Cole et al. (2005)	28 American football players	3-Day diet record	2624	Body mass not reported	43% CHO 19% PRO 38% Fat
Reeves and Collins (2003)	Gaelic football players	7-Day diet record	2954	36	51% CHO 15% PRO 28% FAT
Zieler et al. (2001)	80 Elite figure skaters	3-Day diet record	2329	36	57% CHO 15% PRO 30% FAT
Maughan (1997)	51 Professional soccer players	7-Day weighed diet record	2847	36.8	50% CHO 15% PRO 34% FAT
Fogelholm et al. (1992)	418 Athletes (various sport participation)	122-Item food frequency questionnaire	2866	37.7	54% CHO 15% PRO 31% FAT
Average reported energy intakes			2954	39.1	52% CHO 17% PRO 31% FAT

TABLE 8.4
Self-Reported Energy Intakes of Female Intermittent Sport Athletes

Study	Population	Energy Intake Method	Energy Intake per Day (kcal)	Energy Intake (relative to body weight) (kcal/kg)	Macronutrient Composition
Clark et al. (2003)	13 Collegiate soccer players (preseason)	3-Day diet record	2290	36.9	55% CHO 15% PRO 29% Fat
Clark et al. (2003)	13 Collegiate soccer players (postseason)	3-Day diet record	1865	30.3	57% CHO 13% PRO 31% Fat
Ziegler et al. (2001)	81 Elite figure skaters	3-Day diet record	1545	33	60% CHO 16% PRO 25% FAT
Ebine et al. (2000)	9 Elite synchronized swimmers	7-Day diet record	2129	40.5	Macronutrient composition not reported
Fogelholm et al. (1995)	12 Gymnasts and figure skaters	7-Day diet record	1684	32.6	Macronutrient composition not reported
Fogelholm et al. (1995)	12 National league soccer players	7-Day diet record	2146	35.3	Macronutrient composition not reported
Average reported energy intakes			1943	34.8	57% CHO 15% PRO 28% FAT

TABLE 8.5
Self-Reported Energy Intakes of Male Endurance Athletes

Study	Population	Energy Intake Method	Energy Intake (total) (kcal)	Energy Intake (relative to body weight) (kcal/kg)	Macronutrient Composition
Drenowatz et al. (2012)	15 Endurance athletes	Online food-frequency questionnaire	2575	35.1	51% CHO 16% PRO 33% FAT
Zalcman et al. (2007)	18 Adventure race athletes	3-Day food record	3367	44.6	51% CHO 17% PRO 32% FAT
Nogueira and Da Costa (2004)	38 Brazilian triathletes	24-h food recall and food frequency questionnaire	3680	51.7	57% CHO 6% PRO 27% FAT
Vogt et al. (2005)	11 Professional road cyclists	6-Day weighed diet records	3230	45.5	59% CHO 19% PRO 21% FAT
Onywera et al. (2004)	10 Elite Kenyan runners	7-Day weighed diet records	2987	51	77% CHO 10% PRO 13% FAT
Sigiura, Suzuki, and Kobayashi (1999)	12 Japanese track-and-field athletes	3-Day food record	3532	57.4	50% CHO 15% PRO 34% FAT
Poehlman (1992)	36 Aerobically trained athletes	3-Day food record	3150	45	56% CHO 15% PRO 28% FAT
Average reported energy intakes			2692	47.1	57% CHO 15% PRO 27% FAT

TABLE 8.6
Self-Reported Energy Intakes of Female Endurance Athletes

Study	Population	Energy Intake Method	Energy Intake (total) (kcal)	Energy Intake (relative to body weight) (kcal/kg)	Macronutrient Composition
Zalcman et al. (2007)	6 Adventure race athletes	3-Day dietary record	3064	48.1	60% CHO 18% PRO 24% FAT
Nogueira and Da Costa (2004)	9 Triathletes	24-h Food recall and food frequency questionnaire	2366	42.4	55% CHO 15% PRO 30% FAT
Hill et al. (2002)	7 Elite lightweight rowers	4-Day weighed dietary record	2214	36.7	Not reported
Hassapidou and Manstrantoni (2001)	11 Middle-distance runners (during training)	7-Day weighed dietary record	1816	33.8	48% CHO 14% PRO 39% FAT
Hassapidou and Manstrantoni (2001)	11 Runners (competitive season)	7-Day weighed dietary record	1679	31.3	51% CHO 14% PRO 37% FAT
Sigiura, Suzuki, and Kobayashi (1999)	7 Elite Japanese long-distance runners	3-Day dietary record	2720	45	51% CHO 16% PRO 33% FAT
Edwards et al. (1993)	9 Distance runners	24-h Food recall and 2-day dietary record	2037	36.8	Not reported
Schulz et al. (1992)	9 Elite distance runners	6-Day dietary record	2193	41.8	59% CHO 13% PRO 27% FAT
Average reported energy intakes			2261	39.5	54% CHO 15 % PRO 31% FAT

TABLE 8.7
Self-Reported Energy Intakes of Male Strength/Power Athletes

Study	Population	Energy Intake Method	Energy Intake (total) (kcal)	Energy Intake (relative to body weight) (kcal/kg)	Macronutrient Composition
Faber, Spinnler-Benadé, and Daubitzer (1990)	20 National level throwing athletes	7-Day food diary	3489	36.3	43% CHO 18% PRO 40% FAT
Sugiura et al. (1999)	2 National level team throwing athletes	3-Day food diary	3323	31.9	51% CHO 16% PRO 32% FAT
Sugiura et al. (1999)	10 National level sprinting athletes	3-Day food diary	2578	38.4	53% CHO 16% PRO 31% FAT
Poehlman et al. (1992)	18 Resistance-trained athletes	3-Day food diary	3224	38.3	56% CHO 19% PRO 24% FAT
Average reported energy intakes			3154	36.2	51% CHO 17% PRO 32% FAT

TABLE 8.8
Self-Reported Energy Intakes of Female Strength/Power Athletes

Study	Population	Energy Intake Method	Energy Intake (total) (kcal)	Energy Intake (relative to body weight) (kcal/kg)	Macronutrient Composition
Jonnalagadda, Benardot, and Dill (2000)	28 U.S. national team gymnasts	3-Day food record	1757	36.1	65% CHO 17% PRO 17% FAT
Hassapidou and Manstrantoni (2001)	8 Volleyball players	7-Day weighed food record	1541	23.8	51% CHO 14% PRO 37% FAT
Doyle-Lucas, Akers, and Davy (2010)	15 Elite dancers	4-Day food record	1557	30.0	56% CHO 17% PRO 26% FAT
Kim et al. (2002)	18 Judo athletes	4-Day dietary record	2667	40.9	58% CHO 12% PRO 29% FAT
Sugiura et al. (1999)	11 National sprinting athletes	3-Day food record	2350	43.5	52% CHO 15% PRO 33% FAT
Average reported energy intakes			1974	34.9	56% CHO 15% PRO 29% FAT

BOX 8.1 DOUBLY LABELED WATER TECHNIQUE

When using the doubly labeled water technique, the subject ingests a quantity of water containing a known concentration of the stable isotopes of hydrogen (^{2}H or deuterium) and oxygen (^{18}O or oxygen-18)—hence the term doubly labeled water. After ingesting the doubly labeled water, the ^{2}H and ^{18}O are used as tracers and can be measured in the various body fluids (urine and saliva). Over time (several days or weeks), the labeled isotopes undergo a rate of disappearance from the body, which is measured. ^{2}H leaves the body as water ($^{2}H_2O$) in sweat, urine, and pulmonary water vapor, while labeled oxygen leaves as water ($H_2{}^{18}O$) and carbon dioxide ($C^{18}O_2$) produced during macronutrient oxidation in energy metabolism. The difference in the rate of turnover of $H_2{}^{18}O$ and $^{2}H_2O$ is an estimate of carbon dioxide production during the measurement period. The carbon dioxide production rate is then used to calculate energy expenditure with the knowledge of the fuel mixture oxidized. (In order to calculate the subject's energy expenditure, the mean respiratory quotient [RQ] must be known.)

The main advantage of the doubly labeled water technique is that it does not interfere with the everyday life of the athlete, and measurements in a free-living environment can be obtained. The main disadvantages of this technique are the expense of the enriched ^{18}O and the spectrometric analysis of the two isotopes. Also, the method is best used as a mean value of energy expenditure over a several-week period and is not used to calculate day-to-day variation in energy expenditure.

technique. This technique provides a useful way to estimate total daily energy expenditure in free-living conditions without the constraints imposed by the other methods (McArdle, F. I. Katch, and F. L. Katch 2009; Ekelund et al. 2002; Starling et al. 1998). The doubly labeled water technique is based on the difference in the rates of turnover of $^{2}H_2O$ and $H_2{}^{18}O$ in body water. For a more detailed description of the doubly labeled water method, refer to Box 8.1.

8.5.1 Resting Metabolism

Basal metabolic rate is the energy expended when an individual is lying at complete rest, in the morning, after sleep, in the postabsorptive state. The postabsorptive state describes the condition in which the gastrointestinal tract is empty of nutrients and body stores must supply required energy. Basal metabolic rate is usually measured 12–18 h after the last meal. Other terms that are closely related to and often confused with basal metabolic rate are resting energy expenditure and resting metabolic rate. Resting energy expenditure or resting metabolic rate is measured in subjects at complete rest about 4 h after eating a light meal. Due to the difficulty of achieving the conditions of a true basal metabolic rate assessment, resting energy expenditure or resting metabolic rate is more commonly measured. Resting energy expenditure is

TABLE 8.9
Contribution of Various Organs and Body Tissues to Total Body Weight and Basal Metabolic Rate

Organ/Tissue	Contribution to Body Weight (%)	Contribution to Basal Metabolic Rate (%)
Muscle	40	22
Liver	2.6	21
Brain	2	20
Heart	0.5	9
Kidneys	0.4	8
Adipose tissue	21.4	4
Miscellaneous (bones, intestines, lungs, etc.)	33.1	16

Source: Adapted from Schutz, Y., and E. Jequier. 2004. In *Handbook of Obesity, Etiology and Pathophysiology,* 2nd ed., ed. G. A. Bray and C. Bouchard, 618–622. New York: Marcel Dekker, Inc. and Elia, M. 1992. In *Energy Metabolism. Tissue Determinants and Cellular Corollaries,* ed. J. M. Kinney and H. N. Tucker, 61–79. New York: Raven Press.

within ~10% of basal metabolic rate. About 75% of the variability in basal metabolic rate is predicted by lean body mass (Hill, Saris, and Levine 2004; Dériaz et al. 1992; Ford 1984). Similarly, skeletal muscle mass is the best predictor of resting metabolic rate (Dériaz et al. 1992). Elia (1992) has published an excellent review of the contribution of the various organs and tissues to the basal metabolic rate (Schutz and Jequier 2004). The major contribution of resting metabolic rate stems from organs with high metabolic activity such as the liver, kidneys, brain, and heart, although these account for approximately 5% of the total body weight (Schutz and Jequier 2004). Table 8.9 displays the organs and tissues of the body and their contribution to basal metabolic rate.

Resting metabolic rate is important in the energy balance equation because it represents 60% to 75% of total daily energy expenditure in the average sedentary person (Poehlman 1989). Resting metabolic rate is proportional to the fat-free mass of an individual, and after age 20 it decreases approximately 2% and 3% per decade in women and men, respectively. Females have a lower resting metabolic rate at all ages, due primarily to their lower fat-free mass. However, when resting metabolic rate is expressed per unit of fat-free mass, there is no gender difference. Resting metabolic rate can be expressed in several different ways, including:

- Kilocalories per day
- Kilocalories per kilogram per day
- Kilocalories per kilogram fat-free mass per day
- Kilocalories per minute

Obtaining an accurate measure or estimation of resting metabolic rate is important because this information can be used to measure an individual's daily energy

needs. Specifically, once resting metabolic rate is determined, it can be multiplied by an activity factor. (Refer to Section 8.6, "Estimating Total Daily Energy Needs," for information about activity factors.)

8.5.1.1 Resting Metabolic Rates of Sedentary Versus Active Individuals

Using indirect calorimetry, researchers assessed the resting metabolic rates of sedentary, aerobically trained, and resistance-trained females (Ballor and Poehlman 1992). The average age of the females was 27 and those that were in the exercise groups had been participating regularly in that mode of exercise (either resistance training or aerobic training) for at least 2 years. The sedentary females had a resting metabolic rate of 1375 kcal (0.95 kcal/min; 22.7 kcal/kg), the aerobically trained group had a resting metabolic rate of 1485 kcal (1.03 kcal/min; 25.6 kcal/kg), and the resistance-training group had a resting metabolic rate of 1464 kcal (1.02 kcal/min; 25.1 kcal/kg). The resting metabolic rates for the exercise groups (both the aerobic and resistance-trained groups) were ~7% greater as compared to the sedentary group. When the resting metabolic rates of the groups were compared relative to the fat-free mass of the groups, the resting metabolic rates were 29.2, 30.5, and 29.5 kcal/kg fat-free mass for the sedentary group, aerobic group, and resistance-training group, respectively. Reporting resting metabolic rates in relation to fat-free mass greatly minimizes the perceived differences that are observed when reported in kilocalories per day and kilocalories per kilogram of body mass per day.

A similar study investigated the resting metabolic rates in males who were also sedentary, aerobically trained, or resistance trained (Poehlman et al. 1992). The average age of the males was 24 and those that were in the exercise groups had been participating regularly in that form of exercise for 4 years. The calculated resting metabolic rates of the three groups were as follows:

- Sedentary group = 1714 kcal (1.19 kcal/min; 20.9 kcal/kg)
- Aerobic group = 1843 kcal (1.28 kcal/min; 26.3 kcal/kg)
- Resistance-training group = 1973 kcal (1.37 kcal/min; 23.5 kcal/kg)

When the resting metabolic rates of the groups were compared relative to the fat-free mass of the groups, the resting metabolic rates were 25.7, 28.8, and 25.6 kcal/kg fat-free mass for the sedentary group, aerobic group, and resistance-training group, respectively.

8.5.1.2 Resting Metabolic Rate Prediction Equations

While indirect calorimetry is a very good lab method of measuring basal and resting metabolic rate, it is a laboratory method and may not be available or practical for use in all situations. Because of this, several equations have been proposed to estimate the basal and resting metabolic rates of individuals and athletes (Cunningham 1980; Harris and Benedict 1918; Owen et al. 1986, 1987; Mifflin et al. 1990). These and other resting metabolic rate prediction equations are shown in Table 8.10. An issue with the utilization of many of these prediction equations is that they have not been validated in athletic populations.

Two different investigations have measured resting metabolic rate in male and female athletic populations via indirect calorimetry and then compared this

TABLE 8.10
Resting Metabolic Rate Prediction Equations

Source	RMR Prediction Equation
Cunningham (1980)	RMR = 500 + 22 (fat-free mass in kg)
De Lorenzo et al. (1999)	RMR = –857 + 9 (wt in kg) + 11.7 (ht in cm)
FAO/WHO/UNU (1985):	
• Males (age 18–30)	RMR = 15.4 (wt) – 27 (ht) + 717
• Females (age 18–30)	RMR = 13.3 (wt) + 334 (ht) + 35
Harris and Benedict (1918):	
• Males	RMR = 66.47 + 5 (ht in cm) + 13.75 (wt in kg) – 6.76 (age)
• Females	RMR = 655.1 + 1.85 (ht in cm) + 9.56 (wt in kg) – 4.68 (age)
Mifflin et al. (1990):	
• Males	RMR = 9.99 (wt in kg) + 6.25 (ht in cm) – 4.92 (age) + 5
• Females	RMR = 9.99 (wt in kg) + 6.25 (ht in cm) – 4.92 (age) – 161
Owen et al. (1987):	RMR = 879 + 10.2 (wt in kg)
• Males	RMR = 290 + 22.3 (fat-free mass in kg)
Owen et al. (1986):	
• Female athletes	RMR = 50.4 + 21.1 (wt in kg)

Notes: RMR = resting metabolic rate; kg = kilograms; wt = weight; ht = height; cm = centimeters.

measured value with the predicted value of several published RMR prediction equations. Thompson and Manore (1996) used the prediction equations of Harris and Benedict (1918), Mifflin et al. (1990), Owen (male [1987] and female [1986]), and Cunningham (1980) in their study. The Cunningham equation (1980) was found to be the best prediction equation for both male and female endurance athletes. In male athletes, fat-free mass was found to be the best single predictor of measured resting metabolic rate (energy intake was the single best predictor for the female athletes). Studies have shown that fat-free mass accounts for the largest portion of variation in resting metabolic rate in men (Bader et al. 2005; Speakman and Selman 2003; Mifflin et al. 1990; Owen et al. 1987), which could lead to the conclusion that the use of fat-free mass in a prediction equation (such as that used in the Cunningham equation) would increase the accuracy of the equation (Thompson and Manore 1996).

In contrast to the male endurance athletes, the best single predictor of resting metabolic rate in the female endurance athletes was energy intake (Thompson and Manore 1996). This finding was also reported by other investigators who found that resting metabolic rate in trained subjects was influenced by the total energy flux throughout the body (Bullough et al. 1995). On the other hand, low energy intakes can depress resting metabolic rate (Tremblay et al. 1986). While more research needs to be done in this area, it appears as if individuals who expend a relatively large amount of energy and match this expenditure with a large energy intake have an elevated resting metabolic rate. In contrast, resting metabolic rate is lower when athletes both consume and expend less energy (Thompson and Manore 1996).

In the other study investigating resting metabolic rates, Carlsohn and colleagues (2011) utilized the Harris and Benedict (1918) and Cunningham (1980) equations to predict resting metabolic rate in highly trained male and female rowers and canoeists. To assess the accuracy of these prediction equations, resting metabolic rate was also measured via indirect calorimetry. For the male athletes, measured resting metabolic rate was 2675 kcal. Both the Cunningham (2260 kcal) and the Harris and Benedict (2133 kcal) equations significantly underestimated resting metabolic rate in the male athletes. For the female athletes, the predicted RMRs using either the Cunningham (1734 kcal) or the Harris and Benedict (1737 kcal) equation tended to be higher than (but not significantly different from) the measured resting metabolic rate (1577 kcal).

Both prediction equations significantly underestimated the RMR in males. Specifically, estimations of 2260 kcal (Cunningham equation) and 2133 kcal (Harris and Benedict equation) were far below the measured RMR of 2675 kcal. For both genders, the mean differences between the prediction equations and the measured resting metabolic rate were 133 kcal/day and 202 kcal/day for the Cunningham and Harris and Benedict equations, respectively (Carlsohn et al. 2011). Therefore, the Cunningham equation (1980) was superior to the Harris and Benedict equation (1918) in terms of predicting actual RMR in male and female athletes.

In both of the investigations discussed here, the Cunningham equation best predicted resting metabolic rate for both male and female endurance athletes. Although this equation appears to be better than other equations, it still differs from measured resting metabolic rate. One of the primary reasons why resting metabolic rate prediction equations do not accurately predict resting metabolic rate in athletes is because the populations from which these equations were derived are not from active/athletic populations. In order to address this problem, an investigation was undertaken to create a prediction equation for estimating resting metabolic rate in male athletes (De Lorenzo et al. 1999). In this study, 51 male athletes (22 water polo, 12 judo, and 17 karate athletes) who exercised at least 3 h/day had their resting metabolic rates measured via indirect calorimetry. It was reported that the average resting metabolic rate for all male athletes was approximately 1929 kcal/day (24.7 kcal/kg or 28.8 kcal/kg fat-free mass). Also, relationships between measured resting metabolic rate and the different predictive variables were evaluated and subsequently used to develop a predictive equation for male athletes. The equation that provided the best prediction for resting metabolic rate in this athletic population included both height and weight and is given by the following:

Resting metabolic rate (kilocalories per day) = –857 + 9.0 (weight in kilograms) + 11.7 (height in centimeters)

In addition to formulating a new prediction equation for athletes, the researchers also compared the measured resting metabolic rate with several other prediction equations including:

- FAO/WHO/UNU (1985)
- Harris and Benedict (1918)

- Mifflin et al. (1990)
- Owen et al. (1987) (male prediction equation)
- Cunningham (1980)

All of the prediction equations (with the exception of the Cunningham et al., 1980, equation) significantly underestimated measured resting metabolic rate. The Harris and Benedict (1918) equation was the most accurate (underestimated resting metabolic rate by 49 kcal/day) followed by the Cunningham equation (overestimated resting metabolic rate by 59 kcal/day).

While fat-free mass appears to be the best single predictor of resting metabolic rate, other variables such as age, height, body weight, and energy flux are also important contributors to predicting the variance in resting metabolic rate. Of the available options for prediction equations, the De Lorenzo et al. (1999) or the Cunningham et al. (1980) prediction equations are the best choice for predicting resting metabolic rate in athletes (Carlsohn et al. 2011; Thompson and Manore 1996).

8.5.2 Thermic Effect of Food

The second component of total daily energy expenditure is the thermic effect of food (also called dietary induced thermogenesis) (Kinabo and Durnin 1990a,1990b). The energy that is released from the catabolism of carbohydrates, protein, and fat is approximately 4, 4, and 9 kcal/g, respectively (Livesey 2001). The thermic effect of food is mainly due to the energy cost of digesting, absorbing, transporting, and storing the various macronutrients. The total thermic effect of food over a 24-h period represents ~10% of the total daily energy expenditure in sedentary individuals.

The thermic effect of food actually increases one's metabolism above the normal baseline energy expenditure for a period of time (possibly several hours) after a meal (Tappy 1996). There are two different techniques to measure the thermic effect of food. The better technique is to measure energy expenditure following a meal during a 3- to 5-h period and to compare the values with a control test during the same period of time, after a zero-energy drink is given (Schutz and Jequier 2004). The other method involves measuring a baseline resting energy expenditure in a postabsorptive subject during a 1-h period. Thereafter, a meal is given to the subject and energy expenditure is continuously measured during a 3- to 5-h period. The area under the curve over the baseline represents the thermic effect of the meal (Schutz and Jequier 2004).

Certain macronutrients require more energy than others (i.e., have a higher thermic effect) to digest, absorb, transport, and store. Expressed in percentage of the energy content of the nutrient, values of 8%, 2%, and 20%–30% have been reported for carbohydrate, fat, and protein, respectively (Tappy 1996; Tappy and Jéquier 1993; Jéquier, Acheson, and Schutz 1987). In other words, fat has a relatively low thermic effect, protein has the highest thermic effect, and carbohydrate is in the middle. What accounts for the differences in the thermic effects of the macronutrients? Carbohydrate-induced thermogenesis mainly results from the energy cost of glycogen synthesis and substrate cycling (such as the cycling of glucose to glucose 6-phosphate and back to glucose) (Schutz and Jequier 2004; Jéquier et al. 1987). The

2% increase in thermogenesis for dietary fat is explained by ATP (adenosine triphosphate) consumption in the process of free fatty acid re-esterification to triglyceride (Schutz and Jequier 2004). The ~25% increase in thermogenesis of dietary protein is explained by the two pathways of amino acid metabolism. During digestion, proteins are degraded to amino acids. After absorption, amino acids are deaminated, the amino group is transferred to urea, and their carbon skeleton can be converted into glucose or other metabolites. The second pathway of amino acid metabolism is protein synthesis, which also accounts for ~25% of the energy content of amino acids (Schutz and Jequier 2004).

From a practical perspective, it is important to keep the thermic effect of food in mind when formulating a nutritional strategy to enhance weight gain or weight loss because all calories are not the same biologically. For example, if an athlete consumes 300 kcal of extra dietary protein (such as chicken breast) every day for a year as opposed to 300 kcal of carbohydrate (such as soda) for the same length of time, it could be theorized that they most likely would not have the same effect regarding weight gain due to the thermogenic properties of these different macronutrients. When considering the thermic effect of food, the common phrase "a Calorie is a Calorie" is not true.

8.5.3 Thermic Effect of Activity

The third component of total daily energy expenditure (in addition to resting metabolic rate and the thermic effect of food) is the energy expenditure of physical activity, referred to as the thermic effect of activity or activity thermogenesis. Physical activity is the most variable component of total daily energy expenditure. In contrast to the other components, it can be voluntarily modified by the behavior of the athlete. The range of activity thermogenesis is wide, ranging from ~15% of total daily energy expenditure in sedentary individuals to 50% or more of total daily energy expenditure in highly active individuals, such as athletes (Livingstone et al. 1991; Dauncey 1990). Activity thermogenesis can be divided into two categories: that associated with nonexercise activities (such as the activities of daily living) and that associated with purposeful exercise (such as sport practices and competitions). Given that athletes engage in high amounts of physical activity during sport practices and competition, it is beneficial to have an understanding of how much energy is expended during such periods.

8.5.3.1 Methods for Determining Energy Expenditure of Athletes

The best method of measuring the thermic effect of activity is direct calorimetry. However, most athletes do not have access to the expensive equipment needed for this method. The next best method is indirect calorimetry, which relies on the measurement of oxygen consumption and is less expensive, smaller, and more portable than direct methods. In fact, some manufacturers produce portable indirect calorimetry devices that allow an athlete to wear the calorimeter while participating in his or her respective sport (Figure 8.3). While more practical than direct calorimetry, wearing portable respiratory gas collection equipment may be cumbersome and not ideal during all daily activities. Due to the limitations of direct and indirect calorimetry, less obtrusive (and, unfortunately, less accurate) methods of estimating energy

FIGURE 8.3 Portable indirect calorimetry device. (Photo courtesy of Cosmed USA, Inc.)

expenditure have been developed. The following list provides details about common approaches to estimating energy expenditure during physical activity (Howley and Thompson 2012):

- Accelerometers. These devices are worn on the limbs of the body and track the acceleration of the body and thus provide information about the intensity, duration, and frequency of physical activity. Uniaxial accelerometers tend to overpredict slightly the energy expenditure during those activities that involve ambulation, like level walking or running (McMurray 2011; Bassett et al. 2000; Nichols et al. 1999) and underpredict the energy cost of activities that involve arm movement or external work, like stair climbing or hill walking (McMurray 2011; Jakicic et al. 1999). Also, uniaxial accelerometers are ineffective for measuring energy

expenditure for activities that do not involve ambulation, such as swimming, cycling, and weightlifting (McMurray 2011). Some types of accelerometers, called triaxial or omnidirectional accelerometers, are able to gather physical activity data in three dimensions (the sagittal, frontal, and transverse planes).

- Pedometers. These devices track the number of steps an individual takes and can give information about the distance walked when stride length is programmed into the device (Butte, Ekelund, and Westerterp 2012; Bassett et al. 2008). Drawbacks include not providing information about the intensity or rate of walking and that nonlocomotor activities (such as biking, swimming, and resistance exercise) are not recorded.
- Heart rate monitors. These devices are very practical for use and are a good measure of intensity of the exercise bout. Heart rate monitors are very popular because this relatively inexpensive method allows the athlete to be assessed in a free-living state. Heart rates have the potential to provide information on the pattern of activity as well as a general estimate of energy expenditure (McMurray 2011). However, several limitations of utilizing heart rates to estimate energy expenditure exist. Heart rate only represents metabolic rate when a steady-state condition is achieved. Thus, during anaerobic activities (such as sprinting) in which heart rates are elevated above metabolic rate, the use of heart rate can provide inaccurate results (McMurray 2011). Due to this and other limitations, heart rates are commonly used for minute-by-minute training intensities rather than to estimate energy expenditure (McMurray 2011).

In addition to the aforementioned methods, the MET (metabolic equivalent) is sometimes used to express the energy cost of physical activity. Since the average resting metabolic rate for an adult is close to 3.5 mL/kg min of oxygen, or 1 kcal/kg body weight/h, the energy cost of activities can be expressed as multiples of resting metabolic rate, and they are called METs. The use of METs to estimate energy expenditure takes body weight into account and is a relatively simple approach. A range of activities—from sleeping to engaging in a competitive basketball game—can be categorized by their MET value. Tables have been developed for estimating energy expenditure of most physical activities, including work, transportation, and sports activities (McMurray 2011; Ainsworth et al. 2000). In terms of METs, normal individuals have a maximal capacity of 10–13 METs and highly trained endurance athletes can reach a capacity of 20–24 METs (McMurray 2011). Ainsworth and colleagues (1993, 2000) have published a comprehensive list of MET values of common activities. Table 8.11 summarizes a selection of these MET values.

8.6 ESTIMATING TOTAL DAILY ENERGY NEEDS

Throughout this chapter the energy balance equation has been discussed in relation to its two components: energy intake and energy expenditure. Determining total energy expenditure for athletes can have a positive impact on their health and their ability to train and compete in their respective sports. Also, knowledge of the total daily energy

TABLE 8.11
Specific MET Values for Selected Physical Activities

Light (<3 METs)	Moderate (3–6 METs)	Vigorous (>6 METs)
Lying or sitting quietly (1.0)	Water aerobics (4.0)	Tennis—singles (8.0)
Sitting or standing (1.0)	Track and field—shot, discus (4.0)	Basketball game (8.0)
Riding in a car (1.0)	Walking briskly—3.5 mph (4.0)	Competitive volleyball (8.0)
Standing (2.0)	Golf—walking (4.5)	Jogging at 5 mph (8.0)
Mild stretching (2.5)	Track and field—high/long jump (6.0)	Jogging at 6 mph (10.0)
Walking 2 mph (2.5)	Vigorous weight lifting (6.0)	Running at 7 mph (11.5)

Source: Adapted from Ainsworth, B. E. et al. (2000). *Medicine & Science in Sports & Exercise* 32 (9 Suppl): S498–S504, and Ainsworth, B. E. et al. (1993). *Medicine & Science in Sports & Exercise* 25 (1): 71–80.

expenditure is essential in those situations where planned adjustments are made to the energy balance equation in order to lose weight (fat mass) or to gain lean muscle mass. When utilizing the concept of the energy balance equation in formulating a dietary plan for an athlete, the proper sequences of actions are the following:

1. Measure/estimate total daily energy expenditure.
2. Determine an optimal level of energy intake based upon the goal of the athlete:
   - For energy balance, match energy intake with energy expenditure.
   - For fat loss, set energy intake lower than energy expenditure.
   - For weight gain, set energy intake above energy expenditure.

To measure energy expenditure accurately, resting metabolic rate, the thermic effect of food, and energy expended during physical activity are measured via direct or indirect calorimetry. While this is the preferred method of measuring total daily energy expenditure, it is often not practical. Therefore, more simplified approaches have been put forth to estimate these variables of energy expenditure (i.e., resting metabolic rate prediction equations, assuming a 10% daily energy expenditure due to the thermic effect of food, and the use of indirect methods such as heart rate monitors, pedometers, and accelerometers to estimate the energy expended during physical activity).

Using these simplified approaches can be beneficial and provide approximations in determining total daily energy expenditure. However, another approach that is commonly used to estimate total daily energy expenditure is to express it as a multiple of some baseline value, such as resting metabolic rate. Once a value for resting metabolic rate has been obtained by either measurement or estimation, total daily energy expenditure can be estimated by a variety of methods that differ in labor intensiveness (Manore, Meyer, and Thompson 2009). The least labor-intensive methods multiply the resting metabolic rate by an appropriate activity factor to estimate total daily energy expenditure (Manore et al. 2009). Although no standard activity factors exist, activity factors of 1.3 to 1.5 are commonly used with sedentary people

Estimating total daily energy expenditure of an adult male:
662 – 9.53 (age in years) + PA Factor [15.91 (weight in kgs) + 539.6 (height in meters)]

Estimating total daily energy expenditure of an adult female:
354 – 6.91 (age in years) + PA Factor [9.36 (weight in kgs) + 726 (height in meters)]

PA = Physical Activity factor and is defined as:

1.0 – 1.39	Sedentary, typical daily living activities
1.4 – 1.59	Low active, typical daily living activity plus 30-60 min of daily moderate activity (walking at 3-4 mph)
1.6 – 1.89	Active, typical daily living activities plus 60 min of daily moderate activity
1.9 – 2.5	Very active, typical daily activities plus at least 60 min of daily moderate activity plus an additional 60 min of vigorous activity of 120 min of moderate activity

FIGURE 8.4 DRI method for estimating total daily energy expenditure of males and females. Adapted from Rodriguez, N. R., N. M. DiMarco, S. Langley, American Dietetic Association, Dietitians of Canada, American College of Sports Medicine 2009. Position of the American Dietetic Association, Dietitians of Canada, and the American College of Sports Medicine: Nutrition and athletic performance. *J. Am. Diet. Assoc.* 109 (3): 509–27.

and activity factors greater than 2 can be used with athletes engaging in intense and voluminous training.

As an example, if a collegiate tennis player has a resting metabolic rate of 1400 Calories/day and an activity factor of 1.6, the estimated total daily energy expenditure would be 2240 Calories. An equation that encompasses several variables (including age, weight, and height) to estimate total daily energy expenditure is the dietary reference intake (DRI) method (Institute of Medicine 2005) (Figure 8.4). Another method that utilizes activity factors is shown in Table 8.12. The reason for obtaining a resting metabolic rate measurement and then multiplying this measure by some activity factor is to set a caloric value for the athlete to remain in energy balance. Chapter 9, "Enhancing Body Composition: Gaining Muscle and Losing Fat," discusses the use of resting metabolic rate assessments and activity factors in situations in which the athlete wishes to decrease fat mass or increase lean muscle mass.

8.7 LIMITATIONS OF THE ENERGY BALANCE EQUATION

Weight gain occurs when food intake exceeds energy expenditure for an extended period of time (Martinez 2000; Flatt 1995; Westerterp 1993). Conversely, body mass will decrease when food intake is below energy expenditure for extended periods of time (Idoate et al. 2011; Kerksick et al. 2009). In this context, the energy balance equation functions as a valid and reliable tool, predicting that alterations in energy intake and energy expenditure will change body mass.

When making specific dietary changes, such as decreasing caloric intake by 500 kcal/day, the energy balance equation does not predict actual losses of body mass

TABLE 8.12
Estimation of Total Daily Energy Expenditure Using RMR and Activity Factors

Activity Level	Activity Factor (multiply by RMR)
Very light activity/exercise	~1.3
Light activity/exercise	~1.5 to 1.6
Moderate activity/exercise	~1.7 to 1.8
Heavy activity/exercise	~2.0
Very heavy activity/exercise	~2.4

Instructions: Multiply RMR (in kilocalories) by the activity factor that corresponds to the amount of physical activity/exercise that the athlete engages in on a typical day.

over periods of time (weeks to months). Practitioners in the fitness and nutrition professions have incorrectly used the energy balance equation to predict exact losses of body mass (ideally from fat stores) from specific changes in caloric reductions or increases in energy expenditure.

As an example, consider a female athlete who is 5 ft. 7 in. tall (1.7 m) and weighs 150 lb. (68 kg). This athlete would like to lose 10 lb. (4.5 kg) of fat mass. She will not alter her training and conditioning in any way; rather, her strategy for fat loss will be to reduce her energy intake by 500 kcal/day. Assuming that 1 lb. of stored body fat is equivalent to 3500 kcal, a 10-lb. (4.5-kg) loss of fat is equivalent to a caloric deficit of 35,000 kcal. By reducing her caloric intake by 500 kcal/day, she would have a weekly caloric deficit of 3500 kcal. At this rate, this athlete would reach her goal of creating a 35,000 kcal deficit in 10 weeks. The energy balance equation would accurately predict a general decrease in body weight, but it does not do a very good job of predicting the specific amount of weight that will be lost.

Taking a specific example from published literature, male wrestlers and judo athletes were instructed to reduce energy intake by 1000 kcal/day over a 3-week period (Fogelholm et al. 1993). During the actual study, it was reported that the athletes decreased their energy intake by approximately 750 kcal/day for 21 days, resulting in a caloric deficit of 15,750 kcal. Also, the authors estimated that daily energy expenditure increased by approximately 400 kcal/day, adding 8400 kcal to the total caloric deficit over the 21-day intervention. Using the energy balance equation, the predicted amount of weight loss would be 4.5 lb. (2.04 kg) from the decreases in energy intake and 2.4 lb. (1.09 kg) from the increases in daily energy expenditure, for a total predicted loss of 6.9 lb. (3.13 kg) of body mass. At the end of the 3-week period, it was reported that the male athletes lost an average of 8.1 lb. (3.7 kg) of body mass (Fogelholm et al. 1993). In this example, the energy balance equation correctly predicted that there would be a loss of body mass, but it underpredicted the amount of body mass that was lost by approximately 17%.

In another study (Foster-Schubert et al. 2012), overweight and obese women followed a hypocaloric diet for 12 months. During this dietary period, the overweight females reduced their caloric intake by approximately 250 Calories/day. Over the course of 1 year, this equates to a caloric reduction of about 90,000 Calories. Assuming that 1 lb. of stored body fat is equivalent to 3500 kcal, the energy balance equation predicts the amount of weight loss would be about 26 lb. (11.8 kg). However, the authors reported at the end of the 12-month investigation that the average weight loss for each participant was 15.6 lb. (7.1 kg)—only 60% of the predicted weight loss. In each of these examples (the wrestler weight loss study of Fogelholm et al., 1993, and the overweight female weight loss study of Foster-Schubert et al., 2012), the energy balance equation failed to predict actual weight loss accurately over a period of time.

Why does the energy balance equation not accurately predict specific amounts of body weight that will be lost when following a hypocaloric diet? McDonald (2013) reviewed the problems associated with the energy balance equation's ability to predict weight loss accurately. There are three general issues that partly explain the shortcomings of the energy balance equation:

- There are water balance issues.
- Weight loss comes not only from fat stores.
- Energy balance is not stationary.

(Another issue that needs to be stated is that there is also error in the measurements of the variables of the energy balance equation, including errors in measuring/predicting energy intake, errors in measuring/predicting energy expenditure, and errors in the measurement of body composition.)

For every gram of glycogen stored in the liver and skeletal muscle, there are approximately 3 g of water associated with it. Therefore, when glycogen is depleted during a period of weight loss, there is a corresponding loss of water, which manifests itself as a loss of body weight. Therefore, some of the body mass that is reduced results from water loss, rather than from any storage of energy (i.e., fat mass). The issue of water balance can work in the other direction also. For example, as Calories are reduced, there is a reduction in fat mass over time, but the body mass of the individual may not change due to his or her ability to retain water. Hence, water balance is an aspect that influences body weight but that is not accounted for in the energy balance equation.

It is often assumed that a 3500-kcal reduction results in 1 lb. of weight that will be lost. There are approximately 3,500 Calories in 1 lb. of fat, so this value would be true *if* all weight loss came in the form of fat loss. However, this is not the case. When body mass is reduced, some lean muscle mass is also lost (1 lb, of lean muscle mass is equivalent to approximately 550 Calories). The fact that not all body weight is lost as fat is another reason why the energy balance equation does not specifically predict actual changes in body mass over time.

Another assumption concerning the energy balance equation is that it is a "static" equation. This premise does not consider the effect that the weight gain or weight loss will have on energy expenditure (Swinburn and Ravussin 1993). When body mass

is lost, some fat-free mass is expected to be lost and this is associated with a reduction in resting metabolic rate (Schoeller 2009; Cunningham 1991). Additionally, a loss in body mass will also reduce the energy costs of physical activities of daily life (Schoeller 2009; Schoeller and Jefford 2002; Racette et al. 1995). The reverse is also true: When body weight increases as a result of a chronically elevated energy intake, there is a compensatory increase in the amount of energy used at rest and during daily activities and movements (Powers and Howley 2004).

When applying the energy balance equation to predict actual changes in body mass, it appears as if the equation is not valid. However, the energy balance equation is a valid tool as it is based on one of the fundamental properties of thermodynamics and it has been invaluable in understanding the interactions of energy intake, energy expenditure, and body composition (Schoeller 2009). It is the assumptions that many hold in consideration of the energy balance equation that are not valid. The best utilization of the energy balance equation is to use it as a starting point for planning changes in body composition (whether the goal is to lose fat or gain muscle). After these changes are implemented, routine body composition assessments should be the primary guide in making further changes to the athlete's diet. A detailed discussion of this concept can be found in the next chapter.

8.8 CONCLUSION

An understanding of the energy balance equation requires an understanding of how each of the contributing factors can be modified in order to improve an athlete's body composition. If appropriately applied, the energy balance equation can provide an initial strategy for manipulating energy intakes and physical activity to assist the athlete in losing fat, gaining muscle, or maintaining current body composition. Unlike the general population, the physical activity patterns for an athlete are typically defined and centered on sport-specific practices, resistance training, and competitive events. For this reason, most of the manipulations made to an athlete's total daily energy expenditure will focus on the energy intake side of the energy balance equation. Whenever changes to an athlete's body composition are desired, planned strategies must adhere to the principles of the energy balance equation and the steps outlined in this chapter for successful and timely outcomes. In the next chapter, a specific strategy will be presented that builds upon the basic foundation provided by the energy balance equation in assuring a scientifically sound approach to optimizing body composition.

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9 Enhancing Body Composition
Gaining Muscle and Losing Fat

Bill Campbell, Paul La Bounty, and Elfego Galvan

CONTENTS

9.1 Introduction 240
9.2 Four-Step Plan for Optimizing Body Composition 240
9.2.1 Obtain Baseline Data 241
9.2.1.1 Three-Day Food Record 241
9.2.1.2 Resting Metabolic Rate 241
9.2.1.3 Body Composition 242
9.2.1.4 Performance Measure 242
9.2.2 State the Goal 243
9.2.3 Set Daily Total Caloric Levels 244
9.2.4 Monitor Body Composition and Performance Frequently 246
9.3 Measuring Body Composition 246
9.3.1 Skinfold Method 248
9.3.2 Ultrasound 248
9.4 Principles of Fat Loss for Athletes 251
9.4.1 Principle 1: The Rate of Weight Loss Should Be Slow 251
9.4.2 Principle 2: Do Not Decrease Dietary Protein When Dieting 255
9.4.3 Principle 3: Resistance Train during Energy Restriction 256
9.5 Planned Reductions in Lean Body Mass 257
9.6 Meal Frequency 257
9.7 Principles of Weight Gain for Athletes 259
9.7.1 Importance of Resistance Training 259
9.7.2 Focus on Calories 260
9.8 Conclusion 262
References 263

9.1 INTRODUCTION

"Weight loss" is a phrase many individuals use when describing a decrease in body mass. Weight loss describes a condition in which body mass is decreased with no delineation if the decrease in body mass is derived from fat stores or lean muscle mass. Often, attempts at losing body mass are achieved at the expense of significant losses of lean muscle mass. Athletes invest a lot of time and exert a great deal of effort in obtaining (in the off-season) and then maintaining (during the season) lean muscle mass. With few exceptions, athletes should strive to maintain lean muscle mass gains. Therefore, attempts at decreasing body mass without regard to which bodily compartments the body mass is reduced from serves to undermine the athlete and his or her performance within their respective sports. In consideration of this, the term "weight loss" is contextually too broad. Rather, the term "fat loss" should be used when describing a comprehensive plan of reducing body mass for athletes. Using specific terminology such as this helps the athlete and his or her support staff in designing dietary and training strategies to emphasize fat loss without large losses of muscle mass.

In contrast to a loss of fat mass, some athletes seek to gain body mass. If a body mass-gaining program is not properly planned and executed, there may also be unwanted outcomes, such as increases in fat mass, which may compromise performance. This chapter presents a simple yet comprehensive strategy to guide the athlete and his or her support staff in their attempts to optimize body composition. Throughout this chapter, most of the examples and discussion will revolve around the assumption that the athlete desires to reduce fat mass. However, at times, athletes wish to increase lean body mass. Strategies for increasing lean body mass are discussed toward the end of the chapter in the Section 9.7, "Principles of Weight Gain for Athletes."

9.2 FOUR-STEP PLAN FOR OPTIMIZING BODY COMPOSITION

The strategy for optimizing an athlete's body composition consists of the following four steps:

1. Obtain baseline data.
2. Clearly state the body composition goal.
3. Set daily total caloric levels.
4. Monitor body composition and performance frequently.

While the system for optimizing body composition is simple, with only four steps, it is also methodical and scientific. In the mass media/weight loss culture, there is an aversion to "counting Calories" and monitoring body composition on a frequent basis (steps 3 and 4 in the plan). However, if an athlete is serious about altering body composition and maintaining or improving performance, these steps of weight loss/weight gain should not be avoided.

9.2.1 Obtain Baseline Data

Attempting to make changes to an athlete's body composition should not be taken lightly. In this regard, up to a 2-week period should be planned for when attempting to obtain the baseline data that will be utilized in making the necessary decisions and in formulating a strategy to achieve the athlete's body composition goals. In the baseline data collection period, it is recommended that the following measures be taken:

1. Three-day food record
2. Resting metabolic rate
3. Body composition
4. Performance measure

9.2.1.1 Three-Day Food Record

As was discussed in Chapter 8, "Energy Balance," a 3- to 7-day diet-monitoring period provides reasonably accurate and precise estimations of habitual energy and macronutrient consumption (Magkos and Yannakoulia 2003; Deakin 2000; Black 2001). A 3-day diet record is recommended for two reasons: (1) It is more representative of typical intake than a 1-day diet record, but not as cumbersome as a 7-day diet record, and (2) it is a valid measure of energy intake (Lührmann et al. 1999).

9.2.1.2 Resting Metabolic Rate

In addition to the 3-day food record, a resting metabolic rate measurement should be obtained (Figure 9.1). Such a measurement provides specific information about the athlete's metabolism and will highlight if there are any metabolic issues (such as having a suppressed metabolism). Another advantage of obtaining this measure is that as changes are made to the athlete's diet and training programs, progress can be matched against the athlete's actual resting metabolic rate. If it is determined that the athlete's metabolic rate is declining too much with the current energy intake reduction strategy, changes can be made in regard to energy intake or the training program so that metabolic rates can be optimized. If a resting metabolic rate cannot be obtained, the next best method is to estimate the athlete's resting metabolic rate with the Cunningham or De Lorenzo prediction equations. For more information about these prediction equations, refer to Chapter 8.

Once a 3-day food record and a measure (or estimation) of resting metabolic rate have been determined, the next step is to set the energy intake level that results in energy balance (where body mass is neither gained nor lost). This is accomplished by starting with the resting metabolic rate value and then multiplying this value by an activity factor that most closely matches the athlete's current activity levels. (Refer to Table 8.12 in Chapter 8 for a list that matches potential activity factors with different levels of physical activity.) For a 1- to 2-week period, the energy intake should be set to match the estimated energy expenditure (which was determined by multiplying the resting metabolic rate [RMR] by an appropriate activity factor). The goal during this 1- to 2-week period is to be in energy balance. If the athlete is in energy balance, then body mass will remain stable and there will be neither gain in nor loss of body

FIGURE 9.1 Resting metabolic rate canopy test.

mass. Assuming a successful energy balance period in which body mass remained constant, a specific caloric intake can then be set to accomplish the athlete's body composition goals. This is step 3 of the program and is discussed in Section 9.2.3, "Set Daily Total Caloric Levels."

9.2.1.3 Body Composition

It is essential that an estimation of body composition be made during the baseline measurements. The variables that are important to obtain and monitor over time are body fat percentage, fat mass, and lean body mass. Using either the skinfold method or ultrasound and integrating this into a two-compartment model is recommended for tracking the athlete's body composition due to the low cost, the quickness of the assessment, and the demonstrated accuracy. Three- and four-compartment models of body composition can also be utilized for even greater accuracy, but these methods are more time consuming and not as applicable in the field setting. In Section 9.3, "Measuring Body Composition," more information is provided in regard to the various body composition assessment methods available as well as their advantages and disadvantages.

9.2.1.4 Performance Measure

As body mass is decreased, there may be a loss of lean body mass, which negatively affects performance. By periodically measuring performance, the athlete will know if body composition changes are manifesting in performance gains or in performance decrements. Therefore, it is essential that performance measures be made so that if performance is suffering, changes can be made immediately to the training and dietary program. There are an infinite number of performance measures that can be obtained. However, a performance measure should be selected so that it is

quickly administered, easy to obtain, is nonfatiguing, and does not interfere with the athlete's normal training program.

Assuming that athletes of all types wish to develop as much power as possible, a simple power-to-weight ratio assessment is recommended. Power-to-weight ratio is the amount of power the athlete is able to generate per pound or kilogram of body weight. A simple test to measure an athlete's power-to-weight ratio is the vertical jump. Technically, the vertical jump test by itself does not measure power because it does not consider the weight or mass of the athlete performing the jump. However, the vertical jump test is validated and meets every one of the requisites for a frequent assessment of performance: It is easy to obtain, is nonfatiguing, and does not interfere with the athlete's normal training program. While the vertical jump is highlighted here for the aforementioned reasons, there are many other tests of performance that can be selected that may be appropriate for the athlete within a given sport. For example, a cross-country runner may choose a performance measure such as a 40-m sprint since this type of movement (running) more closely resembles his or her sport. Regardless of which test of performance is used, it is essential that the measure be made frequently, can be administered quickly, and is relatively nonfatiguing.

9.2.2 State the Goal

After the baseline data have been collected, and it is decided that a change in (or maintenance of) body composition is needed, it is important that a clear goal be stated. In totality, there are four goals that can be set for an athlete in terms of body composition:

1. Maintain current body composition.
2. Decrease fat mass.
3. Increase lean muscle mass.
4. Simultaneously increase muscle mass and decrease fat mass.

At times, particularly during the season, athletes will attempt to maintain their current body composition levels and seek neither to gain muscle mass nor decrease fat stores. Even in this regard, the athlete should be clear in stating this goal. At other times, particularly in the off-season, athletes desire to increase muscle mass and decrease fat mass. While this goal is common, it is important to note that attempts to reduce fat mass are more successful when all aspects of the athlete's dietary plan and training program are planned to accomplish the goal of reducing fat mass. Unfortunately, when the primary goal is to reduce fat mass, some lean muscle mass will also likely be lost. An effective strategy is to maximize fat loss while maintaining or reducing the loss of lean muscle mass as much as possible. Similarly, when the primary goal is to increase lean muscle mass, it is probable that some fat mass will be added. An effective strategy is to maximize lean muscle mass gains while maintaining or minimizing the gain of fat mass as much as possible.

Many athletes desire to increase muscle mass and decrease fat mass simultaneously. This is a very difficult proposition in individuals who already possess high levels of lean muscle mass and relatively low levels of fat mass, but it is not impossible. (Section 9.4.1 highlights research that documented a simultaneous increase of

lean body mass and a decrease in fat mass of elite athletes.) The reason that this is difficult is because of the necessary conditions that surround each of these two goals. For example, when attempting to lose body fat, caloric intake needs to be decreased to below maintenance levels (assuming that no changes in the strength and conditioning program are made). In such an environment, when total daily calories are below maintenance levels, it is difficult to increase lean muscle mass. On the other hand, when attempting to gain lean muscle mass, caloric intake levels are elevated above maintenance levels since the process of muscle protein synthesis is endergonic (requires a net input of energy). When caloric intake is above maintenance levels, the likelihood of adding fat mass exists. For these reasons, it is important to state the body composition goal clearly (such as increasing lean muscle mass *or* decreasing fat mass) and then to pursue this goal from a dietary and training program plan.

TOPIC BOX 9.1 FAT FROM THE ATHLETE'S PERSPECTIVE

Fat does not participate directly in the production of mechanical work, but does so indirectly as a source of energy. In this regard, body fat is essential in terms of energy production. However, excess fat, in the form of excessive adipose tissue, is detrimental. Fat mass above that which is necessary for maintaining the athlete's health prevents the athlete from achieving optimal performance.

9.2.3 Set Daily Total Caloric Levels

Once the goal has been clearly stated and baseline data have been collected, the next step for the athlete and his or her support staff is to set a total daily caloric level. If the goal is to reduce fat mass, the only way to reach this goal is to reduce energy intake, increase energy expenditure, or a combination of the two. Given that dietary intake is the primary variable affecting fat mass, the focus of this discussion will center on energy intake. Also, in many instances, the strength and conditioning program is already established and designed to maximize performance at the appropriate times. For this reason, changes to the training program are often not recommended solely for desired changes in body composition. In terms of energy intake, total daily Calories (nutritionist's Calories; see Chapter 8, Section 8.3) must be set to a value below the value that the athlete used to remain in energy balance (which was determined during the baseline data collection period).

There is often disagreement among nutrition professionals in regard to determining the extent to which energy intake levels should be reduced. Regardless of what caloric reduction strategy is chosen, if the decision made was not optimal, the frequent assessments of body composition and performance measures will expose this and adjustments can be made in a very timely manner. Many sports nutrition "experts" recommend decreasing Calories by an absolute amount, such as by 500 or 750 Calories/day. While reducing Calories is the preferred method for decreasing fat mass, setting absolute values is detrimental and does not take into consideration the current body mass and activity levels of individual athletes.

For example, if a female volleyball player has a resting metabolic rate of 1200 Calories and it is determined that her activity factor is 1.5, then to remain in energy balance she would need to consume 1800 Calories/day. If it was decided that this player needed to decrease body fat levels and a caloric deficit of 750 Calories was recommended, this would put her new caloric intake at 1050 Calories—150 Calories below her resting metabolic rate! This drastic reduction of Calories (42% below Calories needed to remain in energy balance) would result in weight loss and fat loss, but it would also certainly reduce lean body mass and ultimately cause her training and performance to suffer.

On the other hand, if an American football player's resting metabolic rate was 2500 Calories and his activity factor was 1.7, a caloric intake value of 4250 Calories would place this athlete in a state of energy balance. A 750-caloric reduction for this athlete would only be a 17.5% reduction in caloric intake and this amount is still considerably above his resting metabolic rate. Such a caloric reduction would result in a reduction in fat mass while minimizing losses to lean body mass. Also, because there would be minimal losses to lean body mass, exercise and sport performance would not be negatively impacted very much, if at all.

Reducing energy intakes based on an absolute caloric basis is to be avoided because it does not account for individual differences in body size, metabolic rates, and activity levels. Instead, reductions in energy intakes should be based on a percentage of energy balance values. Specifically, energy intake should be reduced by 15% to 25% of energy balance values. If there is no urgency to the reduction of fat loss, then a 15% reduction of energy intake is recommended. However, if there is a reason for a more rapid loss of fat mass, a 25% energy intake reduction can be implemented. When in doubt or if there is uncertainty about the rate of fat loss that should be pursued, a 20% energy intake reduction is recommended. Regardless of the extent to which energy intake is reduced, a frequent assessment of body composition and performance will provide invaluable insight into the effectiveness of the initial strategy, and if the results are not optimal, appropriate adjustments to the energy intake strategy can be implemented.

Following are two examples of caloric restriction and its effects on exercise performance in physically fit/athletic populations. In the first study, aerobically active men (having an average of 19% body fat and a VO_2max of 53 mL/kg/min) and women (having an average of 28% body fat and a VO_2max of 49 mL/kg/min) were randomly assigned to a diet group or an energy balance group for a 14-day period (Zachwieja et al. 2001). Body composition, muscular strength, muscular endurance, and endurance performance (5-mile run time) were measured before and after the 14-day dietary intervention, and an anaerobic capacity test (Wingate test) was conducted. During the study, each subject engaged in daily aerobic treadmill exercise and reduced caloric intake by an average of 24% below maintenance levels (for the diet group) and protein intake was approximately 1.46 g/kg body mass. In terms of exercise performance, energy restriction at 24% below maintenance levels did not impair any performance measures. In fact, when compared with baseline measures, several measures were significantly improved after energy restriction (muscle endurance, 5-mile run time, and muscular strength). Performance was also unchanged in the energy balance group. In summary, data from this investigation reveal that a

caloric restriction of 24% does not adversely affect exercise performance in the short term (2-week period) (Zachwieja et al. 2001).

In contrast, when Calories were restricted to levels exceeding 25% in an athletic population, exercise performance was compromised (Filaire et al. 2001). In this study, 11 judo athletes reduced their Calories to 30% below energy balance levels (well above the recommended range of 15%–25%) for a 7-day period. Protein intake was about 1.2 g/kg body mass/day. Performance measures included static strength (hand dynamometer), vertical jump, and repeated maximal jumps. These particular assessments were chosen due to their sport-specific capabilities. Following the 1-week diet intervention, performance was significantly reduced for left-hand grip strength and repeated maximal jump performance (30-s test). Based on the studies summarized here, it appears that a caloric restriction of more than 25% of total daily energy expenditure may be detrimental to athletic performance and thus should be avoided (Zachwieja et al. 2001; Filaire et al. 2001).

9.2.4 Monitor Body Composition and Performance Frequently

The only way to know objectively if fat mass is being lost or lean muscle mass is being gained is to assess these measures periodically. Also, if body composition goals are being met but the athlete's performance is suffering, changes to either the dietary or training program will need to be made. Again, the only way to determine objectively if performance is suffering is to assess performance. It is recommended that body composition and performance be measured as frequently as every other week, but not less than every month. While this may seem frequent, there are no drawbacks to such a recommendation. For example, when setting the daily total caloric levels for the athlete, if these levels were either too severe or not aggressive enough, the frequent body composition and performance assessment will highlight this so that appropriate changes can be made to the diet and training program. Without a frequent assessment of body composition and performance, mistakes made at the level of the program (both dietary and training) implementation will not be recognized until more damage has been done in relation to a failure to meet body composition goals or in the athlete's performance.

9.3 MEASURING BODY COMPOSITION

There are many different methods that can be used to measure/estimate body composition, ranging from simple to very sophisticated measures. For example, body composition can be estimated with hydrodensitometry (underwater weighing), dual-energy x-ray absorptiometry (DXA), skinfolds, air displacement plethysmography (via the BOD POD), ultrasound, and bioelectrical impedance (BIA). Each of these methods has its own advantages and drawbacks in terms of precision, reliability, validity, and practicality.

From a broad perspective, body composition can be estimated using a two-, three-, or four-compartment model. The goal in body composition is to measure one or more of these compartments and assume that the relationship between the compartments is constant to estimate another compartment. In the two-compartment model, the body is divided into fat mass and fat-free mass. Fat-free mass is more commonly

called lean body mass (also referred to as lean muscle mass), even though there is a technical difference: The fat-free mass represents the body mass devoid of all fat, whereas lean body mass contains a small percentage of essential fat. For the purpose of consistency, we will use the terms *lean body mass* or *lean muscle mass* throughout this text. In the three- and four-compartment models, fat mass and total body water are basic components. In the three-compartment model, in addition to fat mass and total body water, the third component is referred to as fat-free dry mass. In the four-compartment model, the fat-free dry mass is broken down into mineral and protein compartments. Figure 9.2 summarizes the two-, three-, and four-compartment models of body composition.

The four-compartment model is often regarded as the reference for estimating body composition. In practice, if a four-compartment model is used, three compartments are measured and the fourth is estimated as the difference between total body weight and the sum of the measured compartments. In the four-compartment model, fat is typically measured by densitometry or air displacement plethysmography, total body water is measured by deuterium dilution, and bone mineral content is measured by DXA. These methods are considered the reference methods for body composition. Even though the four-compartment model is regarded as the reference for estimating body composition, it is rather laborious to use and takes a considerable amount of time. For this reason, more attention will be given to the two-compartment model. In fact, when underwater weighing, BOD POD, or DXA is used on its own to estimate body composition, a two-compartment model is used. The two-compartment model is based on the conversion of body density to body fat percentage. The two classic equations used to predict body fat percentage from body density are (1) the Brozek equation (Brozek et al. 1963):

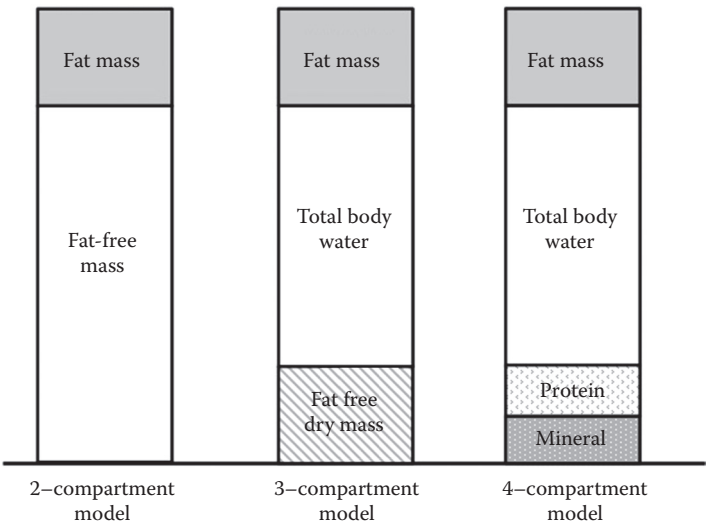

FIGURE 9.2 Two-, three-, and four-compartment models of body composition.

$$\text{Percentage body fat} = (4.57/\text{body density}) - 414.2)$$

and (2) the Siri equation (Siri 1961):

$$\text{Percentage body fat} = (4.95/\text{body density}) - 450$$

These models are based on the assumption that the densities of the fat mass and fat-free mass are the same for all individuals. The two-compartment models provide reasonable estimates of body fat percentage as long as the assumptions of the model are met. With that being said, age, gender, ethnicity, and physical activity level affect the density of the fat-free mass and introduce some error in predicting body fat percentage (Wagner and Heyward 2001; Modlesky et al. 1996; Baumgartner et al.1991). For this reason, two-compartment model equations (population-specific conversion formulas) have been developed for various age and ethnic groups based on the average fat-free mass densities reported for these groups.

9.3.1 Skinfold Method

The most common anthropometric procedure for measuring body composition utilizing the two-compartment model is the skinfold method. The reason why this method is commonly used is because it is cost effective, can be done quickly, and is able to estimate the average body fatness accurately in both male and female athletes (Houtkooper et al. 2001; Hortobágyi et al. 1992; Sinning et al. 1985; Sinning and Wilson 1984). However, the technician performing the measure must be highly skilled and be meticulous with both accurate site location and measurement technique. To become highly skilled in this technique, it is essential that the technician perform repeated assessments over time. Also, when tracking individual athletes over time, such as prior to and after dietary changes, it is important that the same technician collect the data (Hume and Marfell-Jones 2008).

Topic Box 9.2 lists recommended skinfold equations and some general procedures for obtaining skinfold measures. The skinfold method estimates body density to derive body fat percentage, fat mass, and lean body mass. There are many different population-specific equations to predict body density from various combinations of skinfold measures. It is important to use population-specific statistical equations when estimating both body density and body fat from skinfold measures. When using the skinfold method, it is recommended that multiple skinfold sites be measured (i.e., four sites for female athletes and seven sites for male athletes) and that skinfold measures be taken from the upper and lower body.

9.3.2 Ultrasound

Even though the skinfold method is valid, reliable, and relatively easy to perform, there are some drawbacks to using this method. These drawbacks include:

- Some error associated with the compressibility and elasticity of skinfolds

TOPIC BOX 9.2 SKINFOLD METHOD PREDICTION EQUATIONS FOR MALE AND FEMALE ATHLETES

Vivian Heyward and Dale Wagner (2004) authored a comprehensive text on body composition titled *Applied Body Composition Assessment*. In this text, Heyward and Dale recommend the following skinfold equations be used for male (Jackson and Pollock 1978; PMID: 718832) and female (Jackson, Pollock, and Ward 1980; PMID: 7402053) athletes:

Male athletes: body density = 1.112 – 0.00043499 (sum of seven skinfolds) + 0.00000055 (sum of seven skinfolds)^2 – 0.00028826 (age) (seven skinfolds for males include chest, midaxillary, triceps, subscapular, abdomen, suprailiac, and thigh)

Female athletes: body density = 1.096095 – 0.0006952 (sum of four skinfolds) + 0.0000011 (sum of four skinfolds)^2 – 0.0000714 (age) (four skinfolds for females include triceps, thigh, suprailiac, and abdomen)

General procedures to follow when obtaining skinfold measures include:

- Perform all measurements on the right side of the body.
- Grasp the skinfold firmly between the thumb and index finger of the left hand.
- Place the jaws of the caliper perpendicular to the fold.
- Take the measurement a few seconds after the pressure of the calipers is released.
- Take duplicate measures at each site.

- Accurate measurements in the obese (the skinfold thickness often exceeds the width of the caliper's jaws)
- Lack of reproducible results of the technician in consistency of grasping the skinfold for measurement
- The type of skinfold calipers used

Each of these drawbacks can be lessened with the use of an alternative, noninvasive method of surface anthropometry to measure subcutaneous adipose tissue—ultrasound. Specifically, the use of ultrasound for measuring subcutaneous adipose tissue improves the error associated with compressibility and elasticity of skinfolds (Ramirez 1992), improves the accuracy of skinfold measures in the obese (Kuczmarski, Fanelli, and Koch 1987), and eliminates the learned skill and associated errors in pinching/grasping the fold of skin that is needed when using calipers.

The practicality of using ultrasound for estimating body composition has improved due to the availability of more affordable ultrasound equipment and its higher resolution and portability. Figures 9.3 and 9.4 highlight a commercially available portable ultrasound device and the type of body scan that the device provides.

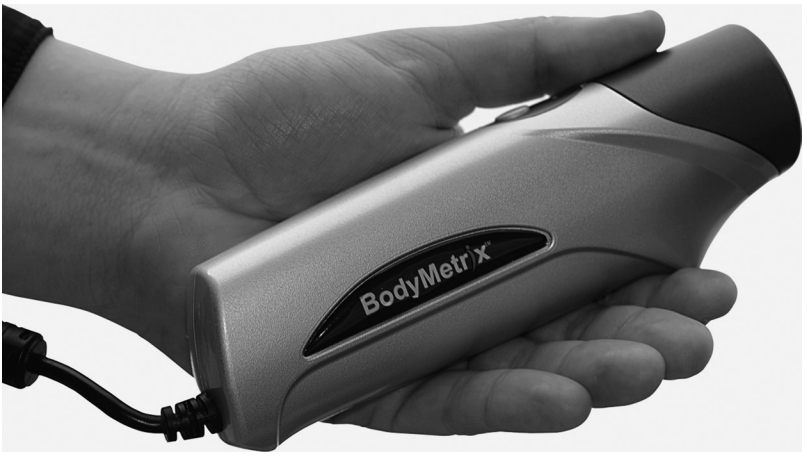

FIGURE 9.3 Ultrasound device used for the measurement of body composition.

The use of ultrasound for estimating body composition has been utilized in athletic populations. Results from these studies indicate that ultrasound technology is comparable to both underwater weighing (Utter and Hager 2008) and DXA (Pineau, Filliard, and Bocquet 2009). In addition to providing accurate body composition values, ultrasound can also provide skeletal muscle cross-sectional area. With this feature, changes to the diet and training programs of the athlete can be monitored in terms of their effects on specific muscle groups. In this manner, if the diet and

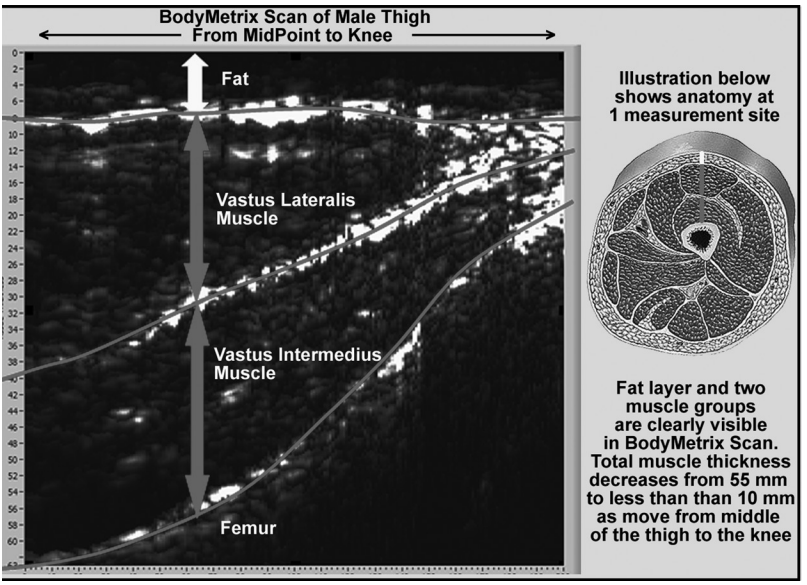

FIGURE 9.4 BodyMetrix scan of male thigh.

training program are focused in a manner to increase lean body mass, this specific variable can be monitored with ultrasound technology.

9.4 PRINCIPLES OF FAT LOSS FOR ATHLETES

There are three principles to which athletes should adhere when attempting to reduce Calories for the goal of losing body fat. Each of these principles seeks to prevent the loss of lean muscle mass and exercise performance:

1. The rate of weight loss should be slow.
2. Do not decrease dietary protein when dieting.
3. Perform resistance training during energy restriction.

9.4.1 Principle 1: The Rate of Weight Loss Should Be Slow

Generally, severe caloric restriction leads to greater body weight reduction; however, this type of approach to weight loss may have a greater negative effect on an athlete's training capacity and competition performance. Caloric restriction diets can be classified by the degree of caloric restriction implemented. Severe Calorie restriction diets are commonly known as very low-Calorie diets, while moderate Calorie-restriction diets are known as low-Calorie diets. A very low-Calorie diet (VLCD) is defined as a hypocaloric diet providing less than 800 kcal/day or less than 12 kcal/kg ideal body weight/day (National Task Force on the Prevention and Treatment of Obesity 1993). Additionally, VLCDs include 100% of the recommended daily allowance (RDA) of vitamins and minerals and typically comprise 70 to 100 g protein/day, 80 g carbohydrate/day, and 15 g fat/day.

This type of dietary approach is designed to induce rapid, short-term weight loss and is usually reserved for obese individuals (body mass index [BMI] ≥ 30 kg/m^2). It is important to understand that BMI is an indirect means for estimating body fat based on weight and height and is not a measure of percentage of body fat. Generally, athletes have a greater ratio of lean body mass than their nonathlete counterparts and thus an athlete may be incorrectly classified as obese. For example, the 2012 Summer Olympic judo heavyweight gold medalist measuring 6 ft. 8 in. (2.03 m) and weighing a lean (low body fat percentage) 290 lb. (~131.5 kg) would be considered obese based on his BMI of 31.9 kg/m^2.

VLCDs are utilized from 3 to 18 weeks (Fricker et al. 1991; Barrows and Snook 1987) and result in an average loss of 2.4–5.5 lb./week (1.1–2.5 kg/week) (National Task Force on the Prevention and Treatment of Obesity 1993). In 12 to 16 weeks on VLCDs, obese patients on average lose 44 lb. (20 kg) (National Task Force on the Prevention and Treatment of Obesity 1993), of which 25% comprised lean body mass (Bryner et al. 1999). The use of VLCDs should occur under the supervision of experienced health care professionals as their use can lead to severe health complications, including death (Tsai and Wadden 2006).

When athletes must reduce body weight, there should be an emphasis on minimizing the loss of lean muscle mass while maximizing fat mass loss; this cannot be accomplished if weight loss is achieved by VLCDs. Because of the loss of lean

body mass associated with VLCDs, these diets should be reserved only for individuals with moderate to severe degrees of obesity. Since lean muscle mass is of great importance to the success of most athletes, it is not advisable for athletes to rely on VLCDs as a modality to reduce body weight. In fact, the loss of lean mass is greater in nonobese than obese subjects, as obese subjects may be more efficient at conserving body nitrogen during severe caloric restriction (Forbes and Drenick 1979). Therefore, the loss of lean body mass associated with VLCDs may be even greater than 25% in athletes as they maintain lesser degrees of adiposity.

A better weight loss strategy (as compared to VLCDs) for athletes involves a moderate caloric-restriction diet as decreases in lean body mass with a low-calorie diet (LCD) are less severe than with VLCDs. LCDs consist of approximately 800 to 1500 kcal/day (Tsai and Wadden 2006) or 12–20 kcal/kg of ideal body weight per day of conventional foods (Atkinson 1989). LCDs are typically composed of 15%–22% protein, 48%–71% carbohydrate, and 13%–30% fat (Kiortsis, Durack, and Turpin 1999; Kraemer et al. 1997; Ballor et al. 1996, Sweeney et al. 1993). Researchers have reported a greater retention of lean body mass when weight loss occurs slowly with moderate Calorie restriction instead of rapidly with severe calorie restriction (Sénéchal et al. 2012; Sweeney et al. 1993).

Research has demonstrated a significantly greater retention of lean body mass (a loss of only 0.9 lb. [0.4 kg] vs. 6.4 lb. [2.9 kg]) and decreases in fat mass (an approximate 13-lb. [6-kg] loss vs. a 7-lb. [3-kg] loss) when a 5% weight reduction of initial body mass occurred slowly (~6 kg in 15 weeks) instead of rapidly (6 kg in 5 weeks) (Sénéchal et al. 2012). Another study reported a significantly greater loss of fat-free mass with VLCDs (~800 kcal/day) than with LCDs (~1400 kcal/day) (Sweeney et al. 1993). Although VLCDs for 24 weeks resulted in significantly greater weight loss (~33 lb. [15 kg] vs. 24 lb. [11 kg]) and body fat (~27 lb. [12 kg] vs. 18 lb. [8 kg]), the LCD group did not lose as much lean muscle mass (~4.4 lb. [2 kg]) as the VLCD group (~6.6 lb. [3 kg]).

Both of these aforementioned studies, which demonstrated a greater maintenance of lean body mass with a slow rate of weight loss, were conducted in obese populations. It is essential to view severe and moderate Calorie-restriction diets as a percent reduction of total daily energy expenditure and not solely on absolute caloric value. For this reason, while an LCD is clearly superior to VLCD in both obese and athletic populations in relation to maintaining lean muscle mass, an LCD is not appropriate for most athletes. For example, an elite junior basketball player with a total daily energy expenditure of ~4200 kcal/day (Silva et al. 2012) would consume only ~35% of maintenance energy requirement (energy balance) if prescribed the upper limit of LCD (i.e., 1500 kcal/day). A diet providing only ~35% of maintenance energy requirement would be classified as a severe Calorie restriction diet. With regard to sport performance (one of the most important factors an athlete should consider), severe Calorie restriction diets are associated with decreases in performance factors such as aerobic endurance capacity and muscular strength and endurance (Eston et al. 1992; Bender and Martin 1986; Horswill et al. 1990; Friedlander et al. 2005). Declines in performance factors will inhibit both training capacity and competition performance.

While these aforementioned studies were conducted in obese populations, the principle that they set forth should be followed by athletes as well: that the rate of weight loss should be slow. For the athlete, it is recommended that the rate of weight loss range from 0.7% to 1% per week. For a 200-lb. athlete, this would equate to 1.5 to 2 lb. (0.7 to 0.9 kg) of weight loss per week. For a 140-lb. athlete, this equates to 1.0 to 1.4 lb. (0.45 to 0.6 kg) of body weight lost per week. Weight losses at this rate (assuming that protein intake is adequate and the athlete is engaging in a resistance-training program) will result in fat loss, but will also prevent large losses of lean muscle mass. In fact, with this approach, it is possible that the athlete can simultaneously lose fat mass and gain lean muscle mass.

A landmark study in elite athletes reported that it is possible to increase muscle mass and decrease fat mass simultaneously (Garthe et al. 2011). In this study, elite athletes (both male and female) were recruited from the Norwegian Olympic Sport Center to participate in a weight loss program. The athletes participated in sports such as soccer, judo, skiing, track and field, cycling, ice hockey, and others. The male athletes were in their early 20s, possessed an average body fat percentage of 17%, had 13 years of experience as athletes, and engaged in 15 h of training per week, including about 3 h of strength training. The female athletes were also in their early 20s, possessed an average body fat percentage of 27%, had 12 years of experience as athletes, and engaged in 15 h of training per week, including about 2.5 h of strength training.

The purpose of the study was to compare changes in body composition, strength, and power during a weekly body-weight loss of 0.7% (slower weight loss group) or 1.0% (faster weight loss group). All of the athletes, regardless of which group they were randomly assigned to, ingested an average of 1.5 g protein/kg body weight (which adheres to principle 2 of weight loss for athletes described in this chapter) and resistance-trained 4 days/week (which adheres to principle 3 of weight loss for athletes described in this chapter). Both groups lost the same amount of body weight: about 9 lb. (4.2 kg). However, it took the slower weight loss group 8.5 weeks to lose this amount of weight as compared to 5.3 weeks for the faster weight loss group.

The impactful findings from this study surface when we look at the composition of the body weight that was lost. The athletes in the slower weight loss group lost significantly more fat mass (~11 lb. [5 kg]) than the athletes in the faster weight loss group (~7 lb. [3 kg]). Also, the slower weight loss group gained significantly more lean body mass (~2 lb. [1 kg]) than the athletes in the faster weight loss group, which lost a trivial amount of lean body mass (0.66 lb. [0.3 kg]). To state this again, the athletes in the slower weight loss group lost fat mass and gained lean muscle mass when reducing their Calories by 20% and losing 0.7% of their body weight per week (Garthe et al. 2011). While this accomplishment is difficult to achieve in athletic populations, this is evidence that elite athletes have the potential to gain lean muscle mass while simultaneously decreasing fat mass.

In addition to body composition, there were several measures of performance conducted in this study, including power measures (40-m sprint and vertical jump) and strength measures (1 RM [repetition maximum] bench press and 1 RM squat). There were basically no changes in the 40-m sprint for either group, but there were significant changes in muscular strength and countermovement jump performance, with the slower weight loss group performing better than the faster weight loss group.

Specifically, vertical jump significantly improved by 6% in the slower weight loss group, but only 1% in the faster weight loss group. In terms of strength measures, 1 RM bench press improved by 12.5% in the slower weight loss group, which was significantly higher than the 3% improvement in the faster weight loss group. Also, the 1 RM squat significantly improved by 9% in the slower weight loss group as compared to a 7.7% improvement in the faster weight loss group. The results reported in this study indicate that not only body composition is improved to a greater extent in a slower weight loss strategy (as compared to a faster weight loss strategy), but also strength and power measures (Garthe et al. 2011).

Three important points need to be made about the results reported in this landmark study. The first point is that the weight loss was designed to occur at a slow rate. Second, each athlete did not reduce protein intake to below recommended intake levels, but ingested an average of 1.5 g protein/kg body mass during the energy-restricted period. Last, the athletes engaged in a resistance-training program that emphasized strength and hypertrophy 4 days/week while maintaining their sport-specific training schedule.

When attempting to lose fat mass, it is recommended that athletes reduce caloric intake by about 20% and lose body weight at a rate of about 0.7% to 1.0% per week. To provide support for this recommendation, Pasiakos and colleagues (2010) reported that when energy intake was reduced by 19% below estimated energy requirements in young, physically active men and women, the rate of weight loss was approximately 1% in the first 7 days of a 10-day weight loss intervention. Protein intake was maintained at 1.5 g/kg body weight during the weight loss period. Unfortunately, body composition changes were not reported. This particular finding is precisely the results that an athlete would find acceptable—a moderate caloric reduction that results in a slow rate of weight loss.

In another study (which used aerobically active males and females), Calories were reduced to levels 24% below energy balance for a 2-week period, and the percent reduction in body weight was 1.75% over the 2-week period, equivalent to about 0.9% per week (Zachwieja et al. 2001). This rate of weight loss is within the recommended ranges, and several measures of exercise performance (such as endurance and muscular strength) actually improved during the 2-week intervention. However, despite these positive reports, the caloric reduction resulted in a loss of 2.6 lb. (1.2 kg) of body mass, of which fat mass accounted for about 39% of the weight loss and lean body mass accounted for 61% of the weight loss. (Remember that lean body mass includes both protein stores and body water, so it is very likely that some of these losses were from body water stores.) Even though performance was not negatively impacted, it is our contention that too much of the reductions in body mass were coming from lean body mass stores. Protein intake during the 2-week diet period was less than 1.5 g/kg body mass (authors reported 1.46 g of protein/kg body mass). Also, the participants in the study did not resistance train during the weight reduction period (the subjects were aerobically active during the study). There is some evidence to support the effects that higher protein intakes and adherence to a proper resistance-training program may have suppressed the losses of lean body mass observed in this study.

When judo athletes reduced their Calories (by 30%) for a 7-day period, the rate of weight loss was nearly 5%—well above the recommended 0.7% to 1.0% rate of weight loss (Filaire et al. 2001). Not only were sport-specific strength and power measures decreased during this period, but also body composition measures were negatively impacted. Specifically, about 7.3 lb. (3.3 kg) of body mass were lost during the 7-day diet period. Of this weight loss, fat mass accounted for about 30% of the weight loss and lean body mass accounted for 70% of the weight loss. While some of the lean body mass was accounted for by water content, it is likely that some of the losses were attributed to skeletal muscle losses. Protein intake was about 1.2 g/kg body mass/day, and there was no report that the judo athletes participated in a resistance-training program during the weight loss intervention. Improvements in protein intake and adherence to a resistance-training program would likely have maintained a greater proportion of lean body mass.

9.4.2 Principle 2: Do Not Decrease Dietary Protein When Dieting

When reducing Calories, it is important to maintain dietary protein intakes at appropriate levels. In obese populations undergoing caloric restriction, elevated protein intakes result in greater maintenance of lean body mass during weight loss (Gordon et al. 2008; Leidy et al. 2007; Garrow et al. 1981). Other investigations, including obese subjects, have also reported greater reductions in fat mass (Treyzon et al. 2008) and improved weight maintenance after body weight loss (Westerterp-Plantenga et al. 2004). In each of these studies, protein intakes were 20% to 100% higher for the group that experienced significant improvements in body composition as compared to the lower dietary protein intake groups.

Unfortunately, there have been only a few scientific investigations in athletic populations focusing on this topic. Taking data that have been reported in obese populations and extrapolating them to athletic populations is not advisable and may lead to inappropriate conclusions. However, it appears that the same benefits apply to athletes as have been demonstrated in obese populations in regard to maintaining lean body mass with increased protein intake during weight loss. A well-designed study compared the influence of a high-protein diet with a normal protein diet on lean body mass and performance in resistance-trained male athletes on a diet (Mettler, Mitchell, and Tipton 2010). Both groups reduced their caloric intake by 40% for a 2-week period. However, the high-protein group ingested 2.3 g protein/kg body mass and the normal protein group ingested 1 g protein/kg body mass. Body composition and measures of performance (which included vertical jump, peak force attained during a vertical jump, 1 RM bench press, and a Wingate test, among others) were conducted prior to and following the 2-week intervention.

Both groups lost the same amount of fat mass, but the normal-protein group lost significantly more lean body mass and total body mass than the high-protein group. Specifically, the normal-protein group lost 3.5 lb. (1.6 kg) of lean body mass while the high-protein group only lost 0.6 lb. (0.3 kg). In terms of performance, there were no significant differences between the two groups on any of the performance tests. Also, none of the performance measures decreased over the 2-week period, even though energy intake was reduced by 40%. It is important to note that the athletes

continued their resistance training during the 2-week period (about five times per week) and that it is likely that the training stimulus contributed to the maintenance of exercise performance during the investigation.

Focusing on the loss of body mass that each group experienced also highlights some interesting conclusions. The normal protein group lost 1.9% of their body mass per week, while the high-protein group lost 0.9% per week. This large difference in the rate of the weight loss was in spite of the fact that both groups reduced their energy intakes by 40%. The ingestion of higher protein intakes not only suppressed the loss of lean body mass, but also contributed to a rate of weight loss that is ideal for maintaining lean body mass (a loss of 0.7% to 1.0% per week).

Based on this and other reports, it appears that, during energy restriction, dietary protein intake should have priority status in order to preserve lean body mass. It is recommended during energy restriction that protein intake range from 1.5 to 2.3 g/kg body mass.

When this principle is adhered to, lean body mass is better maintained (Mettler et al. 2010) or even increased (Garthe et al. 2011) in athletic populations. When dietary protein intake is less than 1.5 g/kg body mass/day during energy restriction, a large amount of lean body mass is lost (61% to 70% of the weight loss coming from the lean body mass compartment) (Zachwieja et al. 2001; Filaire et al. 2001) in physically active and athletic populations.

Knowing that protein intake should not be reduced when decreasing Calories, where should the Calories be reduced? Once a caloric level is set below energy balance levels, it is recommended that fat Calories be reduced first, and then, if necessary, a reduction in carbohydrate intake may be needed to meet the energy intake goals in an effort to lose fat mass. In Chapter 2, "Dietary Fat Strategies for Performance Enhancement," an argument was made not to reduce dietary fat intake to levels less than 20% of total energy intake levels. While the data are lacking, one investigation reported that dietary fat intakes at levels below 20% of total energy intake reduced endurance performance (Horvath et al. 2000).

To put these concepts into a hypothetical example, let us assume that a 200-lb. (91-kg) baseball player needs to lose some fat mass. After he went through all of the baseline measures and following the principles set forth in this chapter for losing fat mass, it was determined that the athlete needed to reduce energy intake by 15%. During the baseline measurement period, it was determined that the athlete was ingesting 3000 Calories, of which 32% comprised dietary fat, 48% carbohydrates (ingesting 4 g carbohydrate/kg body mass/day), and 20% dietary protein (ingesting about 1.7 g protein/kg body mass/day). An energy intake reduction of 15% would set a goal of 2550 Calories on a daily basis. In this example, all of the reduction in caloric intake (all 450 Calories) could come from dietary fat intake. Under this situation, the reduced dietary fat intake would still account for approximately 20% of total energy intake, with no planned reductions in dietary protein or carbohydrate.

9.4.3 Principle 3: Resistance Train during Energy Restriction

The final principle that athletes must adhere to when attempting to lose fat mass is to engage in an appropriate resistance-training program. This aspect should be an

extension of the athlete's normal training and lifestyle due to the benefits that resistance training provides (strength/power improvement and prevention of injuries). When an athlete decreases energy intake to levels below energy balance, the role that resistance training provides is now additive in that it helps to maintain resting metabolic rate and muscular strength. In the studies reviewed in this chapter, several studies were summarized in which the athletes lost body mass. However, if resistance training was not a part of the dieting program, a majority of the body mass lost came from lean body mass stores, rather than fat mass stores (Zachwieja et al. 2001; Filaire et al. 2001). Also, exercise performance was significantly decreased during a period of energy restriction when resistance training was not included as part of the athlete's training regimen (Filaire et al. 2001).

In contrast, when athletes adhered to a resistance-training program, they were able to lose fat mass, retain lean body mass, and improve measures of sport-specific performance (Garthe et al. 2011). Recommending a specific type of resistance-training program is outside the scope of this chapter. However, the resistance-training program should follow a progressive, periodized format with a focus on increasing/maintaining maximal muscular strength.

9.5 PLANNED REDUCTIONS IN LEAN BODY MASS

Achieving weight loss at slow rates, at least in the short term, does not appear to decrease exercise performance (Garthe et al. 2011; Mettler et al. 2010). It is recognized that at times certain athletes may not desire to hold on to lean body mass when losing weight and may perform better with a lower amount of muscle tissue. In these circumstances, two approaches can be taken, both in conjunction with a reduction in energy intake. The athlete can cease resistance training until the ideal body weight (fat mass and lean body mass) is attained. Cessation of resistance training effectively eliminates the stimulus to the body that results in the maintenance or increase of skeletal muscle. This approach should be avoided.

A better approach for the athlete to take would be to continue to resistance train (so that sport-specific performance and strength can be maintained to the greatest extent possible) and to ingest dietary protein at levels less than 1.5 g/kg body mass. If energy and protein intake (less than 1.5 g/kg body mass) are reduced, the athlete will lose body mass, which will include lean body mass losses as well (Mettler et al. 2010). In most circumstances, the athlete will want to lose fat mass only and will want to maintain or even increase lean body mass. To facilitate this, it is important that the athlete lose body weight at a slow rate (0.7% to 1.0% per week), ingest 1.5 to 2.3 g protein/kg body mass on a daily basis, and engage in a resistance-training program.

9.6 MEAL FREQUENCY

Common interventions to improve body composition typically include various exercise and dietary interventions. When it comes to specific nutritional approaches to enhance body mass or body composition, the effects of meal frequency are often discussed and debated. It is commonly purported in fitness-related magazines, on blogs, and by "nutrition experts" that eating smaller but more frequent meals throughout

the day (i.e., 4–≥6 meals with or without snacks) is metabolically advantageous compared to following a more "traditional" eating regimen consisting of two to three larger meals (i.e., breakfast, lunch, and dinner). In other words, if you eat smaller but more frequent meals, it will assist in possessing a lower percentage of body fat.

However, does the preponderance of the existing peer-reviewed research published to date actually support this notion or are these assertions more theoretical in nature? Studies investigating the effects of meal frequency on various markers of health, diet-induced thermogenesis, energy expenditure, appetite, hunger, body weight, and body composition have been published. However, the focus of this section will examine the existing data on the effects that meal frequency has on body weight/body composition specifically.

Several observational studies in nonathletic populations have reported an inverse relationship between meal frequency and body mass/body fat (Fabry et al. 1964; Hejda and Fabry 1964; Metzner et al. 1977; Drummond et al. 1998) and BMI (Ruidavets et al. 2002; Franko et al. 2008; Antonogeorgos et al. 2012; Ritchie 2012). Other observational studies suggest that increased meal frequency alone does not seem to play a substantial role in improving body mass/body fat (Dreon et al. 1988; Kant et al. 1995; Titan et al. 2001). Furthermore, other research shows that eating more frequently may actually increase the chance of becoming overweight (Gunes et al. 2012; Stote et al. 2007). Knowing there are conflicting results, what can one conclude from these previously mentioned observational studies?

Bellisle, McDevitt, and Prentice (1997) noted in their extensive review that the observational studies that concluded decreased meal frequency actually increased body mass/body fat may be flawed due to two important factors: (1) the underreporting of dietary intake and (2) the concept of "reverse causality." These two aforementioned factors may make the interpretation of meal frequency studies difficult to conclude with certainty. For example, overweight people tend to underreport food intake more so than lower weight individuals (Braam et al. 1998). Thus, if someone is overweight or obese, he or she may report eating only two times a day, when in reality the individual may eat five times per day because he or she does not feel comfortable disclosing how much and how often eating actually takes place. Second, "reverse causality" is when an individual that is participating in a observational study initially reports that he or she is eating a certain number of meals per day and actually decreases the number of meals consumed during the study in attempts to mitigate weight gain (Bellisle et al. 1997). At the conclusion of the study, a researcher may falsely conclude that the participant gained weight as a result of eating fewer meals per day. However, in reality, it was just a strategy that might have been used to decrease total caloric intake in an attempt to lose weight.

Several experimental studies in nonathletic populations have also demonstrated that increased meal frequency did not seem to decrease body weight/body fat significantly (Wolfram et al. 1987; Cameron, Cyr, and Doucet 2010). However, less is known regarding the effects of increased meal frequency in athletic populations. As a result, caution must be taken when attempting to extrapolate meal frequency results from nonathletic populations to athletes. Currently, there are limited data examining the effects of meal frequency in athletic populations (La Bounty et al. 2011). Of the limited research, interestingly, two published studies (Iwao, Mori, and

Sato 1996; Deutz et al. 2000) and one published abstract (Benardot et al. 2005) have shown benefits of increased meal frequency on lean muscle retention (Iwao et al. 1996), body composition (Deutz et al. 2000; Benardot et al. 2005), and anaerobic power (Benardot et al. 2005). These limited findings could be significant for an athlete, particularly in weight-restricted sports, such as wrestling, boxing, mixed martial arts, etc., where hypocaloric diets are commonly employed and minimal excessive body fat is typically desired (La Bounty et al. 2011).

Therefore, if the frequency of eating can positively affect protein retention or body composition in athletes, then these outcomes alone may help shape the discussion on how often to eat. Regarding how much and how often one should eat, particularly protein, is a very popular question. Two well-respected researchers in the area of protein needs in athletes (Philips and van Loon 2011), in their recent review, recommend that athletes should ingest ~1.3–1.8 g/kg protein/day spread evenly over three to four meals to optimize muscle protein synthesis. This eating frequency recommendation approximates what is typically done in Western cultures. Thus, eating equal amounts of high-quality protein at breakfast, lunch, and dinner, as well as a postworkout meal, would meet this suggestion and is typically easier to follow/maintain than eating six or more smaller meals per day. It should be noted, however, that if an athlete is attempting to decrease body fat through a hypocaloric diet (i.e., dieting), then this aforementioned recommendation by Philips and van Loon (2011) is increased to 1.8–2.0 g protein/kg body mass per day.

9.7 PRINCIPLES OF WEIGHT GAIN FOR ATHLETES

When attempting to gain weight, the athlete will need to pay special attention to the type of weight that is gained as well as the impact that the weight gain is having on sport performance. The type of weight gain the athlete should focus on is the augmentation of lean muscle mass. For many athletes, the goal of increasing muscle mass will not be an end to itself. Rather, the goal will be to increase muscle mass and then translate this muscle mass into functional strength and power that can be applied to the sport in which the athlete participates. The translation of muscle mass into functional strength is brought about by sport-specific training. In this regard, all phases of the athlete's lifestyle need to be coordinated in such a way that gains in lean body mass can be attained. These phases include the strength and conditioning program, the practices for which the athlete seeks to improve sport-specific skills, and diet. The following sections will discuss the dietary aspect of the athlete's plan, which is primarily focused on increasing Calories to levels that are above energy balance. Also, the importance of resistance training during a weight gain program is essential and will be discussed as well.

9.7.1 Importance of Resistance Training

Resistance training (e.g., weight lifting) is a common exercise modality used by strength and power athletes to peak physical performance through an increase in skeletal muscle mass. Several distinct training program methodologies (e.g., nonperiodization, linear periodization, undulating periodization) exist and are aimed at

achieving maximal strength (Baker, Wilson, and Carlyon 1994). Two different meta-analyses concluded that periodized programs resulted in greater increases in muscular strength and power as well as greater gains in lean body mass when compared to non-periodized programs (Rhea and Alderman 2004; Fleck 1999). Thus, our main focus concerning resistance training will revolve around periodized training programs.

The foundation of periodization programs is to promote continuous long-term muscle development and adaptations in strength, power, hypertrophy, and peak performance while avoiding overtraining and performance decrements (Kraemer, Duncan, and Volek 1998; Fleck 1999). Resistance-training programs can be modified by adjusting numerous training variables (e.g., load, number of sets, number of repetitions per set, intraset rest periods, number of training sessions per day and per week) (Fleck 1999). Training intensity (i.e., load) is a very important aspect of resistance-exercise-induced hypertrophy as inappropriate training intensity can lead to undesirable muscle mechanics (Schoenfeld 2010). Typically, high-intensity (i.e., >65% 1 RM) resistance training with low (1–5 reps) to moderate (6–12 reps) repetitions is more conducive at maximizing muscle hypertrophy when compared to high (15+ reps) repetitions with low intensity (i.e., <65% 1 RM) (Schoenfeld 2010).

The activation of all motor units is imperative for muscular strength as only activated motor units respond and adapt to resistance exercise (Spiering et al. 2008). In 1957, Elwood Henneman, an American neurophysiologist, concluded that motor unit recruitment occurs in a specific order and, as more force is needed, additional motor units are recruited (Henneman 1957). The small, type I motor units are recruited first, while the larger, type II motor units are recruited only after heavy loads, explosive exercises, or significant muscle fatigue (Spiering et al. 2008; Kraemer et al. 1996). This is important to consider as type II muscle fibers have a greater capacity for growth than type I fibers. With that said, low-intensity resistance exercise should not be excluded from a resistance-training program and should be supplemented as part of the periodized regimen as muscular power development is commonly carried out with loads less than 65% 1 RM (Kraemer et al. 1998). In addition to an appropriate resistance-training program, it is important to understand the critical role nutritional intake plays in optimizing skeletal muscle gains.

9.7.2 Focus on Calories

As discussed in Chapter 5, "Protein Metabolism," in order for a gain in muscle mass to occur, net muscle protein balance must be positive. Net muscle protein balance consists of both protein synthesis and protein breakdown, with the goal being to increase protein synthesis and decrease protein breakdown. There is evidence to support that when caloric intake is reduced to levels below energy balance, whole-body protein breakdown increases (Knapik et al. 1991; Hoffer and Forse 1990). When the effects of protein metabolism in athletes and physically active individuals are observed, skeletal muscle protein metabolism is superior to whole-body measures.

Researchers from the University of Connecticut investigated, for the first time, the effects of a moderate Calorie-restricted diet and its effects on skeletal muscle protein synthesis rates in physically active people. In this study, the participants reduced

their Calories by 19% (~80% of estimated energy requirements) for a 9-day period. The energy restriction resulted in a 19% decrease in skeletal muscle protein synthesis. This finding was in spite of the fact that protein intake was 1.5 g/kg body mass/day during the energy restriction period. Based on this study alone, reducing energy intakes below maintenance levels is counterproductive to maximizing protein synthesis. Based on this and other published data, it is recommended that caloric intake be increased to levels above energy balance levels if the goal is to increase lean muscle mass.

Caloric consumption, as well as the macronutrient composition of a diet, can influence the relationship between protein synthesis and protein catabolism. Muscle hypertrophy can only occur when protein synthesis surpasses protein catabolism (Spiering et al. 2008). Nutritional strategies for promoting protein accretion should include caloric consumption of both protein and protein-free calories. When attempting to increase skeletal muscle mass through modifications in diet and exercise, the primary nutritional strategy used by many involves increasing the consumption of daily protein intake. Solely increasing protein intake may not be the most optimal approach because the stimulus that caloric intake has on muscle hypertrophy is vital and just as important as protein intake in determining body nitrogen balance (Butterfield and Calloway 1984; Todd, Butterfield, and Calloway 1984; Calloway and Spector 1954). A positive nitrogen balance is associated with periods of growth, while negative nitrogen balance is commonly associated with periods of fasting, wasting diseases, and serious injuries such as burns.

It appears that positive energy balance must be reached in order to promote positive nitrogen balance and the further increase of excess energy consumption only improves nitrogen retention. Chiang and Huang (1998) studied the effects of three successive energy levels in an ascending (energy maintenance [EM], 15% above energy maintenance [+15%], 30% above energy maintenance [+30%]) and descending (+30%, +15%, EM) series during a fixed protein intake of 1.2 g protein/kg/day. The mean daily nitrogen balance significantly increased by 362.5% (7.2–33.3 mg/kg) in the ascending series of energy intake and significantly decreased by –82.7% (27.8–4.8 mg/kg) in the descending series.

Excessive calorie consumption, typically 500–2000 Calories/day, has been successful in increasing lean body mass as well as total body mass (Kreider 1999; Miller and Mumford 1967; Forbes et al. 1986). The increase in both lean body mass and total body mass with excess caloric consumption has occurred with and without resistance training. In a randomized, inpatient study of 25 healthy men and women, a 56-day diet providing excess kilocalories of ~40% above energy maintenance (~950 kcal/day) resulted in a weight gain of approximately 13.9 lb. (6.3 kg), of which ~49.2% (6.8 lb. [3.1 kg]) was lean body mass (Bray et al. 2012). In a different study, young men and women overfed by ~1200–1800 kcal/day for approximately 21 days gained 9.7 lb. (4.4 kg) in total body weight, with 51% of the total gains consisting of lean body mass (Forbes et al. 1986). The aforementioned studies were able to induce increases in lean body mass with dietary intervention alone.

Rozenek and colleagues (2002) studied the effects of high-calorie supplements on body composition following an 8-week high-intensity resistance-training program. The high-calorie supplement provided approximately 2000 Calories/day and

resulted in significant increases in body mass (6.8 lb. [3.1 kg]) and fat-free mass (~6.8 lb. [3.1 kg]) when compared to the similarly trained group not receiving the high-calorie supplement. The contribution of lean mass (~100%) to total body mass observed in this study is rare. Typically, fat mass contributes approximately 60%–70% of the total weight gain associated with overconsumption (Kreider 1999).

Novel energy intake should be centered on a percentage increase above weight maintenance values rather than on an absolute caloric value. Absolute caloric values should be avoided as this does not consider differences in factors that influence total daily energy expenditure such as age, gender, resting metabolic rate, or sport-specific training. The initial increase in caloric consumption should be 15%–25% above weight maintenance values. Conservative increases in caloric intake may minimize fat mass gains and would allow an athlete to make appropriate nutritional adjustments if there are undesirable gains in fat mass.

It is important to understand that interindividual differences exist and that not all athletes will respond similarly to identical relative increases in caloric intake. In a previously mentioned study, one subject gained 9.5 lb. (4.3 kg) when overfed by 20.5% above weight maintenance values for 3 weeks (Forbes et al. 1986). In this particular subject, fat mass accounted for 100% of the total body mass gains, whereas the other similarly fed subjects gained equal portions of both fat mass and fat-free mass. Caloric intake should be adjusted if desirable weight gain goals are not being reached. Frequent assessment of body composition will provide appropriate feedback that will determine if nutritional modifications are needed in order to avoid undesirable weight gain.

9.8 CONCLUSION

The process of making changes to an athlete's body composition should not be taken lightly. A plan to implement changes in body composition should be both simple and methodical. In this chapter, a four-step plan was introduced as well three principles that should be followed. Using fat loss as an example, the plan included (1) obtaining baseline data, (2) clearly stating the body composition goal (such as losing fat mass), (3) setting daily total caloric levels to below energy balance levels, and (4) monitoring body composition and performance frequently. In conjunction with this four-step plan, three principles were also presented. The rate of weight loss should be slow (a loss of about 0.7% to 1.0% of body mass per week), protein intake should be maintained at 1.5 to 2.3 g/kg body mass/day, and the athlete should participate in a progressive, periodized resistance-training program.

Recommendations were also given for athletes wishing to increase lean body mass. When attempting to increase lean body mass, caloric intake should be elevated above maintenance levels and a progressive, periodized resistance-training program should be followed. Following the plan and principles laid out in this chapter assists the athlete in achieving body composition goals in a timely manner and in a way in which lean body mass is maintained for those attempting to lose fat mass and gains in fat mass are minimized for those athletes attempting to gain lean body mass.

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10 Hydration and Performance

Jennifer Bunn

CONTENTS

10.1 Introduction 269
10.2 Function of Water in the Body 270
10.2.1 Temperature Regulation 271
10.2.2 Electrolyte Balance 273
10.2.3 Hormonal Regulation 275
10.3 Assessment of Hydration in Athletes 276
10.3.1 Dehydration 278
10.3.2 Hyponatremia/Overhydration 280
10.4 Hydration and Aerobic Exercise Performance 283
10.4.1 Prehydration Strategies 283
10.4.2 Hydration Strategies during Exercise 284
10.4.3 Rehydration Strategies 285
10.5 Hydration and Anaerobic Exercise Performance 286
10.5.1 Prehydration Strategies 288
10.5.2 Rehydration Strategies 288
10.6 Conclusion 289
References 289

10.1 INTRODUCTION

Water is an essential component in daily living and in exercise and sport activities. It has several functions in the body, including carrying nutrients in the blood, regulating body temperature, protecting the spinal cord and brain, lubricating, and participating in various biochemical reactions. Because water has several important functions in the body, water balance is very important. The density of various electrolytes like sodium, calcium, magnesium, and chloride determines this balance in the volume of body water, as well as daily body water intake compared to daily body water loss. This concentration of electrolytes or solutes and body water balance determines one's hydration status. Euhydration refers to when the body has an adequate volume of water to meet physiological demands. Hyperhydration refers to a temporary condition when there is too much water volume, and hypohydration refers to when the water volume is too low.

For a sedentary individual in temperate conditions, the required intake of water is approximately 2.5 L per day (Naghii 2000), and as participation in physical activity increases, so does the requirement for water intake, especially for individuals exercising in a hot environment. During exercise, water balance becomes increasingly important because of the role of body water volume in temperature regulation, cardiovascular functioning, and various biochemical and neural reactions. Many coaches and athletes are aware of the physiological and performance effects of poor hydration as it relates to being hypohydrated or dehydrated, but there are also implications to consider in states of hyperhydration, such as hyponatremia. It is important that athletes and coaches understand the multiple uses of water in the body in order to manage hydration status properly for optimal performance capabilities. This chapter will include details on the various roles of water in the body, how body water is assessed, and practical applications for management of water balance prior to training, during exercise, and for rehydration in both aerobic and anaerobic sports.

10.2 FUNCTION OF WATER IN THE BODY

Water helps control the osmotic pressure, or electrolyte balance, within the body, which is important for proper cellular functioning. Water makes up approximately 60% of one's body weight. This level may vary depending on one's lean body mass and adipose tissue because different tissues contain various volumes of water. For example, muscle is about 75% water, adipose tissue is 5% water, bone is about 25% water, and blood is 90% water. The size of a person will also determine total body water. In general, a 70-kg male carries approximately 42 L of water, whereas there are approximately 30 L of water in the average female because females tend to have less body mass than men. Table 10.1 shows the breakdown of water storage for a 70-kg person.

Approximately two-thirds (28 L) of the body's water is found in intracellular fluid, and one-third (14 L) is found in extracellular fluid. The two main areas to find water in extracellular fluid are blood plasma and the interstitial fluid, with trace amounts of water found in the lymph and cerebrospinal fluid. Water in the blood plasma is the principal component of blood. The blood plasma serves to transport nutrients, oxygen, carbon dioxide, hormones, and other substances throughout the body. It also helps with the removal of waste products from metabolism. In addition, the plasma serves as a reservoir for excess water and has a significant role in

TABLE 10.1
Volumes of Water Storage in a 70-kg Person

Body Compartment	Water Volume (L)	Total Body Water (%)
Intracellular fluid	~28	66.6
Extracellular fluid:		
• Blood plasma	~2.8	6.7
• Interstitial fluid	~11.2	26.7

thermoregulation, which is discussed in the next section. The plasma volume also affects the cardiovascular system, and alterations in the volume may influence heart rate, stroke volume, and cardiac output. The role of the interstitial fluid is to surround and protect cells, as well as provide an area of exchange between intracellular fluid and blood plasma.

The amount of water stored in the body tends to decline with age and with increasing body fatness. With age, participation in physical activity tends to decline, resulting in a significant decline in muscle mass and increase in adipose tissue. The body water levels thereby decrease because much of a person's water is held within lean muscle tissue.

10.2.1 Temperature Regulation

The human body constantly makes adjustments to gain or lose heat to maintain an optimal core temperature of 37°C. Heat production is a by-product of metabolism and is increased during exercise. The human body has four primary methods to get rid of heat, including:

- Conduction
- Convection
- Radiation
- Evaporation

Conduction (also called heat diffusion) is the transfer of heat from the body through direct physical contact with another object or person. When an object (such as the human body) is at a different temperature from its surroundings, heat flows so that the body and the surroundings reach the same temperature, at which point they are in thermal equilibrium. Such spontaneous heat transfer always occurs from a region of high temperature to another region of lower temperature, as described by the second law of thermodynamics. Convection is the transfer of heat by movement of air or water around the body. Radiation is the transfer of energy through space (from the body to the surrounding air or vice versa) by means of electromagnetic waves in much the same way as electromagnetic light waves transfer light. Evaporation is when the body loses heat through the evaporation of sweat from the skin or through respiratory evaporation.

At rest, radiation is the primary method for the body to release heat, whereas evaporation becomes the primary method of heat release during exercise. In addition to evaporation, radiation plays a small role in heat loss during exercise, and convection may also play a role depending on the environmental conditions of exercise. When core body temperature increases as a result of energy production with exercise or with exposure to a hot environment, the body works to cool itself by releasing water from the skin in the form of sweat. The cooling actually occurs when the sweat evaporates from the skin. Additionally, when body temperature rises above normal, this results in vasodilation toward the skin to release heat.

It appears that exercising metabolic rate is the larger determinant of body temperature during exercise than hydration status (Noakes 1995). At high exercise

intensities, heat production from energy metabolism may exceed that of heat loss, resulting in an increase in core temperature. High-intensity exercise in a hot and humid environment further exacerbates this increase in core temperature. If exercise intensity is not decreased and heat production continues to exceed heat loss, then the athlete may experience heat-related illness.

TOPIC BOX 10.1 HEAT EXHAUSTION AND HEAT STROKE

Heat stroke and heat exhaustion are two types of heat illness. Of these two illnesses, heat exhaustion is less severe than heat stroke. Heat exhaustion occurs when a person does not get enough liquid, especially water, in very hot, humid weather. Left untreated, heat exhaustion can lead to heat stroke. Heat stroke is caused by the body not being able to regulate its own temperature due to intensive sweating under conditions of high heat and humidity. Specifically, heat stroke is defined as a body temperature of greater than 40.6°C (105.1°F) due to environmental heat exposure with lack of thermoregulation. While most people believe that heat-related illnesses are related to hydration status, some believe that heat-related illnesses occur independently of hydration.

In general, exercise capacity decreases in higher temperatures compared to lower temperatures (Galloway and Maughan 1997), and this limitation can be worsened in states of hypohydration. Dehydration tends to accompany a significant increase in core body temperature compared to when one is euhydrated (Gonzalez-Alonso et al. 1995, 1997; Gonzalez-Alonso 1998; Montain and Coyle 1992) and may increase one's risk for both heat exhaustion (McLellan et al. 1999; Sawka et al. 1992) and heat stroke (Epstein et al. 1999). Hydration status does not appear to affect metabolic rate or core body temperature directly. Rather, the effect of dehydration on temperature regulation during exercise is based on the limited capacity of the body to cool itself through evaporation. As core temperature increases during exercise in hot and humid weather, the need to regulate temperature by sending blood to the skin trumps the need to continue to perform at a high level, so blood flow that was directed toward the working skeletal muscles decreases. This vasodilation toward the skin increases heat loss through evaporation and radiation, and the decrease in blood flow to the working muscle will decrease oxygen to the muscle and subsequently inhibit energy production and heat production in the muscle.

Increased core body temperature and dehydration are of greater concern during exercise in hot and humid weather. In hot weather, the body must sweat more to cool itself through evaporation. This increase in sweat results in a greater loss in plasma volume, causing heart rate to increase even without a change in exercise intensity. In humid conditions, cooling the body through evaporation is hindered because of a lower vapor pressure differential. If exercise intensity is maintained, sweat rate will increase to try to maintain body temperature, resulting in dehydration if an attempt to replenish fluid losses is not made (McLellan et al. 1999).

Participation in regular exercise training at a consistent intensity can help improve temperature regulation. For heat acclimatization, exercise training should be done in a hot environment. The training helps to improve the body's exercise tolerance to heat and improvement in temperature regulation in hot and humid conditions. Exercise training in a hot environment will also increase plasma volume, thereby allowing for greater loss of sweat without the physiological implications on the cardiovascular system. Acclimatization also causes the athlete to sweat sooner during exercise and the sweat is more diluted, containing less sodium than that of an untrained person.

A controversial area of hydration status and temperature regulation is with the incidence of heat stroke. Because heat stroke is a very serious condition that often results in collapse during exercise, medical personnel have been led to believe that all athletes who collapse during or after exercise are likely to be suffering from heat stroke. This appears to be incorrect for those individuals that experience postexercise collapse. As heat stroke is caused by an inability to dissipate heat, an athlete would tend to collapse once core body temperature reaches detrimental levels and therefore hinders exercise *during* the activity rather than *after* the exercise bout. This is especially confusing as one's metabolic rate would begin to decline with the cessation of exercise, and therefore the core body temperature would begin to fall as well.

It is probably more likely that postexercise collapse is caused by postural hypotension rather than heat stroke (Noakes 1995). During exercise there is significant vasodilation to the working skeletal muscles, and blood is forced to return to the heart via the skeletal muscle pump and the cardiorespiratory pressure differential that helps to move the blood against gravity. With the cessation of exercise, both of these mechanisms decrease in activity, causing a decrease in blood pressure and a decrease in blood returning to the heart and head, thereby causing presyncopal symptoms and perhaps syncope. The best treatment for this problem is to place the athlete in a supine position.

10.2.2 Electrolyte Balance

Electrolyte balance refers to the balance between water volume in the body and the concentration of various solutes within the body fluid. The primary solutes of concern are sodium, potassium, calcium, and magnesium. All of these are positively charged electrolytes, or cations. Essentially, water found in the intracellular and extracellular fluid is dynamic, with constant addition or removal of various solutes and water. There is consistent passing of fluid between the extracellular portion and intracellular portions of fluid through the cell membrane. Water tends to move easily through this membrane because cells are freely permeable to water, but there must still be a stimulus to induce this motion. The two major stimuli are hydrostatic and osmotic pressure.

Hydrostatic pressure is a result of a fluid pressure differential between two compartments. Fluid in the body prefers to move from areas of higher pressure to areas of lower pressure. For example, in a hyperhydrated state, fluid will first increase in the blood plasma, increasing the pressure in the blood plasma compared to the interstitial fluid. Water will then move down the pressure gradient from the area of high pressure in the blood plasma to the area of lower pressure in the interstitial fluid.

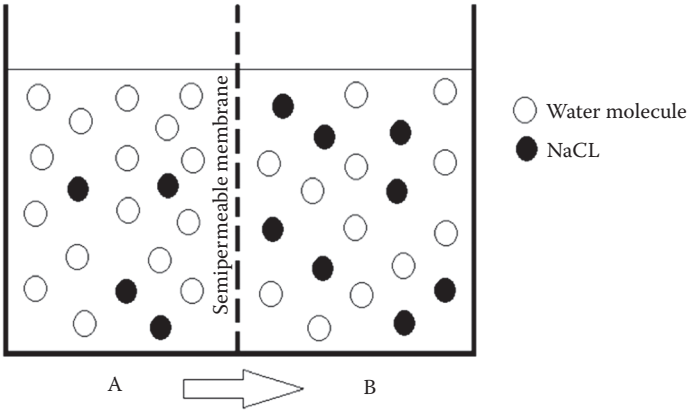

FIGURE 10.1 Osmosis. Sodium chloride (NaCl) is a common and important solute that helps control electrolyte and water balance in the body. Side A is hypotonic compared to side B, which is hypertonic. There would be movement of water from side A to side B in order to even out the tonicity of the two sides.

Once the pressure difference between the two fluid compartments is diminished to zero, there will be no net movement of water. A hypohydrated state will create a hydrostatic pressure differential that will tend to favor movement of water from intracellular fluid to interstitial fluid, and then to blood plasma.

The second stimulus to promote water movement is osmotic pressure, which refers to the concentration of a solute within a solution in one fluid compared to another fluid. When two fluids have the same solute concentration and there is no net movement of water, they are said to be isotonic. Osmosis is the tendency for water to move from an area of high solute concentration to an area of lower solute concentration. In Figure 10.1, solution A has a lower solute density than solution B; therefore, solution A would be considered hypotonic compared to solution B. Vice versa, because of a higher solute density, solution B would be considered hypertonic compared to solution A. This difference in osmotic pressure would result in water movement from solution A into solution B. The water movement would therefore result in equilibration of the osmotic pressures and make the solutions isotonic.

If an athlete enters into a hyperhydrated state during exercise by taking in more fluid than is lost, the water volume of the extracellular fluid increases, subsequently lowering the solute concentration making the blood plasma hypotonic. Generally, the renal system will respond by increasing urinary output, but in some cases the kidneys may not be able to keep up with urinary production. This causes movement of water from the extracellular fluid to the intracellular fluid to balance the solute concentrations between them. This excess fluid intake would cause the cells to swell. In a hypohydrated state, the extracellular fluid is hypertonic compared to the intracellular fluid, causing water to move from the cells to the extracellular fluid. This cellular water loss results in a shrinking of the cells. In both cases of cellular shrinking and cellular swelling, cellular functioning may be impaired and exercise performance is likely to be affected.

Genetic predisposition, heat acclimatization, and body weight all play a role in determining one's sweat rate and metabolic efficiency (Barr and Costill 1989). The concentration of sodium in sweat, or sweat composition, is influenced by rate of sweat, temperature, and diet, but there is still large interindividual variation (Robinson and Robinson 1954). Sodium lost in sweat varies among individuals, ranging from 20–80 mmol/L in concentration (Maughan 1991; Schedl, Maughan, and Gisolfi 1994). These factors all contribute to a large variation in sweat rates among various sports, levels of fitness, and training or competition environments, and between training and competition. For example, male soccer players training in the summer had an average sweat rate of 1.46 ± 0.47 L/h (Shirreffs and Maughan 1998b) compared to those training in the winter, with an average sweat rate of 1.13 ± 0.42 L/h (Maughan et al. 2005). Athletes participating in summer training for American football averaged a much larger sweat rate of 2.14 ± 1.04 L/h (Godek, Bartolozzi, and Godek 2005).

This larger sweat rate in American football players could have occurred because these athletes tend to be larger in stature than soccer players, and the football players consumed an average of 1.42 ± 0.85 L/h in fluid, compared to the soccer players training in the summer, who consumed 0.65 ± 0.49 L/h of fluid. Higher fluid consumption allows for greater sweat rates. Additionally, soccer is a sport that is played continuously with water breaks available for athletes only during half-time or an extended injury timeout, whereas an American football game has frequent breaks that would allow for greater fluid consumption.

10.2.3 Hormonal Regulation

Water and electrolyte balance are essential for peak athletic performance, so the body has several different mechanisms to mitigate any significant disturbances in fluid homeostasis. Several hormones play a significant role during exercise as core body temperature increases from energy production and water is lost through sweat. Renin, angiotensin, aldosterone, and vasopressin (also known as antidiuretic hormone or ADH) are all hormonal respondents for maintaining plasma volume during exercise (Costill et al. 1976) and heat exposure (Kosunen et al. 1976). Exercise, in both hot and temperate conditions, results in a decrease in plasma volume, which sets forth a series of reactions that would work to reverse the change in plasma volume, as shown in Figure 10.2. These hormones all serve to function to increase plasma osmolality (Takamata et al. 1994), assist with greater absorption of water within the kidney tubules, and decrease fluid output from the body.

Renin, angiotensin I, angiotensin II, and aldosterone work as a system to increase water reabsorption in the kidneys and decrease urine production. Low blood pressure, a decrease in plasma volume, and increased osmolality of the blood can all act to trigger this system. When the stimulus is detected, renin is released from the kidneys, triggering the release of angiotensin I from the lungs. Angiotensin I is converted to angiotensin II via the angiotensin converting enzyme, and the presence of angiotensin II triggers the release of aldosterone from the adrenal cortex. Aldosterone acts on the kidneys to foster reabsorption of water and inhibit urinary production. This system also triggers the thirst mechanism to help increase body

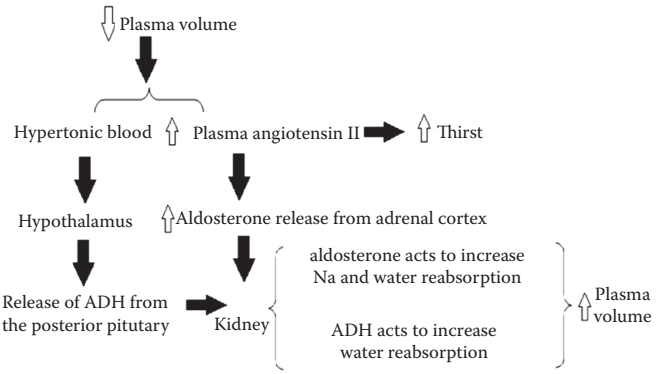

FIGURE 10.2 Mechanisms to counteract a decrease in plasma volume. Schematic indicates the hormonal response to low plasma volume. ADH and aldosterone both function to preserve water in the body during exercise.

water and plasma levels. Once blood pressure and plasma osmolality are restored, this system is slowed or stopped.

Vasopressin or antidiuretic hormone also functions to help keep fluid in the body. Vasopressin is released from the posterior pituitary and functions in the kidney to reabsorb water and produce less urine. Vasopressin is released during exercise, and higher intensity or longer duration exercise causes a larger release and response of vasopressin. Overall, a greater response in vasopressin causes greater water and electrolyte retention and less urine production. However, the response of vasopressin is lower for women than men, which may result in elevated water and electrolyte losses (Stachenfeld et al. 1997).

10.3 ASSESSMENT OF HYDRATION IN ATHLETES

Assessing the hydration status of athletes may serve to be beneficial for optimal performance because of the potential implications of altered water and electrolyte balance on nervous system function and the cardiovascular system. Method selection for hydration assessment is somewhat controversial, as there are several methods that vary based on accuracy, sensitivity, type of dehydration anticipated, and cost. The gold standard for hydration assessment is with doubly labeled water, in which total body water measurement is measured using a trace amount of an isotope, usually deuterium oxide (^2H_2O) (Cheuvront and Sawka 2005). Essentially, a known volume of a concentrated isotope is ingested, and the concentration of the isotope is later measured from a sample of body fluid. Total body water is calculated based on the dilution of the isotope within the body. This method is very accurate with just a 1% error (Ritz 1998), but it is also very expensive to complete and involves the use of expensive and specialized laboratory equipment.

Blood volume and plasma osmolality can vary in the short term in response to exercise, food and fluid intake, postural changes, and several other factors—suggesting that neither should be used to assess hydration status (Armstrong et al. 1994;

Popowski et al. 2001). In contrast to blood volume and blood plasma, several different urinary measures are used to assess hydration status, including:

- Osmolality
- Specific gravity
- Color

Each of these measurements produces results that are not as consistent or reliable as doubly labeled water, but may be more practical and cost efficient. It should be noted that these measures do not correlate well with one's hydration status after exercise (Kovacs, Senden, and Brouns 1999). The first void of the day is recommended for use in all of these measurements to increase the reliability (Armstrong et al. 1994; Shirreffs and Maughan 1998a; Cheuvront and Sawka 2005). Urine osmolality is used to measure the number of dissolved particles per unit of water in the urine. The 24-h urine osmolality should be, on average, 500–800 milli-osmoles per kilogram (mOsm/kg) of water. Greater than normal urine osmolality values indicate a state of dehydration and lower than normal values indicate a state of hyperhydration. Some evidence indicates that pretraining urine osmolality correlated well with the volume of fluid ingested during exercise (Maughan et al. 2005). This suggests that pre-exercise hydration status may be an accurate measurement to estimate how much one should drink during the exercise bout.

Unlike urine osmolality, both the number and size of particles in solution affect urine specific gravity. Normal values range from 1.000 to 1.030. Greater than normal urine specific gravity values indicate a state of dehydration and lower than normal values indicate excessive fluid intake. Urine specific gravity can be measured with a urinary dipstick and is a quantifiable field test. In terms of practicality, it is the preferred method for assessing hydration status in athletes due to the fact that it is extremely easy to travel with and use in field settings. When comparing urine osmolality and urine specific gravity, osmolality is used for more detailed analyses but urine specific gravity is popular because of its convenience.

Another method besides urinary osmolality and urine specific gravity is to assess the color of the urine (Armstrong et al. 1994). Ideally, an athlete would want to have urine that is pale yellow to indicate euhydration. A clear color indicates hyperhydration, and golden to darker shades of yellow or even beige or brownish colored urine indicate varying levels of dehydration. Urinary color can also be affected by several dietary factors. For example, recent consumption of B-complex vitamins or carotene can make the urine appear dark yellow. Consumption of pigmented foods or foods with artificial colors can make the urine appear darker. Also, various medications can make the urine appear with a green or blue tint (Maughan and Shirreffs 2008).

Last, several athletes employ changes in body mass to assess their hydration. Body mass is measured both before and after an exercise bout and, essentially, 1 g of lost mass is equal to 1 mL of lost water (or 1 kg of body mass reduction is equal to 1 L of lost water) (Cheuvront and Sawka 2005). To improve the accuracy of this method, the athlete should remove as much clothing as possible for both measurements, and the amount and mass of water held in the clothes should be considered for the postworkout measurement. While this hydration assessment method is somewhat

rudimentary, it is the most frequently utilized method to measure an athlete's hydration status and is also used to determine fluid intake after an exercise bout.

10.3.1 Dehydration

Sedentary adults lose from 1 to 3 L of water each day, with losses primarily coming from insensible water losses in respiration and feces, as well as sensible losses in urine and sweat evaporation (Sawka, Cheuvront, and Carter 2005). Water loss is greater during exercise because it is used as a cooling mechanism when an athlete sweats. Excessive sweating that would occur with exercise and inadequate fluid replacement during exercise would likely result in dehydration. Athletes should also consider that not all losses in body mass are from fluid lost from sweat, because body mass can also be lost during exercise from insensible fluid losses from the respiratory tract since ventilation is increased during exercise. Additionally, dehydration commonly occurs when exercise is performed in hot and humid conditions or when an athlete begins an exercise bout with a water deficiency without proportionate sodium chloride loss (Sawka and Coyle 1999). Symptoms of dehydration generally include thirst and the inability to spit. In severe dehydration, symptoms may include a decrease or cessation in sweat rate. Additionally, when the athlete is placed in a supine position with the lower extremities elevated, a severely dehydrated athlete would have an increased heart rate and elevated blood pressure.

Both the National Collegiate Athletic Association and the National Federation of High School Associations indicate dehydration as having urine specific gravity greater than 1.020–1.025 (Committee, National Collegiate Athletic Association Wrestling Rules 2003; National Federation of High School Associations 2006). However, a change in body mass is a common and practical method employed to measure the severity of dehydration. Plasma osmolality (a measure of the concentration of substances such as sodium, chloride, potassium, urea, glucose, and other ions in blood) increases by approximately 5 mOsm/kg for every 2% loss in body mass when sweating (Popowski et al. 2001). This decrease in body weight may impair exercise (Cheuvront et al. 2004; Nielsen et al. 1982) and cognition (Edwards et al. 2007; Wilson and Morley 2003), and it may increase physiological strain on the cardiovascular system and may impair thermoregulation (Maughan 2003). For example, dehydration without an increase in core temperature has been shown to reduce stroke volume by 7%–8%, but dehydration resulting in a 1%–2% decline in body mass in a temperate environment of 20°C–21°C was shown to have no effect on exercise lasting less than 90 min (Robinson et al. 1995; McConell, Stephens, and Canny 1999; Bachle et al. 2001).

During a 90-min bout of cycling and swimming, participants experienced a 5%–6% decrease in plasma volume within the first 5–10 min of exercise, with very little change for the remainder of the exercise (Nielsen, Sjogaard, and Bonde-Petersen 1984). Interestingly, cycling exercise resulted in four times the sweat loss compared to the swim exercise, but the gradual cardiovascular drift was the same between groups.

The combination of dehydration and hyperthermia can reduce stroke volume by more than 20% (Gonzalez-Alonso 1998). A review of several studies showed that

exercise performance is attenuated when working in an environment hotter than 30°C and with a state of 2%–7% dehydration (Cheuvront, Carter, and Sawka 2003). Additionally, dehydration of 3% or greater has been shown to impact aerobic exercise even during cold stress (Cheuvront et al. 2005). When hypohydrated by 2.5% body weight, exercise tolerance time has been shown to decrease from 60 to 53 min (McLellan et al. 1999). In opposition, there is no evidence indicating that a state of hyperhydration at the onset of exercise will improve one's ability to tolerate higher core temperatures.

The effects of dehydration have been seen with as little as 1% reduction in body weight causing cardiovascular strain, an increase in plasma osmolality, and a possible effect on intra- and extracellular electrolyte balance (Naghii 2000). Studies also indicate that mild dehydration affects cognitive function, mood, and subjective feelings (Shirreffs et al. 2004; Petri, Dropulic, and Kardum 2006). For every 1% of body mass lost with dehydration, heart rate has been shown to increase five to eight beats per minute, and cardiac output declines as core temperature increases 0.2%–0.3% (Sawka and Coyle 1999; Coyle and Montain 1992; Cheuvront and Haymes 2001b; Sawka, Montain, and Latzka 2001; Cheuvront and Sawka 2005). For each liter of fluid ingested, core body temperature was shown to decrease by 0.3°C, heart rate was reduced by eight beats per minute, and cardiac output increased 1 L/min (Montain and Coyle 1992).

Muscle cramps have also long been associated with long-term physical activity in a hot environment, and ingestion of water and electrolytes has been shown to reduce the intensity and frequency of the cramps (Talbott and Michelsen 1933; Talbott 1935). In fact, recent studies have indicated that cramps tend to occur in players who sweat profusely and in those that have a high concentration of sodium in sweat (Bergeron 2003; Eichner 2007; Stofan et al. 2005). Additionally, beginning exercise in a dehydrated state of 5% of body mass has been shown to increase rectal temperature and heart rate, as well as decrease sweat rate, exercise capacity, and maximal oxygen consumption when compared to normal hydration status (Naghii 2000). All of these physiological strains caused by fluid deficits appear to be less strenuous in laboratory settings compared to field settings (Godek et al. 2006; Noakes 2007).

Traditionally, athletes do not drink enough fluids during exercise to replace losses from sweat. Instead, they drink approximately two-thirds of their water loss from sweat (Pitts and Consolazio 1944; Hubbard et al. 1984). Reasons for this may be that there is not an appropriate opportunity to drink that much fluid, as in several team sports, or that they do not want to slow down to ingest fluid, as in running. One study showed that tennis players only consumed 27% of their fluid losses when drinking ad libitum during practice (Dawson et al. 1985). Another study forced participants to drink at a rate that matched their sweat loss, and results indicated an improvement in performance (Pitts and Consolazio 1944).

Arguments have been made that the signs for dehydration are unclear, and that many athletes are unaware of the small effects of dehydration that result in fatigue, irritability, increased core temperature, and thirst (Murray 2008). However, other researchers argue that this voluntary dehydration is not detrimental to performance in most situations and does not cause any significant physiological harm (Noakes 2012). Data indicate that in several endurance races of marathons and Ironman

triathlons, the fastest times are often completed by those that are the most dehydrated. Because of the potential detrimental physiological effects of dehydration, the general consensus is that athletes should not exceed more than a 2% loss in body mass during an exercise bout (Casa et al. 2000; Noakes and Martin 2002).

10.3.2 Hyponatremia/Overhydration

In an effort to prevent the detrimental physiological and performance effects of dehydration during exercise, athletes may overconsume fluid. At rest, hyperhydration simply results in greater urine production. However, during exercise the hormone vasopressin works to decrease the volume of urine produced, making it difficult for the body to get rid of the excessive fluid. This excessive fluid intake can lead to a condition called hyponatremia. The kidneys regulate urinary output so that the minimum output is 20 mL/h and the maximum output is 100 mL/h (Institute of Medicine 2005). In resting conditions, evidence indicates that if ingestion rates exceed 800 mL/h, total body water will increase and plasma sodium concentrations will decrease in a proportionate manner (Noakes et al. 2001).

Researchers have proposed that hyponatremia may be caused by a limitation of the intestines to absorb water during exercise when ingestion is 750–1000 mL/h (Noakes 1993). When fluid is ingested at these high rates, the unabsorbed fluid would accumulate in the intestine and sodium would then move down its concentration gradient from the extracellular fluid to the intestinal fluid to 40–100 mmol/L, thereby further decreasing the extracellular sodium volume (Gisolfi et al. 1990).

It is proposed, then, that the kidneys have a limited capacity to produce urine with high rates of fluid ingestion, resulting in fluid accumulation in the body and progressive increase in extracellular fluid with a proportionate decrease in sodium. During exercise, urinary output declines because of the decrease in renal blood flow, causing a decrease in glomerular filtration rate. If an athlete overconsumes fluid during prolonged exercise, this may result in a state of hyperhydration and hyponatremia. For a 70-kg individual completing a marathon in 5 h in temperate conditions, the predicted fluid loss is approximately 4 L, or 0.8 L/h (Barr and Costill 1989). To predict fluid loss while running, one of the following equations can be used (Barr and Costill 1989):

$$\text{Predicted hourly fluid loss (L)} = \text{weight (kg)} \times \text{running speed (km/h)}/732$$

$$\text{Predicted hourly fluid loss (oz.)} = \text{weight (lb.)} \times \text{running speed (mph)}/28.5$$

During exercise, sodium plasma concentration should be 130–160 mmol/L to maintain proper electrolyte balance and functions of various cells, tissues, and organs (Coyle 2004). If this level drops below 130 mmol/L, making the plasma a hypotonic solution, this can result in movement of fluid from the plasma into the brain. This movement of water from extracellular fluid to intracellular fluid in the brain could cause the brain to swell. Symptoms related to hyponatremia tend to become apparent when serum sodium levels drop below 125 mmol/L (Barr and Costill 1989) and include headache, feeling strange, confusion, muscle weakness,

odd behavior, difficulty speaking, and wheezy breathing. More severe symptoms like collapse, seizing, and coma are associated with plasma sodium levels below 120 mmol/L (Murray and Eichner 2004). In severe hyponatremia cases, death may result from cardiac arrest secondary to significant cerebral swelling that causes brainstem herniation through the foramen magnum (Ayus, Varon, and Arieff 2000).

Evidence suggests that up to 10% of all participants who complete an ultra-endurance event do not experience symptoms of hyponatremia, but have plasma sodium levels that drop below 135 mmol/L (Noakes 1992; Speedy et al. 1999). Asymptomatic hyponatremia is believed to be caused by consumption of hypotonic beverages during exercise at a low rate that still results in varying degrees of weight loss, so these athletes are often incorrectly assumed to be dehydrated (O'Toole et al. 1995). It is still not well understood if hyponatremia is related to water intoxication or the failure to replace sodium lost in sweat.

Hyponatremia is thought to occur more frequently than heat stroke in endurance athletes (Noakes 1993). Evidence indicates that most of the cases of hyponatremia occur in the United States, where athletes are encouraged to drink as much as tolerable during exercise (Noakes 2003; Hew et al. 2003; Gardner 2002). Excessive fluid consumption and longer exercise times (lower intensity) appear to be the key risk factors for developing hyponatremia. In fact, in the 2000 Houston Marathon, the highest incidence of hyponatremia was detected in runners finishing after the 4:20 mark, and further analyses indicated an inverse relationship between finishing time and serum sodium levels (Hew et al. 2003). In the 2002 Boston Marathon, 13% of runners participating in a research study presented hyponatremic serum sodium levels at the finish line (Almond et al. 2005). Based on these data, the researchers extrapolated that approximately 1,900 of the 15,000 runners completed the race with some degree of hyponatremia, and that approximately 90 finishers had critical hyponatremia, where their serum sodium level was below 120 mmol/L. Hyponatremic conditions in these runners were associated with consumption of more than 3 L of fluids, weight gain, race time of more than 4:00 h, and regular fluid consumption at each mile; most of the runners who experienceed hyponatremia were females.

When runners presented in the medical tent at the 2000 Houston Marathon, the primary symptom differentiating hyponatremia from other conditions that are associated with exercise-induced collapsing was vomiting (Hew et al. 2003). Research also suggests that when exercising in hot conditions, an athlete's intensity decreases, and the athlete may then confuse this feeling of fatigue with dehydration, which may thereby encourage runners to overconsume fluids (Hew et al. 2003). Women may be at a greater risk for developing symptoms associated with hyponatremia because the fluid intake information that has been previously utilized was obtained from sweat loss data from men (Sawka et al. 2007; Hew et al. 2003).

The hydration methods often encouraged for novice exercisers—(1) drinking prior to the onset of thirst, and (2) drinking until urine is clear—appear to be a primary reason for the hyponatremia phenomenon (Hew et al. 2003). Developing an individualized plan that considers body size, sweat rate, and exercise intensity appears to be the most effective method for prevention of both hyponatremia and dehydration. It is also important to note that these conditions developed regardless of

water or sport beverage consumption, as both fluids are hypotonic. One method that endurance and ultra-endurance races can utilize to prevent cases of hyponatremia at the event is to decrease the number of aid stations available to the athletes.

Incidences of hyponatremia have also been addressed by the US military, and medical personnel concluded that four factors contributed to the development of hyponatremic conditions in military patients (O'Brien et al. 2001):

1. Aggressive fluid replacement practices
2. Poor knowledge of overhydration and the medical consequences of hyperhydration in the military leaders
3. Medical and supervisor personnel treating all heat-related illnesses as though they were linked to dehydration
4. No standardized criteria for evacuation for those suspected to have heat-related illnesses

Excessive sweat sodium losses may have also contributed to incidences of hyponatremia. The fluid replacement guidelines for military personnel are based on weather conditions and are shown in Table 10.2. At temperatures below 29.5°C, the military guidelines for fluid replacement are similar to those of the ACSM (0.4–0.8 L/h). However, in warmer temperatures, the guidelines become significantly more aggressive, with intake exceeding 2 L/h at high temperatures. The exercise bouts performed would cause an increase in vasopressin release, resulting in greater water reabsorption in the kidneys, and plasma sodium levels would decline. Further, the military personnel may also experience nausea and vomiting with this volume of water intake, which would further increase the release of vasopressin and exacerbate the volume of water being reabsorbed in the kidneys.

TABLE 10.2
Fluid Replacement Guidelines for Army Personnel

Wet Bulb Globe Temperature (°F)	Water Intake (L/h)	Work/Rest Cycle (min)
25.5–27.7	At least 0.475	Continuous
27.8–29.4	At least 0.475	50/10
29.5–31.1	At least 0.950	45/15
31.2–32.2	At least 2.365	30/30
32.3	More than 1.9	20/40
31.2–32.2	At least 2.365	30/30
32.3	More than 1.9	20/40

Source: Data adapted from O'Brian, K. K. et al. 2001. *Military Medicine* 166 (5): 405–410.

Note: These guidelines were utilized when cases of hyponatremia were reported. The water intake values were converted from quarts per hour to liters per hour.

10.4 HYDRATION AND AEROBIC EXERCISE PERFORMANCE

The primary concern for proper hydration in aerobic exercise performance is based around the cardiovascular system and temperature regulation. Studies indicate that performance of aerobic and related activities may be hindered, with dehydration resulting in cardiovascular strain (Montain et al. 1998; Saltin 1964), heat illness (Sawka et al. 1985), increased core temperature (Nielsen et al. 1982), and altered metabolic function. Some researchers suggest that each athlete should try to match fluid intake with sweat rate (Coyle 2004). However, runners tend to drink only 500 mL/h of fluid, resulting in dehydration if sweat rates are 500–1000 mL/h (Coyle 2004). Interestingly, for runners participating in road races or triathlons, studies indicate that the fastest athletes are the ones that register the greatest amount of body mass loss (Noakes 2012), and that there is a weak relationship between performance and level of dehydration (Cheuvront and Haymes 2001b; Cheuvront et al. 2003).

With this evidence and the negative physiological effects seen with both dehydration and hyponatremia, research suggests that no single fixed drinking schedule will be successful for each athlete, so developing an individualized plan is the best strategy (Maughan and Noakes 1991). The American College of Sports Medicine (ACSM) suggests that fluid intake during exercise should be sufficient to limit body mass loss to <2%, and to avoid drinking so much fluid that one gains weight during exercise (Sawka et al. 2007). However, if the athlete begins exercise in a dehydrated state, this plan does not hold true. In addition to water, athletes exercising longer than 60 min in a single bout should consider consuming fluids with electrolytes and carbohydrates. Without an intake of sodium during prolonged exercise, one may experience an increase in blood osmolality, and a reduction in plasma volume and skin blood flow, which may thereby affect thermoregulation (Sanders, Noakes, and Dennis 2001).

10.4.1 Prehydration Strategies

General recommendations from the ACSM for fluid intake prior to endurance exercise include slowly drinking beverages at least 4 h before the exercise bout. During this time, approximately 5–7 mL/kg should be consumed. If urine is not produced or color is undesirable, consume another 3–5 mL/kg 2 h before the bout (Sawka et al. 2007). The fluid consumed should be kept cold, between 15°C and 21°C to promote faster digestion and absorption from the gastrointestinal tract. Early fluid ingestion during exercise has been shown to attenuate the expected core temperature and heart rate increases compared to when subjects waited 40–80 min to ingest fluid (Montain and Coyle 1993). However, after exercising for longer than 2 h, the effects of early hydration were no longer evident.

Athletes should assess their urine color and volume prior to exercise and throughout the activity when applicable. Additionally, recording the athlete's weight prior to engaging in exercise and again after exercise will help estimate fluid losses. The only time when it is acceptable for the athlete to gain weight during exercise is if he or she begins in a hypohydrated state. Prior dehydration of 1.5%–2% of body mass has been

shown to decrease running performances at 1500, 5000, and 10,000 m (Armstrong, Costill, and Fink 1985).

10.4.2 Hydration Strategies during Exercise

When developing an individualized hydration plan, the general recommendation is to ingest enough fluid during exercise to match sweat rate. The ACSM suggests that athletes who are euhydrated at the beginning of exercise may drink ad libitum and aim for 400–800 mL/h to try to match fluid lost in sweat (Sawka et al. 2007). Research indicated that ingestion of fluid to match sweat rate reduced heart rate and core temperature, as well as improved blood volume and plasma osmolality, but it took 40–60 min for these changes to be realized during a 140-min exercise bout (Montain and Coyle 1993). This lag time represents the time it takes for gastric emptying, intestinal absorption, and osmotic flow (Noakes, Rehrer, and Maughan 1991; Schedl et al. 1994). This therefore indicates that one would not realize the benefits of fluid intake during exercise bouts shorter than 40–60 min. An athlete should pay attention to sweat rates and make fluid intake adjustments accordingly, especially during prolonged exercise. When participating in exercise lasting longer than 3 h, there is an increased risk of a fluid and sweat mismatch, which may lead either to dehydration or hyponatremia (Montain, Cheuvront, and Sawka 2006).

Athletes weighing 70 kg that have an average sodium loss in sweat of 50 mmol/L while exercising for over 4 h can expect to lose approximately 10% of their body's stores of sodium (Coyle 2004). Elite endurance athletes may sweat in excess of 1.5 L/h during competition. Including a beverage with sodium while exercising may help attenuate these sodium reductions, improve palatability of the beverage, and increase voluntary drinking (Wilk and Bar-Or 1996; Wemple, Morocco, and Mack 1997). The standard recommendation is to ingest a sodium beverage containing 20–40 mmol/L during exercise lasting longer than 1 h (Coyle 2004). Cyclists who drank a sodium-containing sports drink during a 3-h ride in warm temperatures maintained plasma sodium levels and had less urine production than those who drank only water (Vrijens and Rehrer 1999). Potassium and magnesium are also lost during exercise, but losses are so small that there is no evidence to indicate that these be included in sports beverages (Powers et al. 1990; Deuster and Singh 1993; Sawka and Montain 2000).

The recommendation that athletes should drink as much as tolerable or that they should drink enough fluid during exercise to replace fluid losses during exercise completely is often made to prevent heat-related illnesses. However, new evidence indicates that the relationship between hydration status and heat illness is not as strong as once thought. Dr. Timothy Noakes from South Africa indicates that the risk of heat-related illness is increased with increased exercise intensity, in athletes with greater body mass that tend to generate more heat during exercise, with increased ambient temperature and humidity, and when methods of convective cooling are decreased (Noakes 2003; Adams et al. 1992). When athletes drink ad libitum during a marathon race, the average weight loss is 2–3 kg (Noakes 1993), which is approximately 3%–4% of their body weight. These elite level runners tend to ingest 200 mL/h,

which is significantly less than the 1200–2000 mL/h that has been recommended in the ACSM guidelines (Sawka et al. 2007; Noakes 2003).

Noakes recommends that following standard hydration guidelines may be detrimental because there are significant differences between individual responsiveness for tolerable fluid intake. Rather, athletes should pay attention to their own sweat rate, which may be determined by metabolic rate, as well as the environmental conditions. Noakes recommends that, during a marathon, runners should aim to consume approximately 400–800 mL/h, with higher levels of intake for the faster, heavier runners exercising in warm conditions and the lower rates for the slower runners in cooler conditions. These rates seem to be consistent with feasible consumption during a marathon, whereas consumption of 1500 mL/h of fluid, both water and sports beverages, is associated with incidence of hyponatremia (Noakes 2002; Hew et al. 2003).

Evidence does not seem to support the guideline for athletes to drink as much as tolerable during exercise, but rather performance can be maintained when drinking ad libitum and with some loss in body mass. According to Noakes, ingestion rates should never exceed 800 mL/h (Noakes 2003). Noakes also argues against taking in too much sodium during exercise (Noakes 2002). High levels of sodium intake may reduce urine production and have also been shown to increase the desire to drink, which may both result in fluid overload. Noakes further warns that high rates of fluid intake of greater than 1.5 L/h for several hours may lead to hyponatremia (Noakes 1995).

10.4.3 Rehydration Strategies

Athletes should assess their postexercise urinary color and volume, as well as any change in body mass to help gauge rehydration strategies. Approximately 1.5 L of fluid should be consumed for every kilogram of body weight lost (Shirreffs and Maughan 1998b). Consumption of fluids should be done slowly to enhance retention and avoid increased urine production (Kovacs et al. 2002; Sawka et al. 2007; Wong et al. 1998). Following a prescribed rehydration plan based on volume of fluid loss may help decrease fast ingestion of fluid and decrease urine production (Wong et al. 1998). With rehydration, one is ideally looking to balance plasma osmolality and sodium concentration that likely increased during exercise. Studies have shown that rehydrating with water or a hypotonic electrolyte beverage causes this desired change and decreases renin, agiostensin II, and aldosterone concentrations (Costill and Sparks 1973; Gonzalez-Alonso, Heaps, and Coyle 1992; Shirreffs et al. 1996; Takamata et al. 1994).

There is some evidence indicating that rehydrating through water alone is difficult because water does not maintain the physiological drive to drink (Murray 2008), so it has been suggested that sodium ingestion with fluid may improve the willingness to drink as well as fluid retention. The National Athletic Trainers Association believes that including sodium in a rehydration beverage will improve both the impulse to drink and blood osmolality (Casa et al. 2000), and several studies indicate that the addition of electrolytes to a beverage is optimal for postexercise body water restoration (Costill and Sparks 1973; Maughan and Leiper 1995; Shirreffs et al. 1996). Combining carbohydrate with an electrolyte beverage containing 22 mmol/L of sodium proved to be more effective for plasma volume recovery when compared to

water alone (Costill and Sparks 1973). It is believed that the addition of carbohydrate may stimulate both sodium and water absorption within the small intestine (Fordtran 1975). Studies have shown that for whole-body rehydration, the optimal sodium level is approximately 25 mmol/L (Mitchell et al. 2000).

Cardiovascular variables returned to pre-exercise values after 3 h of rehydration regardless of the composition of the fluid. However, when athletes were given variable levels of sodium and fluid at different rates, the results showed that the volume of fluid was of greater influence on retention than the presence of sodium was (Mitchell et al. 2000). Most athletes should be able to replenish lost fluids through normal ingestion, so intravenous fluid replacement should only be done in situations of severe dehydration of greater than 7% loss of body mass (Sawka et al. 2007).

If time permits, athletes should consume normal meals and snacks while ingesting sufficient volumes of water to achieve euhydration (Institute of Medicine 2005). Consumption of a mixed meal that contains sodium has been shown to stimulate fluid ingestion and may also be an effective method for rehydration after exercise (Engell 1988; Szlyk et al. 1990; Ray et al. 1998; Maughan, Leiper, and Shirreffs 1996). In fact, some researchers suggest consuming soup or chicken broth after exercise because this has been shown to increase plasma volume with reduced urine volume when compared to water intake (Ray et al. 1998). Additionally, this same study showed that ingesting a carbohydrate–electrolyte beverage was no more effective in restoration of plasma volume than water and resulted in more urine production. Interestingly, the carbohydrate–electrolyte beverage had a higher concentration of sodium, and the soup and chicken broth were more isotonic.

Research indicates that many athletes do not rehydrate properly between exercise bouts, which may be problematic for subsequent performances (Greenleaf and Sargent 1965; Hubbard et al. 1984; Pitts and Consolazio 1944; Maughan et al. 2004; Cheuvront and Haymes 2001a). In the event of repeated aerobic exercise performance, replacement of 50% of fluid losses during a break has been shown to improve subsequent exercises better than when no hydration was allowed (Casa et al. 2000). Cardiovascular response is of large concern with repeated bouts, and studies have shown mixed responses, with higher heart rates measured after 75% rehydration (Nielsen et al. 1986) and normal heart rate with only 62% rehydration (Costill and Sparks 1973). With repeated bouts, rapid ingestion of fluids may be more beneficial for rehydration despite an increased urine production than slow ingestion would be (Kovacs et al. 2002).

10.5 HYDRATION AND ANAEROBIC EXERCISE PERFORMANCE

There is conflicting evidence of how impactful hydration is on anaerobic exercise performance. Some studies indicate that dehydration decreases performance (Viitasalo et al. 1987; Torranin, Smith, and Byrd 1979) and muscular strength (Caterisano et al. 1988), others have shown that it improves performance (Widerman and Hagan 1982), and still others have shown no difference on muscular strength (Institute of Medicine 2005; Greiwe et al. 1998) or anaerobic capacity (Jacobs 1980; Institute of Medicine 2005; Hoffman, Stavsky, and Falk 1995; Watson et al. 2005). Reasons for such

disparity between studies include various methods utilized to induce dehydration and small sample sizes.

In a comprehensive review on how hydration affects muscular performance (Judelson, Maresh, Anderson, et al. 2007), studies were classified as employing masking factors that attenuated the effects of hypohydration or with exacerbating factors that enhanced the effects of hypohydration. The authors indicated that studies with masking factors showed 0.3% decrease in muscular strength, and studies with exacerbating factors showed 3.8% decrease in strength. Overall, there was a 2% decline in strength with 3%–4% hypohydration. Studies with masking factors also showed improved power by 1.8%, whereas studies with exacerbating factors decreased power by 7.7%. On average, a 3%–4% decrease in body mass is thought to reduce power by 3%.

In regard to resistance- or power-based high-intensity endurance, studies with masking factors showed a loss of 6.7% and studies with exacerbating factors showed an endurance loss of 5.6%. On average, the data indicate that 3%–4% hypohydration reduces high-intensity endurance by 10%. This large reduction in high-intensity endurance capabilities may be due to the cardiovascular changes that occur with dehydration (i.e., decreased stroke volume and increased heart rate). The mean changes seen in strength and power with hypohydration would most likely not affect the recreational athlete a great deal, but it would affect his or her ability to maintain high-intensity endurance activities. In all three cases of strength, power, and endurance, there would be a significant effect at the elite level of athletics, especially those that are separated by seconds as seen in running, cycling, and swimming.

Studies have shown no difference in power output during Wingate anaerobic tests between euhydrated and dehydrated conditions (Jacobs 1980; Cheuvront et al. 2006). The data indicate that there was no difference in either mean or peak power output between hydration states. However, there was a significant difference between core body temperatures, with the hypohydrated state resulting in a higher core body temperature. The authors concluded that despite moderate hyperthermia and hypohydration resulting in a 2.7% body mass reduction, there was no difference in anaerobic performance as measured by a 30-s all-out cycling bout.

A similar result was found in track sprint performances with diuretic-induced dehydration (Watson et al. 2005). With more than a 2% reduction in body weight, there was no difference between the diuretic-dehydrated group and the control group in performance times for 50, 200, and 400 m runs and the vertical jump. Sprint capacity was shown to be diminished during repeat 20-s sprints of increasing intensity (Maxwell, Gardner, and Nimmo 1999). The sprint intensity was increased by increasing the incline up to a 10.5% gradient, and a 5:1 rest-to-work ratio was used. Individuals that were euhydrated were able to complete more sprints and sprint time than those that were hypohydrated. Judelson, Maresh, Farrell, et al. (2007) showed no benefit of hypohydration on vertical jump height and reported a reduction in performance during six sets of back squats.

Other data have shown that vertical jump was improved with dehydration of approximately 3% body mass (Viitasalo et al. 1987). Despite this jump study, most of the anaerobic studies indicate that there is no beneficial effect of hypohydration on performance, and in some cases there is a negative effect (Gisolfi and Lamb

1990). When dehydration was induced through sauna exposure, results showed that despite a significant loss in body mass and plasma volume through dehydration, there was no effect on peak torque or time to fatigue 3 h after dehydration (Greiwe et al. 1998).

In skills related to team sports, a state of hypohydration also appears to be detrimental. Soccer athletes who dribbled between a series of seven cones without any rehydration period realized a diminished performance of about 5% (McGregor et al. 1999). Similar results were achieved in a soccer population that underwent hypohydration of 2.4% of body mass and scored worse during a soccer-specific fitness test compared to a trial with limited fluid loss of 0.7% body mass (Edwards et al. 2007). Similarly, dehydration of 2.8% in body mass resulted in a decline in cricket bowling accuracy (Devlin et al. 2001). One important thing to note with team sport athletes is that studies suggest that soccer players tend to arrive at practice or a game in a dehydrated state (Maughan, Shirreffs, and Leiper 2007; Shirreffs et al. 2005). Similar evidence has been shown for professional basketball players (Osterberg, Horswill, and Baker 2009) and for collegiate athletes across a variety of sports (Volpe, Poule, and Bland 2009). All of the measurements used in these studies were from urinary samples.

10.5.1 Prehydration Strategies

Anaerobic or intermittent-based athletes should eat regular meals that are nutritionally balanced 24 h prior to exercise. Water is the only beverage necessary if proper intake of carbohydrates and electrolytes is included in these meals (Hoffman and Maresh 2011). Approximately 2–3 h prior to exercise, it is recommended to drink 500–600 mL of fluid, as well as another 200–300 mL of fluid 10–20 minutes before exercise (Casa et al. 2000).

10.5.2 Rehydration Strategies

Depending on the intensity of the activity and the volume of sweat lost, water may not be the best beverage for rehydration because it may decrease osmolality, decrease the drive to drink, and increase urine output (Hoffman and Maresh 2011). Sports drinks are recommended for athletes competing in multiple competitions in one day that do not have time to eat a meal (Hoffman and Maresh 2011). If the athlete has information about weight loss during the exercise, he or she should consume fluid to equal 150% of the weight loss (Shirreffs et al. 1996). Research has shown that athletes participating in high-intensity sports, such as tennis, over a 3-day period in a warm environment were able to maintain their overall fluid–electrolyte balance by drinking ad libitum (Bergeron et al. 1995). This indicates that listening and adhering to one's thirst mechanism may provide enough fluid and electrolytes to maintain physiological needs. For athletes participating in short-duration, high-intensity events, the primary emphasis should be on rehydration rather than on hydrating during their events (Maughan and Shirreffs 2010).

10.6 CONCLUSION

Many athletes are primarily concerned with their caloric intake and balancing carbohydrate, protein, and fat; water is generally a secondary thought to them. However, with the significant effects of both hyper- and hypohydration, athletes should pay attention to their water intake and losses. The existing recommendations for fluid intake before, during, and after exercise are based on scientific principles, but athletes should consider using these as guidelines rather than as hard and fast rules. These recommendations are better utilized as guidelines because of the wide variation seen with fluid intake and losses, depending upon gender, size, genetic predisposition, level of fitness, clothing worn, and exercise environment. Ultimately, athletes should pay attention to their fluid and sodium intake and listen to the one and only physiological indicator of hydration that humans have: their thirst mechanism. While previous research suggested that the thirst mechanism lags behind the need for fluid and that athletes should attempt to replace any fluid losses during exercise completely, research has also shown that there is no performance benefit that comes from this practice. Instead, athletes should aim to limit their dehydration during exercise to less than 2% of their body weight and prevent any weight gain during exercise. The thirst mechanism is the best physiological guide.

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Appendix A: Overview of Bioenergetics

Bill Campbell

CONTENTS

A.1 Introduction 297
A.2 ATP Resynthesis 299
A.3 Immediate Sources of Energy (Phosphagen System) 299
A.4 Short-Term Sources of Energy (Glycolysis) 301
A.5 Long-Term Sources of Energy (Oxidative Phosphorylation) 302
A.6 Conclusion 306
Bibliography 307

A.1 INTRODUCTION

Regardless of how skilled an athlete is, if the athlete is fatigued his or her performance will suffer. To resist fatigue, the body calls upon several energy systems. The term "energy" is broad, with several kinds of energy existing in the human body, including:

- Electrical energy in nerves and muscles
- Chemical energy in the synthesis of molecules (such as myosin heavy chain proteins in skeletal muscle)
- Mechanical energy in the contraction of muscle
- Thermal (i.e., heat) energy that is derived from all of these aforementioned processes

Inherent with the different kinds of energy that exist in the human body is an interplay between the macronutrients (carbohydrates, protein, and fat) and adenosine triphosphate (ATP) production. ATP is the primary energy source for all biological work, whether the work is electrical, chemical, or mechanical. ATP is formed in the body as macronutrients are catabolized in a manner that conserves most of the energy contained within the bonds of carbohydrates, protein, and fats. During the catabolism of these macronutrients, in addition to ATP production, the simple products CO_2 and H_2O are also formed. Figure A.1 summarizes the formation of ATP from the breakdown of the macronutrients.

In ATP, three phosphates are linked by high-energy bonds. When a bond between the phosphates is broken, energy is released and may be used by the cell. At this

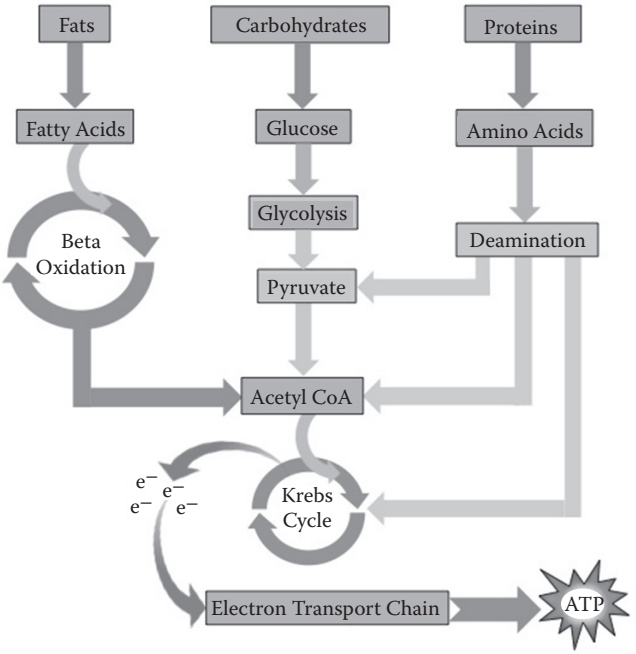

FIGURE A.1 Overview of ATP production.

point, ATP has been reduced to a lower energy state, becoming adenosine diphosphate (ADP) and inorganic phosphate (P_i). During muscular activity, ATP is constantly being broken down into ADP + P_i in order to provide the energy needed to fuel the types of activities undertaken by the athlete (Figure A.2). ATP must be replaced (or resynthesized) as fast as it is used if the skeletal muscles are to continue to generate force quickly (for power athletes) in some instances and for prolonged periods of time (for endurance athletes) in others. Essentially what is happening is that a phosphate group (also known as inorganic phosphate) is added to ADP to form ATP. We use the term *phosphorylation* to describe the process of resynthesizing ATP from ADP.

Given the importance of resynthesizing ATP from ADP in order for muscular activity to continue, an understanding of the various pathways responsible for converting the macronutrients into ATP is needed. Bioenergetics is the study of how energy is captured, transferred, and utilized within biological systems. The transfer of energy is a major limiting factor of human performance. A comprehensive understanding of bioenergetics allows the athlete and his or her support staff to design appropriate training programs that mimic the demands of the athlete's sport. In addition, specific diet programs can be created for the athlete, matching the dominant

$$\text{ATP} \xrightarrow{\text{ATPase}} \text{ADP} + P_i$$

FIGURE A.2 ATP hydrolysis.

types of work that the athlete demands of his or her body with the macronutrients that best meet the physiologic demands.

For example, athletes who are primarily power athletes (shot-putters, gymnasts, high jumpers, etc.) do not need as many carbohydrates in their diets to fuel the types of explosive activities in their daily training and competitions. That is not to say that these athletes do not need carbohydrates, but rather, in comparison to other types of athletes such as endurance athletes and intermittent sport athletes (basketball players, soccer players, hockey players, etc.), carbohydrates will not play as prominent a role. In order to make appropriate decisions about the athlete's diet without fear of doing harm to performance capabilities, the following principles must be understood:

1. The metabolic demands of the sport
2. How the body resynthesizes ATP
3. The rates with which ATP can be resynthesized

This appendix seeks to provide a summary of points 2 and 3.

A.2 ATP RESYNTHESIS

Skeletal muscle cells possess a great ability to replace ATP under a variety of work demands, ranging from a high-intensity 100-m sprint to running a marathon as fast as possible. A logical approach to understanding how energy (i.e., ATP) is supplied for muscle contraction is to view it from the perspective of how rapidly ATP can be resynthesized. In this regard, the energy sources can be divided into the following:

- Immediate sources of energy (also referred to as the phosphagen system)
  - Maximal bursts of effort lasting 1–5 s
- Short-term sources of energy (glycolysis)
  - Involves maximal work lasting about 2 min
- Long-term sources of energy (also referred to as oxidative phosphorylation)
  - Maximal work lasting longer than 2 min and in all submaximal work

A.3 IMMEDIATE SOURCES OF ENERGY (PHOSPHAGEN SYSTEM)

The phosphagen system is the immediate source of energy for skeletal muscle contraction. Three main phosphagens are involved: ATP, ADP, and phosphocreatine (PCr). For ATP to be the important energy currency it is, the concentration in the muscle fiber must be kept well above equilibrium, to the point that the ratio of ATP/ADP is normally greater than 40. During most exercise bouts when ATP is used as an energy source, it is replenished at the same rate that it is utilized, so its concentration does not decrease very much. Only during very severe exercise does the concentration of ATP decline, but not to less than 50% of resting levels. As an example, there is a 20% decrease in ATP concentrations at an exercise intensity of 100% VO_2max for 5–6 min and a 50% decrease for an all-out intensity during a 30-s Wingate test (Sahlin et al. 1987; Norman et al. 2001). Even though these very high intensity exercise bouts result in a decrease of ATP concentrations, it is important to

remember that during most kinds of physical activity (less than maximal effort), the ATP content seldom decreases much below the resting level.

Given the importance of ATP for muscle contraction, one would assume there would be large stores contained in skeletal muscle. This is not the case. There is a very limited supply of readily available ATP stored in the skeletal muscles. In fact, the amount of ATP that is available may meet the energy demands of a maximal effort lasting about 1 s. This means that ATP-generating processes cannot produce ATP at the same rate at which it is hydrolyzed to drive muscle contraction during sprinting. Therefore, if a maximal effort is to last for several seconds, the ADP that is formed from ATP hydrolysis needs to be rapidly rephosphorylated into ATP. To prevent muscle fibers from exhausting their ATP stores at the start of maximal contractions, an alternate energy-rich molecule is capable of regenerating ATP at a very high rate. This phosphagen is known as phosphocreatine (also referred to as creatine phosphate).

PCr is a high-energy phosphate stored in skeletal muscle that provides the most rapid means to resynthesizing ATP. By donating its phosphate molecule to ADP, ATP is resynthesized, allowing skeletal muscle to continue producing force. The enzyme responsible for catalyzing this reaction is creatine kinase (Figure A.3).

During exercise, the concentration of PCr declines while the concentration of free creatine increases. For all-out efforts to fatigue (such as during a Wingate test), PCr levels can decrease by 90% or more. One of the reasons why athletes ingest supplemental creatine is because there is a limited supply of PCr in the skeletal muscle. If an adequate amount of creatine is ingested orally, then creatine will enter the muscle fiber and subsequently more PCr will be formed, thereby increasing the resting concentration of muscle PCr stores. This then results in significant improvements in sprint performance and anaerobic exercise capacity (Buford et al. 2007; Branch 2003; van Loon et al. 2003).

The creatine kinase reaction takes place as fast as the muscle forms ADP. However, the phosphocreatine that is stored in the muscle lasts only about 5 s when the muscle is contracting at maximal levels. Thus, PCr is the primary source of ATP for athletic feats such as the first few seconds of a sprint, repeated vertical jumps in a basketball or volleyball game, and sprinting to first base in a baseball game. During muscle contraction, the forward direction of the creatine kinase reaction is favored in order to regenerate ATP. During recovery or rest periods, the reverse of the creatine kinase reaction is favored to regenerate PCr. In this reverse reaction, there is a phosphate transfer from ATP (produced by oxidative phosphorylation) to creatine forming PCr. The concentration of PCr in skeletal muscle is about three to four times that of ATP. This is enough to act as a temporary ATP buffer until other ATP-generating processes (such as glycolysis) reach maximal rates.

In addition to the creatine kinase reaction, there is another enzymatic reaction that rapidly resynthesizes ATP from ADP. Adenylate kinase (also known as myokinase) resynthesizes ATP from two ADP molecules. Specifically, adenylate

FIGURE A.3 Creatine kinase reaction.

FIGURE A.4 Adenylate kinase reaction.

kinase causes two ADP molecules to interact and produce one ATP and one AMP. This interaction keeps the ADP concentration from building up to the extent that it would otherwise without this reaction. The adenylate kinase reaction is demonstrated in Figure A.4. Both the creatine kinase and the adenylate kinase reactions do not require oxygen and are therefore considered anaerobic energy systems for producing ATP.

A.4 SHORT-TERM SOURCES OF ENERGY (GLYCOLYSIS)

As the muscle depletes its store of available PCr, the muscle fibers break down glucose to produce ATP at a high rate. While the breakdown of glucose is slower than the phosphagen system, it is still considered a fast form of resynthesizing ATP. The process of breaking down glucose to form ATP is referred to as glycolysis. There are four sources of glucose for entering glycolysis:

- Muscle glycogen
- Blood glucose derived from liver glycogen breakdown
- Blood glucose derived from the liver via gluconeogenesis
- Blood glucose derived from ingested carbohydrates

Glycolysis does not require oxygen to produce ATP and hence is also an anaerobic ATP-producing pathway. During glycolysis (which occurs in the sarcoplasm), glucose is broken down into two molecules of pyruvate and in the process ADP is converted into ATP. The starting point of glycolysis is glucose and the ending point of glycolysis is two molecules of pyruvate. The process of converting glucose into two molecules of pyruvate requires the activity of 10 different enzymes. The reactions of glycolysis are shown in Figure A.5.

It was stated earlier that glycolysis starts with glucose and ends with two molecules of pyruvate. While this is the technical definition of glycolysis, glycolysis can start from a point physiologically "above" glucose: muscle glycogen. Also, in certain instances the end point of glycolysis, pyruvate, can be converted into lactate. The conversion of pyruvate to lactate occurs when glycolysis occurs at a high rate, such as during high-intensity exercise. If exercise continues at a high intensity, the lactate that is produced accumulates in the muscle and the blood. As lactate levels continue to accumulate, it correlates with muscular fatigue as lactate production increases the intracellular acidity (decreases the pH levels) of skeletal muscles.

While the normal reactions of glycolysis (the conversion of glucose to two molecules of pyruvate) require 10 enzymatic reactions, the process of starting with muscle glycogen and ending with lactate requires 12 enzymatic reactions. The specific reactions that describe the process of converting muscle glycogen into a glucose unit and pyruvate to lactate are discussed in Chapter 3, "Carbohydrate Metabolism." The

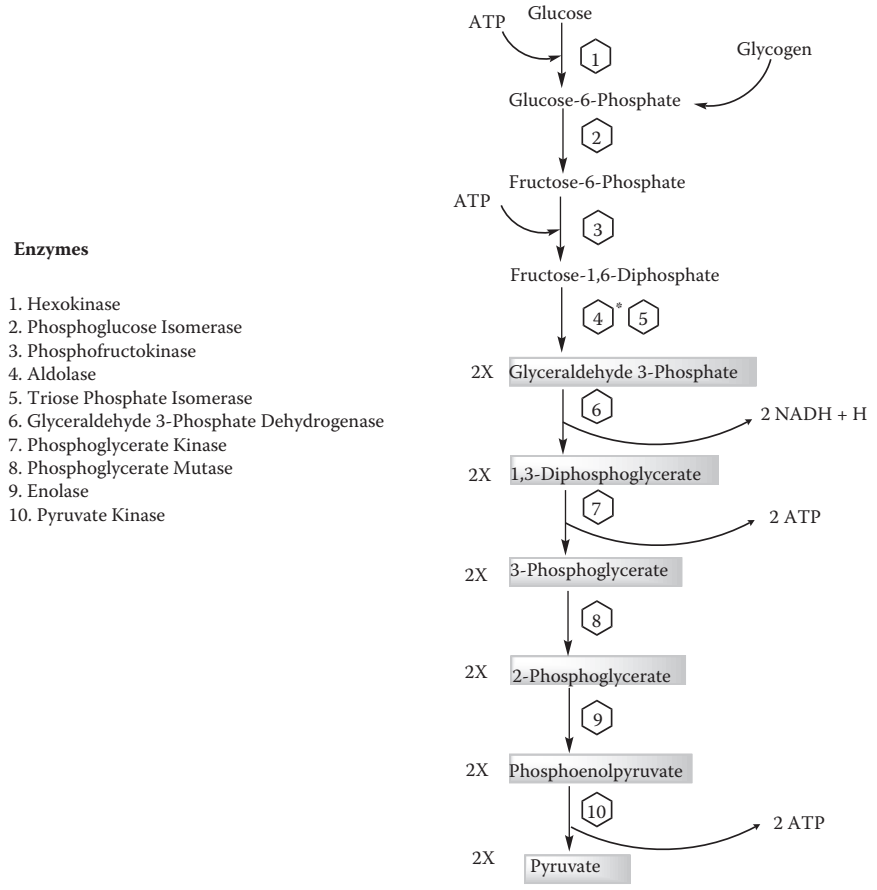

FIGURE A.5 Glycolysis.

reactions of glycolysis supply ATP at a fast rate and are able to supply the needed energy for events involving maximal work for about 2 min.

A.5 LONG-TERM SOURCES OF ENERGY (OXIDATIVE PHOSPHORYLATION)

Oxidative phosphorylation is the formation of ATP from ADP and P_i in association with the transfer of electrons from fuel substrates (carbohydrates, protein, and fat) to coenzymes to oxygen. Because the use of oxygen is needed, these long-term energy sources are aerobic (i.e., requiring oxygen) processes. Products of oxidative phosphorylation are H_2O and CO_2. Other names for oxidative phosphorylation include aerobic metabolism, oxidative metabolism, and cellular respiration. Oxidative phosphorylation takes place in the mitochondria and encompasses the Krebs cycle and the electron transport chain. The substrates that can provide energy aerobically are carbohydrates, protein, and fats. When

compared to the amount of energy that carbohydrates and fats can produce, the amount of energy derived from protein is minimal. Therefore, the discussion of breaking down macronutrients to resynthesize ATP aerobically will focus on carbohydrates and fat.

There are two major metabolic pathways involved in oxidative phosphorylation:

- Krebs cycle (sometimes referred to as the tricarboxylic acid cycle or the citric acid cycle)
- Electron transport chain (ETC)

The Krebs cycle starts with the macronutrient-derived metabolite acetyl-coenzyme A (acetyl-CoA). The acetyl-CoA that enters the Krebs cycle was derived from carbohydrate or fat. The energy originally contained in the carbohydrate or fat is subsequently extracted from the acetyl-CoA molecule and is used to generate ATP in the electron transport chain. During this process, the reduced coenzymes nicotinamide adenine dinucleotide (NADH) and flavine adenine dinucleotide ($FADH_2$) are formed, as well as CO_2. The acetyl-CoA that was generated from carbohydrate (glucose) was specifically derived from the end product of glycolysis: pyruvate. The pyruvate that was formed in the sarcoplasm during glycolysis is taken into the mitochondria, where it is converted into the two-carbon compound acetyl-CoA.

Once formed, acetyl-CoA enters the Krebs cycle where it is completely broken down. Fat breakdown also results in the same intermediate: acetyl-CoA. The first step in fat oxidation/breakdown is lipolysis, which is the breakdown of a triglyceride into three fatty acids and one glycerol molecule. Fatty acids that are broken down to produce ATP basically come from two bodily stores: adipose tissue and intramuscular triglyceride stores. Regardless of the source, the fatty acids are transported into the mitochondria where they are also broken down (via a process termed beta-oxidation) into acetyl-CoA, which is the same two-carbon compound into which carbohydrates are converted. Acetyl-CoA is so important to the Krebs cycle that it is sometimes referred to as the driver of the Krebs cycle.

TOPIC BOX A.1 MITOCHONDRIA

The mitochondria are located in the sarcoplasm of muscle cells and are composed of two lipid bilayers: an inner membrane and an outer membrane. Because of this double-membrane organization, there are five distinct parts to a mitochondrion:

1. Outer mitochondrial membrane (divides the mitochondrion from the sarcoplasm of the muscle cell)
2. Intermembrane space (space between the outer and inner membranes)
3. Inner mitochondrial membrane (where the electron transport chain is located)

4. Cristae space (formed by infoldings of the inner membrane)
5. Matrix (space within the inner membrane)

Oxidative phosphorylation (including the Krebs cycle and the electron transport chain) takes place in the mitochondria. Specifically, all of the enzymes of the Krebs cycle take place within the mitochondrial matrix. The inner membrane is the site of the redox reactions of the electron transport chain. Mitochondrial density (the number of mitochondria present) is very important in terms of synthesizing ATP. The more mitochondria contained in a cell, the greater the amount of ATP that can be produced. Because they generate most of the muscle cell's supply of ATP, the mitochondria are sometimes described as "cellular power plants" or the "power house of the cell."

After acetyl-CoA is formed and enters the Krebs cycle, it first reacts with oxaloacetate to form citrate under the enzymatic control of citrate synthase. This is the first of eight reactions that occur in the Krebs cycle. Figure A.6 displays the eight reactions of the Krebs cycle. As the figure shows, one turn of the Krebs cycle consumes one acetyl group and produces four pairs of electrons (three pairs on NADH and one pair on $FADH_2$), one guanosine triphosphate (GTP), and two CO_2 compounds. The GTP that is produced is very similar to ATP and is an example of substrate level

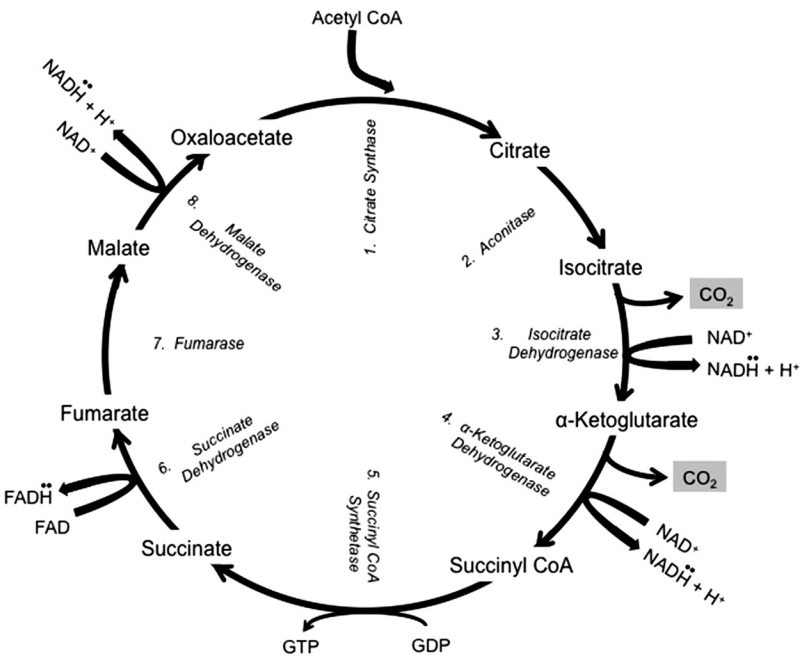

FIGURE A.6 Krebs cycle.

phosphorylation—that is, formation of an energy-rich phosphate without using oxidative phosphorylation. The ATP produced in glycolysis also occurs via substrate-level phosphorylation. The formation of NADH and $FADH_2$ is critical in terms of producing ATP via oxidative phosphorylation.

TOPIC BOX A.2 SUBSTRATE LEVEL PHOSPHORYLATION AND OXIDATIVE PHOSPHORYLATION

There are two mechanisms involved in ATP production: substrate level phosphorylation and oxidative phosphorylation. Substrate level phosphorylation is the conversion of ADP to ATP *without* the use of oxygen; in other words, it is an anaerobic process. Oxidative phosphorylation, on the other hand, requires oxygen for the transfer of a phosphate group to phosphorylate ADP to its triphosphate form and is thus an aerobic process.

In order to appreciate the importance of NADH and $FADH_2$ in resynthesizing ATP in aerobic metabolism, the terms *oxidation* and *reduction* must be understood. Oxidation can take three forms. The first form is to add an oxygen atom to a substance; the second involves the loss of a hydrogen atom from a substance; the third form involves the loss of an electron from a substance. In contrast, reduction involves the loss of an oxygen atom from a substance or the gain of a hydrogen atom or an electron. For the purposes of studying ATP resynthesis, we will focus on the fact that oxidation involves a loss of electrons and reduction involves a gain of electrons.

NAD^+ (nicotinamide adenine dinucleotide) and FAD (flavine adenine dinucleotide) are coenzymes that are able to accept and donate electrons; hence the ability to be reduced and oxidized. During reactions three, four, and eight, NAD^+ (the oxidized form of this coenzyme) removes two hydrogen atoms from its substrate. It is important to remember that a hydrogen atom comprises one proton (H^+) and one electron (e^-). From the two hydrogens that were removed by NAD^+, both electrons but only one proton are accepted by the NAD^+ to produce its reduced form, NADH, plus H^+.

Similarly, in reaction six, FAD (the oxidized form of this coenzyme) removes two hydrogen atoms ($2H^+$ and $2e^-$) from its substrate. Both electrons and both protons are accepted by the FAD to produce its reduced form, $FADH_2$. Since NAD^+ and FAD take two hydrogens and their associated electrons, it is helpful to think of them with having two dots placed above them as a reminder that they are carrying electrons (Figure A.7).

Once NADH and $FADH_2$ are formed, their primary role is to take the electrons they have removed from the various intermediates in the Krebs cycle and deliver them to the electron transport chain. The ETC accomplishes exactly what its name

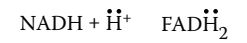

FIGURE A.7 NADH and $FADH_2$.

implies: It transfers the electrons delivered from NADH and $FADH_2$ to oxygen, ultimately resulting in the production of ATP (via the phosphorylation of ADP with P_i). In addition to the ATP that is produced in the electron transport chain, water is formed. In oxidative phosphorylation, electrons are transferred from NADH and $FADH_2$ to oxygen, reducing the oxygen (which combines with two protons [2 H^+] to produce water). At the same time, NADH and $FADH_2$ are oxidized to NAD^+ and FAD, respectively.

The electron transport system consists of five protein–lipid complexes located in the inner mitochondrial membrane. Four of the complexes make up the electron transport chain and the fifth complex is referred to as ATP synthase. The protein complexes that make up the electron transport chain are referred to as cytochromes. One of the first things that occur in the electron transport chain is the arrival of NADH and $FADH_2$ from the Krebs cycle in the mitochondrial matrix. Both NADH and $FADH_2$ are oxidized by removing the hydrogen atoms. These atoms split into protons (H^+) and electrons (e^-). In three of the four complexes that make up the electron transport chain, these protons (H^+) are pumped across the mitochondrial inner membrane from the matrix side to the intermembrane space.

Proton pumping is a form of active transport since the protons are being transferred across a membrane from a region of lower concentration (the matrix) to one that is higher in concentration (the intermembrane space). The energy needed to pump these protons against their concentration gradients is generated by the energy released when the electrons flow from one complex (cytochrome) to another. The ATP synthase, or complex V, couples proton flow down the gradient into the matrix to phosphorylation of ADP with P_i to make ATP. The electrons that are transferred from cytochrome to cytochrome in the electron transport chain are ultimately received by oxygen. Because of this, oxygen is referred to as the final electron acceptor. So, oxygen that has diffused into the mitochondria reacts with the two electrons and two hydrogens to produce a water molecule.

While the ATP production via aerobic mechanisms is slower than the ATP production from immediate and short-term sources of energy, the amount of ATP produced aerobically is vast compared to the limited amounts of ATP that are produced anaerobically. Aerobic production of ATP is the primary means of supplying energy to the muscle in maximal work lasting more than 2 min and in all submaximal work.

A.6 CONCLUSION

Bioenergetics is the study of how energy is captured, transferred, and utilized within biological systems. Athletes and their support staff should have a basic understanding of bioenergetics and should choose modes of training, conditioning, and dietary strategies that complement one another. The phosphagen system is utilized during immediate, high-intensity activities with transition to glycolysis and aerobic metabolism as exercise becomes more prolonged. The three energy systems are constantly working together to maintain ATP levels. ATP levels are never depleted; rather, if the energy systems cannot keep up with energy demand, fatigue occurs. An understanding of bioenergetics and knowledge of how to design conditioning and dietary

programs that support the energy systems primarily utilized by the athlete will increase the potential for continuous, improved performance.

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Nutrition

SPORTS NUTRITION

Enhancing Athletic Performance

Edited by

BILL I. CAMPBELL

With the constant flow of information related to sports nutrition coming from scholarly journals, it is difficult to sift through it all and determine what is relevant. *Sports Nutrition: Enhancing Athletic Performance* helps in this endeavor, with more than 1,000 references from top academic journals, offering critical knowledge concerning nutrient ingestion for enhancing exercise and sports performance. This book offers a clear focus on scientifically based sports nutrition advice to maximize performance. It also addresses exercise metabolism, which governs how nutrients exert physiologic effects that lead to increased athletic potential.

The book examines the three key macronutrients: fat, carbohydrate, and protein. It discusses various aspects of macronutrient metabolism, including differences between a body at rest and during high-intensity exercise. Topics covered in the text include the following:

- Nutrient timing
- Leucine threshold to optimize muscle protein synthesis
- Carbohydrate manipulations for better endurance- and resistance-exercise performance
- Dietary fat intake recommendations for improving performance
- Carbohydrate loading strategies
- Optimal amounts of protein to ingest on a meal-by-meal basis
- Pre-exercise dietary fat intake strategies
- Comparison of high-quality proteins

In addition to enhancing performance, the book describes how to improve body composition, presenting a scientifically based strategy for losing body fat while maintaining precious lean muscle mass. Four principles of fat loss are set forth that are integral for success in optimizing body composition. This book presents both performance nutrition principles and exercise biochemistry, addressing the key questions of what, when, and how much to ingest for improved performance and training recovery.

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ISBN: 978-1-4665-1358-7

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