

COMPUTATIONAL MODELING for ANTHROPOMETRY

Zahra Hojjati Zidashti, PhD
Soheila Yavarmasroor
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LIST OF ABBREVIATIONS

ACB	Spanish Basketball League
ANS	Autonomic Nervous System
BMI	Body Mass Index
DH	Distance Hopped
FAO	Food and Agriculture Organization
HR	Heart Rate
HRV	Heart Rate Variability
IAAF	International Association of Athletics Federation
LSI	Life Style Inventory
NIOSH	National Institute for Occupational Safety and Health
SPSS	Statistical Product and Service Solutions
UNU	United Nations University
WHO	World Health Organization

LIST OF SYMBOLS

T	Time (sec)
V	Velocity (m/s)
S	Stature (m)
P	Runner Blood Pressure (mm-Hg)
S	Runner Stature (cm)
BMI	Weight (kg)
H	Runner heart rate

Subscripts

Min.	Minimum
Max.	Maximum
Lab.	Laboratory

PREFACE

This book provides a broad understanding of the main computational techniques used for anthropometric data. The theoretical background to a number of techniques is introduced and general data analysis techniques and examining the application of techniques in a sport setting, including current practices and current research, are considered. The book also provides practical experience of females' physical activity. This book offers scope for academics, researchers, and sport professionals to present their research and development works that have potential for applications in several disciplines of any orthopedic injury that would inhibit physical activity. Chapters range from new methods to novel applications of existing methods to gain an understanding of the material and/or structural behavior of new and advanced systems. This book provides innovative chapters on the growth of educational, scientific, and research activities among athletes and provides a medium for mutual communication between international sports academia. This book publishes significant research reporting new methodologies and important applications in the fields of anthropometric measuring and software. Software and anthropometric-based research findings to date contribute to the scholarly debates over mortality trends, the nature of slavery, and the outcomes of industrialization and economic development. On the other hand, this idea was used to perform a proper analysis to provide a dynamic response to the shortcomings of the body motion. This book also uses high technology and high-speed detectors equipment to determine the operational procedures to avoid hazards on human health from the points of economics and human biology. Consequently, the results of this book will help to reduce the health risk of sport activities.

INTRODUCTION

This book, *Computational Modeling For Anthropometry*, shows a dynamic and computational modeling for anthropometry of athletes. According to this technique, an improved modeling based on measurements was classified and compared with a special type of skulls of sportswomen with other anthropometric classes of sportswomen. The disciplines of anthropometry and biomechanics have a specialized vocabulary of terms with specific meanings for designating points and distances of measurement, range, direction of motion, and mass. Height itself has a direct benefit on economic success or an increased standard of living. Rod Usher's "a tall story for our time" shows that one's tallness is a product of favorable living conditions. Thus, growth in human height within a designated area could well be an accurate measurement of economic growth and development. Many years ago some works compared the skulls of men with those of other animals. Some of them claimed that antique statues presented an angle of 90° , Europeans of 80° , Black people of 70° , and the Orangutan of 58° . Swedish Professor of Anatomy Anders Retzius first used the cephalic index in physical anthropology to classify ancient human remains found in Europe. He classed skulls in three main categories—long and thin, short and broad, and intermediate length and width. Paleoanthropologists still rely upon craniofacial anthropometry to identify species in the study of fossilized hominid bones. Specimens of *Homo erectus* and athletic specimens of *Homo sapiens*, for example, are virtually identical from the neck down but their skulls can easily be told apart. In 1856, workers found in a limestone quarry the skull of a Neanderthal man. The discovery was jointly announced in 1857, giving rise to the discipline of paleoanthropology. A multitude of factors affects physical performance. The effects of both physiological and anthropometric factors vary, depending on the intensity, duration, mode, and some other peculiarities of physical exercise. Some researchers found a significant correlation between height and performance in 45 m-sprint test. It seems in healthy matured athletes there

may be a correlation between height (stretch stature), blood pressure, and Running Time. Some researchers and coaches can use a regression line by these variables for their selection and predict exercise blood pressures of their athletes from height or sprint Running Time.

This book compares the anthropometric data for blood pressure of runners related to Runner Velocity (Running Time), Runner Stature, runner weight. Consequently, the results of this work will help to reduce the risk of sportswomen activities.

— **Zahra Hojjati Zidashti, PhD, Soheila Yavarmasroor,
and Kaveh Hariri Asli, PhD**

CHAPTER 1

FORMULATION OF REGRESSION MODEL IN SPORT AND ENGINEERING

CONTENTS

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1.1 INTRODUCTION

Regression model and Scatter diagram; correlation; regression and curve fit relates the runner dynamic blood pressure to running velocity. The autocorrelations procedure for blood pressure shows by estimating regression statistics and producing related plots for field tests model with the Regression equation. The effects of both physiological and anthropometric factors vary depending on the intensity, duration, mode and some other peculiarities of physical exercise.

It seems in healthy athletes there may be a correlation between weight, blood pressure and Running Time.

1.2 MATERIALS AND METHODS

In this work, dateline for field tests data collection was at 10:00 a.m., 10/05/2012 until 09/06/2012. Locaton of work field tests model was at Rasht city in the north of Iran. Following are considerations that was made when using and applying anthropometric data for blood pressure of runner related to Runner Velocity (Running Time) and Runner Stature. Blood pressure of runner was measured in two cases: first at the running state and second at the before of running. Twenty healthy and young sprint runner females participate in this study. They signed a written/informed consent. All subjects completed a medical questionnaire to ensure that they were not taking any medication, and were free cardiac, respiratory, renal or metabolic diseases. They didn't have any orthopaedic injury that would inhibit physical activity. The subjects were tested in the follicular phase of the menstrual cycle based on their previous menses. All participants consumed their last meal at least 2 h before the test and refrained from intensive exercise in the 24 h period preceding testing. All anthropometric measures were recorded in the morning by the same experienced anthropometrist. Stretch stature was measured using a wall-mounted stadiometer (± 0.1 cm). The exercise systolic and diastolic blood pressures were measured on left arm of each participant using cuffs of appropriate size (Table 1).

TABLE 1 Anthropometric model for dependent variable.

No.	Age	Height (cm)	BMI	WHR	100 (m) Running Time (sec)
1	19	165	18.38	0.71	13.45
2	16	159	20.63	0.75	13.66
3	18	168	16066	0.73	15.03
4	16	158	20.08	0.74	13.67
5	14	164	19.44	0.71	14.3
6	14	166	16	0.76	14.73
7	14	161	20.84	0.7	17.1
8	20	161	17.76	0.73	17.55
9	21	159	19.04	0.75	16.28
10	14	167	18.7	0.76	16.03
11	18	168	19.85	0.74	16.27
12	15	64	20.52	0.68	16.77
13	23	154	18.98	0.77	17.35
14	22	170	19.37	0.71	15.22
15	21	174	19.53	0.72	14.98
16	21	151	24.12	0.74	14.9
17	22	170	21.45	0.73	14.51
18	28	158	22.08	0.73	13.98
19	22	165	20.58	0.71	14.45
20	24	166	22.54	0.8	16.98

The measurements were recorded just after 100 m intense running in standing position [1–19]. Curve estimation procedure has used these data, which have been detected in field tests procedure (Table 1).

These data were compared by blood pressure of runner and Runner Velocity and stature data, which were collected from actual systems (field tests).

The model was calibrated by using one set of data, without changing parameter values [20–21].

Thus, we obtain the following model to approximate the variables:
Linear function:

$$y = a_0 - a_1x \quad (1)$$

Logarithmic function:

$$\log y = \log(-1.242) + b \log x \quad (2)$$

Compound function:

$$A = Ce^{kt} \quad (3)$$

Quadratic function:

$$y = a_0 + a_1x + a_2x^2 \quad (4)$$

Growth function:

$$(dA/dT) = KA \quad (5)$$

Exponential function:

$$y = ab^x + g \quad (6)$$

Logistic function:

$$y = ax^b + g \quad (7)$$

Regression model (Table 1) was built based on field tests data. Scatter diagram; correlation; regression and curve fit were defined for runner dy-

dynamic blood pressure against Running Time. The autocorrelations procedure for blood pressure was showed by estimating regression statistics and producing related plots for field tests model with the following three assumptions in Eqs. (10)–(12):

Assumption (1):

$$p = f(S), \quad (8)$$

Assumption (2):

$$p = f(V), \quad (9)$$

Assumption (3):

$$p = f(V, S), \quad (10)$$

V – Runner Velocity is the most important variable, S – Runner Stature. Dependent variable: P – Blood pressure (mm-Hg) for starting point of running condition. The independent variable is Running Time (sec) and Runner Stature (cm).

The purpose of this study was although to determine the relationship between Runner Velocity and important anthropometric variables. Variables were selected for the study on the basis of theoretical modeling or previous research regarding the relationship between study variables and Runner Velocity range. The specific research question addressed was whether a significant relationship exists between Runner Velocity range and some combination of the following variables: age, sex, height, weight, torso height, percentage body fat, arm length, thigh girth, calf girth, pelvic width, and pelvic girth.

Following are considerations was made when using and applying anthropometric data for heart rate of runner related to Runner Velocity (Running Time) and runner weight. Heart rate of runner was measured in two cases: first at the running state (dynamic heart rate) and second at the before of running (static heart rate). Twenty healthy and young sprint runner females (Tables 1, and 2) participate in this study. They signed a written/informed consent.

TABLE 2 Model for formulation and regression.

No.	Age	Running speed (m/sec)	Stature (cm)	Weight (kg)	Dynamic Heart Rate	Static Heart Rate
1	19	7.43	165	50	176	74
2	16	7.32	159	52	173	72
3	18	6.65	168	47	174	70
4	16	7.31	158	50	111	70
5	14	6.99	164	52	173	79
6	14	6.78	166	44	178	77
7	14	5.84	161	54	191	111
8	20	5.69	161	46	133	83
9	21	6.14	159	48	160	82
10	14	6.23	167	52	150	79
11	18	6.14	168	56	153	75
12	15	5.96	64	55	166	81
13	23	5.76	154	45	140	94
14	22	6.57	170	56	154	73
15	21	6.67	174	59	164	79
16	21	6.71	151	55	177	77
17	22	6.89	170	62	93	75
18	28	7.15	158	55	170	68
19	22	6.92	165	56	166	70
20	24	5.88	166	62	168	83

All subjects completed a medical questionnaire to ensure that they were not taking any medication, and were free cardiac, respiratory, renal or metabolic diseases. They didn't have any orthopaedic injury that would inhibit physical activity. The subjects were tested in the follicular phase

of the menstrual cycle based on their previous menses. All participants consumed their last meal at least 2 h before the test and refrained from intensive exercise in the 24 h period preceding testing. All anthropometric measures were recorded in the morning by the same experienced anthropometrist. The weight was measured using a wall-mounted stadiometer (± 0.1 cm). The exercise systolic and diastolic heart rates were measured on left arm of each participant using cuffs of appropriate size (Table 2).

1.3 RESULTS AND DISCUSSION

Conclusions were drawn on the basis of experiments and calculations for the three assumptions:

Assumption (1):

$$p = f(S), \quad (11)$$

The most important effects that were observed based on regression for model summary were showed.

The auto-regression procedure accounts for first-order auto-correlated residuals. It provides reliable estimates of both goodness of-fit measures and significant levels of chosen predictor variables.

Present work compared the anthropometric data for blood pressure of runner related to Runner Velocity (Running Time) and Runner Stature in two cases [22–24]:

First: at the running state (dynamic condition);

Second: before the running state (static condition).

Dependent variable: P – blood pressure with nomenclature “ Y ”.

Independent variable with nomenclature “ X ” such as:

V – Runner Velocity is the most important variable, S – Runner Stature.

The independent variable is velocity (m.s^{-1}) and Runner Stature (cm).

Assumption (2):

$$p = f(V), \quad (12)$$

For second assumption, the curve estimation procedure allows quick estimating regression statistics and producing related plots for different models.

Hence the auto-regression procedure by regression software “SPSS 10.0.5” was selected for the curve estimation procedure in the present work. The regression model was defined based on field test data [23–26]. Regression software “SPSS” fitted the function curve and provided regression analysis. So, the regression model has been found in the final procedure. This model has compared field test results using Lab model results.

Assumption (3):

$$p = f(V, S), \quad (13)$$

For third assumption, the curve estimation procedure was illustrated. The runner blood pressure was formed by estimating regression statistics are listed in Table 1. Although related plots for the field test model were produced [27–38]. Regression software “SPSS 10.0.5” performs multidimensional scaling of proximity data to find least-squares representation of the objects in a low-dimensional space.

1.3.1 COMPARISON WITH OTHER WORKS

Rahmani-Nia and Hojjati obtained the effect of exercise training on body composition and aerobic power in sedentary college age females. Comparison showed similarity in present work and the results of that works [39–100].

1.4 CONCLUSIONS

The literature indicates that relatively little research is available to describe the relationship between functional running tasks and characteristics of individuals who perform these tasks. Considerable research does exist, however, indicating significant relationships between the strength of specific muscle groups and anthropometric and demographic variables.

Several studies, however, indicate the value of using multiple variables in regression analyses to predict computational modeling for anthropometric of athletes. The results of present work raise the question of whether a

similar approach might be useful in predicting normal functional-running Materials and Methods range on the basis of models that include multiple anthropometric and demographic predictor variables.

NOMENCLATURES

P Runner Blood Pressure (mm-Hg)

t Running Time (sec)

V Velocity (m/s)

S Runner Stature (cm)

KEYWORDS

- anthropometric
- body motion
- computational model
- physical modeling
- regression

REFERENCES

1. Hurst Thomas, D. Skull Wars Kennewick Man, Archaeology and the Battle for Native American Identity, Washington University: School of Law, Anheuser-Busch Hall, 2001; 38–41.
2. Nabieh, A.; Mohamed, I. Anthropometric Measurements as a Significant for Choosing Juniors in Both Volleyball and Handball Sports (Factorial Analysis Study), *World J. Sport Sci.*, **2010**, 3(4), 277–289.
3. Rogana, S.; Hilfikerd, R.; Clarysb, P.; Clijsenc, R.; Taeymansac, J. Position-Specific and Team-Ranking-Related Morphological Characteristics In German Amateur Soccer Players – A Descriptive Study, *Int. J. Appl. Sports Sci.*, **2011**, 23(1), 168–182.
4. Luigi P. W.; Bercades, T. Somatotypes of National Elite Combative Sport Athletes, *Braz. J. Biomotricity*, **2009**, 3(1), 21–30.
5. Dacres-Mannings, S.; Rochester, S.; Frail, H. Anthropometric Profiles of Australian Rugby Institute, Club and State Level Rugby Union Players, **2010**.

6. Gaurav, V.; Singh, M.; Singh, S. Anthropometric characteristics, somatotyping and body composition of volleyball and basketball players, *J. Phys. Educ. Sports Manage.*, **2010**, *1*(3), 28–32, Available online <http://www.acadjourn.org/jpesm>
7. Beat K.; Kohler, G. Influence of anthropometry on race performance in ultra-endurance triathletes in the longest triathlon in North America, *Int. SportMed J.*, **2007**, *8*(2), 87–96, <http://www.ismj.com>
8. Joseph, J.; Greene, M. S.; Timothy, A.; McGuine, A. T. C.; Levenson, G.; Thomas M. Anthropometric and Performance Measures for High School Basketball Players, *J. Athl. Training*, **1998**, *33*(3), 229–232
9. Amatya, D. L. Comparative Study of Somatotype of Nepalese Sportsmen, National Association for Sports Health and Fitness. **1999**.
10. Sang Hong, K. Kinanthropometric Study of Korean and Nepalese Marathon Runners, keimyung university Diwakar Lal Amatya, MSC-Athletics and Sports Science-India, Sports Kinanthropometrist-Australia, Sports Expert-National Sports Council, January. **2008**.
11. Gaunt, B. W. Anthropometric and Demographic Factors Affecting Distance Hopped-aid Limb Symmetry Index for the Crossover Hop for distance test in High School athletes, *J. Orthop. Sport Phys.*, **2001**, *31*(3), 145–151
12. Gross, M.; Dailey, E. S.; Melissa, D.; Dalton, A.; lee, K.; Wendy, I.; Ashley, C. Relationship Between Lifting Capacity and Anthropometric Measures, *J. Orthop. Sport Phys.*, **2000**, *30*(5), 237–247, 258–262.
13. Roberto, C, Jose, A.; Perez, J.; Cortell, m. Juan, J.; Rivas, J. Correlations Among Anthropometric Parameters, Jump Power, And Position In Professional Basketball Players, **2008**.
14. Pui, W. K.; Hendrik H. Anthropometric, gait and strength characteristics of Kenyan distance runners, *J. Sports Sci. Med.* **2008**, *7*, 499–504, <http://www.jssm.org>
15. Jawis, M. N.; Singh, R.; Singh, H. J.; Yassin, M. N. Anthropometric and physiological profiles of sepak takraw players, *Br. J. Sports Med.*; **2005**, *39*, 825–829. doi: 10. 1136/bjbm.2004.016915.
16. Lephart, S. M.; Abt, J. P.; Ferris, C. M.; Sell, T. C.; Nagai, T.; Myers, J. B.; Irrgang, J. Neuromuscular and biomechanical characteristic changes in high school athletes: a plyometric versus basic resistance program, *Br. J. Sports Med.* **2005**, *39*, 932–938. doi: 17.1136/bjbm.2005.019083.
17. Darr, K. C.; Bassett, D. R.; Morgan, B. J.; Thomas, D. P. Effects of age and training status on heart rate recovery after peak exercise. *Am. J. Physiol.* **1988**, *254*, H340-H343.
18. Engerman, S. ‘The Height of U.S. Slaves’, *Local Population Studies*, **1976**, *16*(1), 45–50.
19. Sunder, M. The Making of Giants in a Welfare State: The Norwegian Experience in the Twentieth Century, *Economics and Biology*, **2004**, 75–86.
20. Woitek, U. Height Cycles in the Eighteenth and Nineteenth Centuries’, *Econ. Hum. Biol.*, **2003**, 243–258.
21. Fogel, R. ‘Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the making of Economic Policy’, *Am. Econ. Rev.*, **1994**, 369–394.

22. Goonetilleke, R. S.; Ho, Cheuk Fan E.; So, R. H. Y. Foot Anthropometry in Hong Kong. **1997**, 81–88. Proceedings of the ASEAN 97 Conference, Kuala Lumpur, Malaysia.
23. Baten, J. “Economic Development and the Distribution of Nutritional Resources in Bavaria, 1797–1839, in *J. Income Distrib.* 9, **2000**, 89–106.
24. Hariri Asli K. Water Hammer Research; Advances in Nonlinear Dynamics Modeling, **2012**, 88–121. Canada, USA: Published by Apple Academic Press, Inc., Exclusive worldwide distribution by CRC Press, a Taylor and Francis Group, Print ISBN: 9781926895314, eBook: 978-1-46-656887-7.
25. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. “Some Aspects of Physical and Numerical Modeling of Water Hammer in Pipelines,” *Nonlinear Dynamics An International Journal of Nonlinear Dynamics and Chaos in Engineering Systems*, ISSN: 1573–269X (electronic version) Journal /1071 Springer, Netherlands, **2009**, ISSN: 0924–090X (Print Version), Heidelberg, Germany, Springer, Volume 60, Number 4 / June, **2010**, 677–701.
26. Heather, E. Collins-Schramm and others, “Markers that Discriminate Between European and African Ancestry Show Limited Variation Within Africa,” *Human Genetics*, **2002**, 111, 566–569.
27. Komlos, J.; Baur, M “From the Tallest to One of the Fattest: The Enigmatic Fate of the Size of the American Population in the Twentieth Century,” *Econ. Hum. Biol.* . **2004**, 2(1), 57–74.
28. Komlos, J.; Kriwy, P. **2003**, “The Biological Standard of Living in the Two Germanies,” *German Econ. Rev.* 4, 493–507.
29. Levy, W. C.; Cerqueira, M. D.; Harp, G. D.; Johannessen, K. A.; Abrass, I. B.; Schwartz, R. S.; Stratton, J. R. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. *Am. J. Cardiol.*, **1998**, 82, 1236–1241.
30. Margo, R. The Heights of American Slaves. *Social Science History*, **1982**, 6, 516–538.
31. Rahmani-Nia, F.; Hojjati, Z. Effect of selected training on body composition and aerobic power of Females College. *J. Harkat*, Iran, **2004**, 1, 18–21.
32. Rahmani-Nia, F.; Rahnama, N.; Hojjati, Z.; And Soltani, B. Acute effects of aerobic and resistance exercise on serum leptin and some risk factors of coronary heart disease in obese females. *Sport sciences for health*, **2008**, 2(3), 118–124.
33. Rahmani-Nia, F.; Hojjati, Z.; Rahnama, N.; Soltani, B. Leptin, Heart Disease and Exercise. *World J. Sport Sci.*, Iran, **2009**, 2(1), 13–20.
34. Rushton, J. P. Race, Brain Size, and Intelligence: A Rejoinder to Cain and Vanderwolf, *Personality and Individual Differences*, **2004**, 11, 785–794.
35. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. A Computational Approach To Study Fluid Movement, *Nanomaterials Yearbook – 2009*, From Nanostructures, Nanomaterials and Nanotechnologies to Nanoindustry, **2010**, 181–196. USA: Nova Science Publications. ISBN: 978-1-60876-451-8.16.
36. Young, J. W. Anthropometric and Mass Distribution Characteristics of the Adult Female FAA Civil Aeromedical Institute FAA, **2008**, 9–83.
37. Bartlett, R. Introduction to Sports Biomechanics, 2nd Edition. Taylor andFrancise-Library, **2007**.

38. Hariri Asli, K.; Haghi, A. K. A Numerical Study on Fluid Flow and Pressure Drop in Microtubes, *J. Balkan Tribological Association*, Tribotechnics and Tribomechanics, Sofia, Bulgaria, **2010**, *16*(3), 382–392.
39. Rahmani-Nia, F.; Hojjati, Z. The effect of exercise training on body composition and aerobic power in sedentary college age females, *Int. J. Fitness*, India, **2005**, *(1)*, 24–31.
40. Unal, M.; Unal, D. D. O.; Baltaci, A. K.; Mgulkoc, R. Investigation of serum leptin levels and $\text{Vo}_{2\text{max}}$ value in trained young male athletes and healthy males. *Acta Physiol. Hungary* **2005**, *92*, 173–179
41. Askari, H.; Tykodi, G.; Liu, J.; Dagogo-Jack S. Fasting plasma leptin level is a surrogate measure of insulin sensitivity. *J. Clin. Endocrinol. Metab.*; **2010**, *95*(8), 3836–3843.
42. Reseland, J. E.; Andessen, S. A.; Solvoll, K.; Hjermann, I.; Urdal, P.; Holme, I.; Drevon, C. A. Effect of long-term change in diet and exercise on plasma leptin concentration. *Clin. Nutr.* **2001**, *73*, 240–245.
43. Mokdad, A. H.; Ford, E. S.; Bowman, B. A.; Dietz, W. H.; Vinicor, F.; Bales, V. S.; Marks, J. S. Prevalence of obesity, *Diabetes* and obesity-related health risk factors, 2001. *JAMA* **2003**, *289*, 76–79.
44. Mendosa-Nunez, V. M.; Garcia-Sanchez, A.; Sanchez-Rodriguez, M.; Galvan-Duart, R. E.; Fonseca-Yerena, E. F. Overweight, waist circumference, age, gender, and insulin resistance as risk factors for Hyperleptinemia. *Obes. Res.* **2002**, *10*, 253–259.
45. Gippini, A.; Mato, A.; Peino, R.; Lage, M.; Dieguez, C.; Casanueva, F. F. Effect of resistance exercise (body building) training on serum leptin levels in young men. Implications for relationship between body mass index and serum leptin. *J. Endocrinol. Invest.* **1999**, *22*, 824–828.
46. Thong FSL, Hudson, R.; Ross, R.; Janssen, I.; Grahnan, T. E. Plasma leptin in moderately obese males: independent effects of weight loss and aerobic exercise. *Am. J. Physiol. Endocrinol. Metabol.* **2000**, *279*, E307-E313.
47. Schulze, P. C.; Kratzsch, J.; Linke, A.; Schoene, N.; Adams, V.; Gielen, S. Elevated serum levels of leptin and leptin and leptin receptor in patients with advanced chronic heart failure. *Eur. J. Heart Fail.* **2003**, *5*, 33–40.
48. Sesso, H. D.; Buring, J. E.; Rifai, N.; Blake, G. J.; Gaziano, J. M.; Ridker, P. M. **2003**, C-reactive protein and the risk of developing hypertension. *J. Am. Med. Assoc.* *200*, 2945–2951.
49. Konstantinides, S.; Schafer, K.; Koschnick, S.; Loskutoff, D. J. Leptin-dependent Platelet aggregation and arterial thrombosis suggests a mechanism for atherothrombotic disease in obesity. *J. Clin. Invest.* **2001**, *108*, 1533–1540.
50. Kyearsiiazis, G. A.; Caplan, J. D.; Lowndes, J.; Carpenter, R. L.; Dennis, K. E.; Sivo, S. A.; Angelopoulos, T. J. Moderate exercise-induced energy expenditure does not alter leptin levels in sedentary obese men, *Clin. J. Sport Med.* **2007**, *17*, 49–51.
51. Mildred, T.; Consuelo, L. Correlates of body image satisfaction among economically depressed urban Filipino women. *Philippine J. Sci.*, **2009**, 67–74.
52. Tamer, L.; Ercan, B.; Unlu, A.; Sucu, N.; Pekdemir, H.; Eskandari, G.; Atik, U. The relationship between leptin and lipids in atherosclerosis. *Indian Heart J.* **2002**, *54*, 692–696.

53. McGill, H. C.; McMahan, A.; Hederick, E. E.; Zieske, A. W.; Malcom, G. T.; Tracy, R. E.; Strong, J. P. Obesity accelerates the progression of coronary atherosclerosis in young men. *Circulation* **2002**, *105*, 2712–2717.
54. Tam, J.; Fukumura, D.; Jain, R. K. A mathematical model of murine metabolic regulation by leptin: energy balance and defence of a stable body weight. *Cell Metab.* **2010**, *7*, 9(1), 52–63.
55. Corsonello, A.; Malara, A.; Ientile, R. **2002**, Leptin enhances adenosine diphosphate-induced platelet aggregation in healthy subjects. *Obes. Res.* *10*, 306–317.
56. Maughan, R. J.; Leiper, J. B.; Vist, G. E. Gastric emptying and fluid availability after ingestion of glucose and soy protein hydrolysate solutions in man. *Exp physiol*, **2004**, *89*(1), 101–108.
57. Shamsuzzaman, A. S. M.; Winnicki, M.; Wolk, R.; Svatikova, A. A.; Phillips, B. G.; Davison, D. E.; Berger, P. B.; Somers, V. K. Independent association between plasma leptin and C-reactive protein in healthy humans. *Circulation*, **2004**, *109*, 2181–2185.
58. Mc Connell, G.; Snow, R. J.; Proietto, J.; Hargreaves M. Muscle metabolism during prolonged exercise in humans: influence of carbohydrate availability. *J. Appl. Physiol.* **1999**, *87*, 1083–1086.
59. Klein, S.; Horowitz, J. F.; Landt, M.; Goodrick, S. J.; Mohamed-Ali, V.; Coppack, S. W. Leptin production during early starvation in lean and obese women. *Am. J. Endocrinol. Metabol.* **2000**, *278*, E280–E284.
60. Knudson, J. D.; Incer, U. D.; Dick, G. M.; Shibata, H.; Akahane, R.; Saito, M.; Tune, J. D. Leptin resistance extends to the coronary vasculature in prediabetic dogs and provides protective adaptation against endothelial dysfunction. *Am. J. Physiol. Heart Circulation Physiol.* **2005**, *289*, H1038–H1046.
61. Maughan R. J.; Murray R. Sports Drinks; Basic Science and Practical Aspects CRS Press, LIC, Boca Ranton, FL, USA, 2001.
62. Bosch A. N.; Dennis S. C.; Noakes T. D. influence of carbohydrate ingestion of faecal substrate turnover and oxidation during prolonged exercise *J. Appl. Physiol.* **1994**, *76*, 2364–2372.
63. Hamed-Nia, M.; Rezaei S. Some risk factors associated with physical activity and body fat percentage cardio-vascular university faculty of Medical sciences and Health Services Blog, The Eleventh: **2007**, 34–40. (In Persian with English abstract).
64. Reseland, J. E.; Andessen, S. A.; Solvoll, K.; Hjermann, I.; Urdal, P.; Holme, I.; Drevon, C. A. Effect of long-term change in diet and exercise on plasma leptin concentration. *Clin. Nutr.* **2001**, *73*, 240–245.
65. Hamilton M. T.; Gonzalez -Alonso J.; Montoin S. J.; Cotle, E. F. Fluid replacement and glucose during exercise prevent cardiovascular drift *J. Appl. Physiol.* **1991**, *71*, 871–877.
66. Livshits, G.; Patsulaia, I.; Gerber, L. M. Association of leptin levels with obesity and blood pressure: possible common genetic variation. *Int. J. Obes.* **2005**, *29*, 85–92.
67. Leal-Cerro, A.; Garcia-Luna, P. P.; Astorga R. Serum leptin levels in male marathon athletes before and after the marathon run. *J. Clin. Endocrinol. Metabol.* **1998**, *83*, 2376–2379.
68. Hartman, J. W.; et al., Consumption of fat – free fluid milk after resistance exercise promotes greater lean mass accretion than does consumption of soy or carbohydrate in young, novice. Male weightlifters. *Am. Clin. Nutr.*, **2007**, *86*(2), 313–381.

69. Iolo, B. G.; Williams, B. D.; Fleming, R. Y.; Wolfe, R. R. Insulin action on muscle protein kinetics and amino acid transport during recovery after resistance exercise. *Diabetes* **1999**, *48*, 949–957.
70. Pritvhet, K. L.; Bishop, P. A.; Pritchett, R. C.; Green, J. M.; Katica C. Acute effects of chocolate milk and a commercial recovery beverage on post-exercise muscle damage and cycling performance. *Appl. Phys. Nutr. Metab.* **2009**, *34*(6), 1017–1022.
71. Kanaley, J. A.; Fenicchia, L. M.; Miller, C. S.; Ploutz Synder, L. L.; Weinstock, R. S.; Carhart, R.; Azevedo, J. L. Resting leptin responses to acute and chronic resistance training in type 2 diabetic males and females. *Int. J. Obes.* **2001**, *25*, 1474–1480.
72. Wallace, A. M.; McMahon, A. D.; Pakard, C. J.; Mibiol, A. K.; Shephard, J.; Gaw, A.; Sattar, N. Plasma leptin and the risk of cardiovascular disease in the west of Scotland coronary prevention study. *Circulation* **2001**, *104*, 3052–3060.
73. Paquot, N.; Tappy, L. Adipocytokines: link between obesity, type 2 Diabetes and atherosclerosis. *Rev. Med. Liege.* **2005**, *60*, 369–373.
74. Ishii, T.; Yamakita, T.; Yamagami, K.; Yamamoto, T.; Miyamoto, M.; Kawasaki, K.; Hosoi, M.; Yashioka, K.; Sato, T.; Tanaka, S.; Fujii, S. Effects of exercise training on serum leptin levels in type 2 diabetic patients. *Metabolism*, **2001**, *50*, 1136–1140.
75. Weltman, A.; Pritzlaff, C. J.; Wideman, L.; Considine, V.; Fryburg, A.; Gutgesell, M. E.; Hartman, M. L.; Veldhuis, J. D. Intensity of acute exercise does not affect serum leptin concentrations in young males. *Med. Sci. Sports Exer.* **2000**, *32*, 1556–1561.
76. Kraemer, K. K.; Chu, H.; Castracane, V. D. Leptin and exercise. *Exp. Biol. Med.* **2002**, *227*, 701–708.
77. Zolanz, J. A.; Konturek, S. J.; Duda, K.; Majerczak, J.; Sliwowski, Z.; Grandys, M.; Bielanski, W. Effect of moderate incremental exercise, performed in fed and fasted state on cardiorespiratory variables and leptin and ghrelin concentration in young healthy men. *J. Physiol. Pharmacol.* **2005**, *56*, 63–85.
78. Kraemer, R. R.; Acevedo, A. O.; Synovitz, L. B.; Herbert, E. P.; Gimpel, T.; Castracane, V. D. Leptin and steroid hormone responses to exercise in adolescent females runners over a 7 week season. *Eur. J. Appl. Physiol.* **2001**, *86*, 85–91.
79. Olive, J. L.; Miller, G. D. Differential effects of maximal- and moderate-intensity run on plasma leptin in healthy trained subjects. *Nutrition* **2001**, *17*, 365–369.
80. Zhang, Y.; Proenca, R.; Maffei, M.; Barone, M.; Leopold, M.; Friedman, J. M. Positional cloning of the mouse obese gene and its human homologue. *Nature* **1994**, *373*, 425–432.
81. Barbato, K. B.; Martins Rde, C.; Rodrigues Mde, L.; Braga, J. U.; Francischetti, E. A.; Genelhu, V. Effects of greater than 5% weight reduction on hemodynamic, metabolic and neuroendocrine profiles of grade 1 obese subjects. *Cardiology*, **2006**, *87*, 12–21.
82. Wilkinson, S. B.; et al., Consumption of fluid skin milk promotes greater muscle protein accretion after resistance exercise than does consumption of an isonitrogenous and isoenergetic soy – protein beverage. *Am. J. Clin. Nutr.* **2007**, *85*(4), 1031–1040.
83. Bouassida, A.; Zalleg, D.; Bouassida, S.; Zaouali, M.; Feki, Y.; Zbidi, A.; Tabka, Z. Leptin, its implication in physical exercise and training: a short review. *J. Sport Sci. Med.* **2006**, *5*, 172–181.
84. Hynes, W. G.; Sivitiz, W. I.; Morgan, D. A.; Walsh, S. A.; Mark, A. L. **1997**, Sympathetic and cardiorenal actions of leptin. *Hypertension* *30*, 619–623.

85. Bouassida, A.; Zalleg, D.; Bouassida, S.; Zaouali, M.; Feki, Y.; Zbidi, A.; Tabka, Z. **2006**, Leptin, its implication in physical exercise and training: a short review. *J. Sport Sci. Med.* **5**, 172–181.
86. Zafeiridis, A.; Smilios, I.; Conisidine, V.; Tokmakidis, S. P. Serum leptin responses after acute resistance exercise protocols. *J. Appl. Physiol.* **2003**, *94*, 591–597.
87. Elloit, T. A.; et al, Milk ingestion stimulates net muscle protein synthesis following resistance exercise. *Med. Sci. Sport Exerc.*, **2006**, *38*(4), 667–74.
88. Okazaki, T.; Himeno, E.; Manri, H.; Ogata, H.; Ikeda, M. Effects of mild aerobic exercise and mild Hypocaloric diet on plasma leptin in sedentary females. *Clin. Exp. Pharmacol. Physiol.* **1999**, *26*, 415–420.
89. Krauss, R. M.; Winston, M.; Flecher, B. J.; Grundy, S. M. Obesity: impact on cardiovascular disease. *Circulation* **1998**, *98*, 10–16.
90. Singhal, A.; Farooqi, I. S.; Cole, T. J.; O’Rahilly, S.; Fewtrell, M.; Kattenhorn, M.; Lucas, A. D.; Eanfield, J. Influence of leptin on arterial distensibility: a novel link between obesity and cardiovascular disease? *Circulatin* **2002**, *106*, 1919–1926.
91. Essig, D. A.; Alderson, N. L.; Ferguson, M. A.; Bartolli, W. P.; Durstine, J. L. Delayed effects of exercise on the plasma leptin concentration. *Metabolism* **2000**, *49*, 395–399.
92. Enas, E. A. Coronary artery disease epidemic in Indians: a cause for alarm and far action. *J. Indian Med. Assoc.*, **2000**, *98*, 694–702.
93. Hall, J. E.; Kuo, J. J. da Silva, A. A. de Paula, R. B.; Liu, J.; Tallam, L. Obesity -associated hypertension and kidney disease. *Curr. Opin. Nephrol. Hypertens.* **2003**, *12*, 195–299.
94. Hilton, L. K.; Loucks, A. B. Low energy availability, not exercise stress, suppress the durenal rhythm of leptin in healthy young women. *Am. J. Physiol. Endocrinol. Metabol.* **2000**, *278*, E43–E49.
95. Gharakhanloo, R.; Gaeyni, A.; Peyghoon, A. Standardization waist-to-hip ratio for men over 40years to the city of Ahvaz and its association with cardiovascular risk factor-*Diabetes* and cardiovascular desease. *Olympic J. Iran*, **2002**, *3–4*, 59–73. (In Persian with English abstract).
96. Gaeeni, A.; Lameyi, T. The relationship between percent body fat (BF) and Body mass index (BMI), and the ratio of waist to hip circumference (WHR) women over fifteen years in Tehran, Harkat J., **2003**, *17*, 95–105. (In Persian with English abstract).
97. Gupta, R. P.; Rastogi, et al., Body mass index, waist-size, waist-hip ratio and cardiovascular Risk factors in urban subjects. *JAPI*, **2007**, *55*, 621–627.
98. Houmard, J. A.; Cox, J. H.; Maclean, P. S.; Barakat, H. A. Effect of short-term exercise training on leptin and insulin action. *Metabolism* **2000**, *49*, 858–861.
99. Lee, J. K.; et al., Effects of milk ingestion on prolonged exercise capacity in young, healthy men, *Nutrition*, **2008**, *24*(4), 340–347.
100. Landt, M.; Lawson, G. M.; Helgeson, J. M.; Davila-Roman, V. G.; Ladenson, J. H.; Jaffe, A. S.; Hickner, R. C. Prolonged exercise decreases serum leptin concentrations. *Metabolism* **1997**, *46*, 1109–1112.

CHAPTER 2

STATIC AND DYNAMIC MODELING FOR ANTHROPOMETRY

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2.1 INTRODUCTION

The division of the body weight into various components can well be conceived by considering the major parts of the body, i.e., fat mass, muscle mass, and bone mass. Knowing and understanding the effect of training and competition on body composition can help athletes control weight and alter body composition safely. Following body composition trends in specific sports enable coaches and athletes to accurately prepare athletes for specific events/positions. A multitude of factors effects physical performance [1–9].

Historical anthropometrics is the study of patterns in human body size and their correlates over time. While social researchers, public health specialists and physical anthropologists have long utilized anthropometric measures as indicators of well-being, only within the past three decades have historians begun to use such data extensively. Adult stature is a cumulative indicator of net nutritional status over the growth year, and thus reflects command over food and access to healthful surroundings. Since expenditures for these items comprised such a high percentage of family income for historical communities, mean stature can be used to examine changes in a population's economic circumstances over time and to compare the well-being of different groups with similar genetic height potential. Anthropometric measures are available for portions of many national populations as far back as the early 1700s. While these data often serve as complements to standard economic indicators, in some cases they provide the only means of assessing historical economic well-being, as “conventional” measures such as per capita GDP, wage and price indices, and income inequality measures have been notoriously spotty and problematic to develop. Anthropometric-based research findings to date have contributed to the scholarly debates over mortality trends, the nature of slavery, and the outcomes of industrialization and economic development. Height has been the primary indicator utilized to date. Other indicators include height-standardized weight indices, birth weight, and age at menarche. Potentially even more important, historical anthropometrics broadens the understanding of “well-being” beyond the one-dimensional “ruler” of income, providing another lens through which the quality of historical life can be viewed [10–16].

Athletes in this context have to face many adverse environmental conditions, the most important being hypoxia with its incidence on physical performance. To counter these problems, the body generates immediate and complex compensations in respiration, metabolism, and hemodynamic regulations for the redistribution of blood flow to vital organs and maintenance of systemic oxygenation, which is critical for survival.

2.2 MATERIALS AND METHODS

Present work shows a physical modeling for anthropometric of athletes. According to this technique, an improved modeling based on measurements was classified and compared with a special type of weights of sportswomen with other anthropometric classes of sportswomen. On the other hand present work compares the anthropometric data for blood pressure of runner related to Runner Velocity (Running Time) and runner weight in two cases.

During dynamic exercise, it is generally assumed that heart rate increases due to both a parasympathetic withdrawal and an augmented sympathetic activity. However, because some authors disagree with the former statement and the fact that during exercise there is also a technical problem related to the non-stationary signals, a critical look at interpretation of results is needed. It is strongly suggested that, when presenting reports on HRV studies related to exercise physiology in general or concerned with athletes, a detailed description should be provided on analysis methods, as well as concerning population, and training schedule, intensity and duration. Most studies concern relatively small numbers of study participants, diminishing the power of statistics. Therefore, multicenter studies would be preferable.

In order to further develop this fascinating research field, we advocate prospective, randomized, controlled, long-term studies using validated measurement methods. Finally, there is a strong need for basic research on the nature of the control and regulating mechanism exerted by the autonomic nervous system on cardiovascular function in athletes,

preferably with a multidisciplinary approach between cardiologists, exercise physiologists, pulmonary physiologists, coaches and biomedical engineers.

Consequently, the results of this work will help to reduce the risk of sportswomen activities. In this work, dateline for field tests data collection was at 12:00 a.m., 10/06/2012 until 30/06/2012. Locaton of work field tests model was at Rasht city in the north of Iran. Following are considerations was made when using and applying anthropometric data for blood pressure of runner related to Runner Velocity (Running Time) and Runner Stature. Blood pressure of runner was measured in two cases: first at the running state and second at the before of running. Twenty healthy and young sprint runner females (Table 2) participate in this study. They signed a written/informed consent. All subjects completed a medical questionnaire to ensure that they were not taking any medication, and were free cardiac, respiratory, renal or metabolic diseases [17–21].

They didn't have any orthopaedic injury that would inhibit physical activity. The subjects were tested in the follicular phase of the menstrual cycle based on their previous menses.

All participants consumed their last meal at least 2 h before the test and refrained from intensive exercise in the 24 h period preceding testing. All anthropometric measures were recorded in the morning by the same experienced anthropometrist. Stretch stature was measured using a wall-mounted stadiometer (± 0.1 cm). The exercise systolic and diastolic blood pressures were measured on left arm of each participant using cuffs of appropriate size (Table 1).

The measurements were recorded just after 100m intense running in standing position. In present work, for professional runner at fast running, blood pressure of runner, Runner Velocity and weight were recorded. Curve estimation procedure has used these data which were detected field tests (Tables 1, and 2). These data have been compared by blood pressure of runner and Runner Velocity and weight data, which were collected from actual systems (field tests). The model was calibrated using one set of data, without changing parameter values. It was used to match a different set of results [22–27].

TABLE 1 Field tests and regression anthropometric model for blood pressure.

No.	Running Time (sec)	Runner Stature (cm)	Dynamic Blood pressure (mm-Hg)	Static Blood Pressure (mm-Hg)
1	13.45	165	111	108
2	13.66	159	120	96
3	15.03	168	114	94
4	13.67	158	100	96
5	14.3	164	118	90
6	14.73	166	109	96
7	17.1	161	126	117
8	17.55	161	142	102
9	16.28	159	120	97
10	16.03	167	134	112
11	16.27	168	148	111
12	16.77	64	134	125
13	17.35	154	139	102
14	15.22	170	120	96
15	14.98	174	110	106
16	14.9	151	116	103
17	14.51	170	126	120
18	13.98	158	113	96
19	14.45	165	113	102
20	16.98	166	119	86

TABLE 2 Physical modeling for anthropometric of sportswomen.

No.	Age	Height (cm)	Weight (kg)	BMI	Hip Circumference	Waist Circumference	WHR
1	19	165	50	18.38	90	64	0.71
2	16	159	52	20.63	94	71	0.75
3	18	168	47	16066	88	65	0.73
4	16	158	50	20.08	91	68	0.74
5	14	164	52	19.44	92	66	0.71
6	14	166	44	16	82	63	0.76
7	14	161	54	20.84	93	66	0.7
8	20	161	46	17.76	88	65	0.73
9	21	159	48	19.04	92	69	0.75
10	14	167	52	18.7	92	70	0.76
11	18	168	56	19.85	94	70	0.74
12	15	64	55	20.52	98	67	0.68
13	23	154	45	18.98	88	68	0.77
14	22	170	56	19.37	90	64	0.71
15	21	174	59	19.53	95	69	0.72
16	21	151	55	24.12	98	73	0.74
17	22	170	62	21.45	97	71	0.73
18	28	158	55	22.08	95	70	0.73
19	22	165	56	20.58	92	66	0.71
20	24	166	62	22.54	97	78	0.8

Regression model (Tables 3–5) has been built based on field tests data.

TABLE 3 Model Summary and regression anthropometric model for blood pressure.

Model	R	R-square	Adjusted R-square	Std. Error of the Estimate
1	.654(a)	.428	.361	10.41122

^a Predictors: (Constant), Running Time (sec), Runner weight (kg).

^b Dependent Variable: Blood Pressure (mm-Hg).

TABLE 4 Residuals Statistics for blood pressure.

	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	108.5796	135.1232	120.6000	8.52276	20
Residual	−17.70139	21.81707	.00000	9.84802	20
Std. Predicted Value	−1.410	1.704	.000	1.000	20
Std. Residual	−1.700	2.096	.000	.946	20

^a Dependent Variable: Blood Pressure (mm-Hg).

TABLE 5 Autocorrelations for blood pressure.

Series: Running Time (sec)					
Lag	Autocorrelation	Std. Error(a)	Box-Ljung Statistic		
			Value	Df	Sig.(b)
1	.004	.212	.000	1	.987
2	−.152	.206	.545	2	.762
3	−.214	.200	1.687	3	.640
4	.124	.194	2.098	4	.718
5	.178	.187	3.004	5	.699
6	−.227	.181	4.580	6	.599
7	−.164	.173	5.478	7	.602
8	−.002	.166	5.478	8	.705
9	−.050	.158	5.578	9	.781

TABLE 5 (Continued)

Lag	Autocorrelation	Std. Error(a)	Box-Ljung Statistic		
			Value	Df	Sig.(b)
10	.042	.150	5.656	10	.843
11	−.308	.142	10.385	11	.496
12	.019	.132	10.406	12	.580
13	.226	.123	13.806	13	.388
14	.055	.112	14.047	14	.446
15	−.016	.100	14.074	15	.520
16	−.150	.087	17.053	16	.382

^a The underlying process assumed is independence (white noise).

^b Based on the asymptotic chi-square approximation.

Scatter diagram, correlation (Figs. 1–3) and autocorrelations procedure (Fig. 4) for blood pressure was formed by estimating regression statistics and producing related plots (Figs. 5–7) for field tests model (Tables 1–2) with the following three assumptions Eqs. (1)–(3):

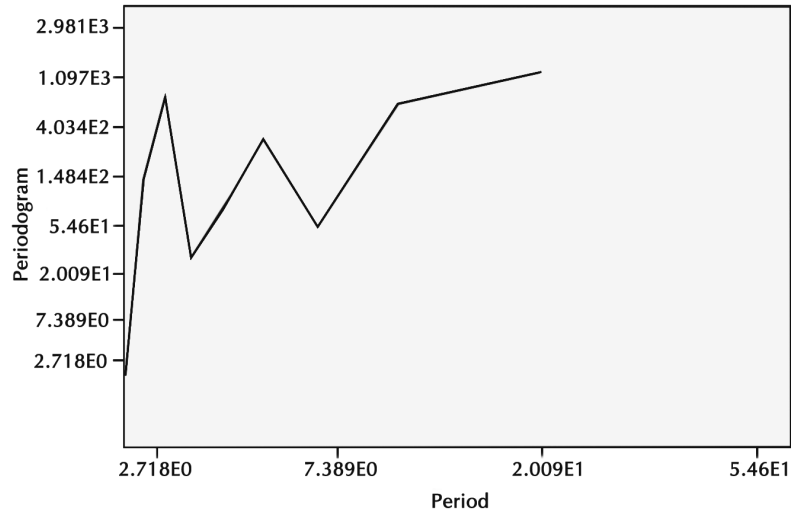


FIGURE 1 Periodogram for dynamic blood pressure against Running Time.

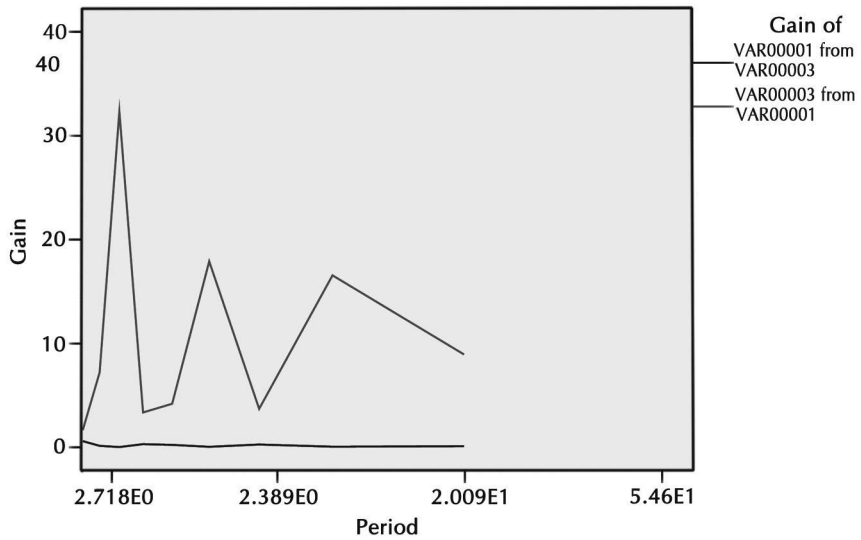


FIGURE 2 Gain of weight against Running Time.

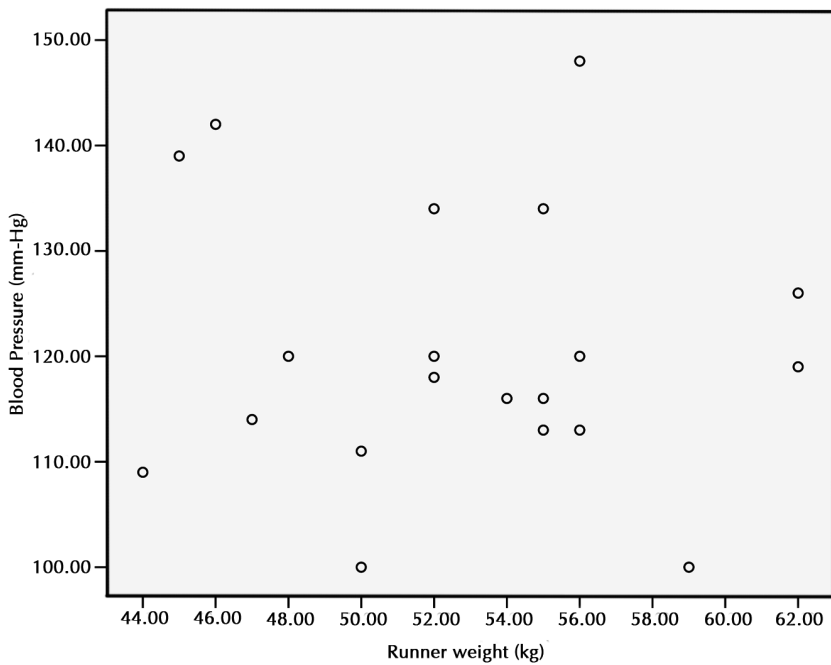


FIGURE 3 Scatter diagram for runner dynamic blood pressure against runner weight.

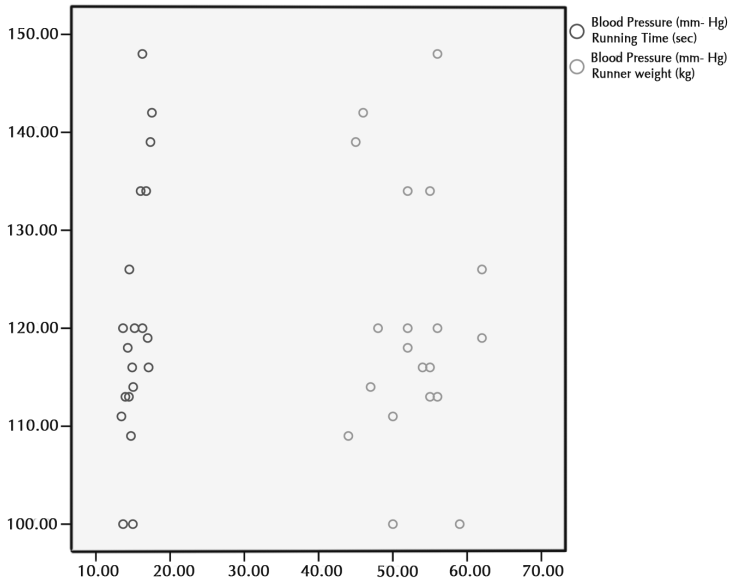


FIGURE 4 Runner dynamic blood pressure against runner weight.

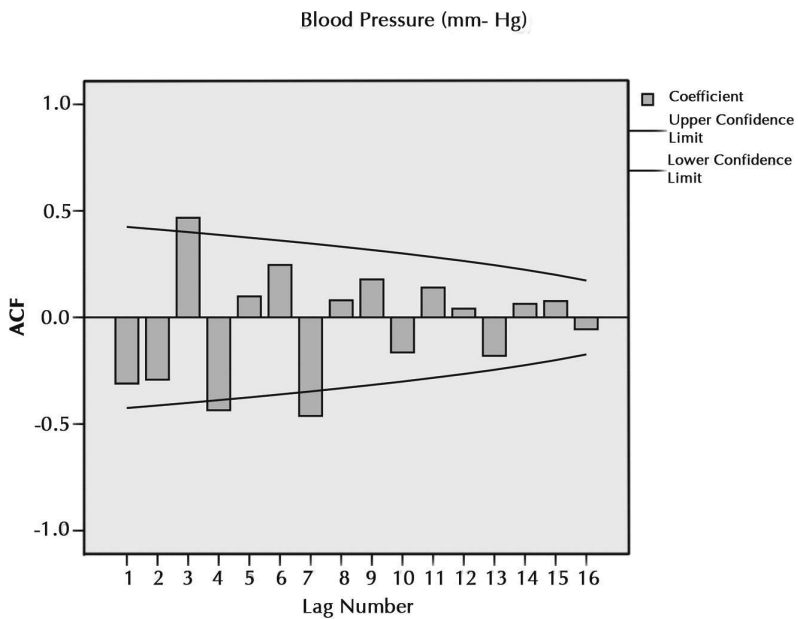


FIGURE 5 Runner dynamic blood pressure against Running Time.

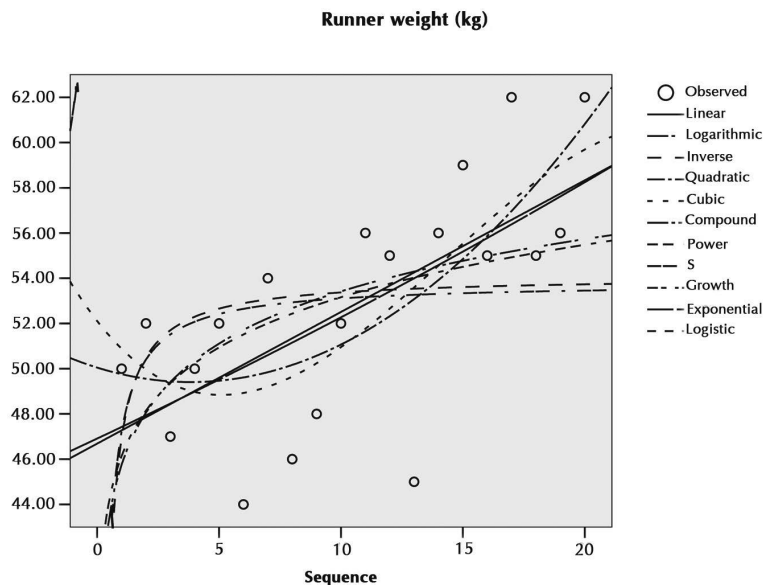


FIGURE 6 Curve fit for runner dynamic blood pressure.

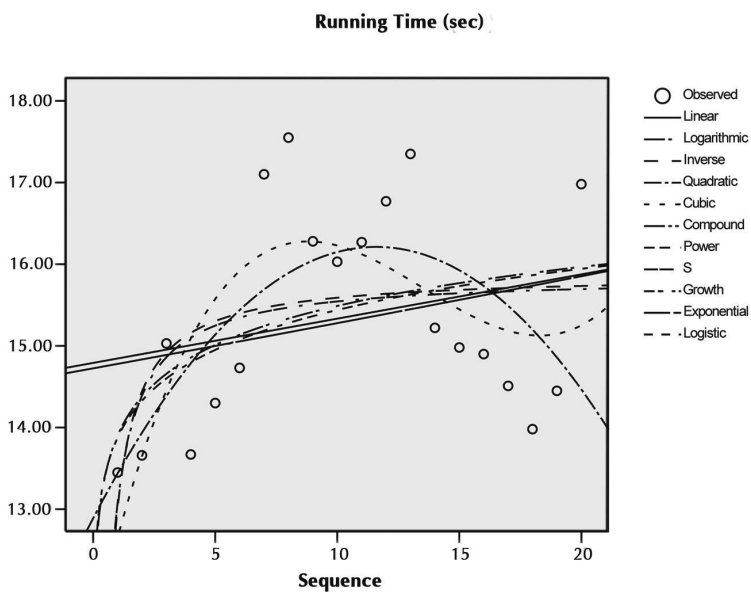


FIGURE 7 Regression and curve fit for runner static blood pressure against Running Time.

Assumption (1): $p = f(V)$, (1)

Assumption (2): $p = f(W)$, (2)

Assumption (3): $p = f(V, W)$, (3)

V – Runner Velocity is the most important variable,

W – runner weight.

Dependent variable: P – Blood pressure (mm-Hg) for starting point of running condition.

The independent variable is Running Time (sec) and runner weight (kg).

2.3 RESULTS AND DISCUSSION

In this work conclusions were drawn on the basis of experiments and calculations for the three assumptions:

Assumption (1):

$$p = f(V), \quad (1)$$

The most important effects that were observed based on regression for model summary (Tables 3, and 4) are as follows (Figs. 1, 2, 5, 7, 8, and 9).

2.3.1 REGRESSION MODEL DUE TO FIELD TEST FOR ANTHROPOMETRIC MODEL OF BLOOD PRESSURE

The auto-regression procedure (Table 5) accounts for first-order auto-correlated residuals. It provides reliable estimates of both goodness of-fit measures and significant levels of chosen predictor variables.

Present work compared the anthropometric data for blood pressure of runner related to Runner Velocity (Running Time) and Runner Stature in two cases.

The dependent and independent variables of runner were measured in the following cases:

First: at the running state (dynamic) (Fig. 6)

Second: at the before of running (static) (Figs. 7–8)

Dependent variable:

P – blood pressure with nomenclature “ Y ,” independent variable with nomenclature “ X ” such as:

V – Runner Velocity is the most important variable, S – runner weight. The independent variable is velocity (m.s^{-1}) and runner weight (kg).

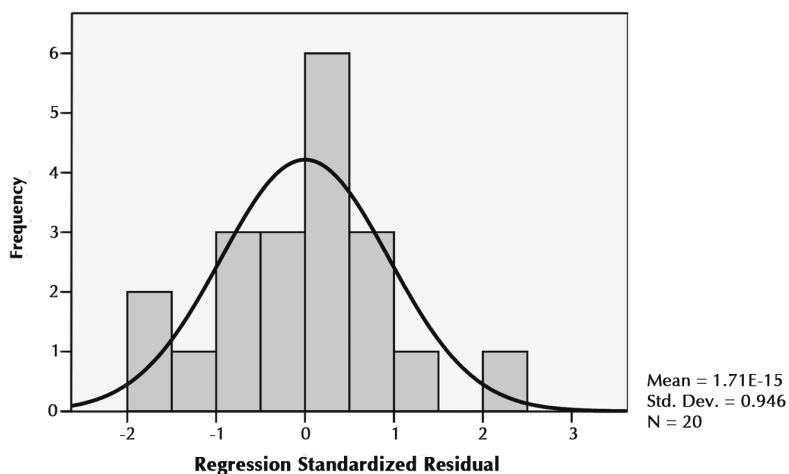


FIGURE 8 Curve fit for histogram of runner static blood pressure.

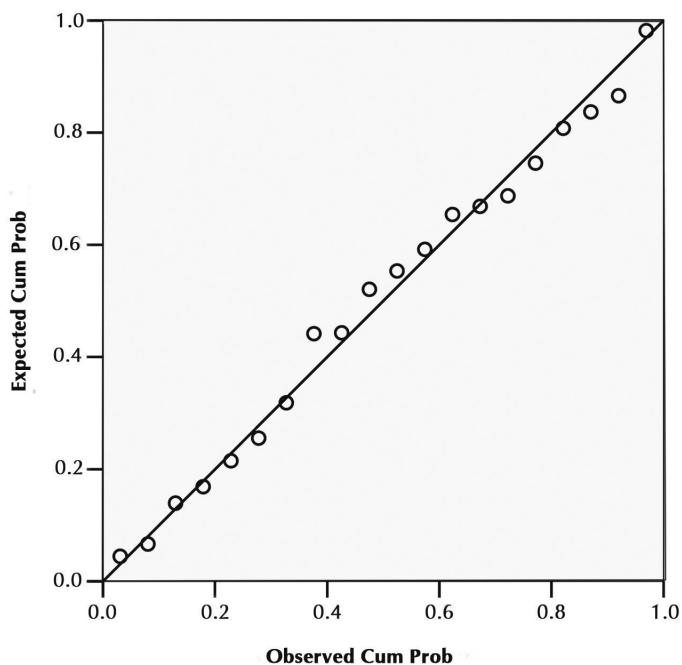


FIGURE 9 Normal P-P plot of regression standardized residual.

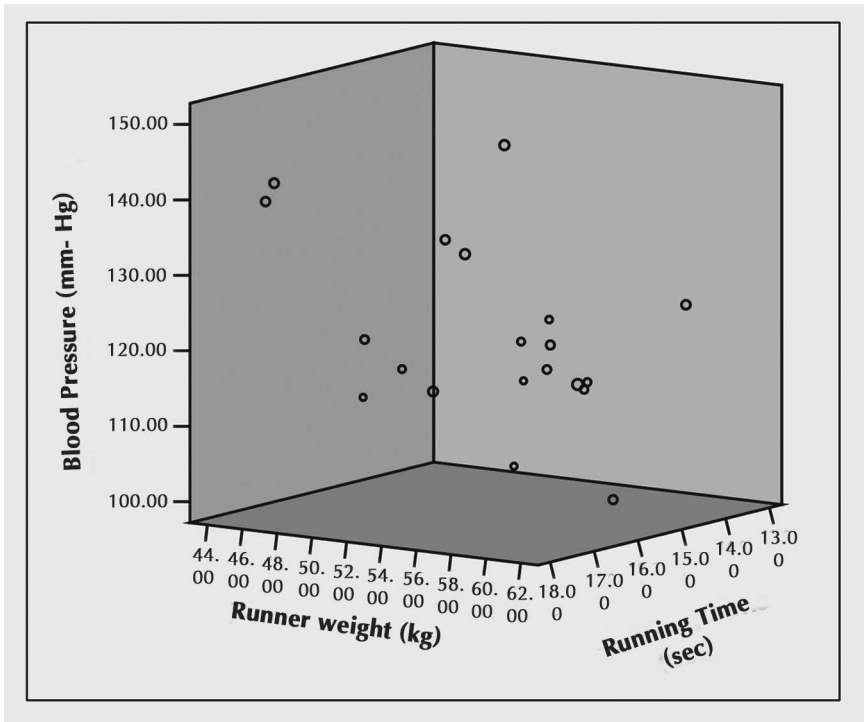


FIGURE 10 D Scatter diagram for dynamic blood pressure against runner weight and Running Time.

Assumption (2):

$$p = f(W), \quad (2)$$

For second assumption the curve estimation procedure (Figs. 3, 4, 6, and 8) allows quick estimating regression statistics, and producing related plots for different models. Hence the auto-regression procedure by regression software “SPSS 10.0.5” was selected for the curve estimation procedure in the present work. The regression model has been built based on field test data. Regression software “SPSS” has fitted the function curve and provided regression analysis. So, the regression model was found in the final procedure. This model has compared field test results using Lab model results [28–34].

Assumption (3):

$$p = f(V, W), \quad (3)$$

For third assumption the curve estimation procedure was illustrated in (Fig. 10). Besides, the runner blood pressure was formed by estimating regression statistics are listed in Table 6. Although related plots for the field test model were produced. Regression software “SPSS 10.0.5” performs multidimensional scaling of proximity data to find least-squares representation of the objects in a low-dimensional space [35–101].

TABLE 6 Regression residuals statistics.

	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	108.5796	135.1232	120.6000	8.52276	20
Residual	−17.70139	21.81707	.00000	9.84802	20
Std. Predicted Value	−1.410	1.704	.000	1.000	20
Std. Residual	−1.700	2.096	.000	.946	20

^a Dependent Variable: Blood Pressure (mm-Hg).

2.3.2 COMPARISON WITH OTHER WORKS

Rahmani-Nia, and Hodjati obtained the effect of exercise training on body composition and aerobic power in sedentary college age females. Comparison showed similarity in present work and the results of that works [102].

2.4 CONCLUSIONS

Present work showed that “regression model” is accurate model for modeling for anthropometric of body motion. Hence in order to presentation for importance of Runner Velocity on blood pressure phenomenon, it was compared the models for laboratory; computational and field tests experiments. As long as these procedures, it was showed that the regression based model for anthropometric of body motion at the running state (dynamic)

and before of running (static). On the other hand, this idea were included the proper analysis to provide a dynamic response to the shortcomings of the body motion. It also performed the high technology and high-speed detectors equipment's to determine the operational procedures to avoid hazards on human health; economics and human biology. Consequently, the results of this work will help to reduce the risk of sport women activities.

NOMENCLATURES

- P Runner Blood Pressure (mm-Hg)
 t Running Time (sec)
 V Velocity (m/s)
 S Runner Stature (cm)

KEYWORDS

- anthropometric
- body motion
- computational model
- physical modeling
- regression

REFERENCES

1. Hurst Thomas, D. Skull Wars Kennewick Man, Archaeology and the Battle for Native American Identity, Washington University: School of Law – Anheuser-Busch Hall, **2001**, 38–41.
2. Nabieh, A.; Mohamed, I. Anthropometric Measurements as a Significant for Choosing Juniors in Both Volleyball and Handball Sports (Factorial Analysis Study), *World J. Sport Sci.* **2010**, *3*(4), 277–289.
3. Rogana, S.; Hilfiker, R.; Clarys, P.; Clijsen, R.; Taeymans, J. Position-specific and Team-ranking-related Morphological Characteristics in German Amateur Soccer Players – a Descriptive Study, *Int. J. Appl. Sports Sci.*, **2011**, *23*(1), 168–182.

4. Luigi P. W.; Bercades, T. somatotypes of national elite combative sport athletes, *Braz. J. Biomotricity*, **2009**, 3(1), 21–30.
5. Dacres-Mannings, S.; Rochester, S.; Frail, H. Anthropometric profiles of Australian Rugby Institute, club and state level Rugby Union players. **2010**.
6. Gaurav, V.; Singh, M.; Singh, S. Anthropometric characteristics, somatotyping and body composition of volleyball and basketball players, *J. Phys. Educ. Sports Manage.* **2010**, 1(3), 28–32, Available online <http://www.acadjourn.org/jpessm>
7. Beat K.; Kohler, G. Influence of anthropometry on race performance in ultra-endurance triathletes in the longest triathlon in North America, *Int. SportMed J.*, **2007**, 8(2), 87–96, <http://www.ismj.com>
8. Joseph, J.; Greene, M. S.; Timothy, A.; McGuine, A. T. C.; Levenson, G.; Thomas M. Anthropometric and Performance Measures for High School Basketball Players, *J. Athl. Training*, **1998**, 33(3), 229–232
9. Amatya, D. L. Comparative Study of Somatotype of Nepalese Sportsmen, National Association for Sports Health and Fitness. **1999**.
10. Sang Hong, K. Kinanthropometric Study of Korean and Nepalese Marathon Runners, keimyung university Diwakar Lal Amatya, MSC, Athletics and Sports Science, India, Sports Kinanthropometrist, Australia, Sports Expert, National Sports Council, January. **2008**.
11. Gaunt, B. W. Anthropometric and Demographic Factors Affecting Distance Hopped-aid Limb Symmetry Index for the Crossover Hop for distance test in High School athletes, *J. Orthop. Sport Phys.*, **2001**, 31(3), 145–151
12. Gross, M.; Dailey, E. S.; Melissa, D.; Dalton, A.; lee, K.; Wendy, I.; Ashley, C. Relationship Between Lifting Capacity and Anthropometric Measures, *J. Orthop. Sport Phys.*, **2000**, 30(5), 237–247, 258–262.
13. Roberto, C, Jose, A.; Perez, J.; Cortell, m. Juan, J.; Rivas, J. Correlations among anthropometric parameters, jump power, and position in professional basketball players, **2008**.
14. Pui, W. K.; Hendrik, H. Anthropometric, gait and strength characteristics of Kenyan distance runners, *J. Sports Sci. Med.* **2008**, 7, 499–504, <http://www.jssm.org>
15. Jawis, M. N.; Singh, R.; Singh, H. J.; Yassin, M. N. Anthropometric and physiological profiles of sepak takraw players, *BRJ. Sports Med.* **2005**, 39, 825–829. doi: 10.1136/bjism.2004.016915.
16. Lephart, S. M.; Abt, J. P.; Ferris, C. M.; Sell, T. C.; Nagai, T.; Myers, J. B.; Irrgang, J. Neuromuscular and biomechanical characteristic changes in high school athletes: a plyometric versus basic resistance program, *Br. J. Sports Med.* **2005**, 39, 932–938. doi: 17.1136/bjism.2005.019083.
17. Darr, K. C.; Bassett, D. R.; Morgan, B. J.; Thomas, D. P. Effects of age and training status on heart rate recovery after peak exercise. *Am. J. Physiol.* **1988**, 254, H340–H343.
18. Engerman, S. ‘The Height of U. S. Slaves’, *Local Population Studies*, **1976**, 16(1), 45–50.
19. Sunder, M. The Making of Giants in a Welfare State: The Norwegian Experience in the Twentieth Century’, *Economics and Biology*, **2004**, 75–86.

20. Woitek, U. Height Cycles in the Eighteenth and Nineteenth Centuries', *Econ. Hum. Biol.*, **2003**, 243–258.
21. Fogel, R. 'Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the making of Economic Policy', *Am. Econ. Rev.*, **1994**, 369–394.
22. Goonetilleke, R. S.; Ho, Cheuk Fan E.; So, R. H. Y. Foot Anthropometry in Hong Kong. (pp. 81–88) Proceedings of the ASEAN 97 Conference, Kuala Lumpur, Malaysia. **1997**.
23. Baten, J. "Economic Development and the Distribution of Nutritional Resources in Bavaria, **2000**, 1797–1839, *J. Income Distrib.* 9, 89–106.
24. Hariri Asli K. Water Hammer Research; Advances in Nonlinear Dynamics Modeling, (pp. 88–121). Canada, USA: Published by Apple Academic Press, Inc., Exclusive worldwide distribution by CRC Press, a Taylor and Francis Group, **2012**, Print ISBN: 9781926895314, eBook: 978-1-46-656887-7.
25. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. "Some Aspects of Physical and Numerical Modeling of Water Hammer in Pipelines," *Nonlinear Dynamics An International Journal of Nonlinear Dynamics and Chaos in Engineering Systems*, ISSN: 1573–269X (electronic version) Journal /1071 Springer, Netherlands, **2009**, ISSN: 0924–090X (print version), Heidelberg, Germany, Springer, Volume 60, Number 4 / June, **2010**, 677–701.
26. Heather, E. Collins-Schramm and others, "Markers that Discriminate Between European and African Ancestry Show Limited Variation Within Africa," *Human Genetics* **2002**, 111, 566–9.
27. Komlos, J.; Baur, M. "From the Tallest to One of the Fattest: The Enigmatic Fate of the Size of the American Population in the Twentieth Century," *Econ. Hum. Biol.* **2004**, 2(1), 57–74.
28. Komlos, J.; Kriwy, P. "The Biological Standard of Living in the Two Germanies," *German Econ. Rev.* **2003**, 4, 493–507.
29. Levy, W. C.; Cerqueira, M. D.; Harp, G. D.; Johannessen, K. A.; Abrass, I. B.; Schwartz, R. S.; Stratton, J. R. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. *Am. J. Cardiol.*, **1998**, 82, 1236–1241.
30. Margo, R. The Heights of American Slaves. *Social Science History*, **1982**, 6, 516–538.
31. Rahmani-Nia, F.; Hojjati, Z. Effect of selected training on body composition and aerobic power of Females College. *J. Harkat*, Iran, VI, **2004**, 18–21.
32. Rahmani-Nia, F.; Rahnama, N.; Hojjati, Z.; And Soltani, B. Acute effects of aerobic and resistance exercise on serum leptin and some risk factors of coronary heart disease in obese females. *Sport sciences for health*, **2008**, 2(3), 118–124.
33. Rahmani-Nia, F.; Hojjati, Z.; Rahnama, N.; Soltani, B." Leptin, heart disease and exercise. *World J. Sport Sci.*, Iran, **2009**, 2(1), 13–20.
34. Rushton, J. P. Race, Brain Size, and Intelligence: A Rejoinder to Cain and Vanderwolf, *Personality and Individual Differences*, **2004**, 11, 785–794.
35. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. A computational approach to study fluid movement, *Nanomaterials Yearbook* – **2009**, From Nanostructures, Nanomaterials and

- Nanotechnologies to Nanoindustry, (2010, 181–196). USA: Nova Science Publications. ISBN: 978-1-60876-451-8.16
36. Young, J. W. Anthropometric and Mass Distribution Characteristics of the Adult Female FAA Civil Aeromedical Institute FAA, **2008**, 9–83.
 37. Bartlett, R. Introduction to Sports Biomechanics, 2nd Edition. Taylor and Francis-Library. **2007**.
 38. Hariri Asli, K.; Haghi, A. K. A Numerical Study on Fluid Flow and Pressure drop in Microtubes, *J. Balkan Tribological Association*, Tribotechnics and tribomechanics, Sofia, Bulgaria, **2010**, 16(3), 382–392.
 39. Lee, J. K. et al., Effects of milk ingestion on prolonged exercise capacity in young, healthy men, *Nutrition*, **2008**, 24(4), 340–347.
 40. Unal, M.; Unal, D. D. O; Baltaci, A. K.; Mgulkoc, R. Investigation of serum leptin levels and $\dot{V}O_{2\max}$ value in trained young male athletes and healthy males. *Acta Physiol. Hungary* **2005**, 92, 173–179
 41. Askari, H.; Tykodi, G.; Liu, J.; Dagogo-Jack S. Fasting plasma leptin level is a surrogate measure of insulin sensitivity. *J. Clin. Endocrinol. Metab.*; **2010**, 95(8), 3836–3843.
 42. Reseland, J. E.; Andessen, S. A.; Solvoll, K.; Hjermann, I.; Urdal, P.; Holme, I.; Drevon, C. A. Effect of long-term change in diet and exercise on plasma leptin concentration. *Clin. Nutr.* **2001**, 73, 240–245.
 43. Mokdad, A. H.; Ford, E. S.; Bowman, B. A.; Dietz, W. H.; Vinicor, F.; Bales, V. S.; Marks, J. S. Prevalence of obesity, Diabetes and obesity-related health risk factors, 2001. *JAMA* **2003**, 289, 76–79.
 44. Mendosa-Nunez, V. M.; Garcia-Sanchez, A.; Sanchez-Rodriguez, M.; Galvan-Duart, R. E.; Fonseca-Yerena, E. F. Overweight, waist circumference, age, gender, and insulin resistance as risk factors for Hyperleptinemia. *Obes. Res.* **2002**, 10, 253–259.
 45. Frank, L. L.; Sorensen, B. E.; Yasui, Y.; Tworoger, S. S.; Schwartz, R. S.; Ulrich, C. M.; Irwin, M. L.; Rudolph, R. E.; Rajan, K. B.; Stanczyk, F.; Bowen, D.; Weigle, D. S.; Potter, J. D.; McTiernan A. Effects of exercise on metabolic risk variables in overweight postmenopausal women: a randomized clinical trial. *Obes. Res.* **2005**, 13, 615–625.
 46. Racette, S. B.; Coppack, S. W.; Landt, M.; Klein, S. Leptin production during moderate-intensity aerobic exercise. *J. Clin. Endocrinol. Metabol.* **1997**, 82, 2275–2277.
 47. Altman, R. Risk factors in coronary atherosclerosis athero-inflammation: the meeting point. *Thrombosis J.*; **2003**, 1, 4–7.
 48. Stefanie, L.; Martina, E. Physical activity and endogenous sex hormones in postmenopausal women. *Cancer Causes Control*, **2011**, 22, 81–89.
 49. Akbarzadeh, R. Relationship between the ratio of waist circumference to hip circumference and risk factors and increased lipids and blood pressure in patients with insulin-dependent Diabetes referred to the Endocrinology Institute in Tehran in 1995 and 1996. A Master's thesis, Iran University of Medical Sciences; Faculty of Nursing. **1996**.
 50. Ashwell, M.; Lejeane, S. Ratio of waist circumference to height maybe better indicator need for weight management. *BMI*: **1990**, 312–377.

51. McMahon, M.; Skaggs, B. J.; Sahakian, L.; Grossman, J.; FitzGerald, J.; Ragavendra, N.; Charles-Schoeman, C.; Chernishof, M.; Gorn, A.; Witztum, J. L.; Wong, W. K.; Weisman, M.; Wallace, D. J.; La Cava, A.; Hahn BH. High plasma leptin levels confer increased risk of atherosclerosis in women with systemic lupus erythematosus, and are associated with inflammatory oxidised lipids. *Ann. Rheum. Dis.* **2011**, *70*(9), 1619–1624.
52. Delavar A Research methods in psychology and educational sciences; 1st edition; Tehran; Payame Noor University Press. **2000**.
53. McHugh, P. R.; T. H. Moran, Calories and gastric emptying: a regulatory capacity with implications for feeding. *Am. J. Physiol.* **1979**, *236*(5), 254–260.
54. Shirreff, S. M.; P. Watson, and R. J. Maughan, Milk and effective post-exercise rehydration drink. *Br. J. Nutr.*, **2007**, *98*11, 173–80.
55. Merson S. J.; Shirreff S. M.; Leiper J. B.; Maughan R. J: Changes in blood plasma and red cell volume after ingestion of hypotonic and hypertonic solutions. *Proc. Nutr. Soc.* (In the Press), 2013.
56. Melov, S. et al., Resistance exercise reverses aging in human skeletal muscle. *PLOS One*. **2007**, *2*(5), E465.
57. Miller, S. L. et al., Metabolic response to provision of mixed protein – carbohydrate supplementation during endurance exercise. *Int. J. Sport Nutr. Exerc. Metab.*, **2002**. *12*(4), 384–97.
58. Miller S. L.; et al., The effects of nutritional supplementation throughout an endurance run on leucine kinetics during recovery. *Int. J. Sport Nutr. Metab.*, **2007**, *17*(5), 456–467.
59. Cooke, J. P.; Oka, R. K. Does leptin cause vascular disease? *Circulation* **2002**, *106*, 1904–1905.
60. Canavan, B.; Salem RO, Schurgin, P. K.; Lipinska, I.; Laposata, M.; Grinspoon, S. Effect of physiological leptin administration on markers of inflammation, platelet activation, and platelet aggregation during caloric deprivation. *J. Clin. Endocrinol. Metabol.* **2005**, *90*(10), 5779–5785.
61. Cooke, J. P.; Oka, R. K. Does leptin cause vascular disease? *Circulation* **2002**, *106*, 1904–1905.
62. Ahima, A. S.; Flier, J. S. Adipose tissue as an endocrine organ. *Trends Endocrinol. Metabol.* **2000**, *11*, 327–331.
63. Altman, R. **2003**, Risk factors in coronary atherosclerosis athero-inflammation: the meeting point. *Thrombosis J. 1*, 4–7.
64. Konstantinides, S.; Schafer, K.; Koschnick, S.; Loskutoff, D. J. Leptin-dependent Platelet aggregation and arterial thrombosis suggests a mechanism for atherothrombotic disease in obesity. *J. Clin. Invest.* **2001**, *108*, 1533–1540.
65. Kaye, S. A.; Fdosom, A. R.; Jacobs, D. R. Psychosocial correlates of body fat distribution in Black and white young adult. *Int. J. Obes.*, **1995**, *17*, 271–277
66. Montain S. J.; Coyle E. F.: Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J. Appl. Physiol.* **1992a**, *73*, 1340–1350.

67. Meier, U.; Gressner, A. M. Endocrine regulation of energy metabolism: review of pathobiology and clinical chemical aspects of leptin, ghrelin and adiponectin, and resistin. *Cli. Chem.* **2004**, *50*, 1511–1525.
68. Mokdad, A. H.; Ford, E. S.; Bowman, B. A.; Dietz, W. H.; Vinicor, F.; Bales, V. S.; Marks, J. S. Prevalence of obesity, *Diabetes* and obesity-related health risk factors, 2001. *J. Am. Med. Assoc.* **2003**, *289*, 76–79.
69. Ostlund, R. E.; Jr. Yang, S.; Gingerich, R. Relation between plasma leptin concentration and body fat, gender, diet, age, and metabolic covariates. *J. Clin. Endocrinol. Metabol.* **1996**, *81*, 3909–3913.
70. Baratta, M. Leptin from a signal of adiposity to a hormone mediator in peripheral tissues. *Med. Sci. Monit.* **2002**, *8*, RA282–RA292.
71. Gomez-Merino, D, Chennaoui, M, Drogou, C, Bonneau, D, Guezennec, CY, **2002**, Decrease in serum leptin after prolonged physical activity in men, *Med. Sci. Sports Exerc.*; *34*, 1594–1599.
72. Boden, G.; Chen, X.; Mozzoli, M.; Ryan, I. Effect of fasting on serum leptin in normal human subjects. *J. Clin. Endocrinol. Metabol.* **1996**, *81*, 3419–3423.
73. Brabeau, P.; Gutin, B.; Litaker, M. S.; Ramsey, L. T.; Cannady, W. E.; Allison, J.; Lemmon, C. R.; Owens, S. Influence of physical training on plasma leptin in obese youths. *Canadian J. Appl. Physiol.* **2003**, *28*, 382–396.
74. Houmard, J. A.; Cox, J. H.; Maclean, P. S.; Barakat, H. A. Effect of short-term exercise training on leptin and insulin action. *Metabolism* **2000**, *49*, 858–861.
75. Kukuljan, S. et al., Effects of resistance exercise and fortified milk on skeletal muscle mass, muscle size, and functional performance in middle-aged and older men: an 18-mo randomized controlled trial. *J. Appl. Physiol.*, **2009**, *707*(6), , 18, 64–73.
76. Hickey, M. S.; Considine, R. V.; Israel, R. G. Leptin is related to body fat content in male distance runners. *Am. J. Physiol.* **1996**, *271*, E938–E940.
77. Sihna, M. K.; Caro, J. F. Clinical aspects of leptin. *Vitamins and Hormones* **1998**, *54*, 1–30.
78. Spicer, L. J. Leptin: a possible metabolic signal affecting reproduction. *Domestic Animal Endocrinology* **2001**, *21*, 215–250.
79. Kersick, C. M.; Wismann-Bunn, J.; Fogt, D.; Thomas, A. R.; Taylor, L.; Campbell, B. I.; Wilborn, C. D.; Harvey, T.; Roberts, M. D.; La Bounty, P.; Galbreath, M.; Marcello, B.; Rasmussen, C. J.; Kreidercorresponding RB. Changes in weight loss, body composition and cardiovascular disease risk after altering macronutrient distributions during a regular exercise program in obese women, *Nutr. J.* **2010**, *9*, 59.
80. Hammer, L. D. and Wilson, B. M. Impact of pubertal development on body fat distribution among white, *Hispanic J. Pediatr.*, **1991**, *88*, 975–980.
81. Kraemer, R. R.; Acevedo, A. O.; Synovitz, L. B.; Herbert, E. P.; Gimpel, T.; Castracane, V. D. Leptin and steroid hormone responses to exercise in adolescent females runners over a 7 week season. *Eur. J. Appl. Physiol.* **2001**, *86*, 85–91.
82. Olive, J. L.; Miller, G. D. Differential effects of maximal- and moderate-intensity run on plasma leptin in healthy trained subjects. *Nutrition* **2001**, *17*, 365–369.

83. Zhang, Y.; Proenca, R.; Maffei, M.; Barone, M.; Leopold, M.; Friedman, J. M. Positional cloning of the mouse obese gene and its human homologue. *Nature* **1994**, *373*, 425–432.
84. Barbato, K. B.; Martins Rde, C.; Rodrigues Mde, L.; Braga, J. U.; Francischetti, E. A.; Genelhu, V. Effects of greater-than 5% weight reduction on hemodynamic, metabolic and neuroendocrine profiles of grade 1 obese subjects. *Cardiology* **2006**, *87*, 12–21.
85. Wilkinson, S. B.; et al., Consumption of fluid skin milk promotes greater muscle protein accretion after resistance exercise than does consumption of an isonitrogenous and isoenergetic soy – protein beverage. *Am. J. Clin. Nutr.* **2007**, *85*(4), 1031–1040.
86. Bouassida, A.; Zalleg, D.; Bouassida, S.; Zaouali, M.; Feki, Y.; Zbidi, A.; Tabka, Z. Leptin, its implication in physical exercise and training: a short review. *J. Sport Sci. Med.* **2006**, *5*, 172–181.
87. Hynes, W. G.; Sivitz, W. I.; Morgan, D. A.; Walsh, S. A.; Mark, A. L. Sympathetic and cardiorenal actions of leptin. *Hypertension* **1997**, *30*, 619–623.
88. Bouassida, A.; Zalleg, D.; Bouassida, S.; Zaouali, M.; Feki, Y.; Zbidi, A.; Tabka, Z. Leptin, its implication in physical exercise and training: a short review. *J. Sport Sci. Med.* **2006**, *5*, 172–181.
89. Zafeiridis, A.; Smilios, I.; Conisidine, V.; Tokmakidis, S. P. Serum leptin responses after acute resistance exercise protocols. *J. Appl. Physiol.* **2003**, *94*, 591–597.
90. Elloit, T. A.; et al, Milk ingestion stimulates net muscle protein synthesis following resistance exercise. *Med. Sci. Sport Exerc.*, **2006**, *38*(4), 667–674.
91. Okazaki, T.; Himeno, E.; Manri, H.; Ogata, H.; Ikeda, M. Effects of mild aerobic exercise and mild Hypocaloric diet on plasma leptin in sedentary females. *Clin. Exp. Pharmacol. Physiol.* **1999**, *26*, 415–420.
92. Krauss, R. M.; Winston, M.; Flecher, B. J.; Grundy, S. M. Obesity: impact on cardiovascular disease. *Circulation* **1998**, *98*, 10–16.
93. Singhal, A.; Farooqi, I. S.; Cole, T. J.; O’Rahilly, S.; Fewtrell, M.; Kattenhorn, M.; Lucas, A. D.; Eanfield, J. Influence of leptin on arterial distensibility: a novel link between obesity and cardiovascular disease? *Circulatin* **2002**, *106*, 1919–1926.
94. Essig, D. A.; Alderson, N. L.; Ferguson, M. A.; Bartolli, W. P.; Durstine, J. L. Delayed effects of exercise on the plasma leptin concentration. *Metabolism* **2000**, *49*, 395–399.
95. Enas, E. A. Coronary artery disease epidemic in Indians: a cause for alarm and far action. *J. Indian Med. Assoc.*, **2000**, *98*, 694–702.
96. Hall, J. E.; Kuo, J. J. da Silva, A. A. de Paula, R. B.; Liu, J.; Tallam, L. Obesity -associated hypertension and kidney disease. *Curr. Opin. Nephrol. Hypertens.* **2003**, *12*, 195–299.
97. Hilton, L. K.; Loucks, A. B. Low energy availability, not exercise stress, suppress the durenal rhythm of leptin in healthy young women. *Am. J. Physiol. Endocrinol. Metabol.* **2000**, *278*, E43–E49.
98. Gharakhanloo, R.; Gaeyni, A.; Peyghoon, A. Standardization waist -to-hip ratio for men over 40years to the city of Ahvaz and its association with cardiovascular risk factor-*Diabetes* and cardiovascular disease. *Olympic J. Iran*, **2002**, *3–4*, 59–73. (In Persian with English abstract).

99. Gaeeni, A.; Lameyi, T. The relationship between percent body fat (%BF) and Body mass index (BMI), and the ratio of waist to hip circumference (WHR) in women over fifteen years in Tehran. *Harkat J.*, **2003**, *17*, 95–105. (In Persian with English abstract).
100. Gupta, R. P.; Rastogi, et al. Body mass index, waist-size, waist-hip ratio and cardiovascular Risk factors in urban subjects. *JAPI*, **2007**, *55*, 621–627.
101. Houmard, J. A.; Cox, J. H.; Maclean, P. S.; Barakat, H. A. Effect of short-term exercise training on leptin and insulin action. *Metabolism* **2000**, *49*, 858–861.
102. Rahmani-Nia, F.; Hojjati, Z. The effect of exercise training on body composition and aerobic power in sedentary college age females, *Int. J. Fitness*, India, **2005**, *1*, 24–31.

CHAPTER 3

SOME ASPECTS OF PHYSICAL MODELING OF HUMAN HEALTH

CONTENTS

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3.1 INTRODUCTION

It is controversial about the best method of weight reduction for health benefits. The purpose of this study was to determine leptin and some coronary heart disease risk factors responses in young females after aerobic and resistance exercise training and low caloric diet [1–4].

Reduced functional ability and decreased capability to perform daily activities relate to increased kyphosis angle in elderly. Aging is a phenomenon that can be considered as the natural course of human life. Aging causes some changes in the functions of physiological systems, which are involved in the balance. Age-related changes in posture commonly include a forward head, rounded shoulders, increased thoracic kyphosis, reduced lumbar lordosis and flexed hips and knees. These changes are generally attributed to gradual changes in the structure and mechanics of connective tissues, which result in a loss of elasticity and inability to effectively counteract the gravitational torque that pulls the body into a forward bent position. Certainly, muscle weakness can also affect postural alignment [5–14]. The aging process changes normal postural direction and kyphotic posture often increases with age. Insufficiency in back extensor muscles and decreased shoulder and hip range of motion have been correlated with kyphotic posture and are characterized by an increased curvature of back spine (kyphosis), forward head posture, and decrease in stature. Postural kyphosis may occur in both old and young people and as much posterior curve (arch) of the spine (usually the thoracic spine) is defined [15–20]. Ageing kyphosis is a kind of phenomenon that with increasing age, changes in the curvature of the spine, particularly the thoracic curve reveals. The prevalence and incidence of hyper kyphosis in older persons is probably between 20% and 40%. A longitudinal study of 100 healthy males and females aged 50 years or older (mean age, 62 years), reported a mean thoracic angle increase of 3° per decade. For example, one study of men and women reported mean thoracic kyphosis angles of 26° in persons in their 20 s, 53° in those 60 to 74 years of age, and 66° in those older than 75 years of age. Some researchers have suggested that the posterior arch is accelerating in the seventh decade of life. The normal kyphosis is 20–40 degrees, if this angle is greater than 40 degrees is considered as abnormality. Studies

have shown that an increase in angle of kyphosis in adults is related to decline physical function, impaired balance, slow gait, decreased functional ability and also impaired ability to perform daily duties[21–34].

Balance, or the ability to control postural sway, worsens with age, and this age-related decline has been associated with increased fall risk. The second most common cause of death in elderly people aged 65 years or older is accidental falls. One-third of person's age 65 or older will have one or more falls per year. Many factors may affect to an older adult falling. A number of studies have demonstrated a strong positive correlation between poor balance, abnormalities of gait and falling among elderly adults. Decline in postural stability control is influenced by inactivity or low physical activity. The common method to prevent or treatment of this problem in the seniors (impaired balance) is practice of physical activities in rehabilitation. Studies have shown that in aged people, exercise therapy training can help to maintain and promote their balance ability significantly [35–40]. Exercise therapy is a treatment in which the active exercise (by a person) and non-active (coach) is accomplished. The purpose of the exercise, is prevention and rehabilitation following the occurrence of physical abnormalities and maintain proper postural stability. The most important targets of therapeutic exercises are relieving pain, improving physical function and promoting vital capacity. Previous investigations have shown that daily physical activity and exercise training can reduce the incidence and risk of falls [41–44]. Lazowski et al., demonstrated that general physical training has a positive influence on balance in institutionalized frail elderly subjects, while Rubenstein et al., showed that physical training based on strength, endurance and mobility can progress balance in fall prone elderly subjects. Brown and Holloszy reported that following three months of flexibility and strength training, adults age 60 and older showed no change in measures of gait, and only women improved their balance with eyes opened whereas Crilly and colleagues designed a 12-week exercise program to improve postural stability among 50 older females. Following treatment, the participants of exercise group demonstrated no improvement in postural sway over controls [45–54]. Katzman and colleagues reported that, following 12-week training there was a significant improvement in kyphosis angle but no changes in balance in elder women. Despite extensive studies in the field of postural correction in

older people, it seems there is less attention to the same effects of exercise therapy on posture and balance. However, the relationships between physical activity and balance ability in aged people still remain unclear. Therefore, the purpose of this study was to investigate the effect of exercise therapy on posture and balance in elderly kyphotic women [55–64].

3.2 MATERIALS AND METHODS

Thirty-six inactive obese girls volunteered to participate in the study. They were divided to 4 equal aerobic training (age = 21.5 ± 1.2 years, BMI = 32.5 ± 1.8 kg/m²) resistance training (age = 21.1 ± 1.5 years, BMI = 31.9 ± 1.7 kg/m²), low caloric diet (age = 21.1 ± 1.8 years, BMI = 32.2 ± 1.2 kg/m²) and control (age = 21.2 ± 1.3 years, BMI = 32.7 ± 1.4 kg/m²) groups. Fast blood samples were collected at baseline, 4 weeks and 8 weeks after. The exercise programs were for 8 weeks and 3 sessions aerobic (60 minutes cycling at 60 to 70% of $\text{Vo}_{2\text{max}}$) and resistance (12 exercises, 4 sets, 15 repetitions, 60% of 1RM) training per week. Body composition (BI method) and $\text{Vo}_{2\text{max}}$ (standard exercise test and gas analyzer) were assessed at the same time of blood sampling. Data were compared with a 4 (group) $\times$ 3 (before, 4 weeks and 8 weeks of investigation) repeated measures ANOVA. One-way ANOVA was used for determining between group differences. LSD post hoc was used to determine specific differences when a significant interaction or group effect ($p \leq 0.05$) was obtained [65–70].

3.3 RESULTS AND DISCUSSION

Significant decrease was observed in low caloric diet group from pretest to 4 weeks and 8 weeks, but there was not any significant difference in serum leptin, within other groups ($p \leq 0.05$). There was a significant difference between low caloric diet and control group in serum leptin from pretest to post test ($p \leq 0.05$). No significant differences ($p \leq 0.05$) were observed between groups on serum insulin, low-density lipoprotein (LDL), high-density lipoprotein (HDL), total cholesterol (TC), and glucose (Glue) at any trails ($p \leq 0.05$). Significant decrease in glucose (Glue) was observed

within diet group from pre to 4 weeks and 8 weeks test ($p \leq 0.05$). There was significant difference in triglycerides from pre to 4-week test in diet group ($p \leq 0.05$). Waist to hip ratio (WHR), BMI and body fat percentage (BF) significantly decreased with aerobic and resistance training and low caloric diet ($p \leq 0.05$). $Vo_{2\max}$ increased after 4 weeks and 8 weeks trails compare to baseline in aerobic and resistance groups ($p \leq 0.05$).

3.4 CONCLUSION

Main finding of this study is that 8 weeks aerobic and resistance training was not sufficient to product favorite changes on serum leptin and some risk factors of coronary heart disease. It seems that low caloric diet like this study may reduce serum leptin. Aerobic and resistance training can increase $Vo_{2\max}$ and all three methods able to produce favorite changes on body composition.

NOMENCLATURES

P Runner Blood Pressure (mm-Hg)

t Running Time (sec)

V Velocity (m/s)

S Runner Stature (cm)

KEYWORDS

- exercise training
- heart disease
- leptin
- low caloric diet
- obesity

REFERENCES

1. Tam, J.; Fukumura, D.; Jain RK. A mathematical model of murine metabolic regulation by leptin: energy balance and defense of a stable body weight. *Cell Metab.* **7**; **2010**, *9(1)*, 52–63.
2. Ren J. Leptin and hyperleptinemia from friend to foe for cardiovascular function. *J. Endocrinol.*; **2004**, *181*, 1–10.
3. Rahmouni, K.; Haynes WG. Leptin and the cardiovascular system. *Recent Progress in Hormone Research*; **2004**, *59*, 224–244.
4. Rabe, K.; Lehrke, M.; Parhofer, K. G.; Broedl, U. C. Adipokines and Insulin Resistance. *Mol. Med.* **2008**, *14*, 741–751.
5. Konstantinides, S.; Schafer, K.; Koschnick, S.; Loskutoff, D. J. Leptin-dependent Platelet aggregation and arterial thrombosis suggests a mechanism for atherothrombotic disease in obesity. *J. Clin. Invest.* **2001**, *108*, 1533–1540.
6. Rahmani-Nia, F.; Rahnama, N.; Hojjati Z. Soltani B. Acute effects of aerobic and resistance exercise on serum leptin and some risk factors of coronary heart disease in obese females. *Sport sciences for health*; **2008**, *3*, 118–124.
7. Altman, R.; Risk factors in coronary atherosclerosis athero-inflammation: the meeting point. *Thrombosis J.*; **2003**, *1*, 4–7.
8. Singhal, A.; Farooqi, I. S.; Cole, T. J.; O’Rahilly, S.; Fewtrell, M.; Kattenhorn, M.; Lucas, A.; Deanfield J. Influence of leptin on arterial distensibility: a novel link between obesity and cardiovascular disease. *Circulation*; **2002**, *106*, 1919–1926.
9. Dardeno, T. A.; Chou, S. H.; Moon, H. S.; Chamberland, J. P.; Fiorenza, C. G. and Mantzoros CS. Leptin in Human Physiology and Therapeutics. *Front Neuroendocrinol*; **2010**, *31(3)*, 377–393.
10. Jurimae, J., Jurimae, T.; Leptin responses to short term exercise in college level male rowers; *British J. Sports Med.*, **2005**, *39*, 6–9.
11. McMahon, M.; Skaggs, B. J.; Sahakian, L.; Grossman, J.; FitzGerald, J.; Ragavendra, N.; Charles-Schoeman, C.; Chernishof, M.; Gorn, A.; Witztum, J. L.; Wong, W. K.; Weisman, M.; Wallace, D. J.; La Cava, A.; Hahn BH. High plasma leptin levels confer increased risk of atherosclerosis in women with systemic lupus erythematosus, and are associated with inflammatory oxidised lipids. *Ann. Rheum. Dis.* **2011**, *70(9)*, 1619–1624.
12. Frank, L. L.; Sorensen, B. E.; Yasui, Y.; Tworoger, S. S.; Schwartz, R. S.; Ulrich, C. M.; Irwin, M. L.; Rudolph, R. E.; Rajan, K. B.; Stanczyk, F.; Bowen, D.; Weigle, D. S.; Potter, J. D.; McTiernan, A. Effects of exercise on metabolic risk variables in overweight postmenopausal women: a randomized clinical trial. *Obes. Res.*; **2005**, *13*, 615–625.
13. Webber J. Energy balance in obesity. *Proc. Nutr. Soc.*; **2003**, *62*, 539–543.
14. Reseland, JE, Andessen, S. A.; Solvoll, K.; Hjermann, I.; Urdal, P.; Holme, I.; Drevon CA. Effect of long-term change in diet and exercise on plasma leptin concentration. *Clin. Nutr.*; **2001**, *73*, 240–245.
15. Kyezi, G. A.; Caplan, J. D.; Lowndes, J.; Carpenter, R. L.; Dennis, K. E.; Sivo, S. A.; Angelopoulos TJ. Moderate exercise- induced energy expenditure does not alter leptin levels in sedentary obese men, *Clin. J. Sport Med.*; **2007**, *17*, 49–51.

16. Boden, G.; Chen, X.; Mozzoli, M.; Ryan I. Effect of fasting on serum leptin in normal human subjects. *J. Clin. Endocrinol. Metabol.*; **1996**, *81*, 3419–3423.
17. Thong, F. S. L.; Hudson, R.; Ross, R.; Janssen, I.; Grahan, T. E. Plasma leptin in moderately obese males: independent effects of weight loss and aerobic exercise, *Am. J. Physiol.*, Endocrinology and Metabolism; **2000**, *279*, E307–E313.
18. Kraemer, K. K.; Chu, H.; Castracane, V. D. Leptin and exercise. *Exp. Biol. Med.*; **2002**, *227*, 701–708.
19. Rahmani-Nia, F.; Hojjati, Z.; Rahnama, N.; Soltani, B. Leptin, heart disease and exercise. *World J. Sport Sci.*; **2009**, *1*, 13–20.
20. Pasman, W. J.; Westerterp-Plantegna, M. S.; Saris, W. H. M. The effect of exercise training on plasma leptin levels in obese male. *Am. J. Physiol.*; **1998**, *274*, E280–E286.
21. Fatouros, I. G.; Tournis, S.; Leontsini, D.; Jamurtas, A. Z.; Sxina, M.; Thomakos, P.; Manousaki, M.; Douroudos, I.; Taxildaris, K.; Mitrakou A. Leptin and adiponectin responses in overweight inactive elderly following resistance training and detraining are intensity related. *The J. Clin. Endocrinol. Metabol.*; **2005**, *90*, 5970–5977.
22. Fatouros, I. G.; Chatzinikolaou, A.; Tournis, S.; Nikolaidis, M. G.; Jamurtas, A. Z.; Douroudos, I. I.; Papassotiropoulos, I.; Thomakos, P. M.; Taxildaris, K.; Mastorakos, G.; Mitrakou A. Intensity of Resistance Exercise Determines Adipokine and Resting Energy Expenditure Responses in Overweight Elderly Individuals, *Diabetes Care*; **2009**, *32(12)*, 2161–2167.
23. Wisse BE. The inflammatory syndrome: The role of adipose tissue cytokines in metabolic disorders linked to obesity. *J. Am. Soc. Nephrol.*; **2004**, *15*, 2792–2800.
24. Li MD. Leptin and Beyond: An Odyssey to the Central Control of Body Weight. *Yale J. Biol. Med.*; **2011**, *84(1)*, 1–7.
25. Askari, H.; Tykodi, G.; Liu, J.; Dagogo-Jack S. **2010**, Fasting plasma leptin level is a surrogate measure of insulin sensitivity. *J. Clin. Endocrinol. Metab.*; *95(8)*, 3836–3843.
26. Hilton, L. K.; Loucks, A. B. Low energy availability, not exercise stress, suppress the diurnal rhythm of leptin in healthy young women, *Am. J. Physiol. Endocrinol. Metabol.*; **2000**, *278*, E43–E49.
27. Gomez-Merino, D.; Chennaoui, M.; Drogou, C.; Bonneau, D.; Guezennec, CY, Decrease in serum leptin after prolonged physical activity in men, *Med. Sci. Sports Exerc.*; **2002**, *34*, 1594–1599.
28. Hall, J. E.; Kuo, J. J.; da Silva, A. A.; de Paula, R. B.; Liu, J.; Tallam L. Obesity-associated hypertension and kidney disease. *Curr. Opin. Nephrol. Hypertens.*; **2003**, *12*, 195–299.
29. Bouassida, A.; Zalleg D. Bouassida, S.; Zaouali, M.; Feki, Y.; Zbidi, A.; Tabka Z. Leptin, its implication in physical exercise and training: a short review. *J. Sport Sci. Med.*; **2006**, *5*, 172–181.
30. Kersick, C. M.; Wismann-Bunn, J.; Fogt, D.; Thomas, A. R.; Taylor, L.; Campbell, B. I.; Wilborn, C. D.; Harvey, T.; Roberts, M. D.; La Bounty, P.; Galbreath, M.; Marcelllo, B.; Rasmussen, C. J.; Kreidercorresponding RB. Changes in weight loss, body composition and cardiovascular disease risk after altering macronutrient distributions during a regular exercise program in obese women, *Nutr. J.*; **2010**, *9*, 59.

31. Hurst Thomas, D. Skull Wars Kennewick Man, Archaeology and the Battle for Native American Identity, Washington University: School of Law - Anheuser-Busch Hall, **2001**, 38–41.
32. Nabieh, A.; Mohamed, I. Anthropometric Measurements as a Significant for Choosing Juniors in Both Volleyball and Handball Sports (Factorial Analysis Study), *World J. Sport Sci.* **2010**, *3*(4), 277–289.
33. Rogana, S.; Hilfiker, R.; Clarys, P.; Clijsen, R.; Taeymans, J. Position-specific and Team-ranking-related Morphological Characteristics in German Amateur Soccer Players – a Descriptive Study, *Int. J. Appl. Sports Sci.*, **2011**, *23*(1), 168–182.
34. Luigi P. W.; Bercades, T. Somatotypes of national elite combative sport athletes, *Braz. J. Biometricity*, **2009**, *3*(1), p. 21–30.
35. Dacres-Mannings, S.; Rochester, S.; Frail, H. Anthropometric profiles of Australian Rugby Institute, club and state level Rugby Union players. **2010**.
36. Gaurav, V.; Singh, M.; Singh, S. Anthropometric characteristics, somatotyping and body composition of volleyball and basketball players, *J. Phys. Educ. Sports Manage.* **2010**, *1*(3), 28–32, Available online <http://www.acadjourn.org/jpesm>
37. Beat K.; Kohler, G. Influence of anthropometry on race performance in ultra-endurance triathletes in the longest triathlon in North America, *Int. SportMed J.*, **2007**, *8*(2), 87–96, <http://www.ismj.com>
38. Joseph, J.; Greene, M. S.; Timothy, A.; McGuine, A. T. C.; Leverson, G.; Thomas M. Anthropometric and Performance Measures for High School Basketball Players, *J. Athl. Training*, **1998**, *33*(3), 229–232
39. Amatya, D. L. Comparative Study of Somatotype of Nepalese Sportsmen, National Association for Sports Health and Fitness. **1999**.
40. Sang Hong, K. Kinanthropometric Study of Korean and Nepalese Marathon Runners, keimyung university Diwakar Lal Amatya, MSC-Athletics and Sports Science-India, Sports Kinanthropometrist-Australia, Sports Expert-National Sports Council, January. **2008**.
41. Gaunt, B. W. Anthropometric and Demographic Factors Affecting Distance Hopped-aid Limb Symmetry Index for the Crossover Hop-for distance test in High School athletes, *J. Orthop. Sport Phys.*, **2001**, *31*(3), 145–151
42. Gross, M.; Dailey, E. S.; Melissa, D.; Dalton, A.; Lee, K.; Wendy, I.; Ashley, C. Relationship Between Lifting Capacity and Anthropometric Measures, *J. Orthop. Sport Phys.*, **2000**, *30*(5), 237–247, 258–262.
43. Roberto, C.; José, a.; Pérez, J.; Cortell, m. Juan, J.; Rivas, J. Correlations among anthropometric parameters, jump power, and position in professional basketball players, **2008**.
44. Pui, W. K.; Hendrik H. Anthropometric, gait and strength characteristics of Kenyan distance runners, *J. Sports Sci. Med.* **2008**, *7*, 499–504, <http://www.jssm.org>
45. Jawis, M. N.; Singh, R.; Singh, H. J.; Yassin, M. N. Anthropometric and physiological profiles of sepak takraw players, *Br. J. Sports Med.* **2005**, *39*, 825–829. doi: 10.1136/bjism.2004.016915.
46. Lephart, S. M.; Abt, J. P.; Ferris, C. M.; Sell, T. C.; Nagai, T.; Myers, J. B.; Irrgang, J. Neuromuscular and biomechanical characteristic changes in high school athletes: a plyometric versus basic resistance program, *Br. J. Sports Med.* **2005**, *39*, 932–938. doi: 10.1136/bjism.2005.019083.

47. Darr, K. C.; Bassett, D. R.; Morgan, B. J.; Thomas, D. P. Effects of age and training status on heart rate recovery after peak exercise. *Am. J. Physiol.* **1988**, *254*, H340-H343.
48. Engerman, S. 'The Height of U. S. Slaves', *Local Population Studies*, **1976**, *16(1)*, 45–50.
49. Sunder, M. The Making of Giants in a Welfare State: The Norwegian Experience in the Twentieth Century', *Economics and Biology*, **2004**, 75–86.
50. Woitek, U. Height Cycles in the Eighteenth and Nineteenth Centuries', *Econ. Hum. Biol.*, **2003**, 243–258.
51. Fogel, R. 'Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the making of Economic Policy', *Am. Econ. Rev.*, **1994**, 369–394.
52. Goonetilleke, R. S.; Ho, Cheuk Fan E.; So, R. H. Y. Foot Anthropometry in Hong Kong. (pp. 81–88) *Proceedings of the ASEAN 97 Conference*, Kuala Lumpur, Malaysia. **1997**.
53. Baten, J. "Economic Development and the Distribution of Nutritional Resources in Bavaria, 1797–1839, in *J. Income Distrib.* **2000**, *9*, 89–106.
54. Hariri Asli K. Water Hammer Research; *Advances in Nonlinear Dynamics Modeling*, (2012, 88–121). Canada, USA: Published by Apple Academic Press, Inc., Exclusive worldwide distribution by CRC Press, a Taylor and Francis Group, Print ISBN: 9781926895314, eBook: 978-1-46-656887-7.
55. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. "Some Aspects of Physical and Numerical Modeling of Water Hammer in Pipelines," *Nonlinear Dynamics An International Journal of Nonlinear Dynamics and Chaos in Engineering Systems*, ISSN: 1573–269X (electronic version) *Journal /1071 Springer*, Netherlands, **2009**, ISSN: 0924–090X (print version), Heidelberg, Germany, Springer, Volume 60, Number 4 / June, **2010**, 677–701.
56. Heather, E. Collins-Schramm and others, "Markers that Discriminate Between European and African Ancestry Show Limited Variation Within Africa," *Human Genetics* **2002**, *111*, 566–9.
57. Komlos, J.; Baur, M. "From the Tallest to One of the Fattest: The Enigmatic Fate of the Size of the American Population in the Twentieth Century," *Econ. Hum. Biol.* **2004**, *2(1)*, 57–74.
58. Komlos, J.; Kriwy, P. "The Biological Standard of Living in the Two Germanies," *German Econ. Rev.* **2003**, *4*, 493–507.
59. Levy, W. C.; Cerqueira, M. D.; Harp, G. D.; Johannessen, K. A.; Abrass, I. B.; Schwartz, R. S.; Stratton, J. R. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. *Am. J. Cardiol.*, **1998**, *82*, 1236–1241.
60. Margo, R. The Heights of American Slaves. *Social Science History*, **1982**, *6*, 516–538.
61. Rahmani-Nia, F.; Hojjati, Z. Effect of selected training on body composition and aerobic power of Females College. *J. Harkat*, Iran, **2004**, *1*, 18–21.
62. Rahmani-Nia, F.; Rahnama, N.; Hojjati, Z.; Soltani, B. Acute effects of aerobic and resistance exercise on serum leptin and some risk factors of coronary heart disease in obese females. *Sport sciences for health*, **2008**, *2(3)*, 118–124.
63. Rahmani-Nia, F.; Hojjati, Z.; Rahnama, N.; Soltani, B. Leptin, heart disease and exercise. *World J. Sport Sci.*, Iran, **2009**, *2(1)*, 13–20.

64. Rushton, J. P. Race, Brain Size, and Intelligence: A Rejoinder to Cain and Vanderwolf, *Personality and Individual Differences*, **2004**, *11*, 785–794.
65. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. A computational approach to study fluid movement, *Nanomaterials Yearbook – 2009*, From Nanostructures, Nanomaterials and Nanotechnologies to Nanoindustry, (**2010**, 181–196). USA: Nova Science Publications. ISBN: 978-1-60876-451-8.16
66. Young, J. W. Anthropometric and Mass Distribution Characteristics of the Adult Female FAA Civil Aeromedical Institute FAA 9-83. **2008**.
67. Bartlett, R. Introduction to Sports Biomechanics, 2nd Edition. Taylor and Francise- Library. **2007**.
68. Hariri Asli, K.; Haghi, A. K. A Numerical Study on Fluid Flow and Pressure drop in Microtubes, *J. Balkan Tribological Association*, Tribotechnics and tribomechanics, Sofia, Bulgaria, **2010**, *16*(3), 382–392.
69. Rahmani-Nia, F.; Hojjati, Z. The effect of exercise training on body composition and aerobic power in sedentary college age females, *Int. J. Fitness*, India, **2005**, *1*, 24–31.
70. Fenkci, S.; Sarsan, A.; Rota, S.; Ardic F. Effects of resistance or aerobic exercises on metabolic parameters in obese women who are not on a diet. *Adv. Therapeutic*; **2006**, *23*, 404–413.

CHAPTER 4

RISK REDUCTION OF SPORTSWOMEN ACTIVITIES

CONTENTS

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4.1 INTRODUCTION

The anthropometric parameter of athletes itself has a direct benefit on economic success or an increased standard of living. The variety of human physique plays an important role to attain better performance in particular sports. Every game requires a specific type of body where as unsuitable body types in relation to the sports may build great stumbling block in the progress of the sports performance. Besides the body size, the constitutional make up of body composition components are also important.

4.2 MATERIALS AND METHODS

Present book shows a dynamic and computational modeling for anthropometric of athletes. According to this technique, in this work an improved modeling based on measurements was classified and compared with a special type of skulls of sportswomen with other Anthropometric classes of sportswomen. On the other hand present work compares the anthropometric data for Blood pressure of Runner related to Runner Velocity (Running Time) and Runner Stature in two cases. Consequently, the results of this work will help to reduce the risk of sportswomen activities. In this work, dateline for field tests data collection was at 11:00 a.m., 10/09/2012 until 29/09/2012. Locaton of work field tests model was at Rasht city in the north of Iran. In this work the following are considerations was made when using and applying anthropometric data for blood pressure of runner related to Runner Velocity (Running Time) and Runner Stature. Blood pressure of runner was measured in two cases: first at the running state and second at the before of running. Twenty healthy and young sprint runner females participate in this study. They signed a written/informed consent [1–24]. All subjects completed a medical questionnaire to ensure that they were not taking any medication, and were free cardiac, respiratory, renal or metabolic diseases. They didn't have any orthopaedic injury that would inhibit physical activity. The subjects were tested in the follicular phase of the menstrual cycle based on their previous menses. All participants consumed their last meal at least 2 h before the test and refrained from intensive exercise in the 24 h period preceding testing. All anthropometric measures were recorded

in the morning by the same experienced anthropometrist. Stretch stature was measured using a wall-mounted stadiometer (± 0.1 cm). The exercise systolic and diastolic blood pressures were measured on left arm of each participant using cuffs of appropriate size (Table 1). The measurements were recorded just after 100 m intense running in standing position. Curve estimation procedure has used these data, which have been detected field tests (Table 1). These data have been compared by blood pressure of runner and Runner Velocity and stature data, which have been collected from actual systems (field tests). The model was calibrated using one set of data, without changing parameter values. It was used to match a different set of results [25–28]. Regression model (Table 1) has been built based on field tests data. Scatter diagram, Correlation (Fig. 1) and Autocorrelations procedure (Fig. 1) for blood pressure was formed by estimating regression statistics and producing related plots (Fig. 1) for field tests model with the following three assumptions Eqs. (1)–(3):

Assumption (1):

$$p = f(S), \quad (1)$$

Assumption (2):

$$p = f(V), \quad (2)$$

Assumption (3):

$$p = f(V, S), \quad (3)$$

V – Runner Velocity is the most important variable, S – Runner Stature. Dependent variable: P – blood pressure (mm-Hg) for starting point of running condition. The independent variable is Running Time (sec) and Runner Stature (cm).

TABLE 1 Field tests and regression anthropometric model.

No.	Age	Height (m)	Weight (KG)	Heart rate	Blood Pressure	100 (m)
					(mm-Hg)	Running Time (sec)
1	19	165	50	176	111–71	13.45
2	16	159	52	173	120–68	13.66
3	18	168	47	174	114–83	15.03
4	16	158	50	111	100–75	13.67

TABLE 1 (Continued)

No.	Age	Height (m)	Weight (KG)	Heart rate	Blood Pressure	100 (m)
					(mm-Hg)	Running Time (sec)
5	14	164	52	173	118–71	14.3
6	14	166	44	178	109–81	14.73
7	14	161	54	191	116–73	17.1
8	20	161	46	133	142–89	17.55
9	21	159	48	160	120–77	16.28
10	14	167	52	150	134–83	16.03
11	18	168	56	153	148–93	16.27
12	15	64	55	166	134–83	16.77
13	23	154	45	140	139–104	17.35
14	22	170	56	154	120–65	15.22
15	21	174	59	164	100–64	14.98
16	21	151	55	177	116–74	14.9
17	22	170	62	93	126–107	14.51
18	28	158	55	170	113–84	13.98
19	22	165	56	166	113–74	14.45
20	24	166	62	168	119–83	16.98

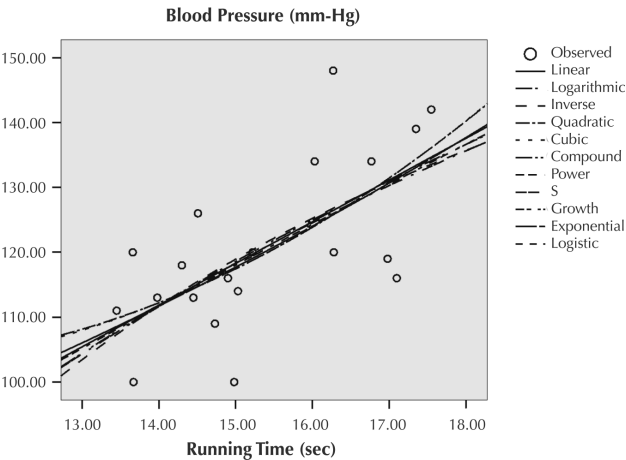


FIGURE 1 Regression and curve fit for runner dynamic blood pressure against Running Time.

4.3 RESULTS AND DISCUSSION

In this work conclusions were drawn on the basis of experiments and calculations for the three assumptions:

Assumption (1):

$$p = f(S), \quad (1)$$

The most important effects that were observed based on regression for model summary (Fig. 2).

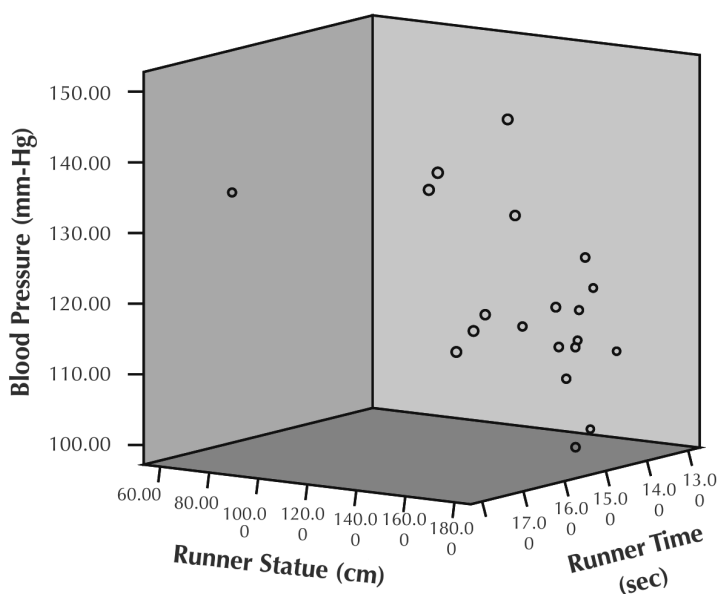


FIGURE 2 Regression and 3-D Scatter diagram for dynamic blood pressure against Runner Statue and Running Time.

4.3.1 REGRESSION MODEL DUE TO FIELD TEST FOR ANTHROPOMETRIC MODEL OF BLOOD PRESSURE

The auto-regression procedure accounts for first-order auto-correlated residuals. It provides reliable estimates of both goodness of-fit measures and significant levels of chosen predictor variables [25–28].

Present work compared the anthropometric data for blood pressure of runner related to Runner Velocity (Running Time) and Runner Stature in two cases.

The dependent and independent variables of runner were measured in the following cases:

First: at the running state (dynamic)

Second: at the before of running (static)

Dependent variable: P – blood pressure with nomenclature “ Y ,” independent variable with nomenclature “ X ” such as:

V – Runner Velocity is the most important variable, S – Runner Stature. The independent variable is velocity (m.s^{-1}) and Runner Stature (cm). Assumption (2):

$$p = f(V), \quad (2)$$

For second assumption the curve estimation procedure allows quick estimating regression statistics and producing related plots for different models. Hence the auto-regression procedure by regression software “SPSS 10.0.5” was selected for the curve estimation procedure in the present work. The regression model has been built based on field test data.

Regression software “SPSS” has fitted the function curve and provided regression analysis. So, the regression model has been found in the final procedure. This model has compared field test results using Lab model results [29–31].

Assumption (3):

$$p = f(V, S), \quad (3)$$

For third assumption the curve estimation procedure is illustrated in besides, the runner blood pressure was formed by estimating regression statistics are listed in Table 1. Although related plots for the field test model were produced. Regression software “SPSS 10.0.5” performs multidimensional scaling of proximity data to find least-squares representation of the objects in a low-dimensional space [32–90].

4.3.2 COMPARISON WITH OTHER WORKS

Rahmani–Nia, and Hodjati Obtained the effect of exercise training on body composition and aerobic power in sedentary college age females. Comparison showed similarity in present work and the results of that works [91].

4.4 CONCLUSIONS

High Technology and high-speed detectors were investigated as long as this work. By field tests, this work investigated the effects of the Runner Velocity as the most important variable and Runner Stature on runner blood pressure.

Present work showed that “Regression model” is accurate model for Modeling for Anthropometric of Body Motion. Hence in order to presentation for importance of Runner Velocity on blood pressure phenomenon, it was compared the models for laboratory; computational and field tests experiments. As long as these procedures, it was showed that the Regression based model for Anthropometric of Body Motion at the running state (dynamic) and before of running (static). On the other hand, this idea were included the proper analysis to provide a dynamic response to the shortcomings of the Body Motion. It also performed the High Technology and high-speed detectors equipment’s to determine the operational procedures to avoid hazards on human health; Economics and Human Biology. Consequently, the results of this work will help to reduce the risk of sport women activities.

NOMENCLATURES

P	Runner Blood Pressure (mm-Hg)
t	Running Time (sec)
V	Velocity (m/s)
S	Runner Stature (cm)

KEYWORDS

- anthropometric
- body motion
- computational model
- dynamic modeling
- regression

REFERENCES

1. Hurst Thomas, D. Skull Wars Kennewick Man, Archaeology and the Battle for Native American Identity, Washington University: School of Law - Anheuser-Busch Hall, **2001**, 38–41.
2. Nabieh, A.; Mohamed, I. Anthropometric Measurements as a Significant for Choosing Juniors in Both Volleyball and Handball Sports (Factorial Analysis Study), *World J. Sport Sci.* **2010**, 3(4), 277–289.
3. Rogana, S.; Hilfigerd, R.; Clarysb, P.; Clijsenc, R.; Taeymansac, J. Position-specific and Team-ranking-related Morphological Characteristics in German Amateur Soccer Players – a Descriptive Study, *Int. J. Appl. Sports Sci.*, **2011**, 23(1), 168–182.
4. Luigi P. W.; Bercades, T. Somatotypes of national elite combative sport athletes, *Braz. J. Biomotricity*, **2009**, 3(1), 21–30.
5. Dacres-Mannings, S.; Rochester, S.; Frail, H. Anthropometric profiles of Australian Rugby Institute, club and state level Rugby Union players. **2010**.
6. Gaurav, V.; Singh, M.; Singh, S. Anthropometric characteristics, somatotyping and body composition of volleyball and basketball players, *J. Phys. Educ. Sports Manage.* **2010**, 1(3), 28–32, Available online <http://www.acadjourn.org/jpesm>
7. Beat K.; Kohler, G. Influence of anthropometry on race performance in ultra-endurance triathletes in the longest triathlon in North America, *Int. SportMed J.*, **2007**, 8(2), 87–96, <http://www.ismj.com>
8. Joseph, J.; Greene, M. S.; Timothy, A.; McGuine, A. T. C.; Leverson, G.; Thomas M. Anthropometric and Performance Measures for High School Basketball Players, *J. Athl. Training*, **1998**, 33(3), 229–232
9. Amatya, D. L. Comparative Study of Somatotype of Nepalese Sportsmen, National Association for Sports Health and Fitness. **1999**.
10. Sang Hong, K. Kinanthropometric Study of Korean and Nepalese Marathon Runners, keimyung university Diwakar Lal Amatya, MSC-Athletics and Sports Science-India, Sports Kinanthropometrist-Australia, Sports Expert-National Sports Council, January. **2008**.

11. Gaunt, B. W. Anthropometric and Demographic Factors Affecting Distance Hopped-aid Limb Symmetry Index for the Crossover Hop-for distance test in High School athletes, *J. Orthop. Sport Phys.*, **2001**, *31*(3), 145–151
12. Gross, M.; Dailey, E. S.; Melissa, D.; Dalton, A.; lee, K.; Wendy, I.; Ashley, C. Relationship Between Lifting Capacity and Anthropometric Measures, *J. Orthop. Sport Phys.*, **2000**, *30*(5), 237–247, 258–262.
13. Roberto, C, Jose, A.; Perez, J.; Cortell, m. Juan, J.; Rivas, J. Correlations among anthropometric parameters, jump power, and position in professional basketball players, **2008**.
14. Pui, W. K.; Hendrik H. Anthropometric, gait and strength characteristics of Kenyan distance runners, *J. Sports Sci. Med.* **2008**, *7*, 499–504, <http://www.jssm.org>
15. Jawis, M. N.; Singh, R.; Singh, H. J.; Yassin, M. N. Anthropometric and physiological profiles of sepak takraw players, *Br. J. Sports Med.* **2005**, *39*, 825–829. doi: 10.1136/bjism.2004.016915.
16. Lephart, S. M.; Abt, J. P.; Ferris, C. M.; Sell, T. C.; Nagai, T.; Myers, J. B.; Irrgang, J. Neuromuscular and biomechanical characteristic changes in high school athletes: a plyometric versus basic resistance program, *Br. J. Sports Med.* **2005**, *39*, 932–938. doi: 17.1136/bjism.2005.019083.
17. Darr, K. C.; Bassett, D. R.; Morgan, B. J.; Thomas, D. P. Effects of age and training status on heart rate recovery after peak exercise. *Am. J. Physiol.* **1988**, *254*, H340-H343.
18. Engerman, S. ‘The Height of U. S. Slaves’, *Local Population Studies*, **1976**, *16*, 1, 45–50.
19. Sunder, M. The Making of Giants in a Welfare State: The Norwegian Experience in the Twentieth Century’, *Economics and Biology*, **2004**, 75–86.
20. Woitek, U. Height Cycles in the Eighteenth and Nineteenth Centuries’, *Econ. Hum. Biol.*, **2003**, 243–258.
21. Fogel, R. ‘Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the making of Economic Policy’, *Am. Econ. Rev.*, **1994**, 369–394.
22. Goonetilleke, R. S.; Ho, Cheuk Fan E.; So, R. H. Y. Foot Anthropometry in Hong Kong. (1997, 81–88) Proceedings of the ASEAN 97 Conference, Kuala Lumpur, Malaysia.
23. Baten, J. “Economic Development and the Distribution of Nutritional Resources in Bavaria, 1797–1839, *J. Income Distrib.* **2000**, *9*, 89–106.
24. Hariri Asli K. Water Hammer Research; Advances in Nonlinear Dynamics Modeling, (2012, 88–121). Canada, USA: Published by Apple Academic Press, Inc., Exclusive worldwide distribution by CRC Press, a Taylor and Francis Group, Print ISBN: 9781926895314, eBook: 978-1-46-656887-7.
25. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. “Some Aspects of Physical and Numerical Modeling of Water Hammer in Pipelines,” *Nonlinear Dynamics An International Journal of Nonlinear Dynamics and Chaos in Engineering Systems*, ISSN: 1573–269X (electronic version) Journal /1071 Springer, Netherlands, **2009**, ISSN: 0924–090X (print version), Heidelberg, Germany, Springer, Volume 60, Number 4 / June, **2010**, 677–701.

26. Heather, E. Collins-Schramm and others, "Markers that Discriminate Between European and African Ancestry Show Limited Variation Within Africa," *Human Genetics* **2002**, *111*, 566–9.
27. Komlos, J.; Baur, M. "From the Tallest to One of the Fattest: The Enigmatic Fate of the Size of the American Population in the Twentieth Century," *Econ. Hum. Biol.* **2004**, *2(1)*, 57–74.
28. Komlos, J.; Kriwy, P. "The Biological Standard of Living in the Two Germanies," *German Econ. Rev.* **2003**, *4*, 493–507.
29. Levy, W. C.; Cerqueira, M. D.; Harp, G. D.; Johannessen, K. A.; Abrass, I. B.; Schwartz, R. S.; Stratton, J. R. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. *Am. J. Cardiol.*, **1998**, *82*, 1236–1241.
30. Margo, R. The Heights of American Slaves. *Social Science History*, **1982**, *6*, 516–538.
31. Rahmani-Nia, F.; Hojjati, Z. Effect of selected training on body composition and aerobic power of Females College. *J. Harkat, Iran*, **2004**, *1*, 18–21.
32. Rahmani-Nia, F.; Rahnama, N.; Hojjati, Z.; And Soltani, B. Acute effects of aerobic and resistance exercise on serum leptin and some risk factors of coronary heart disease in obese females. *Sport sciences for health*, **2008**, *2(3)*, 118–124.
33. Rahmani-Nia, F.; Hojjati, Z.; Rahnama, N.; Soltani, B. Leptin, heart disease and exercise. *World J. Sport Sci.*, Iran, **2009**, *2(1)*, 13–20.
34. Rushton, J. P. Race, Brain Size, and Intelligence: A Rejoinder to Cain and Vanderwolf, Personality and Individual Differences, **2004**, *11*, 785–794.
35. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. A computational approach to study fluid movement, *Nanomaterials Yearbook – 2009*, From Nanostructures, Nanomaterials and Nanotechnologies to Nanoindustry, (**2010**, 181–196). USA: Nova Science Publications. ISBN: 978-1-60876-451-8.16
36. Young, J. W. Anthropometric and Mass Distribution Characteristics of the Adult Female FAA Civil Aeromedical Institute FAA 9–83. **2008**.
37. Bartlett, R. Introduction to Sports Biomechanics, 2nd Edition. Taylor and Francise- Library. **2007**.
38. Hariri Asli, K.; Haghi, A. K. A Numerical Study on Fluid Flow and Pressure drop in Microtubes, *J. Balkan Tribological Association*, Tribotechnics and tribomechanics, Sofia, Bulgaria, **2010**, *16(3)*, 382–392.
39. Corsonello, A.; Malara, A.; Ientile, R. Leptin enhances adenosine diphosphate-induced platelet aggregation in healthy subjects. *Obes. Res.* **2002**, *10*, 306–317.
40. Unal, M.; Unal, D. D. O; Baltaci, A. K.; Mgulkoc, R. Investigation of serum leptin levels and $\dot{V}O_{2max}$ value in trained young male athletes and healthy males. *Acta Physiol. Hungary* **2005**, *92*, 173–179
41. Askari, H.; Tykodi, G.; Liu, J.; Dagogo-Jack S. Fasting plasma leptin level is a surrogate measure of insulin sensitivity. *J. Clin. Endocrinol. Metab.*; **2010**, *95(8)*, 3836–3843.
42. Reseland, J. E.; Andessen, S. A.; Solvoll, K.; Hjermann, I.; Urdal, P.; Holme, I.; Drevon, C. A. Effect of long-term change in diet and exercise on plasma leptin concentration. *Clin. Nutr.* **2001**, *73*, 240–245.
43. Mokdad, A. H.; Ford, E. S.; Bowman, B. A.; Dietz, W. H.; Vinicor, F.; Bales, V. S.; Marks, J. S. Prevalence of obesity, *Diabetes* and obesity-related health risk factors, 2001. *JAMA* **2003**, *289*, 76–79.

44. Mendosa-Nunez, V. M.; Garcia-Sanchez, A.; Sanchez-Rodriguez, M.; Galvan-Duart, R. E.; Fonseca-Yerena, E. F. Overweight, waist circumference, age, gender, and insulin resistance as risk factors for Hyperleptinemia. *Obes. Res.* **2002**, *10*, 253–259.
45. Frank, L. L.; Sorensen, B. E.; Yasui, Y.; Tworoger, S. S.; Schwartz, R. S.; Ulrich, C. M.; Irwin, M. L.; Rudolph, R. E.; Rajan, K. B.; Stanczyk, F.; Bowen, D.; Weigle, D. S.; Potter, J. D.; McTiernan A. Effects of exercise on metabolic risk variables in overweight postmenopausal women: a randomized clinical trial. *Obes. Res.*; **2005**, *13*, 615–625.
46. Racette, S. B.; Coppack, S. W.; Landt, M.; Klein, S. Leptin production during moderate-intensity aerobic exercise. *J. Clin. Endocrinol. Metabol.* **1997**, *82*, 2275–2277.
47. Altman, R.; Risk factors in coronary atherosclerosis athero-inflammation: the meeting point. *Thrombosis J.*; **2003**, *1*, 4–7.
48. Stefanie, L.; Martina, E. Physical activity and endogenous sex hormones in postmenopausal women. *Cancer Causes Control*, **2011**, *22*, 81–89.
49. Akbarzadeh, R. Relationship between the ratio of waist circumference to hip circumference and risk factors and increased lipids and blood pressure in patients with insulin-dependent *Diabetes* referred to the Endocrinology Institute in Tehran in 1995 and 1996. A Master's thesis, Iran University of Medical Sciences; Faculty of Nursing. **1996**.
50. Ashwell, M.; Lejeane, S. Ratio of waist circumference to height maybe better indicator need for weight management. *BMI*: **1990**, 312–377.
51. McMahon, M.; Skaggs, B. J.; Sahakian, L.; Grossman, J.; FitzGerald, J.; Ragavendra, N.; Charles-Schoeman, C.; Chernishof, M.; Gorn, A.; Witztum, J. L.; Wong, W. K.; Weisman, M.; Wallace, D. J.; La Cava, A.; Hahn BH. High plasma leptin levels confer increased risk of atherosclerosis in women with systemic lupus erythematosus, and are associated with inflammatory oxidised lipids. *Ann. Rheum. Dis.* **2011**, *70*(9), 1619–1624.
52. Delavar, A. Research methods in psychology and educational sciences; 1st edition; Tehran; Payame Noor University Press. **2000**.
53. McHugh, P. R.; T. H. Moran, Calories and gastric emptying: a regulatory capacity with implications for feeding. *Am. J. Physiol.* **1979**, *236*(5), R 254–60.
54. Shirreff, S. M.; P. Watson, and R. J. Maughan, Milk and effective post – exercise rehydration drink. *Br. J. Nutr.*, **2007**, *98*(11), 173–80.
55. Merson S. J.; Shirreff S. M.; Leiper J. B.; Maughan R. J: Changes in blood plasma and red cell volume after ingestion of hypotonic and hypertonic solutions. *Proc. Nutr. Soc.* (In the Press), 2002.
56. Melov, S. et al., Resistance exercise reverses aging in human skeletal muscle. *PLOS One*. **2007**, *2*(5), E 465.
57. Miller, S. L.; et al., Metabolic response to provision of mixed protein – carbohydrate supplementation during endurance exercise. *Int. J. Sport Nutr. Exerc. Metab.*, 2002. *12*(4): p 384–97.
58. Miller S. L.; et al., The effects of nutritional supplementation throughout an endurance run on leucine kinetics during recovery. *Int J Sport Nutr Metab*, **2007**, *17*(5), 456–67.
59. Cooke, J. P.; Oka, R. K. **2002**, Does leptin cause vascular disease? *Circulation* *106*, 1904–1905.

60. Canavan, B.; Salem, R. O.; Schurgin, P. K.; Lipinska, I.; Laposata, M.; Grinspoon S **2005**, Effect of physiological leptin administration on markers of inflammation, platelet activation, and platelet aggregation during caloric deprivation. *The J. Clin. Endocrinol. Metabol.* 90(10), 5779–5785.
61. Cooke, J. P.; Oka, R. K. **2002**, Does leptin cause vascular disease? *Circulation* 106, 1904–1905.
62. Ahima, A. S.; Flier, J. S. **2000**, Adipose tissue as an endocrine organ. *Trends Endocrinol. Metabol.* 11, 327–331.
63. Altman, R. **2003**, Risk factors in coronary atherosclerosis athero-inflammation: the meeting point. *Thrombosis J.* 1, 4–7.
64. Konstantinides, S.; Schafer, K.; Koschnick, S.; Loskutoff, D. J. **2001**, Leptin-dependent Platelet aggregation and arterial thrombosis suggests a mechanism for atherothrombotic disease in obesity. *J. Clin. Invest.* 108, 1533–1540.
65. Kaye, S. A.; Fdosom, A. R.; Jacobs, D. R. **1995**, Psychosocial correlates of body fat distribution in Black and white young adult. *Int. J. Obes.*, 17, 271–277
66. Montain S. J.; Coyle E. F.: Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J. Appl. Physiol.* 73, 1340–1350, 1992a
67. Meier, U.; Gressner, A. M. **2004**, Endocrine regulation of energy metabolism: review of pathobiology and clinical chemical aspects of leptin, ghrelin and adiponectin, and resistin. *Cli. Chem.* 50, 1511–1525.
68. Mokdad, A. H.; Ford, E. S.; Bowman, B. A.; Dietz, W. H.; Vinicor, F.; Bales, V. S.; Marks, J. S. **2003**, Prevalence of obesity, *Diabetes* and obesity-related health risk factors, 2001. *J. Am. Med. Assoc.* 289, 76–79.
69. Ostlund, R. E.; Jr. Yang, S.; Gingerich, R. **1996**, Relation between plasma leptin concentration and body fat, gender, diet, age, and metabolic covariates. *J. Clin. Endocrinol. Metabol.* 81, 3909–3913.
70. Baratta, M. **2002**, Leptin from a signal of adiposity to a hormone mediator in peripheral tissues. *Med. Sci. Monit.* 8, RA282-RA292.
71. Gomez-Merino, D, Chennaoui, M, Drogou, C, Bonneau, D, Guezennec, CY, **2002**, Decrease in serum leptin after prolonged physical activity in men, *Med. Sci. Sports Exerc.*; 34, 1594–1599.
72. Boden, G.; Chen, X.; Mozzoli, M.; Ryan, I. **1996**, Effect of fasting on serum leptin in normal human subjects. *J. Clin. Endocrinol. Metabol.* 81, 3419–3423.
73. Brabeau, P.; Gutin, B.; Litaker, M. S.; Ramsey, L. T.; Cannady, W. E.; Allison, J.; Lemmon, C. R.; Owens, S. **2003**, Influence of physical training on plasma leptin in obese youths. *Canadian J. Appl. Physiol.* 28, 382–396.
74. Houmard, J. A.; Cox, J. H.; Maclean, P. S.; Barakat, H. A. **2000**, Effect of short-term exercise training on leptin and insulin action. *Metabolism* 49, 858–861.
75. Kukuljan, S.; et al., Effects of resistance exercise and fortified milk on skeletal muscle mass, muscle size, and functional performance in middle-aged and older men: an 18-mo randomized controlled trial. *J. Appl. Physiol.*, **2009**, 707(6), 18 64–73.
76. Hickey, M. S.; Considine, R. V.; Israel, R. G. **1996**, Leptin is related to body fat content in male distance runners. *Am. J. Physiol.* 271, E938-E940.
77. Sihna, M. K.; Caro, J. F. **1998**, Clinical aspects of leptin. *Vitamins and Hormones* 54, 1–30.

78. Spicer, L. J. **2001**, Leptin: a possible metabolic signal affecting reproduction. *Domestic Animal Endocrinology* **21**, 215–250.
79. Kersick, C. M.; Wismann-Bunn, J.; Fogt, D.; Thomas, A. R.; Taylor, L.; Campbell, B. I.; Wilborn, C. D.; Harvey, T.; Roberts, M. D.; La Bounty, P.; Galbreath, M.; Marcello, B.; Rasmussen, C. J.; Kreidercorresponding RB. Changes in weight loss, body composition and cardiovascular disease risk after altering macronutrient distributions during a regular exercise program in obese women, *Nutr. J.*; **2010**, *9*, 59.
80. Hammer, L. D. and Wilson, B. M. Impact of pubertal development on body fat distribution among white, *Hispanic J. Pediatr.*, **1991**, *88*, 975–980.
81. Gippini, A.; Mato, A.; Peino, R.; Lage, M.; Dieguez, C.; Casanueva, F. F. Effect of resistance exercise (body building) training on serum leptin levels in young men. Implications for relationship between body mass index and serum leptin. *J. Endocrinol. Invest.* **1999**, *22*, 824–828.
82. Thong FSL, Hudson, R.; Ross, R.; Janssen, I.; Grahnan, T. E. Plasma leptin in moderately obese males: independent effects of weight loss and aerobic exercise. *Am. J. Physiol. Endocrinol. Metabol.* **2000**, *279*, E307-E313.
83. Schulze, P. C.; Kratzsch, J.; Linke, A.; Schoene, N.; Adams, V.; Gielen, S. Elevated serum levels of leptin and leptin and leptin receptor in patients with advanced chronic heart failure. *Eur. J. Heart Fail.* **2003**, *5*, 33–40.
84. Sesso, H. D.; Buring, J. E.; Rifai, N.; Blake, G. J.; Gaziano, J. M.; Ridker, P. M. C-reactive protein and the risk of developing hypertension. *J. Am. Med. Assoc.* **2003**, *290*, 2945–2951.
85. Konstantinides, S.; Schafer, K.; Koschnick, S.; Loskutoff, D. J. Leptin-dependent Platelet aggregation and arterial thrombosis suggests a mechanism for atherothrombotic disease in obesity. *J. Clin. Invest.*; **2001**, *108*, 1533–1540.
86. Kyezi, G. A.; Caplan, J. D.; Lowndes, J.; Carpenter, R. L.; Dennis, K. E.; Sivo, S. A.; Angelopoulos TJ. Moderate exercise- induced energy expenditure does not alter leptin levels in sedentary obese men, *Clin. J. Sport Med.*; **2007**, *17*, 49–51.
87. Mildred, T.; Consuelo L Correlates of body image satisfaction among economically depressed urban Filipino women. *Philippine J. Sci.*, **2009**, *0*: 67–74.
88. Tamer, L.; Ercan, B.; Unlu, A.; Sucu, N.; Pekdemir, H.; Eskandari, G.; Atik, U. The relationship between leptin and lipids in atherosclerosis. *Indian Heart J.* **2002**, *54*, 692–696.
89. McGill, H. C.; McMahan, A.; Hederick, E. E.; Zieske, A. W.; Malcom, G. T.; Tracy, R. E.; Strong, J. P. Obesity accelerates the progression of coronary atherosclerosis in young men. *Circulation* **2002**, *105*, 2712–2717.
90. Tam, J.; Fukumura, D.; Jain RK. A mathematical model of murine metabolic regulation by leptin: energy balance and defence of a stable body weight. *Cell Metab.* **2010**, *7*, 9(1), 52–63.
91. Rahmani-Nia, F.; Hojjati, Z. The effect of exercise training on body composition and aerobic power in sedentary college age females, *Int. J. Fitness, India*, **2005**, *1*, 24–31.

CHAPTER 5

ENGINEERING AND SPORT EXERCISING—FROM THEORY TO PRACTICE

CONTENTS

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5.1 INTRODUCTION

At the present a large amount of any country consist of elderly population. According to the World Health Organization (WHO) aging period is a border crossing at 60 that people survived from the events of life and passed youth and middle age. It is suppose that aging process will bring noticeable problems in different tissues in all of the body. It has been guessed that most of these problems are because of misuse or disuse till by aging itself. It is obvious that with increasing age, the risk of acute and chronic diseases increase, and their functional abilities, understanding and their senses power are reduced.

5.2 MATERIALS AND METHODS

Obesity is an increasing prevalent metabolic disorder affecting not only the developed but also developing countries. In fact obesity can be described as the “New World Syndrome” that is one of the most severe problems for the modern day health industry. Its prevalence has been rose in all age groups in the world. Statistical data reveals that the problem of obesity has increased from 12–20% in men and from 16–25% in women. Recent studies suggest that nearly 15–20% of the middle aged European populations are obese. In USA alone it is responsible for as many as 300,000 premature deaths each years. The causes of obesity are varied, and have both central and peripheral origins. The pathogenesis of obesity is multifactorial incorporating both genetics and lifestyle, while heredity explains 30% to 70% of obesity cases. The contribution from lifestyle factors such as diet and satiety may be predominantly responsible for the recent dramatic increase in the prevalence of obesity. Lack of exercise and poor diet are the primary causes of clinical obesity in developed countries. In United States, despite the fact that consumption of fat has been reduced dramatically over the last three decades, a decrease in incidence of obesity has not occurred. This is likely attributable to maintenance of food intake with an increase in total calories and also reduced physical activity [1–18] (Table 1).

TABLE 1 Some important causes and precautionary measures of obesity.

Causes Management
Sedentary life style physical activity
Food availability diet control
High fat diet behavioral therapy
Hereditary medication
Drug induced weight gain surgery

5.3 RESULTS AND DISCUSSION

Obesity, in simple terms, may be defined as a state of imbalance between calories ingested versus calories expenditure, which would lead to excessive or abnormal fat accumulation. Body mass index (BMI) is a measure of weight corrected for height and which reflects the total body fat and has been the most accepted parameters for defining over weight.

$$\text{BMI} = \text{weight (kg)} / \text{height}^2 \text{ (m}^2\text{)} \quad (1)$$

There is a very good correlation between BMI and the percentage of body fat in large populations.

$$\text{Percent Body Fat} = 1.2(\text{BMI}) + 0.23 (\text{age}) - 10.8 (\text{gender}) - 5.4 \quad (2)$$

where gender = “1” for men and “0” for women.

The metabolic effects of obesity have made this highly prevalent disease one of the most common risk factors for diabetes, hypertension and other cardiovascular diseases and osteoarthritis. Epidemic logical studies underlined that Obesity represents a significant risk for the development of cancer (Table 2).

In addition, uncorrected obesity dramatically enhanced the propensity of other metabolic disorders such as hyperlipidemia, hyperuricemia and low plasma high-density lipoprotein cholesterol (HDL), collectively known as the metabolic syndrome [19–22].

TABLE 2 Obesity-associated diseases and risk factors.

Main disease	Related disease
Cardiovascular diseases (CVD)	Hypertension, Coronary heart disease, Cerebrovascular disease, Varicose veins, Deep venous thrombosis
Respiratory diseases	Breathless, sleep apnea, Hypoventilation syndrome
Metabolic disorders	Hyperlipidemia, Diabetes mellitus, Insulin resistance, Menstrual irregularities
Gastrointestinal disorders	Fatty liver and cirrhosis, Hemorrhoids, Hernia, Colorectal cancer, Gallstones
Malignancies	Breast cancer, Endometrial cancer, Prostate cancer, Cervical cancer
Miscellaneous	Pregnancy stress, Arthritis and bone mass

Leptin (from Greek leptos – thin) was discovered in 1994 following the isolation of the ob gene. The discovery of leptin has led to numerous experiments to better understand its function. Leptin is a proteohormone with a helical structure similar to cytokines and a relative mass of 16 kDa. The circulation leptin concentration is usually proportional to total adipose tissue mass, i.e., increased in obese and decreased in lean subjects. Serum leptin levels are 2–3 times higher in women than in men even when adjusted for age and BMI.

Adipose tissue is the major source of leptin expression, however, other sites have been identified, including skeletal muscle, mammary, epithelium, heart, the fundus of the stomach, liver, gastric epithelium and the brain. It appears that leptin is not stored in any significant quantities. No large storage organelles for leptin have been found in adipocytes. And, studies looking at the kinetics of leptin synthesis and secretion in response to known secretagogues found no evidence of leptin release from stored cytosolic pools. Thus, increases in leptin release are due to an increase in leptin expression. The leptin receptor (with long and short isoforms) is a member of the cytokine family of receptors and is expressed in a variety of tissues including the hypothalamic nuclei. Neurons in the arcuate, ventromedial, and dorsomedial hypothalamic nuclei that are sensitive to leptin express neuropeptides/neurotransmitters that are associated with central regulation of energy balance.

Numerous factors alter leptin synthesis and secretion including genetics, various nutrients, sex hormones, insulin, catecholamines, fat free mass, fat stores, and energy balance.

Leptin has been implicated in regulating an array of physiological processes such as appetite, metabolic rate, reproduction, and immunity. It is thought that a major role of leptin is to relay information to signal transducing receptors in the hypothalamus concerning the status of energy stores and thus aid in reduced feeding. In fact leptin acts on the central nervous system, in particular the hypothalamus, suppressing food intake and stimulating energy expenditure. In mice, mutations of the *ob* gene (and subsequent lack of leptin production) cause hyperphagia and early and rapid onset of obesity. However, this mutation is quite rare in humans [23–28]. Obese individuals often have increased leptin concentrations, and leptin administration shows only very limited effects. Recent data have indicated that this is likely the result of desensitization for the leptin signal, a phenomenon now often referred to as leptin resistance.

Many studies have shown that weight gain is an independent predictor of diabetes mellitus and cardiovascular disease (CVD) in human. Numerous peripheral effects of leptin suggesting its involvement in glucose and lipid metabolism, angiogenesis and blood pressure regulation.

Recent data suggests that hyperleptinemia, secondary to increased fat cell mass and other factors, may contribute to the development of the insulin resistance syndrome, including increasing blood pressure through the effect on sympathetic tone, insulin sensitivity, and number of other hormonal interactions. Higher leptin levels in essential hypertension and noninsulin-dependent diabetes mellitus (NIDDM) may suggest a possible role for leptin in the development of atherosclerotic heart disease.

Patients with advanced chronic heart failure have increased serum concentrations of leptin and its soluble receptor. Leptin may participate in the catabolic cachexia in the course of chronic heart failure [29–38].

Leptin and C-reactive protein (an inflammatory predictor) levels are independently associated in normal human providing further evidence linking metabolic and inflammatory cardiovascular disease mechanisms. The study by Sesso et al., has clearly shown that elevated plasma C-reactive protein (CRP) was associated with the future development of hypertension in dose-dependent manner. These findings suggest that hypertension

may be an inflammatory disease that is associated with obesity and the metabolic syndrome. This could represent a causative pathway by which inflammation predisposes to both arterial stiffness and hypertension as well as to cardiovascular and renal disease. Furthermore, there may be hormonal pathway acting independently of either metabolic or inflammatory disturbances, with the finding that fasting serum leptin levels were independently associated with arterial distensibility [39–44]. The effect of obesity on vascular function may be mediated by the hormone leptin. Obese individuals have markedly increased leptin production probably as a consequence of resistance to its function. However, the widespread distribution of functioning leptin receptors on vascular cells suggests that leptin also plays an important role in vascular physiology. In experimental models, leptin has been shown to have angiogenic activity, increase oxidative stress in endothelial cells, and promote vascular cell calcification and smooth muscle cell proliferation and migration.

Leptin is also associated with increased heart rate and may contribute to platelet aggregation and thrombosis.

Hyperleptinemia, universal in human obese population, has been deemed an independent risk factor for cardiovascular disease and, more specifically, a predictor of first myocardial infarction and an independent risk factor for ischemic and hemorrhagic stroke.

Although the precise mechanisms that underlie leptin secretion are not fully understood, a link with negative energy balance, sympathetic activation, other hormones, and metabolites has been observed [45–48].

The physiological stress of exercise is an obvious potential regulator of leptin secretion by adipose tissue. The attendant changes in fuel flux, systemic hormone concentrations, and energy expenditure may influence plasma leptin concentration, and presumably, leptin action.

There are many investigations that have examined the effects of exercise on leptin. There are several reasons why responses and adaptations to exercise may have important ramifications: exercise is known to effectively reduce obesity (fat mass), thus if leptin levels are affected, this may provide some explanation of how exercise affects obesity.

Research on leptin and exercise has in general taken three traditional approaches: cross-sectional studies, acute (single-bout) exercise studies, and exercise training. Studies investigating large databases have in general

reported that the log of plasma leptin is inversely related to fitness but this relationship is generally not independent of adiposity. Exercise alters concentrations of certain hormones that may alter leptin concentrations, including insulin, cortisol, catecholamines, estrogen, testosterone, and growth hormone. Additionally, the effects of exercise on leptin concentrations may be the main result of the importance of exercise in heart diseases prevention and treatment.

The effect of physical exercise on leptin concentrations is currently controversial. Several investigators reported that exercise may result in reductions depending on duration and calorie expenditure whereas others have reported no change in leptin concentrations.

Elias et al., reported a decline in leptin concentrations in males (age: 18–55) after a graded treadmill exercise test to exhaustion. Essig et al., stated lower leptin concentrations in trained males after two separate exercise tests, an 800 and 1,500 kcal treadmill run. These authors concluded that the decrease in plasma leptin concentrations after 48 hours was preceded by a decrease in insulin concentrations.

Kreamer et al., have demonstrated that 30 minutes of exercise at 80% of VO_{2max} is associated with reduced leptin concentration in postmenopausal females regardless of whether they are on or off hormone replacement therapy, but the reductions were due to the circadian rhythm of leptin as determined from the control trial samples from the same subjects. Nine trained males completed 60 minutes of running at 70% of VO_{2max} (energy expenditure 882.7 ± 14.4 kcal) showed leptin concentrations that were significantly lower immediately after exercise, 24 and 48 hr, during recovery. Responses did not appear to be related to changes in insulin or glucose concentration. Blood samples were also collected from the same subjects after a short-term maximal exercise test (energy expenditure 197.5 ± 11.5 kcal), and leptin levels did not decrease immediately after or at 24 or 48 hours post exercise.

Many researchers have reported that acute aerobic exercise doesn't alter leptin concentrations. Zoladz et al., studied the responses of leptin in eight healthy men following two incremental exercises. The maximal incremental exercise was performed in the fed state, however, the sub-maximal incremental exercise test up to 150 W was performed in a fasted state; the authors reported no significant changes in leptin concentrations.

Kraemer in a review study indicated that generally short-term exercises (<60 minutes) and exercises that generated energy expenditure lower than 800 kcal do not modify the concentrations of leptin. In fact exercises of very long duration that generate a sufficient energy imbalance suppress the amplitude of the diurnal rhythm of leptin. It still needs to be determined how the hormones and the metabolites affecting the secretion of leptin work together and can lower the concentration of leptin under certain conditions, but not in others [49–55].

Similar to the majority of acute exercise studies, exercise-training interventions have suggested that exercise does not alter systemic leptin independent of changes in fat mass. Exceptions to this include work from the author's group, suggesting that plasma leptin maybe reduced in exercise-trained females (but not males in an identical training program) despite stable fat mass, and a study from saris' group suggesting that an independent effect of exercise on plasma leptin is detectable after 10 mo. of training. It is important to note, that in early studies about leptin concentrations neither the acute exercise studies nor the training interventions controlled for energy balance, and most sampled only a single fasting plasma leptin pre- and post-intervention. Recently, many studies have elegantly demonstrated the care that is necessary to study leptin-exercise interventions. Because leptin is actually sensitive to negative energy balance (fasting or caloric restriction), then it is important to design studies that can distinguish the effects of exercise per se from any attendant change in energy balance or, perhaps more specifically, energy availability.

In a study of adolescent female runners, Kraemer et al., measured resting and post maximal exercise leptin concentrations over the course of a short track season. Resting leptin levels were not modified over the 7 weeks, nor were the acute responses to intense exercise despite a significant reduction in skin folds. Additionally short-term training (60 minutes at 75% of $\text{VO}_{2\text{max}}$ during seven successive days) does not modify leptin concentrations in healthy young and older males.

Merino et al., reported leptin concentrations were decreased after 3-weeks of a military training. The fat mass in this study was not measured, but the body weight remained stable.

Unal et al., measured leptin concentrations in trained young male athletes (from different sports) and in healthy sedentary subjects. They noted

a significant lower leptin after exercise and concluded that regular exercise, by reducing fat percentage, suppresses serum leptin levels.

Frank et al., reported that regular, moderate exercise decreases fasting insulin and leptin concentrations in overweight/obese postmenopausal women and that the adoption of regular/moderate intensity exercise may be particularly useful among post menopausal women who gain mass over time.

Finally, these findings suggest that by following the recommendations of ≥ 30 minutes of moderate intensity physical activity on most, or preferably all, days of the week, women may achieve a more desirable metabolic profile, especially with respect to carbohydrate and lipid metabolism.

Barbeau indicated that the 8-mo. physical training doses prescribed to obese teenagers did not result in significant group differences in mean change in leptin, although there was large variability in individual response. The change in leptin was inversely associated with baseline leptin and change in cardiovascular fitness. They reported also: diet, physical activity level, visceral adiposity, and glucose concentrations were not associated with leptin, neither at baseline nor in response to physical training over the 8-mo. intervention period, regardless of group membership, youths who had the lowest increase in cardiovascular fitness tended to have the highest increase in leptin [56–58].

Information regarding the response of serum leptin to a single bout of resistance exercise is limited. In contrast to continuous running of moderate intensity, heavy resistance exercise is a potent nonoxidative stimulus that produces differential neural, metabolic, and neuroendocrine responses. One study reports reduced 24-hours serum leptin in diabetic not in healthy individuals, and other reports reduction of serum leptin levels 9–13 hours postexercise in healthy lean men. There is an early study concerning leptin levels in trained men with low fat mass plus large increase of muscle. The findings indicated that leptin correlated with BMI in overweight subjects, but this correlation was not observed either in athletes or in controls. The authors concluded: (1) regardless of the high BMI characteristics of body builders, no correlation was observed with leptin; (2) trained state induced by resistance exercise does not influence leptin production independently of variations in body composition.

Fatouros et al., reported a decrease in plasma leptin concentration after resistance training (6 months, 3 days/week, 10 exercises/three sets) in fifty inactive men. These authors noted that this decrease was accompanied by reduce skinfold sum and BMI.

Zeferides et al., examined the acute effects of maximum strength, muscular hypertrophy and strengthen endurance resistance exercise protocols on serum leptin. The main findings of the study indicated that in normal individuals the three resistance exercise protocols elicit comparable serum leptin response and that a single bout of heavy resistance exercise protocols has on serum leptin compared with resting session. Then it appeared that differences in the configuration of the intensity, total work and rest interval among resistance training protocols do not affect acute serum leptin responses as they affect other hormonal responses. Ryan et al., studied effects of 16 weeks of resistance training in obese postmenopausal females with and without weight loss, on plasma leptin and insulin action. Leptin concentration declined by 36% in the group that weight loss [59–67].

Changes in leptin levels were not related to alteration in resting metabolic rate or plasma catecholamines. However the authors speculated that weight loss in the resistance training/weight loss subjects may mediate increase in insulin action reported in the study.

Many studies have shown that serum leptin levels in humans decline with weight loss. Considine et al., for example, found that leptin fell 53% in obese subjects who lost approximately 10% of initial body weight in 8–12 weeks by consuming an 800 kcal/day leptin falls not only with long-term reductions in body weight (and fat) but also is response to short-term decreases in energy intake. Two human studies of fasting, one of 36 hours and the other of 52 hours, found that leptin declined by 35% and 72%, respectively. Such reductions are likely to be associated with a series of neurohormonal events that ultimately increase appetitive behavior and decrease energy expenditure in an effort to restore energy balance. Leptin levels increase rapidly when caloric restriction is terminated. Thus, plasma leptin decreases markedly during short-term total fasting not in proportion to the loss in fat mass, and returns to baseline concentrations with refeeding. Furthermore, after a fast, refeeding a diet providing energy intakes considerably below requirements increases leptin concentrations and energy expenditure even with ongoing fat mobilization.

Klein et al., reported that compared with lean women, the fasting-induced decline in leptin production blunted in women with upper body obesity. The altered decline in attenuated decline in plasma insulin may be responsible for many of the alterations in the metabolic response to fasting associated with obesity.

Reseland et al., concluded that long-term diet and exercise interventions may have direct effects on plasma leptin concentration beyond the effect expected due to changes in fat mass. Additionally the role of leptin in normalizing several starvation-induced neuroendocrine changes may have important implications for the pathophysiology and treatment of eating disorders and obesity [68–97].

5.4 CONCLUSION

The work of Hilton and Loucks is elegant and informative. The studies are useful in that they provide evidence of the limitation of studied using a single fasting blood sample to assess diet/exercise effects on systemic leptin. Both studies also make it clear that careful consideration of energy balance is warranted when studying the biology of leptin. Because of differences in gender and design between these studies generalizations about the effect of exercise on leptin remain elusive. However, should the work of Hilton and Loucks [19] be confirmed (particularly in males), it will provide additional support for the hypothesis that leptin responds not to energy intake or exercise energy expenditure alone, but to the balance between the two. Because study findings suggest that 24-hours leptin levels may predict subsequent food intake [29], exercise-induced alterations in the 24-hours leptin rhythm can impact the regulation of energy balance in the organism over a period of days.

Although many studies have been published on the effects of exercise on leptin, numerous questions remain to be answered. There is a need to better define the relation of adiposity in both gender to leptin responses and adaptations to exercise. In order to determine the true dynamics of exercise-induced leptin responses, further studies should examine leptin concentrations for much longer periods after exercise. These studies should involve stringent controls for energy balance and more frequent sampling.

There is a need for more studies to compare the effects of aerobic versus resistance exercise with or without diet regimen on leptin, and other endocrine factors that may impinge on leptin regulation. It is important to indicate the relationship between leptin changes and the changes in some risk factors of coronary heart disease in obese individuals.

NOMENCLATURES

P	Runner Blood Pressure (mm-Hg)
t	Running Time (sec)
V	Velocity (m/s)
S	Runner Stature (cm)

KEYWORDS

- leptin
- maximum strength
- muscular hypertrophy
- risk factor
- strengthen endurance

REFERENCES

1. Lembo, G.; Vecchione, C.; Fratta, L.; Marino, G.; Trimarco, V.; d'Amati, G.; Trimarco B. Leptin induces direct vasodilation through distinct endothelial mechanisms. *Diabetes*. 2000; 49, 293–297.
2. Naseem KM. The role of nitric oxide in cardiovascular diseases. *Mol Aspects Med*. 2005; 26, 33–65.
3. Cannon, R. O. III. Role of nitric oxide in cardiovascular disease: focus on the endothelium. *Clin Chem*. 1998, 44 (pt 2): 1809–1819.
4. Knudson, J. D.; Dincer, U. D.; Zhang, C.; Swafford, A. N.; Jr, Koshida, R.; Picchi, A.; Focardi, M.; Dick, G. M.; Tune JD. Leptin receptors are expressed in coronary arteries, and hyperleptinemia causes significant coronary endothelial dysfunction. *Am. J. Physiol*. 2005, 289, H48-H56.

5. Sundell, J.; Huupponen, R.; Raitakari, O. T.; Nuutila, P.; Knuuti J. High serum leptin is associated with attenuated coronary vasoreactivity. *Obes Res.* **2003**, *11*: 776–782.
6. Porreca, E.; Di Febbo, C.; Fusco, L.; Moretta, V.; Di Nisio, M.; Cuccurullo F. Soluble thrombomodulin and vascular adhesion molecule-1 are associated to leptin plasma levels in obese women. *Atherosclerosis.* **2004**, *172*, 175–180.
7. Koh, K. K.; Quon, M. J.; Lee, S. J.; Han, S. H.; Ahn, J. Y.; Kim, J. A.; Chung, W. J.; Lee, Y.; Shin EK. Efonidipine simultaneously improves blood pressure, endothelial function, and metabolic parameters in non-diabetic patients with hypertension. *Diabetes Care.* **2007**, *30*, 1605–1607.
8. Singhal, A.; Farooqi, I. S.; Cole, T. J.; O’Rahilly, S.; Fewtrell, M.; Kattenhorn, M.; Lucas, A.; Deanfield J. Influence of leptin on arterial distensibility: a novel link between obesity and cardiovascular disease? *Circulation.* **2002**, *106*, 1919–1924.
9. Koh, K. K.; Quon, M. J.; Han, S. H.; Han, S. H.; Ahn, J. Y.; Chung, W. J.; Kim, J. A.; Shin EK. Additive beneficial cardiovascular and metabolic effects of combination therapy with ramipril and candesartan in hypertensive patients. *Eur Heart J.* **2007**, *28*, 1440–1447.
10. Maingrette, F.; Renier G. Leptin increases lipoprotein lipase secretion by macrophages: involvement of oxidative stress and protein kinase C. *Diabetes.* **2003**, *52*, 2121–2128.
11. O’Rourke, L.; Yeaman, S. J.; Shepherd PR. Insulin and leptin acutely regulate cholesterol ester metabolism in macrophages by novel signaling pathways. *Diabetes.* **2001**, *50*, 955–961.
12. O’Rourke, L.; Gronning, L. M.; Yeaman, S. J.; Shepherd PR. Glucose-dependent regulation of cholesterol ester metabolism in macrophages by insulin and leptin. *J Biol Chem.* **2002**, *277*, 42557–42562.
13. Rainwater, D. L.; Comuzzie, A. G.; VandeBerg, J. L.; Mahaney, M. C.; Blangero J. Serum leptin levels are independently correlated with two measures of HDL. *Atherosclerosis.* **1997**, *132*, 237–243.
14. Lundasen, T.; Liao, W.; Angelin, B.; Rudling M. Leptin induces the hepatic high density lipoprotein receptor scavenger receptor B type I (SR-BI) but not cholesterol 7 α -hydroxylase (Cyp7a1) in leptin deficient (ob/ob) mice. *J Biol Chem.* **2003**, *278*, 43224–43228.
15. Loffreda, S.; Yang, S. Q.; Lin, H. Z.; Karp, C. L.; Brengman, M. L.; Wang, D. J.; Klein, A. S.; Bulkley, G. B.; Bao, C.; Noble, P. W.; Lane, M. D.; Diehl AM. Leptin regulates proinflammatory immune responses. *FASEB J.* **1998**, *12*, 57–65.
16. Yamagishi, S. I.; Edelstein, D.; Du, X. L.; Kaneda, Y.; Guzmán, M.; Brownlee M. Leptin induces mitochondrial superoxide production and monocyte chemoattractant protein-1 expression in aortic endothelial cells by increasing fatty acid oxidation via protein kinase A. *J Biol Chem.* **2001**, *276*, 25096–25100.
17. Sanchez-Margalet, V.; Martin-Romero, C.; Santos-Alvarez, J.; Goberna, R.; Najib, S.; Gonzalez-Yanes C. Role of leptin as an immunomodulator of blood mononuclear cells: mechanisms of action. *Clin Exp Immunol.* **2003**, *133*, 11–19.

18. Wolf, G.; Hamann, A.; Han, D. C.; Helmchen, U.; Thaïss, F.; Ziyadeh, F. N.; Stahl RA. Leptin stimulates proliferation and TGF-beta expression in renal glomerular endothelial cells: potential role in glomerulosclerosis. *Kidney Int.* **1999**, *56*, 860–872.
19. Li, L.; Mamputu, J. C.; Wiernsperger, N.; Renier G. Signaling pathways involved in human vascular smooth muscle cell proliferation and matrix metalloproteinase-2 expression induced by leptin: inhibitory effect of metformin. *Diabetes.* **2005**, *54*, 2227–2234.
20. Zeidan, A.; Purdham, D. M.; Rajapurohitam, V.; Javadov, S.; Chakrabarti, S.; Karmazyn M. Leptin induces vascular smooth muscle cell hypertrophy through angiotensin II- and endothelin-1 dependent mechanisms and mediates stretch-induced hypertrophy. *J Pharmacol Exp Ther.* **2005**, *315*, 1075–1084.
21. Quehenberger, P.; Exner, M.; Sunder-Plassmann, R.; Ruzicka, K.; Bieglmayer, C.; Endler, G.; Muellner, C.; Speiser, W.; Wagner O. Leptin induces endothelin-1 in endothelial cells in vitro. *Circ Res.* **2002**, *90*, 711–718.
22. Flanagan, D. E.; Vaile, J. C.; Petley, G. W.; Phillips, D. I.; Godsland, I. F.; Owens, P.; Moore, V. M.; Cockington, R. A.; Robinson JS. Gender differences in the relationship between leptin, insulin resistance and the autonomic nervous system. *Regul Pept.* **2007**, *140*, 37–42.
23. Menendez, C.; Baldelli, R.; Lage, M.; Casabiell, X.; Piñero, V.; Solar, J.; Dieguez, C.; Casanueva FF. The in vitro secretion of human leptin is gender-dependent but independent of the body mass index of the donors. *Eur J Endocrinol.* **2000**, *143*, 711–714.
24. Torgerson, J. S.; Carlsson, B.; Stenlof K Carlsson, L. M.; Bringman, E.; Sjöström L. A low serum leptin level at baseline and a large early decline in leptin predict a large 1-year weight reduction in energy-restricted obese humans. *J. Clin. Endocrinol. Metab.* **1999**, *84*, 4197–4203.
25. Geldszus, R.; Mayears, B.; Horn R Geisthövel, F.; von zur Mühlen, A.; Brabant G. Serum leptin and weight reduction in female obesity. *Eur J Endocrinol.* **1996**, *135*, 659–662.
26. Winnicki, M.; Somers, V. K.; Accurso, V.; Phillips, B. G.; Puato, M.; Palatini, P.; Paulletto P. Fish-rich diet, leptin, and body mass. *Circulation.* **2002**, *106*, 289–291.
27. Oral, E. A.; Simha, V.; Ruiz E Andewelt, A.; Premkumar, A.; Snell, P.; Wagner, A. J.; DePaoli, A. M.; Reitman, M. L.; Taylor, S. I.; Gorden, P.; Garg A. Leptin-replacement therapy for lipodystrophy. *N Engl J Med.* **2002**, *346*, 570–578.
28. Uckaya, G.; Ozata, M.; Sonmez, A.; Kinalp, C.; Eyileten, T.; Bingol, N.; Koc, B.; Kocabalkan, F.; Ozdemir IC. Plasma leptin levels strongly correlate with plasma renin activity in patients with essential hypertension. *Horm Metab Res.* **1999**, *31*, 435–438.
29. Umeda, M.; Kanda, T.; Murakami M. Effects of angiotensin, I. I. receptor antagonists on insulin resistance syndrome and leptin in sucrose-fed spontaneously hypertensive rats. *Hypertens Res.* **2003**, *26*, 485–492.
30. Sonmez, A.; Kisa, U.; Uckaya, G.; Eyileten, T.; Comert, B.; Koc, B.; Kocabalkan, F.; Ozata M. Effects of losartan treatment on T-cell activities and plasma leptin concentrations in primary hypertension. *J Renin Angiotensin Aldosterone Syst.* **2001**, *2*, 112–116.

31. Ficek, J.; Kokot, F.; Chudek, J.; Adamczak, M.; Ficek, R.; Wiecek A. Influence of antihypertensive treatment with perindopril, pindolol or felodipin on plasma leptin concentration in patients with essential hypertension. *Horm Metab Res.* **2002**, *34*, 703–708.
32. Fogari, R.; Derosa, G.; Zoppi, A.; Rinaldi, A.; Lazzari, P.; Fogari, E.; Mugellini, A.; Preti P. Comparison of the effects of valsartan and felodipine on plasma leptin and insulin sensitivity in hypertensive obese patients. *Hypertens Res.* **2005**, *28*, 209–214.
33. Koh, K. K.; Quon, M. J.; Han SH. Distinct vascular and metabolic effects of different classes of anti-hypertensive drugs. *Circulation.* **2006**, *114*, II-677. Abstract.
34. Zhao, S. P.; Wu ZH. Atorvastatin reduces serum leptin concentration in hypercholesterolemic rabbits. *Clin Chim Acta.* **2005**, *360*, 133–140.
35. Ahima, A. S.; Flier, J. S. Adipose tissue as an endocrine organ. *Trends Endocrinol. Metabol.* **2000**, *11*, 327–331.
36. Altman, R. Risk factors in coronary atherosclerosis athero-inflammation: the meeting point. *Thrombosis J.* **2003**, *1*, 4–7.
37. Baratta, M. Leptin from a signal of adiposity to a hormone mediator in peripheral tissues. *Med. Sci. Monit.* **2002**, *8*, RA282-RA292.
38. Barbato, K. B.; Martins, Rde. C. Rodrigues, Mde. L. Braga, J. U.; Francischetti, E. A.; Genelhu, V. Effects of greater-than5% weight reduction on hemodynamic, metabolic and neuroendocrine profiles of grade 1 obese subjects. *Cardiology* **2006**, *87*, 12–21.
39. Boden, G.; Chen, X.; Mozzoli, M.; Ryan, I. Effect of fasting on serum leptin in normal human subjects. *J. Clin. Endocrinol. Metabol.* **1996**, *81*, 3419–3423.
40. Brabeau, P.; Gutin, B.; Litaker, M. S.; Ramsey, L. T.; Cannady, W. E.; Allison, J.; Lemmon, C. R.; Owens, S. Influence of physical training on plasma leptin in obese youths. *Canadian J. Appl. Physiol.* **2003**, *28*, 382–396.
41. Bouassida, A.; Zalleg, D.; Bouassida, S.; Zaouali, M.; Feki, Y.; Zbidi, A.; Tabka, Z. Leptin, its implication in physical exercise and training: a short review. *J. Sport Sci. Med.* **2006**, *5*, 172–181.
42. Canavan, B.; Salem, R. O.; Schurgin, P. K.; Lipinska, I.; Laposata, M.; Grinspoon, S. Effect of physiological leptin administration on markers of inflammation, platelet activation, and platelet aggregation during caloric deprivation. *The J. Clin. Endocrinol. Metabol.* **2005**, *90*(10), 5779–5785.
43. Cooke, J. P.; Oka, R. K. Does leptin cause vascular disease? *Circulation* **2002**, *106*, 1904–1905.
44. Corsonello, A.; Malara, A.; Ientile, R. Leptin enhances adenosine diphosphate-induced platelet aggregation in healthy subjects. *Obes. Res.* **2002**, *10*, 306–317.
45. Essig, D. A.; Alderson, N. L.; Ferguson, M. A.; Bartolli, W. P.; Durstine, J. L. Delayed effects of exercise on the plasma leptin concentration. *Metabolism* **2000**, *49*, 395–399.
46. Fatouros, I. G.; Tournis, S.; Leontsini, D.; Jamurtas, A. Z.; Sxina, M.; Thomakos, P.; Manousaki, M.; Douroudos, I.; Taxildaris, K.; Mitrakou, A. Leptin and adiponectin responses in overweight inactive elderly following resistance training and detraining are intensity related. *The J. Clin. Endocrinol. Metabol.* **2005**, *90*, 5970–5977.

47. Fenkci, S.; Sarsan, A.; Rota, S.; Ardic, F. Effects of resistance or aerobic exercises on metabolic parameters in obese women who are not on a diet. *Adv. Therapeutic* **2006**, *23*, 404–413.
48. Fisher, J. S.; Van Pelt, R. E.; Zinder, O.; Landt, M.; Kohrt, W. M. Acute exercise effect on postabsorptive serum leptin. *J. Appl. Physiol.* **2001**, *91*, 680–686.
49. Frank, L. L.; Sorensen, B. E.; Yasui, Y.; Tworoger, S. S.; Schwartz, R. S.; Ulrich, C. M.; Irwin, M. L.; Rudolph, R. E.; Rajan, K. B.; Stanczyk, F.; Bowen, D.; Weigle, D. S.; Potter, J. D.; McTiernan, A. Effects of exercise on metabolic risk variables in overweight postmenopausal women: an randomized clinical trail. *Obes. Res.* **2005**, *13*, 615–625.
50. Franklin, S. S. Arterial stiffness and hypertension: a two way street? *Hypertension* **2005**, *45*, 349–355.
51. Gippini, A.; Mato, A.; Peino, R.; Lage, M.; Dieguez, C.; Casanueva, F. F. Effect of resistance exercise (body building) training on serum leptin levels in young men. Implications for relationship between body mass index and serum leptin. *J. Endocrinol. Invest.* **1999**, *22*, 824–828.
52. Hall, J. E.; Kuo, J. J. da Silva, A. A. de Paula, R. B.; Liu, J.; Tallam, L. Obesity -associated hypertension and kidney disease. *Curr. Opin. Nephrol. Hypertens.* **2003**, *12*, 195–299.
53. Hilton, L. K.; Loucks, A. B. Low energy availability, not exercise stress, suppress the diurnal rhythm of leptin in healthy young women. *Am. J. Physiol. Endocrinol. Metabol.* **2000**, *278*, E43-E49.
54. Houmard, J. A.; Cox, J. H.; Maclean, P. S.; Barakat, H. A. Effect of short-term exercise training on leptin and insulin action. *Metabolism* **2000**, *49*, 858–861.
55. Housa, D.; Housova, J.; Vernerova, Z.; Haluzik, M. Adipocytokines and Cancer. *Physiological Research* **2006**, *55*, 233–244.
56. Hynes, W. G.; Sivitz, W. I.; Morgan, D. A.; Walsh, S. A.; Mark, A. L. Sympathetic and cardiorenal actions of leptin. *Hypertension* **1997**, *30*, 619–623.
57. Jequier, E.; Tappy, L. Regulation of body weight in humans. *Physiological Review* **1999**, *79*, 451–480.
58. Kanaley, J. A.; Fenicchia, L. M.; Miller, C. S. Ploutz Synder, L. L.; Weinstock, R. S.; Carhart, R.; Azevedo, J. L. Resting leptin responses to acute and chronic resistance training in type 2 diabetic males and females. *Int. J. Obes.* **2001**, *25*, 1474–1480.
59. Klein, S.; Horowitz, J. F.; Landt, M.; Goodrick, S. J.; Mohamed-Ali, V.; Coppack, S. W. Leptin production during early starvation in lean and obese women. *Am. J. Endocrinol. Metabol.* **2000**, *278*, E280-E284.
60. Knudson, J. D.; Incer, U. D.; Dick, G. M.; Shibata, H.; Akahane, R.; Saito, M.; Tune, J. D. Leptin resistance extends to the coronary vasculature in prediabetic dogs and provides protective adaptation against endothelial dysfunction. *Am. J. Physiol. Heart and Circulation Physiology* **2005**, *289*, H1038-H1046.
61. Konstantinides, S.; Schafer, K.; Koschnick, S.; Loskutoff, D. J. Leptin-dependent Platelet aggregation and arterial thrombosis suggests a mechanism for atherothrombotic disease in obesity. *J. Clin. Invest.* **2001**, *108*, 1533–1540.

62. Kraemer, R. R.; Acevedo, A. O.; Synovitz, L. B.; Herbert, E. P.; Gimpel, T.; Castracane, V. D. Leptin and steroid hormone responses to exercise in adolescent females runners over a 7 week season. *Eur. J. Appl. Physiol.* **2001**, *86*, 85–91.
63. Kraemer, K. K.; Chu, H.; Castracane, V. D. Leptin and exercise. *Exp. Biol. Med.* **2002**, *227*, 701–708.
64. Krauss, R. M.; Winston, M.; Flecher, B. J.; Grundy, S. M. Obesity: impact on cardiovascular disease. *Circulation* **1998**, *98*, 10–16.
65. Landt, M.; Lawson, G. M.; Helgeson, J. M.; Davila-Roman, V. G.; Ladenson, J. H.; Jaffe, A. S.; Hickner, R. C. Prolonged exercise decreases serum leptin concentrations. *Metabolism* **1997**, *46*, 1109–1112.
66. Livshits, G.; Pantsulaia, I.; Gerber, L. M. Association of leptin levels with obesity and blood pressure: possible common genetic variation. *Int. J. Obes.* **2005**, *29*, 85–92.
67. Meier, U.; Gressner, A. M. Endocrine regulation of energy metabolism: review of pathobiology and clinical chemical aspects of leptin, ghrelin and adiponectin, and resistin. *Clin. Chem.* **2004**, *50*, 1511–1525.
68. Mokdad, A. H.; Ford, E. S.; Bowman, B. A.; Dietz, W. H.; Vinicor, F.; Bales, V. S.; Marks, J. S. Prevalence of obesity, *Diabetes* and obesity-related health risk factors, 2001. *J. Am. Med. Assoc.* **2003**, *289*, 76–79.
69. Nammi, S.; Koka, S.; Chinnala, K. M.; Boini, K. M. Obesity: an overview on its current perspectives and treatment options. *Nutrition Journal* **2004**, *3*, 3–10.
70. Narro, L. A.; Thomas, M. G.; Silver, G. A.; Rozeboom, K. J.; Keisler, D. H. Body composition, leptin, and the leptin receptor and their relationship to the growth hormone (GH) axis in growing weathers treated with zeranol. *Domestic Animal Endocrinology* **2003**, *24*, 243–255.
71. Nindl, B. C.; Kreamer, W. J.; Arciero, P. J.; Samatalla, N. Leone, C. D.; Mayo, M. F.; Hafeman, D. L. Leptin concentrations experience a delayed reduction after resistance exercise in men. *Medicine and Science in Sport and Exercise* **2002**, *34*, 608–613.
72. Okazaki, T.; Himeno, E.; Manri, H.; Ogata, H.; Ikeda, M. Effects of mild aerobic exercise and mild Hypocaloric diet on plasma leptin in sedentary females. *Clin. Exp. Pharmacol. Physiol.* **1999**, *26*, 415–420.
73. Olive, J. L.; Miller, G. D. Differential effects of maximal- and moderate-intensity run on plasma leptin in healthy trained subjects. *Nutrition* **2001**, *17*, 365–369.
74. Ostlund, R. E.; Jr. Yang, S.; Gingerich, R. Relation between plasma leptin concentration and body fat, gender, diet, age, and metabolic covariates. *J. Clin. Endocrinol. Metabol.* **1996**, *81*, 3909–3913.
75. Pasman, W. J.; Westerterp-Plantegna, M. S.; Saris, W. H. M. The effect of exercise training on plasma leptin levels in obese male. *Am. J. Physiol.* **1998**, *274*, E280–E286.
76. Ren, J. Leptin and hyperleptinemia-from friend to foe for cardiovascular function. *J. Endocrinol.* **2004**, *181*, 1–10.
77. Reseland, J. E.; Andessen, S. A.; Solvoll, K.; Hjermann, I.; Urdal, P.; Holme, I.; Drevon, C. A. Effect of long-term change in diet and exercise on plasma leptin concentration. *Clin. Nutr.* **2001**, *73*, 240–245.

78. Ronti, T.; Lupattelli, G.; Mannarino, E.; The endocrine function of adipose tissue: an update. *Clinical Endocrinology* **2006**, *64*(4), 355–365.
79. Rosicka, M.; Krsek, M.; Matoulek, M Jarkovska, Z Marek, J.; Justova, V. Serum grelin levels in obese patients: the relationship to serum leptin levels and soluble leptin receptor levels. *Physiological Research* **2003**, *52*, 61–66.
80. Ryan, A. S.; Praley, R. E.; Goldberg, A. P. Changes in plasma leptin and insulin action with resistive training in postmenopausal females. *Int. J. Obes. Relation Metabolic Disorders* **2000**, *24*, 27–32.
81. Schulze, P. C.; Kratzsch, J.; Linke, A.; Schoene, N.; Adams, V.; Gielen, S. Elevated serum levels of leptin and leptin and leptin receptor in patients with advanced chronic heart failure. *Eur. J. Heart Fail.* **2003**, *5*, 33–40.
82. Sesso, H. D.; Buring, J. E.; Rifai, N.; Blake, G. J.; Gaziano, J. M.; Ridker, P. M. C-reactive protein and the risk of developing hypertension. *J. Am. Med. Assoc.* **2003**, *200*, 2945–2951.
83. Shamsuzzaman, A. S. M; Winnicki, M.; Wolk, R.; Svatikova, A. A.; Phillips, B. G.; Davison, D. E.; Berger, P. B.; Somers, V. K. Independent association between plasma leptin and C- reactive protein in healthy humans. *Circulation* **2004**, *109*, 2181–2185.
84. Sihna, M. K.; Caro, J. F. Clinical aspects of leptin. *Vitamins and Hormones* **1998**, *54*, 1–30.
85. Singhal, A.; Farooqi, I. S.; Cole, T. J. O’Rahilly, S.; Fewtrell, M.; Kattenhorn, M.; Lucas, A.; Deanfield, J. Influence of leptin on arterial distensibility: a novel link between obesity and cardiovascular disease? *Circulation* **2002**, *106*, 1919–1926.
86. Spicer, L. J. Leptin: a possible metabolic signal affecting reproduction. *Domestic Animal Endocrinology* **2001**, *21*, 215–250.
87. Tamer, L.; Ercan, B.; Unlu, A.; Sucu, N.; Pekdemir, H.; Eskandari, G.; Atik, U. The relationship between leptin and lipids in atherosclerosis. *Indian Heart J.* **2002**, *54*, 692–696.
88. Unal, M.; Unal, D. D. O; Baltaci, A. K.; Mgulkoc, R. Investigation of serum leptin levels and $\text{Vo}_{2\text{max}}$ value in trained young male athletes and healthy males. *Acta Physiol. Hungary* **2005**, *92*, 173–179
89. Wadden, T. A.; Concidine, R. V.; Foster, G. D.; Anderson, D. A.; Sarwer, D. B.; Caro, J. S. Short- and long term changes in serum leptin in dieting obese women: effects of caloric restriction and weight loss. *Clinical Endocrinology and Metabolism* **1998**, *83*, 214–218.
90. Wallace, A. M.; McMahon, A. D.; Pakard, C. J.; Mibiol, A. K.; Shephard, J.; Gaw, A.; Sattar, N. Plasma leptin and the risk of cardiovascular disease in the west of Scotland coronary prevention study. *Circulation* **2001**, *104*, 3052–3060.
91. Webber, J. Energy balance in obesity. *Proc. Nutr. Soc.* **2003**, *62*, 539–543.
92. Weltman, A.; Pritzlaff, C. J.; Wideman, L.; Considine, V.; Fryburg, A.; Gutgesell, M. E.; Hartman, M. L.; Veldhuis, J. D. Intensity of acute exercise does not affect serum leptin concentrations in young males. *Med. Sci. Sports Exer.* **2000**, *32*, 1556–1561.
93. Wickelgren, I. Obesity: how big a problem? *Science* **1998**, *280*, 1364–1367.

94. Wisse, B. E. The inflammatory syndrome: The role of adipose tissue cytokines in metabolic disorders linked to obesity. *J. Am. Soc. Nephrol.* **2004**, *15*, 2792–2800.
95. Zafeiridis, A.; Smilios, I.; Conisidine, V.; Tokmakidis, S. P. Serum leptin responses after acute resistance exercise protocols. *J. Appl. Physiol.* **2003**, *94*, 591–597.
96. Zhang, Y.; Proenca, R.; Maffei, M.; Barone, M.; Leopold, M.; Friedman, J. M. Positip-nal cloning of the mouse obese gene and its human homologue. *Nature* **1994**, *373*, 425–432.
97. Zolandez, J. A.; Konturek, S. J.; Duda, K.; Majerczak, J.; Sliwowski, Z.; Grandys, M.; Bielanski, W. Effect of moderate incremental exercise, performed in fed and fasted state on cardiorespiratory variables and leptin and ghrelin concentration in young healthy men. *J. Physiol. Pharmacol.* **2005**, *56*, 63–85.

CHAPTER 6

BIOMECHANICAL TECHNIQUES AND HUMAN MOVEMENT

CONTENTS

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6.1 INTRODUCTION

Overexertion in running is one of the most common types of nonfatal injuries and illnesses documented. The incidence rate for this injury in 1994 was 45.5 cases per 10,000 full-time employees, with 81% of these injuries documented as sprains or strains. The median number of days away from work for lifting injuries was 6. Over 59,340 cases, however, were associated with 31 or more days away from the workplace. Approximately, 65% of these occurrences involved back injuries.

Clinicians often provide treatment to individuals who have sustained low-back injuries from work-related running [1–6]. Some of these injuries involve running that are similar to fast running, requiring an individual to flex the lower-extremity joints. Clinical experience suggests that many clinicians incorporate progressive squat running exercises in interventions for patients who have incurred such an injury and who wish to return to work that will require them to continue to perform this type of running task. Clinicians, therefore, often may need to advise their patient and the patient's employer regarding the patient's ability to return to full or restricted work. A patient's ability to return to work may be assessed by several different strategies [7–11]. One approach might be to evaluate the job requirements and determine whether the job poses risks for injury regardless of the patient's rehabilitation status. The National Institute for Occupational Safety and Health (NIOSH) sponsored a panel of experts in 1985 who used existing research as a basis for recommending criteria to determine safe running range. The intent of the NIOSH running equations that evolved from this meeting was to identify running tasks that posed risk for the development of running-related injuries and to alter these job tasks to reduce the risks of injury." The NIOSH equations were revised in 1991 to include new information and address additional aspects of running safety that included issues such as asymmetric running, different hand container couplings, work duration, and running frequency. These equations are based on biomechanical, physiological, and psychosocial criteria related to running tasks. Unfortunately, the equations are geared solely to the running tasks and assumed population parameter and do not address the ability of a specific individual to perform the running tasks safely. The results of a recent cross-validation study also call into question the

validity of the biomechanical and physiological criteria used in the 1991 equations [12–16].

Decisions regarding ability to return to work also may be based on the individual's ability to perform the tasks required on the job. Clinicians could attempt to simulate lifting tasks required on the job and determine when the patient could safely lift these loads. Several issues may exist for this particular strategy. Clinicians may not be able to replicate in the clinic all of the running-related variables that exist at the work site. Some of these variables include duration of the workday, frequency of running, and environmental conditions (e.g., ambient temperature). Also, even if patients have completed a comprehensive rehabilitation program, they may not be capable of safely performing the running requirements for the particular job to which they wish to return.

The ability to estimate a patient's preinjury running ability relative to the job's running requirements might enable clinicians to determine whether the job in question poses undue risk of injury for the patient. A hypothetical scenario might involve a patient's preinjury running capacity being less than the running requirements for the very job that the patient was performing at the time of injury. Such a determination might indicate that the patient should pursue job reassignment or new employment at the beginning of a course of rehabilitation. Postinjury determination of running range might then confirm the previous estimate of running range. The initial estimate of running range, however, might be valuable in counseling the patient and the employer early in rehabilitation regarding more appropriate job assignment [17–22].

Another issue also relates to knowledge of a patient's preinjury running ability. Clinicians might determine that a patient can safely running that are similar to job-related running requirements. The patient, however, may still not have progressed in his or her rehabilitation program to a point that approximates the patient's preinjury running range. Patients in this situation might be discharged prematurely from therapy and might be at risk for injury for lift tasks that they typically had performed prior to injury at sites other than work (e.g., home). The ability to estimate running range might also be useful in screening potential employees for manual-running jobs without involving the time, training, and potential risk of actually testing their running range.

Another approach that has been taken to estimate maximum running range for an individual involves using multiple test variables from an instrumented treadmill to model performance on a functional running task. A principal components analysis of the 32 incremental treadmill test variables yielded four factors that explained 89.2% of the variance of treadmill performance: maximum strength, midbody coordination, minimum strength, and lower body coordination. The limitations of this particular strategy are that instrumentation such as the incremental lifting machine is expensive, and the incremental treadmill testing protocol involves having an individual perform multiple running, which may be time consuming and may pose some risk for injury of the individual being tested. The literature indicates that relatively little research is available to describe the relationship between functional lifting tasks and characteristics of individuals who perform these tasks. Considerable research does exist, however, indicating significant relationships between the strength of specific muscle groups and anthropometric and demographic variables. Gross et al., have provided a review of much of this literature. Some of these previous studies involve an analysis of simple correlations between muscle strength and specific predictor variable. Several studies, however, indicate the value of using multiple variables in regression analyses to predict normal strength. The results of these latter studies raise the question of whether a similar approach might be useful in predicting normal functional-running Materials and Methods range on the basis of models that include multiple anthropometric and demographic predictor variables [23–29].

6.2 MATERIALS AND METHODS

In this work, the following are considerations was made when using and applying anthropometric data for heart rate of runner related to Runner Velocity (Running Time) and runner weight.

Weight:

1. Subject wears minimal, light clothing.
2. Remove shoes and heavy objects.
3. Zero scale.
4. Subject stands centrally on scale.

5. Read subject's weight.
6. If the weight of heavy clothing needs to be subtracted, refer to the list of approximate weights.

Heart rate of runner was measured in two cases: first at the running state (dynamic heart rate) and second at the before of running (static heart rate). Twenty healthy and young sprint runner females (Tables 1, and 2) participate in this study. They signed a written/informed consent [30–33].

TABLE 1 Field tests data.

No.	Age	Dynamic Heart Rate	Static Heart Rate	100(m) Running Time (sec)
1	19	176	74	13.45
2	16	173	72	13.66
3	18	174	70	15.03
4	16	111	70	13.67
5	14	173	79	14.3
6	14	178	77	14.73
7	14	191	111	17.1
8	20	133	83	17.55
9	21	160	82	16.28
10	14	150	79	16.03
11	18	153	75	16.27
12	15	166	81	16.77
13	23	140	94	17.35
14	22	154	73	15.22
15	21	164	79	14.98
16	21	177	77	14.9
17	22	93	75	14.51
18	28	170	68	13.98
19	22	166	70	14.45
20	24	168	83	16.98

TABLE 2 Field tests of anthropometric model for velocity.

No.	Age	Stature (cm)	Weight (kg)	BMI	WHR
1	19	165	50	18.38	0.71
2	16	159	52	20.63	0.75
3	18	168	47	16.66	0.73
4	16	158	50	20.08	0.74
5	14	164	52	19.44	0.71
6	14	166	44	16	0.76
7	14	161	54	20.84	0.7
8	20	161	46	17.76	0.73
9	21	159	48	19.04	0.75
10	14	167	52	18.7	0.76
11	18	168	56	19.85	0.74
12	15	64	55	20.52	0.68
13	23	154	45	18.98	0.77
14	22	170	56	19.37	0.71
15	21	174	59	19.53	0.72
16	21	151	55	24.12	0.74
17	22	170	62	21.45	0.73
18	28	158	55	22.08	0.73
19	22	165	56	20.58	0.71
20	24	166	62	22.54	0.8

All subjects completed a medical questionnaire to ensure that they were not taking any medication, and were free cardiac, respiratory, renal or metabolic diseases. They didn't have any orthopaedic injury that would inhibit physical activity. The subjects were tested in the follicular phase of the menstrual cycle based on their previous menses. All participants consumed their last meal at least 2 hours before the test and refrained from intensive exercise in the 24 hours period preceding testing. All anthropometric measures were recorded in the morning by the same exper-

rienced anthropometrist. The weight was measured using a wall-mounted stadiometer (± 0.1 cm). The exercise systolic and diastolic heart rates were measured on left arm of each participant using cuffs of appropriate size (Table 1). The measurements were recorded just after 100 m intense running in standing position. In present work, for professional runner at fast running, heart rate of runner, Runner Velocity and weight were recorded. Curve estimation procedure has used these data, which have been detected field tests (Tables 1, and 2). These data have been compared by heart rate of runner and runner velocity, which have been collected from actual systems (field tests). The model was calibrated using one set of data, without changing parameter values [34–36].

It was used to match a different set of results. Regression model (Table 1) has been built based on field tests data. Scatter diagram, correlation (Figs. 1–5) and autocorrelations procedure for blood pressure was formed by estimating regression statistics and producing related plots for field tests model (Tables 1, and 2).

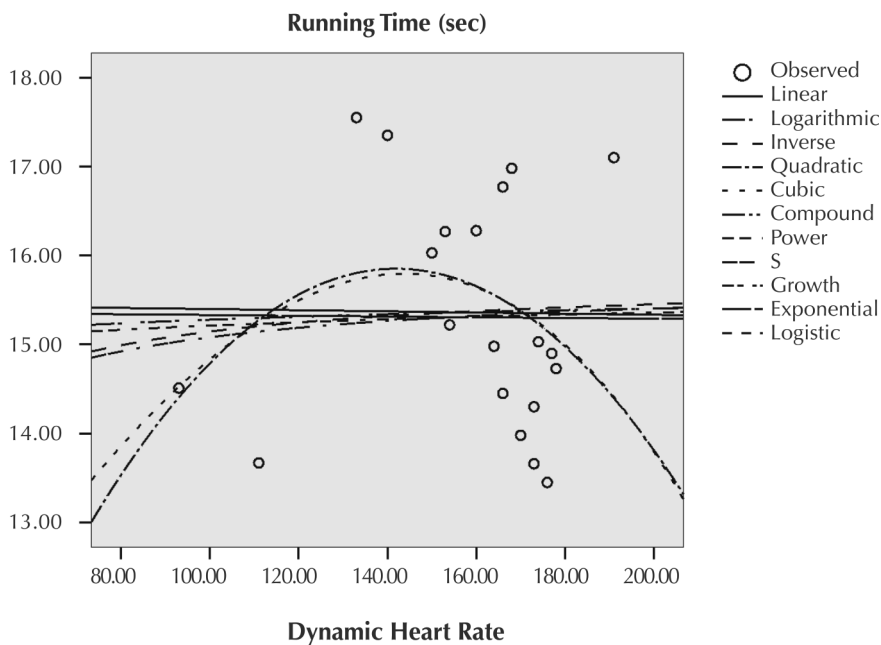


FIGURE 1 Curve fit for Running Time against dynamic heart rate.

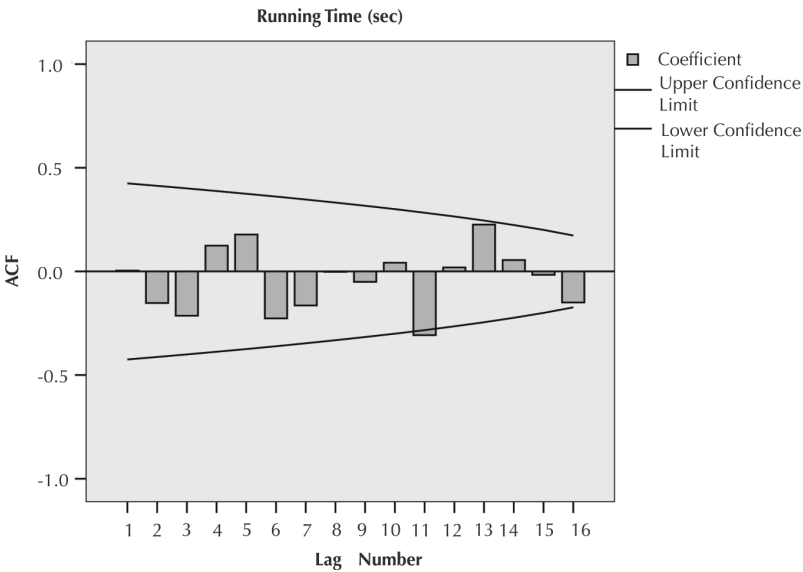


FIGURE 2 Autocorrelations for Running Time against dynamic heart rate.

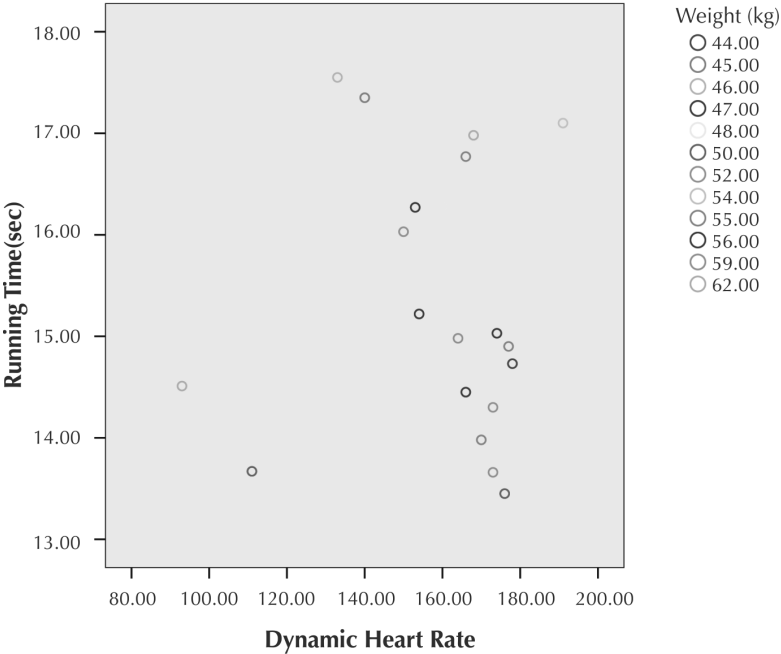


FIGURE 3 Scatter diagram for Running Time against dynamic heart rate.

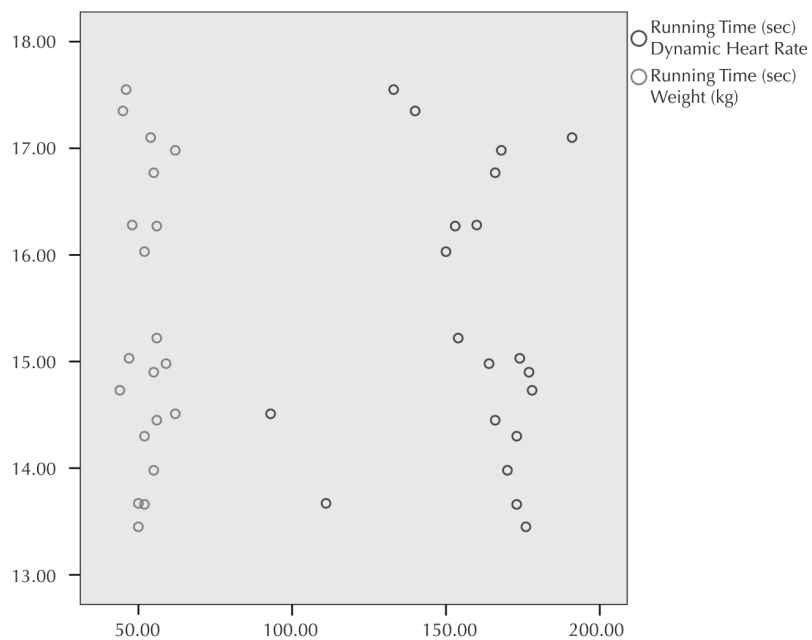


FIGURE 4 Correlation for Running Time against dynamic heart rate.

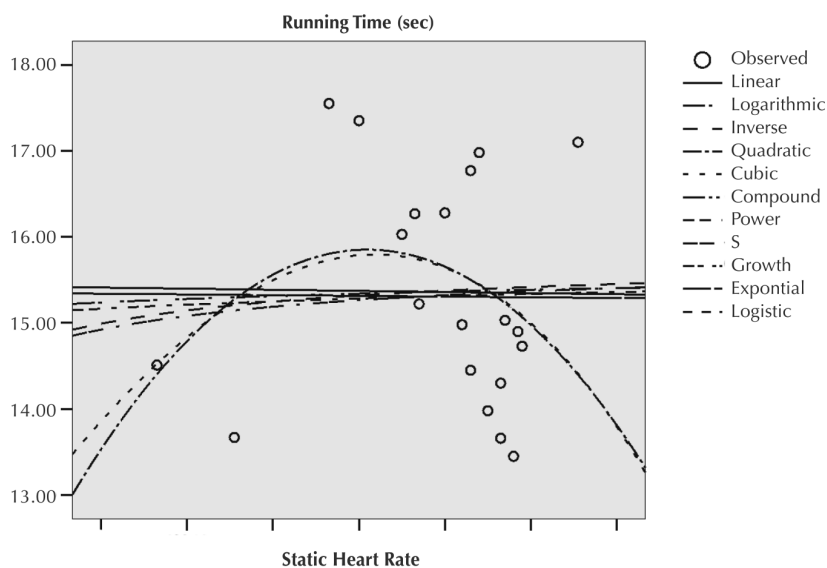


FIGURE 5 Correlation for Running Time against static heart rate.

6.3 RESULTS AND DISCUSSION

In this work, conclusions were drawn on the basis of experiments and calculations for independent variables (running weight and runner heart rate) based on the two cases:

Case 1:

$$V = f(W) \quad (1)$$

Case 2:

$$V = f(H) \quad (2)$$

Dependent variable: V – Runner Velocity (Running Time), with nomenclature “ Y ” for first: at the running state and the second: before of running condition.

Independent variable with nomenclature “ X ” such as:

W – Runner weight (kg) is the most important variable, H – Runner heart rate.

Case 1:

$$V = f(W) \quad (1)$$

The most important effects that were observed based on regression for model summary (Table 3) are as follows (Fig. 1).

Present work compared the anthropometric data for blood pressure of runner related to Runner Velocity (Running Time) and runner heart rate in two cases. The dependent and independent variables of runner were measured in the following conditions:

First: at the running state at dynamic condition (Figs. 2–4).

Second: at the before of running (Table 6) at static condition (Fig. 5).

Case 2:

The independent variable is heart rate.

$$V = f(H) \quad (2)$$

In second assumption for static heart rate against Running Time and for runner dynamic heart rate against runner weight, the curve estimation procedure (Figs. 1–5) allows quick estimating regression statistics (Tables 3–5), and producing related plots for different models. Hence, the auto-regression procedure by regression software “SPSS 10.0.5” was selected for the curve estimation procedure in the present work. The regression model has been built based on field test data. Regression software “SPSS” has fitted the function curve and provided regression analysis. So, the regression model has been found in the final procedure. Although related plots for the field test model were produced. Regression software “SPSS 10.0.5” performs multidimensional scaling of proximity data to find least-squares representation of the objects in a low-dimensional space. The curve estimation procedure allows quickly estimating regression statistics and producing related plots for different models.

TABLE 3 Parameter estimates for Running Time.

Equation	Dependent Variable: Running Time (sec)					Parameter Estimates			
	R-square	F	df1	df2	Sig.	Constant	b1	b2	b3
Linear	.006	.110	1	18	.744	16.410	-.020		
Logarithmic	.009	.156	1	18	.697	20.281	-1.242		
Inverse	.012	.210	1	18	.652	13.933	74.681		
Quadratic	.082	.761	2	17	.482	50.686	-1.328	.012	
Cubic	.082	.761	2	17	.482	50.686	-1.328	.012	.000
Compound	.005	.083	1	18	.776	16.240	.999		
Power	.007	.125	1	18	.728	20.344	-.072		
S	.010	.173	1	18	.682	2.644	4.396		
Growth	.005	.083	1	18	.776	2.787	-.001		
Exponential	.005	.083	1	18	.776	16.240	-.001		
Logistic	.005	.083	1	18	.776	.062	1.001		

The independent variable is Weight (kg).

TABLE 4 Model summary for Running Time.

Dependent Variable: Running Time (sec)									
Equation	Model Summary					Parameter Estimates			
	R-square	F	df1	df2	Sig.	Constant	b1	b2	b3
Linear	.000	.002	1	18	.961	15.464	-.001		
Logarithmic	.001	.010	1	18	.921	14.437	.183		
Inverse	.004	.067	1	18	.799	15.760	-61.517		
Quadratic	.097	.910	2	17	.421	3.672	.172	-.001	
Cubic	.082	.762	2	17	.482	8.095	.080	.000	-1.30E-006
Compound	.000	.001	1	18	.975	15.373	1.000		
Power	.001	.014	1	18	.907	14.273	.014		
S	.004	.075	1	18	.787	2.756	-4.226		
Growth	.000	.001	1	18	.975	2.733	-2.74E-005		
Exponential	.000	.001	1	18	.975	15.373	-2.74E-005		
Logistic	.000	.001	1	18	.975	.065	1.000		

The independent variable is Dynamic Heart Rate.

TABLE 5 Autocorrelations for Running Time.

Series: Running Time (sec)					
Lag	Autocorrelation	Std.Error (a)	Box.Ljung Statistic		
			Value	Df	Sig. (b)
1	.004	.212	.000	1	.987
2	-.152	.206	.545	2	.762
3	-.214	.200	1.687	3	.640
4	.124	.194	2.098	4	.718
5	.178	.187	3.004	5	.699
6	-.227	.181	4.580	6	.599

TABLE 5 (Continued)

Lag	Autocorrelation	Std.Error (a)	Box-Ljung Statistic		
			Value	Df	Sig. (b)
7	-.164	.173	5.478	7	.602
8	-.002	.166	5.478	8	.705
9	-.050	.158	5.578	9	.781
10	.042	.150	5.656	10	.843
11	-.308	.142	10.385	11	.496
12	.019	.132	10.406	12	.580
13	.226	.123	13.806	13	.388
14	.055	.112	14.047	14	.446
15	-.016	.100	14.074	15	.520
16	-.150	.087	17.053	16	.382

^aThe underlying process assumed is independence (white noise).

^bBased on the asymptotic chi-square approximation.

TABLE 6 Model Summary and Parameter Estimates for Running Time against static heart rate.

Equation	Model Summary					Parameter Estimates			
	R-square	F	df1	df2	Sig.	Constant	b1	b2	b3
Linear	.000	.002	1	18	.961	15.464	-.001		
Logarithmic	.001	.010	1	18	.921	14.437	.183		
Inverse	.004	.067	1	18	.799	15.760	-61.517		
Quadratic	.097	.910	2	17	.421	3.672	.172	-.001	
Cubic	.082	.762	2	17	.482	8.095	.080	.000	-1.30E-006
Compound	.000	.001	1	18	.975	15.373	1.000		
Power	.001	.014	1	18	.907	14.273	.014		
S	.004	.075	1	18	.787	2.756	-4.226		

TABLE 6 (Continued)

Equation	Model Summary					Parameter Estimates			
	R-square	F	df1	df2	Sig.	Constant	b1	b2	b3
Growth	.000	.001	1	18	.975	2.733	-2.74E-005		
Exponential	.000	.001	1	18	.975	15.373	-2.74E-005		
Logistic	.000	.001	1	18	.975	.065	1.000		

The independent variable is Static Heart Rate / Dependent Variable: Running Time (sec).

Curve estimation is most appropriate when the relationship between the dependent variable(s) and the independent variable is not necessarily linear (Table 6). Linear regression is used to model the value of a dependent scale variable based on its linear relationship to one or more predictors. Nonlinear regression is appropriate when the relationship between the dependent and independent variables is not intrinsically linear. Binary logistic regression is most useful in modeling of the event probability for a categorical response variable with two outcomes. The auto regression procedure is an extension of ordinary least-squares regression analysis specifically designed for time series.

One of the cases underlying ordinary least-squares regression is the absence of autocorrelation in the model residuals.

Time series, however, often exhibit first-order autocorrelation of the residuals. In the presence of auto-correlated residuals, the linear regression procedure gives inaccurate estimates of how much of the series variability is accounted for by the chosen predictors. This can adversely affect choice of predictors and hence the validity of the model. The auto regression procedure accounts for first-order auto correlated residuals [37–97].

It provides reliable estimates of both goodness-of-fit measures and significance levels of chosen predictor variables. The auto regression procedure by regression software “SPSS 10.0.5” has been selected for the curve estimation procedure in present work. The regression model includes:

$$\text{Case 1: } V = f(W) \quad (1)$$

$$\text{Case 2: } V = f(H) \quad (2)$$

It has been built based on field tests data and in the final procedure they were compared by them.

6.3.1 COMPARISON WITH OTHER WORKS

Rahmani-Nia and Hojjati obtained the effect of exercise training on body composition and aerobic power of females. Comparison showed similarity in present work and the results of that works [98].

6.4 CONCLUSIONS

This book shows a computational modeling for anthropometric of athletes. According to this technique, in this work an improved modeling based on measurements was classified and compared with a special type of weights of sportswomen with other anthropometric classes of sportswomen. On the other hand present work compares the anthropometric data for Runner Velocity (Running Time) related to heart rate and runner weight in two cases. Consequently, the results of this work will help to reduce the risk of sportswomen activities.

NOMENCLATURES

P	Runner Blood Pressure (mm-Hg)
t	Running Time (sec)
V	Velocity (m/s)
S	Runner Stature (cm)

KEYWORDS

- **anthropometric**
- **blood pressure**
- **body motion**
- **computational modeling**
- **regression**

REFERENCES

1. Hurst Thomas, D.; Skull Wars Kennewick Man, Archaeology and the Battle for Native American Identity, Washington University: School of Law – Anheuser-Busch Hall, 2001; 38–41.
2. Nabieh, A.; Mohamed, I. **2010**, Anthropometric Measurements as a Significant for Choosing Juniors in Both Volleyball and Handball Sports (Factorial Analysis Study), *World J. Sport Sci.* 3(4), 277–289.
3. Rogana, S.; Hilfikerd, R.; Clarysb, P.; Clijsenc, R.; Taeymansac, J. **2011**, Position-specific and Team-ranking-related Morphological Characteristics in German Amateur Soccer Players – a Descriptive Study, *Int. J. Appl. Sports Sci.*, 23(1), 168–182.
4. Luigi P. W.; Bercades, T. **2009**, Somatotypes of National Elite Combative Sport Athletes, *Braz. J. Biomotricity*, 3(1), 21–30.
5. Dacres-Mannings, S.; Rochester, S.; Frail, H. **2010**, Anthropometric profiles of Australian Rugby Institute, club and state level Rugby Union players.
6. Gaurav, V.; Singh, M.; Singh, S. **2010**, Anthropometric characteristics, somatotyping and body composition of volleyball and basketball players, *J. Phys. Educ. Sports Manage.* 1(3), 28–32, Available online <http://www.acadjourn.org/jpesm>
7. Beat K.; Kohler, G. **2007**, Influence of anthropometry on race performance in ultra-endurance triathletes in the longest triathlon in North America, *Int. SportMed J.*, 8(2), 87–96, <http://www.ismj.com>
8. Joseph, J.; Greene, M. S.; Timothy, A.; McGuine, A. T. C.; Leverson, G.; Thomas M. **1998**, Anthropometric and Performance Measures for High School Basketball Players, *J. Athl. Training*, 33(3), 229–232
9. Amatya, D. L. **1999**, Comparative Study of Somatotype of Nepalese Sportsmen, National Association for Sports Health and Fitness.
10. Sang Hong, K. **2008**, Kinanthropometric Study of Korean and Nepalese Marathon Runners, keimyung university Diwakar Lal Amatya, MSC-Athletics and Sports Science-India, Sports Kinanthropometrist-Australia, Sports Expert-National Sports Council, January.

11. Gaunt, B. W. Anthropometric and Demographic Factors Affecting Distance Hopped-aid Limb Symmetry Index for the Crossover Hop-for distance test in High School athletes, *J. Orthop. Sport Phys.*, **2001**, *31*(3), 145–151
12. Gross, M.; Dailey, E. S.; Melissa, D.; Dalton, A.; lee, K.; Wendy, I.; Ashley, C. Relationship Between Lifting Capacity and Anthropometric Measures, *J. Orthop. Sport Phys.*, **2000**, *30*(5), 237–247, 258–262.
13. Roberto, C, Jose, A.; Perez, J.; Cortell, m. Juan, J.; Rivas, J. correlations among anthropometric parameters, jump power, and position in professional basketball players, **2008**.
14. Pui, W. K.; Hendrik H. Anthropometric, gait and strength characteristics of Kenyan distance runners, *J. Sports Sci. Med.* **2008**, *7*, 499–504, <http://www.jssm.org>
15. Jawis, M. N.; Singh, R.; Singh, H. J.; Yassin, M. N. Anthropometric and physiological profiles of sepak takraw players, *Br. J. Sports Med.* **2005**, *39*, 825–829. doi: 10.1136/bjbm.2004.016915.
16. Lephart, S. M.; Abt, J. P.; Ferris, C. M.; Sell, T. C.; Nagai, T.; Myers, J. B.; Irrgang, J. Neuromuscular and biomechanical characteristic changes in high school athletes: a plyometric versus basic resistance program, *Br. J. Sports Med.* **2005**, *39*, 932–938. doi: 17.1136/bjbm.2005.019083.
17. Darr, K. C.; Bassett, D. R.; Morgan, B. J.; Thomas, D. P. Effects of age and training status on heart rate recovery after peak exercise. *Am. J. Physiol.* **1988**, *254*, H340–H343.
18. Engerman, S. ‘The Height of U. S. Slaves’, *Local Population Studies*, **1976**, *16*(1) 45–50.
19. Sunder, M. The Making of Giants in a Welfare State: The Norwegian Experience in the Twentieth Century’, *Economics and Biology*, **2004**, 75–86.
20. Woitek, U. Height Cycles in the Eighteenth and Nineteenth Centuries’, *Econ. Hum. Biol.*, **2003**, 243–258.
21. Fogel, R. ‘Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the making of Economic Policy’, *Am. Econ. Rev.*, **1994**, 369–394.
22. Goonetilleke, R. S.; Ho, Cheuk Fan E.; So, R. H. Y. Foot Anthropometry in Hong Kong. (1997, 81–88) Proceedings of the ASEAN 97 Conference, Kuala Lumpur, Malaysia.
23. Baten, J. “Economic Development and the Distribution of Nutritional Resources in Bavaria, 1797–1839, in *J. Income Distrib.* **2000**, *9*, 89–106.
24. Hariri Asli K. Water Hammer Research; Advances in Nonlinear Dynamics Modeling, (2012, 88–121). Canada, USA: Published by Apple Academic Press, Inc., Exclusive worldwide distribution by CRC Press, a Taylor and Francis Group, Print ISBN: 9781926895314, eBook: 978-1-46-656887-7.
25. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. “Some Aspects of Physical and Numerical Modeling of Water Hammer in Pipelines,” *Nonlinear Dynamics An International Journal of Nonlinear Dynamics and Chaos in Engineering Systems*, ISSN: 1573–269X (electronic version) Journal /1071 Springer, Netherlands, **2009**, ISSN: 0924–090X (print version), Heidelberg, Germany, Springer, Volume 60, Number 4 / June, **2010**, 677–701.

26. Heather, E. Collins-Schramm and others, "Markers that Discriminate Between European and African Ancestry Show Limited Variation Within Africa," *Human Genetics* **2002**, *111*, 566–9.
27. Komlos, J.; Baur, M. "From the Tallest to One of the Fattest: The Enigmatic Fate of the Size of the American Population in the Twentieth Century," *Econ. Hum. Biol.* **2004**, *2(1)*, 57–74.
28. Komlos, J.; Kriwy, P. "The Biological Standard of Living in the Two Germanies," *German Econ. Rev.* **2003**, *4*, 493–507.
29. Levy, W. C.; Cerqueira, M. D.; Harp, G. D.; Johannessen, K. A.; Abrass, I. B.; Schwartz, R. S.; Stratton, J. R. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. *Am. J. Cardiol.*, **1998**, *82*, 1236–1241.
30. Margo, R. The Heights of American Slaves. *Social Science History*, **1982**, *6*, 516–538.
31. Rahmani-Nia, F.; Hojjati, Z. The effect of exercise training on body composition and aerobic power in sedentary college age females, *Int. J. Fitness*, India, **2005**, *V1*, 24–31.
32. Rahmani-Nia, F.; Rahnama, N.; Hojjati, Z.; And Soltani, B. Acute effects of aerobic and resistance exercise on serum leptin and some risk factors of coronary heart disease in obese females. *Sport sciences for health*, **2008**, *2(3)*, 118–124.
33. Rahmani-Nia, F.; Hojjati, Z.; Rahnama, N.; Soltani, B. "Leptin, heart disease and exercise. *World J. Sport Sci.*, Iran, **2009**, *2(1)*, 13–20.
34. Rushton, J. P. Race, Brain Size, and Intelligence: A Rejoinder to Cain and Vanderwolf, *Personality and Individual Differences*, **2004**, *11*, 785–794.
35. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. A computational approach to study fluid movement, *Nanomaterials Yearbook – 2009*, From Nanostructures, Nanomaterials and Nanotechnologies to Nanoindustry, (**2010**, 181–196). USA: Nova Science Publications. ISBN: 978-1-60876-451-8.16
36. Young, J. W. Anthropometric and Mass Distribution Characteristics of the Adult Female FAA Civil Aeromedical Institute **2008**, FAA 9–83.
37. Bartlett, R. *Introduction to Sports Biomechanics*, 2nd Edition. Taylor and Francis Library. **2007**.
38. Hariri Asli, K.; Haghi, A. K. A Numerical Study on Fluid Flow and Pressure drop in Microtubes, *J. Balkan Tribological Association*, Tribotechnics and tribomechanics, Sofia, Bulgaria, **2008**, *16(3)*, 382–392.
39. Rahmani-Nia, F.; Hojjati, Z. **2004**, Effect of selected training on body composition and aerobic power of Females College. *J. Harkat*, Iran, *V1*, 18–21.
40. Unal, M.; Unal, D. D. O; Baltaci, A. K.; Mgulkoc, R. Investigation of serum leptin levels and Vo_{2max} value in trained young male athletes and healthy males. *Acta Physiol. Hungary* **2005**, *92*, 173–179
41. Askari, H.; Tykodi, G.; Liu, J.; Dagogo-Jack S. Fasting plasma leptin level is a surrogate measure of insulin sensitivity. *J. Clin. Endocrinol. Metab.*; **2010**, *95(8)*, 3836–3843.
42. Reseland, J. E.; Andessen, S. A.; Solvoll, K.; Hjermann, I.; Urdal, P.; Holme, I.; Drevon, C. A. Effect of long-term change in diet and exercise on plasma leptin concentration. *Clin. Nutr.* **2001**, *73*, 240–245.

43. Mokdad, A. H.; Ford, E. S.; Bowman, B. A.; Dietz, W. H.; Vinicor, F.; Bales, V. S.; Marks, J. S. Prevalence of obesity, *Diabetes* and obesity-related health risk factors, 2001. *JAMA* **2003**, *289*, 76–79.
44. Mendosa-Nunez, V. M.; Garcia-Sanchez, A.; Sanchez-Rodriguez, M.; Galvan-Duart, R. E.; Fonseca-Yerena, E. F. Overweight, waist circumference, age, gender, and insulin resistance as risk factors for Hyperleptinemia. *Obes. Res.* **2002**, *10*, 253–259.
45. Frank, L. L.; Sorensen, B. E.; Yasui, Y.; Tworoger, S. S.; Schwartz, R. S.; Ulrich, C. M.; Irwin, M. L.; Rudolph, R. E.; Rajan, K. B.; Stanczyk, F.; Bowen, D.; Weigle, D. S.; Potter, J. D.; McTiernan A. Effects of exercise on metabolic risk variables in overweight postmenopausal women: a randomized clinical trial. *Obes. Res.*; **2005**, *13*, 615–625.
46. Racette, S. B.; Coppack, S. W.; Landt, M.; Klein, S. Leptin production during moderate-intensity aerobic exercise. *J. Clin. Endocrinol. Metabol.* **1997**, *82*, 2275–2277.
47. Altman, R. Risk factors in coronary atherosclerosis athero-inflammation: the meeting point. *Thrombosis J.*; **2003**, *1*, 4–7.
48. Stefanie, L.; Martina, E. Physical activity and endogenous sex hormones in postmenopausal women. *Cancer Causes Control*, **2011**, *22*, 81–89.
49. Akbarzadeh, R. Relationship between the ratio of waist circumference to hip circumference and risk factors and increased lipids and blood pressure in patients with insulin-dependent *Diabetes* referred to the Endocrinology Institute in Tehran in 1995 and 1996. A Master's thesis, Iran University of Medical Sciences; Faculty of Nursing. **1996**.
50. Ashwell, M.; Lejeane, S. Ratio of waist circumference to height maybe better indicator need for weight management. *BMI*: **1990**, 312–377.
51. McMahon, M.; Skaggs, B. J.; Sahakian, L.; Grossman, J.; FitzGerald, J.; Ragavendra, N.; Charles-Schoeman, C.; Chernishof, M.; Gorn, A.; Witztum, J. L.; Wong, W. K.; Weisman, M.; Wallace, D. J.; La Cava, A.; Hahn, B. H. High plasma leptin levels confer increased risk of atherosclerosis in women with systemic lupus erythematosus, and are associated with inflammatory oxidised lipids. *Ann. Rheum. Dis.* **2011**, *70*(9), 1619–1624.
52. Delavar, A. Research methods in psychology and educational sciences; 1st edition; Tehran; Payame Noor University Press. **2000**.
53. McHugh, P. R.; Moran, T. H. Calories and gastric emptying: a regulatory capacity with implications for feeding. *Am. J. Physiol.* **1979**, *236*(5), R 254–260.
54. Shirreff, S. M.; Watson, P.; Maughan, R. J. Milk and effective post – exercise rehydration drink. *Br. J. Nutr.*, **2007**, *98*(11): P. 173–80.
55. Merson S. J.; Shirreff S. M.; Leiper J. B.; Maughan R. J: Changes in blood plasma and red cell volume after ingestion of hypotonic and hypertonic solutions. *Proc. Nutr. Soc.* (In the Press), **2013**.
56. Melov, S. et al., Resistance exercise reverses aging in human skeletal muscle. *PLOS One*. **2007**, *2*(5), E 465.
57. Miller, S. L.; et al., Metabolic response to provision of mixed protein – carbohydrate supplementation during endurance exercise. *Int. J. Sport Nutr. Exerc. Metab.*, **2002**, *12*(4), 384–397.
58. Miller S. L.; et al., The effects of nutritional supplementation throughout an endurance run on leucine kinetics during recovery. *Int J Sport Nutr Metab*, **2007**, *17*(5), 456–467.

59. Cooke, J. P.; Oka, R. K. Does leptin cause vascular disease? *Circulation* **2002**, *106*, 1904–1905.
60. Canavan, B.; Salem, R. O.; Schurgin, P. K.; Lipinska, I.; Laposata, M.; Grinspoon, S. Effect of physiological leptin administration on markers of inflammation, platelet activation, and platelet aggregation during caloric deprivation. *J. Clin. Endocrinol. Metabol.* **2005**, *90*(10), 5779–5785.
61. Cooke, J. P.; Oka, R. K. Does leptin cause vascular disease? *Circulation* **2002**, *106*, 1904–1905.
62. Ahima, A. S.; Flier, J. S. Adipose tissue as an endocrine organ. *Trends Endocrinol. Metabol.* **2000**, *11*, 327–331.
63. Altman, R. Risk factors in coronary atherosclerosis athero-inflammation: the meeting point. *Thrombosis J.* **2003**, *1*, 4–7.
64. Konstantinides, S.; Schafer, K.; Koschnick, S.; Loskutoff, D. J. Leptin-dependent Platelet aggregation and arterial thrombosis suggests a mechanism for atherothrombotic disease in obesity. *J. Clin. Invest.* **2001**, *108*, 1533–1540.
65. Kaye, S. A.; Fdosom, A. R.; Jacobs, D. R. Psychosocial correlates of body fat distribution in Black and white young adult. *Int. J. Obes.*, **1995**, *17*, 271–277.
66. Montain S. J.; Coyle E. F.: Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J. Appl. Physiol.* **1992a**, *73*, 1340–1350.
67. Meier, U.; Gressner, A. M. Endocrine regulation of energy metabolism: review of pathobiology and clinical chemical aspects of leptin, ghrelin and adiponectin, and resistin. *Cli. Chem.* **2004**, *50*, 1511–1525.
68. Mokdad, A. H.; Ford, E. S.; Bowman, B. A.; Dietz, W. H.; Vinicor, F.; Bales, V. S.; Marks, J. S. Prevalence of obesity, *Diabetes* and obesity-related health risk factors, 2001. *J. Am. Med. Assoc.* **2003**, *289*, 76–79.
69. Ostlund, R. E.; Jr. Yang, S.; Gingerich, R. Relation between plasma leptin concentration and body fat, gender, diet, age, and metabolic covariates. *J. Clin. Endocrinol. Metabol.* **1996**, *81*, 3909–3913.
70. Baratta, M. Leptin from a signal of adiposity to a hormone mediator in peripheral tissues. *Med. Sci. Monit.* **2002**, *8*, RA282–RA292.
71. Gomez-Merino, D, Chennaoui, M, Drogou, C, Bonneau, D, Guezennec, CY, Decrease in serum leptin after prolonged physical activity in men, *Med. Sci. Sports Exerc.*; **2002**, *34*, 1594–1599.
72. Boden, G.; Chen, X.; Mozzoli, M.; Ryan, I. **1996**, Effect of fasting on serum leptin in normal human subjects. *J. Clin. Endocrinol. Metabol.* *81*, 3419–3423.
73. Brabeau, P.; Gutin, B.; Litaker, M. S.; Ramsey, L. T.; Cannady, W. E.; Allison, J.; Lemon, C. R.; Owens, S. Influence of physical training on plasma leptin in obese youths. *Canadian J. Appl. Physiol.* **2003**, *28*, 382–396.
74. Houmard, J. A.; Cox, J. H.; Maclean, P. S.; Barakat, H. A. Effect of short-term exercise training on leptin and insulin action. *Metabolism* **2000**, *49*, 858–861.
75. Kukuljan, S.; et al., Effects of resistance exercise and fortified milk on skeletal muscle mass, muscle size, and functional performance in middle-aged and older men: an 18-mo randomized controlled trial. *J. Appl. Physiol.*, **2009**, *707*(6), 18 64–73.
76. Hickey, M. S.; Considine, R. V.; Israel, R. G. **1996**, Leptin is related to body fat content in male distance runners. *Am. J. Physiol.* *271*, E938-E940.

77. Sihna, M. K.; Caro, J. F. **1998**, Clinical aspects of leptin. *Vitamins and Hormones* **54**, 1–30.
78. Spicer, L. J. Leptin: a possible metabolic signal affecting reproduction. *Domestic Animal Endocrinology* **2001**, *21*, 215–250.
79. Kersick, C. M.; Wismann-Bunn, J.; Fogt, D.; Thomas, A. R.; Taylor, L.; Campbell, B. I.; Wilborn, C. D.; Harvey, T.; Roberts, M. D.; La Bounty, P.; Galbreath, M.; Marcello, B.; Rasmussen, C. J.; Kreidercorresponding, R. B. Changes in weight loss, body composition and cardiovascular disease risk after altering macronutrient distributions during a regular exercise program in obese women, *Nutr. J.*; **2010**, *9*, 59.
80. Hammer, L. D. and Wilson, B. M. Impact of pubertal development on body fat distribution among white, *Hispanic J. Pediatr.*, **1991**, *88*, 975–980.
81. Gippini, A.; Mato, A.; Peino, R.; Lage, M.; Dieguez, C.; Casanueva, F. F. Effect of resistance exercise (body building) training on serum leptin levels in young men. Implications for relationship between body mass index and serum leptin. *J. Endocrinol. Invest.* **1999**, *22*, 824–828.
82. Thong FSL, Hudson, R.; Ross, R.; Janssen, I.; Grahan, T. E. Plasma leptin in moderately obese males: independent effects of weight loss and aerobic exercise. *Am. J. Physiol. Endocrinol. Metabol.* **2000**, *279*, E307–E313.
83. Schulze, P. C.; Kratzsch, J.; Linke, A.; Schoene, N.; Adams, V.; Gielen, S. Elevated serum levels of leptin and leptin and leptin receptor in patients with advanced chronic heart failure. *Eur. J. Heart Fail.* **2003**, *5*, 33–40.
84. Sesso, H. D.; Buring, J. E.; Rifai, N.; Blake, G. J.; Gaziano, J. M.; Ridker, P. M. C-reactive protein and the risk of developing hypertension. *J. Am. Med. Assoc.* **2003**, *200*, 2945–2951.
85. Konstantinides, S.; Schafer, K.; Koschnick, S.; Loskutoff, D. J. Leptin-dependent Platelet aggregation and arterial thrombosis suggests a mechanism for atherothrombotic disease in obesity. *J. Clin. Invest.*; **2001**, *108*, 1533–1540.
86. Kyeasiazis, G. A.; Caplan, J. D.; Lowndes, J.; Carpenter, R. L.; Dennis, K. E.; Sivo, S. A.; Angelopoulos, T. J. Moderate exercise- induced energy expenditure does not alter leptin levels in sedentary obese men, *Clin. J. Sport Med.*; **2007**, *17*, 49–51.
87. Mildred, T.; Consuelo, L. Correlates of body image satisfaction among economically depressed urban Filipino women. *Philippine J. Sci.*, **2009**, 67–74.

CHAPTER 7

MATHEMATICAL AND COMPUTATIONAL MODELING FOR ENGINEERING AND SPORT

CONTENTS

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7.1 INTRODUCTION

Anthropometric data for blood pressure of runner related to Runner Velocity or Running Time and Runner Stature are as an anthropometric parameters. Several physiological factors show an association with endurance performance. Some of these, such as Runner Velocity and stature data. The effects of altitude on physical performance are extensively discussed and divisive for specialists of environmental considerations in sport. Increase in altitude, results in more intense solar radiation and reduction of barometric pressure, ambient temperature, relative humidity, air density and partial pressure of all gases including oxygen.

7.2 MATERIALS AND METHODS

In this work, dateline for field tests data collection was: at 10:00 a.m., 11/09/2012 until 26/09/2012.

Location of work field tests model was at Rasht city in the north of Iran. In this work the following are considerations was made when using and applying anthropometric data for blood pressure of runner related to Runner Velocity (Running Time) and Runner Stature. Blood pressure of runner was measured in two cases: first at the running state and second at the before of running. Twenty healthy and young sprint runner females (Table 2) participate in this study. They signed a written/informed consent. All subjects completed a medical questionnaire to ensure that they were not taking any medication, and were free cardiac, respiratory, renal or metabolic diseases. They didn't have any orthopaedic injury that would inhibit physical activity. The subjects were tested in the follicular phase of the menstrual cycle based on their previous menses. All participants consumed their last meal at least 2 hours before the test and refrained from intensive exercise in the 24 hours period preceding testing [1–15]. All anthropometric measures were recorded in the morning by the same experienced anthropometrist. Stretch stature was measured using a wall-mounted stadiometer (± 0.1 cm). The exercise systolic and diastolic blood pressures were measured on left arm of each participant using cuffs of appropriate size (Table 1). The measurements were recorded just after 100m

intense running in standing position. Curve estimation procedure has used these data, which have been detected in field tests procedure (Table 1). These data were compared by blood pressure of runner and Runner Velocity and stature data, which were collected from actual systems (field tests). The model was calibrated by using one set of data, without changing parameter values. Regression model (Table 1) was built based on field tests data. Scatter diagram; correlation; regression and curve fit were defined for runner dynamic blood pressure against Running Time (Fig. 1).

TABLE 1 Anthropometric model for blood pressure.

No.	Dynamic Blood pressure (mm-Hg)	Static Blood pressure (mm-Hg)
1	111	108
2	120	96
3	114	94
4	100	96
5	118	90
6	109	96
7	126	117
8	142	102
9	120	97
10	134	112
11	148	111
12	134	125
13	139	102
14	120	96
15	110	106
16	116	103
17	126	120
18	113	96
19	113	102
20	119	86

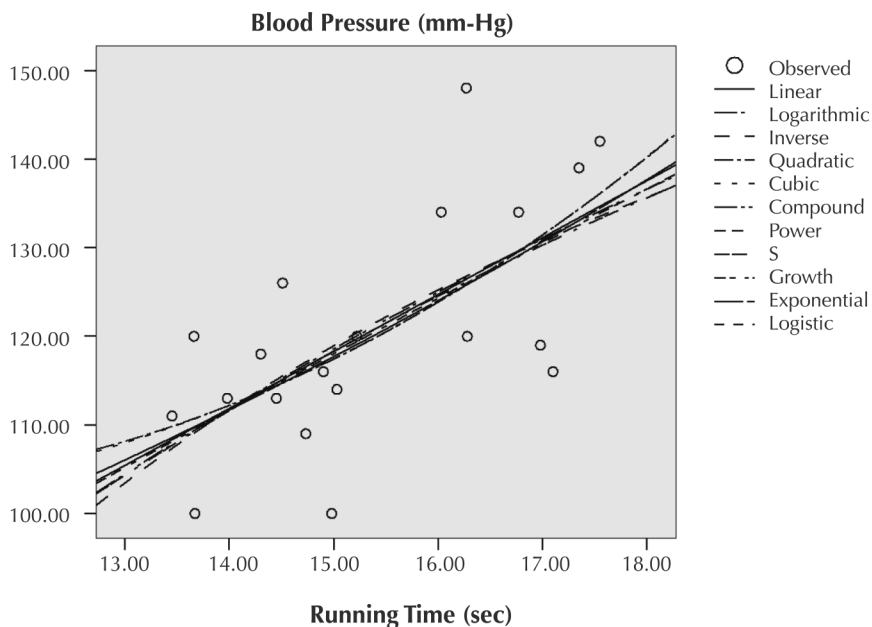


FIGURE 1 Runner dynamic blood pressure against Running Time.

The autocorrelations procedure for blood pressure was showed by estimating regression statistics and producing related plots for field tests model (Table 2) with the following three assumptions in Eqs. (1)–(3):

Assumption (1):

$$p = f(S) \quad (1)$$

Assumption (2):

$$p = f(V) \quad (2)$$

Assumption (3):

$$p = f(V, S) \quad (3)$$

where V – Runner Velocity is the most important variable, S – Runner Stature. Dependent variable: P – blood pressure (mm-Hg) for starting point of running condition. The independent variable is Running Time (sec) and Runner Stature (cm) [16–19].

TABLE 2 Field tests and regression anthropometric model for blood pressure.

No.	Age	Height (cm)	Weight (kg)	BMI	Hip circum- ference	Waist circum- ference	WHR	Heart rate	100 m Running Time (sec)
1	19	165	50	18.38	90	64	0.71	176	13.45
2	16	159	52	20.63	94	71	0.75	173	13.66
3	18	168	47	16.066	88	65	0.73	174	15.03
4	16	158	50	20.08	91	68	0.74	111	13.67
5	14	164	52	19.44	92	66	0.71	173	14.3
6	14	166	44	16	82	63	0.76	178	14.73
7	14	161	54	20.84	93	66	0.7	191	17.1
8	20	161	46	17.76	88	65	0.73	133	17.55
9	21	159	48	19.04	92	69	0.75	160	16.28
10	14	167	52	18.7	92	70	0.76	150	16.03
11	18	168	56	19.85	94	70	0.74	153	16.27
12	15	64	55	20.52	98	67	0.68	166	16.77
13	23	154	45	18.98	88	68	0.77	140	17.35
14	22	170	56	19.37	90	64	0.71	154	15.22
15	21	174	59	19.53	95	69	0.72	164	14.98
16	21	151	55	24.12	98	73	0.74	177	14.9
17	22	170	62	21.45	97	71	0.73	93	14.51
18	28	158	55	22.08	95	70	0.73	170	13.98
19	22	165	56	20.58	92	66	0.71	166	14.45
20	24	166	62	22.54	97	78	0.8	168	16.98

7.3 RESULTS AND DISCUSSION

In this work conclusions were drawn on the basis of experiments and calculations for the three assumptions:

Assumption (1):

$$p = f(S) \quad (1)$$

The most important effects that were observed based on regression for model summary were showed in (Fig. 2).

The auto-regression procedure accounts for first-order auto-correlated residuals. It provides reliable estimates of both goodness of-fit measures and significant levels of chosen predictor variables [20–23].

Present work compared the anthropometric data for blood pressure of runner related to Runner Velocity (Running Time) and Runner Stature in two cases:

First: at the running state (dynamic condition)

Second: before the running state (static condition)

Dependent variable: P – blood pressure with nomenclature “ Y .”

Independent variable with nomenclature “ X ” such as:

V – Runner Velocity is the most important variable, S – Runner Stature.

The independent variable is velocity (m.s^{-1}) and Runner Stature (cm).

Assumption (2):

$$p = f(V) \quad (2)$$

For second assumption the curve estimation procedure allows quick estimating regression statistics and producing related plots for different models. Hence the auto-regression procedure by regression software “SPSS 10.0.5” was selected for the curve estimation procedure in the present work. The regression model was defined based on field test data. Regression software “SPSS” fitted the function curve and provided regression analysis. So, the regression model has been found in the final procedure. This model has compared field test results using Lab. model results [24–32].

Assumption (3):

$$p = f(V, S) \quad (3)$$

For third assumption the curve estimation procedure was illustrated. The runner blood pressure was formed by estimating regression statistics are listed in Table 1. Although related plots for the field test model were produced. Regression software “SPSS 10.0.5” performs multidimensional scaling of proximity data to find least-squares representation of the objects in a low-dimensional space [33–98].

7.3.1 COMPARISON WITH OTHER WORKS

Rahmani-Nia, and Hojjati obtained the effect of exercise training on body composition and aerobic power in sedentary college age females. Comparison showed similarity in present work and the results of that works [99].

7.4 CONCLUSIONS

Present book shows a dynamic and computational modeling for anthropometric of athletes. According to this technique, in this work an improved modeling based on measurements was classified and compared with a special type of skulls of sportswomen with other anthropometric classes of sportswomen. On the other hand present work compares the anthropometric data for blood pressure of runner related to Runner Velocity (Running Time) and Runner Stature in two cases. Consequently, the results of this work will help to reduce the risk of sportswomen activities.

NOMENCLATURES

P	Runner Blood Pressure (mm-Hg)
t	Running Time (sec)
V	Velocity (m/s)
S	Runner Stature (cm)

KEYWORDS

- **anthropometric**
- **blood pressure**
- **body motion**
- **dynamic modeling**
- **regression**

REFERENCES

1. Hurst Thomas, D. Skull Wars Kennewick Man, Archaeology and the Battle for Native American Identity, Washington University: School of Law – Anheuser-Busch Hall, 2001; 38–41.
2. Nabieh, A.; Mohamed, I. Anthropometric Measurements as a Significant for Choosing Juniors in Both Volleyball and Handball Sports (Factorial Analysis Study), *World J. Sport Sci.* **2010**, *3*(4), 277–289.
3. Rogana, S.; Hilfigerd, R.; Clarysb, P.; Clijsenc, R.; Taeymansac, J. Position-specific and Team-ranking-related Morphological Characteristics in German Amateur Soccer Players – a Descriptive Study, *Int. J. Appl. Sports Sci.*, **2011**, *23*(1), 168–182.
4. Luigi P. W.; Bercades, T. somatotypes of national elite combative sport athletes, *Braz. J. Biomotricity*, **2009**, *3*(1), p. 21–30.
5. Dacres-Mannings, S.; Rochester, S.; Frail, H. Anthropometric profiles of Australian Rugby Institute, club and state level Rugby Union players. **2010**.
6. Gaurav, V.; Singh, M.; Singh, S. Anthropometric characteristics, somatotyping and body composition of volleyball and basketball players, *J. Phys. Educ. Sports Manage.* **2010**, *1*(3), 28–32, Available online <http://www.acadjourn.org/jpesm>
7. Beat K.; Kohler, G. Influence of anthropometry on race performance in ultra-endurance triathletes in the longest triathlon in North America, *Int. SportMed J.*, **2007**, *8*(2), 87–96, <http://www.ismj.com>
8. Joseph, J.; Greene, M. S.; Timothy, A.; McGuine, A. T. C.; Leverson, G.; Thomas M. Anthropometric and Performance Measures for High School Basketball Players, *J. Athl. Training*, **1998**, *33*(3), 229–232
9. Amatya, D. L. Comparative Study of Somatotype of Nepalese Sportsmen, National Association for Sports Health and Fitness. **1999**.
10. Sang Hong, K. Kinanthropometric Study of Korean and Nepalese Marathon Runners, keimyung university Diwakar Lal Amatya, MSC-Athletics and Sports Science-India, Sports Kinanthropometrist-Australia, Sports Expert-National Sports Council, January. **2008**.

11. Gaunt, B. W. Anthropometric and Demographic Factors Affecting Distance Hopped-aid Limb Symmetry Index for the Crossover Hop-for distance test in High School athletes, *J. Orthop. Sport Phys.*, **2001**, 31(3), 145–151
12. Gross, M.; Dailey, E. S.; Melissa, D.; Dalton, A.; lee, K.; Wendy, I.; Ashley, C. Relationship Between Lifting Capacity and Anthropometric Measures, *J. Orthop. Sport Phys.*, **2000**, 30(5), 237–247, 258–262.
13. Roberto, C, Jose, A.; Perez, J.; Cortell, M. Juan, J.; Rivas, J. correlations among anthropometric parameters, jump power, and position in professional basketball players, **2008**.
14. Pui, W. K.; Hendrik H. Anthropometric, gait and strength characteristics of Kenyan distance runners, *J. Sports Sci. Med.* **2008**, 7, 499–504, <http://www.jssm.org>
15. Jawis, M. N.; Singh, R.; Singh, H. J.; Yassin, M. N. Anthropometric and physiological profiles of sepak takraw players, *Br. J. Sports Med.* **2005**, 39, 825–829. doi: 10.1136/bjism.2004.016915.
16. Lephart, S. M.; Abt, J. P.; Ferris, C. M.; Sell, T. C.; Nagai, T.; Myers, J. B.; Irrgang, J. Neuromuscular and biomechanical characteristic changes in high school athletes: a plyometric versus basic resistance program, *Br. J. Sports Med.* **2005**, 39, 932–938. doi: 17.1136/bjism.2005.019083.
17. Darr, K. C.; Bassett, D. R.; Morgan, B. J.; Thomas, D. P. Effects of age and training status on heart rate recovery after peak exercise. *Am. J. Physiol.* **1988**, 254, H340-H343.
18. Engerman, S. ‘The Height of U. S. Slaves’, *Local Population Studies*, **1976**, 16(1), 45–50.
19. Sunder, M. The Making of Giants in a Welfare State: The Norwegian Experience in the Twentieth Century’, *Economics and Biology*, **2004**, 75–86.
20. Woitek, U. Height Cycles in the Eighteenth and Nineteenth Centuries’, *Econ. Hum. Biol.*, **2003**, 243–258.
21. Fogel, R. ‘Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the making of Economic Policy’, *Am. Econ. Rev.*, **1994**, 369–394.
22. Goonetilleke, R. S.; Ho, Cheuk Fan E.; So, R. H. Y. Foot Anthropometry in Hong Kong. (1997, 81–88) Proceedings of the ASEAN 97 Conference, Kuala Lumpur, Malaysia.
23. Baten, J. “Economic Development and the Distribution of Nutritional Resources in Bavaria, 1797–1839, in *J. Income Distrib.* **2000**, 9, 89–106.
24. Hariri Asli K. Water Hammer Research; Advances in Nonlinear Dynamics Modeling, (2012, 88–121). Canada, USA: Published by Apple Academic Press, Inc., Exclusive worldwide distribution by CRC Press, a Taylor and Francis Group, Print ISBN: 9781926895314, eBook: 978-1-46-656887-7.
25. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. “Some Aspects of Physical and Numerical Modeling of Water Hammer in Pipelines,” *Nonlinear Dynamics An International Journal of Nonlinear Dynamics and Chaos in Engineering Systems*, ISSN: 1573–269X (electronic version) Journal /1071 Springer, Netherlands, **2009**, ISSN: 0924–090X (print version), Heidelberg, Germany, Springer, Volume 60, Number 4 / June, **2010**, 677–701.

26. Heather, E. Collins-Schramm and others, "Markers that Discriminate Between European and African Ancestry Show Limited Variation Within Africa," *Human Genetics* **2002**, 111, 566–9.
27. Komlos, J.; Baur, M. "From the Tallest to One of the Fattest: The Enigmatic Fate of the Size of the American Population in the Twentieth Century," *Econ. Hum. Biol.* **2004**, 2(1), 57–74.
28. Komlos, J.; Kriwy, P. "The Biological Standard of Living in the Two Germanies," *German Econ. Rev.* **2003**, 4, 493–507.
29. Levy, W. C.; Cerqueira, M. D.; Harp, G. D.; Johannessen, K. A.; Abrass, I. B.; Schwartz, R. S.; Stratton, J. R. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. *Am. J. Cardiol.*, **1998**, 82, 1236–1241.
30. Margo, R. The Heights of American Slaves. *Social Science History*, **1982**, 6, 516–538.
31. Rahmani-Nia, F.; Hojjati, Z. Effect of selected training on body composition and aerobic power of Females College. *J. Harkat, Iran*, **2004**, 1, 18–21.
32. Rahmani-Nia, F.; Rahnama, N.; Hojjati, Z.; And Soltani, B. Acute effects of aerobic and resistance exercise on serum leptin and some risk factors of coronary heart disease in obese females. *Sport sciences for health*, **2008**, 2(3), 118–124.
33. Rahmani-Nia, F.; Hojjati, Z.; Rahnama, N.; Soltani, B. Leptin, heart disease and exercise. *World J. Sport Sci.*, Iran, **2009**, 2(1), 13–20.
34. Rushton, J. P. Race, Brain Size, and Intelligence: A Rejoinder to Cain and Vanderwolf, Personality and Individual Differences, **2004**, 11, 785–794.
35. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. A computational approach to study fluid movement, *Nanomaterials Yearbook – 2009*, From Nanostructures, Nanomaterials and Nanotechnologies to Nanoindustry, (2010, 181–196). USA: Nova Science Publications. ISBN: 978-1-60876-451-8.16
36. Young, J. W. Anthropometric and Mass Distribution Characteristics of the Adult Female FAA Civil Aeromedical Institute **2008**, FAA 9–83.
37. Bartlett, R. Introduction to Sports Biomechanics, 2nd Edition. Taylor and Francise- Library. **2007**.
38. Hariri Asli, K.; Haghi, A. K. A Numerical Study on Fluid Flow and Pressure drop in Microtubes, *J. Balkan Tribological Association*, Tribotechnics and tribomechanics, Sofia, Bulgaria, **2010**, 16(3), 382–392.
39. Hamed-Nia, M.; Rezaei, S. Some risk factors associated with physical activity and body fat percentage cardio-vascular university faculty of Medical sciences and Health Services Blog, The Eleventh: **2007**, 34–40. (In Persian with English abstract).
40. Unal, M.; Unal, D. D. O; Baltaci, A. K.; Mgulkoc, R. Investigation of serum leptin levels and Vo_{2max} value in trained young male athletes and healthy males. *Acta Physiol. Hungary* **2005**, 92, 173–179
41. Askari, H.; Tykodi, G.; Liu, J.; Dagogo-Jack, S. Fasting plasma leptin level is a surrogate measure of insulin sensitivity. *J. Clin. Endocrinol. Metab.*; **2010**, 95(8), 3836–3843.
42. Reseland, J. E.; Andessen, S. A.; Solvoll, K.; Hjermann, I.; Urdal, P.; Holme, I.; Drevon, C. A. Effect of long-term change in diet and exercise on plasma leptin concentration. *Clin. Nutr.* **2001**, 73, 240–245.

43. Mokdad, A. H.; Ford, E. S.; Bowman, B. A.; Dietz, W. H.; Vinicor, F.; Bales, V. S.; Marks, J. S. Prevalence of obesity, *Diabetes* and obesity-related health risk factors, 2001. *JAMA* **2003**, *289*, 76–79.
44. Mendosa-Nunez, V. M.; Garcia-Sanchez, A.; Sanchez-Rodriguez, M.; Galvan-Duart, R. E.; Fonseca-Yerena, E. F. Overweight, waist circumference, age, gender, and insulin resistance as risk factors for Hyperleptinemia. *Obes. Res.* **2002**, *10*, 253–259.
45. Frank, L. L.; Sorensen, B. E.; Yasui, Y.; Tworoger, S. S.; Schwartz, R. S.; Ulrich, C. M.; Irwin, M. L.; Rudolph, R. E.; Rajan, K. B.; Stanczyk, F.; Bowen, D.; Weigle, D. S.; Potter, J. D.; McTiernan A. Effects of exercise on metabolic risk variables in overweight postmenopausal women: a randomized clinical trial. *Obes. Res.*; **2005**, *13*, 615–625.
46. Racette, S. B.; Coppack, S. W.; Landt, M.; Klein S Leptin production during moderate-intensity aerobic exercise. *J. Clin. Endocrinol. Metabol.* **1997**, *82*, 2275–2277.
47. Altman, R.; Risk factors in coronary atherosclerosis athero-inflammation: the meeting point. *Thrombosis J.*; **2003**, *1*, 4–7.
48. Stefanie, L.; Martina, E. Physical activity and endogenous sex hormones in postmenopausal women. *Cancer Causes Control*, **2011**, *22*, 81–89.
49. Akbarzadeh, R Relationship between the ratio of waist circumference to hip circumference and risk factors and increased lipids and blood pressure in patients with insulin-dependent *Diabetes* referred to the Endocrinology Institute in Tehran in 1995 and 1996. A Master's thesis, Iran University of Medical Sciences; Faculty of Nursing. **1996**.
50. Ashwell, M.; Lejeane, S. Ratio of waist circumference to height maybe better indicator need for weight management. *BMI*: **1990**, 312–377.
51. McMahon, M.; Skaggs, B. J.; Sahakian, L.; Grossman, J.; FitzGerald, J.; Ragavendra, N.; Charles-Schoeman, C.; Chernishof, M.; Gorn, A.; Witztum, J. L.; Wong, W. K.; Weisman, M.; Wallace, D. J.; La Cava, A.; Hahn, B. H. High plasma leptin levels confer increased risk of atherosclerosis in women with systemic lupus erythematosus, and are associated with inflammatory oxidised lipids. *Ann. Rheum. Dis.* **2011**, *70*(9), 1619–1624.
52. Delavar, A. Research methods in psychology and educational sciences; 1st edition; Tehran; Payame Noor University Press. **2000**.
53. McHugh, P. R.; Moran, T. H. Calories and gastric emptying: a regulatory capacity with implications for feeding. *Am. J. Physiol.* **1979**, *236*(5), R 254–260.
54. Shirreff, S. M.; Watson, P.; Maughan, R. J. Milk and effective post – exercise rehydration drink. *Br. J. Nutr.*, **2007**, *98*(11), 173–80.
55. Merson S. J.; Shirreff S. M.; Leiper J. B.; Maughan R. J: Changes in blood plasma and red cell volume after ingestion of hypotonic and hypertonic solutions. *Proc. Nutr. Soc.* (In the Press), **2013**.
56. Melov, S. et al., Resistance exercise reverses aging in human skeletal muscle. *PLOS One*. **2007**, *2*(5), E 465.
57. Miller, S. L.; et al., Metabolic response to provision of mixed protein – carbohydrate supplementation during endurance exercise. *Int. J. Sport Nutr. Exerc. Metab.*, **2002**, *12*(4), 384–97.
58. Miller S. L.; et al., The effects of nutritional supplementation throughout an endurance run on leucine kinetics during recovery. *Int J Sport Nutr Metab*, **2007**, *17*(5), 456–67.

59. Cooke, J. P.; Oka, R. K. Does leptin cause vascular disease? *Circulation* **2002**, *106*, 1904–1905.
60. Canavan, B.; Salem, R. O.; Schurgin, P. K.; Lipinska, I.; Laposata, M.; Grinspoon, S. Effect of physiological leptin administration on markers of inflammation, platelet activation, and platelet aggregation during caloric deprivation. *The J. Clin. Endocrinol. Metabol.* **2005**, *90*(10), 5779–5785.
61. Cooke, J. P.; Oka, R. K. Does leptin cause vascular disease? *Circulation* **2002**, *106*, 1904–1905.
62. Ahima, A. S.; Flier, J. S. Adipose tissue as an endocrine organ. *Trends Endocrinol. Metabol.* **2000**, *11*, 327–331.
63. Altman, R. Risk factors in coronary atherosclerosis athero-inflammation: the meeting point. *Thrombosis J.* **2003**, *1*, 4–7.
64. Konstantinides, S.; Schafer, K.; Koschnick, S.; Loskutoff, D. J. Leptin-dependent Platelet aggregation and arterial thrombosis suggests a mechanism for atherothrombotic disease in obesity. *J. Clin. Invest.* **2001**, *108*, 1533–1540.
65. Kaye, S. A.; Fdosom, A. R.; Jacobs, D. R. Psychosocial correlates of body fat distribution in Black and white young adult. *Int. J. Obes.*, **1995**, *17*, 271–277.
66. Montain S. J.; Coyle E. F.: Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J. Appl. Physiol.* **1992a**, *73*, 1340–1350.
67. Meier, U.; Gressner, A. M. Endocrine regulation of energy metabolism: review of pathobiology and clinical chemical aspects of leptin, ghrelin and adiponectin, and resistin. *Cli. Chem.* **2004**, *50*, 1511–1525.
68. Mokdad, A. H.; Ford, E. S.; Bowman, B. A.; Dietz, W. H.; Vinicor, F.; Bales, V. S.; Marks, J. S. Prevalence of obesity, *Diabetes* and obesity-related health risk factors, 2001. *J. Am. Med. Assoc.* **2003**, *289*, 76–79.
69. Ostlund, R. E.; Jr. Yang, S.; Gingerich, R. Relation between plasma leptin concentration and body fat, gender, diet, age, and metabolic covariates. *J. Clin. Endocrinol. Metabol.* **1996**, *81*, 3909–3913.
70. Baratta, M. Leptin from a signal of adiposity to a hormone mediator in peripheral tissues. *Med. Sci. Monit.* **2002**, *8*, RA282-RA292.
71. Gomez-Merino, D, Chennaoui, M, Drogou, C, Bonneau, D, Guezennec, CY, Decrease in serum leptin after prolonged physical activity in men, *Med. Sci. Sports Exerc.*; **2002**, *34*, 1594–1599.
72. Boden, G.; Chen, X.; Mozzoli, M.; Ryan, I. Effect of fasting on serum leptin in normal human subjects. *J. Clin. Endocrinol. Metabol.* **1996**, *81*, 3419–3423.
73. Brabeau, P.; Gutin, B.; Litaker, M. S.; Ramsey, L. T.; Cannady, W. E.; Allison, J.; Lemmon, C. R.; Owens, S. Influence of physical training on plasma leptin in obese youths. *Canadian J. Appl. Physiol.* **2003**, *28*, 382–396.
74. Houmard, J. A.; Cox, J. H.; Maclean, P. S.; Barakat, H. A. Effect of short-term exercise training on leptin and insulin action. *Metabolism* **2000**, *49*, 858–861.
75. Kukuljan, S.; et al., Effects of resistance exercise and fortified milk on skeletal muscle mass, muscle size, and functional performance in middle -aged and older men: an 18-mo randomized controlled trial. *J. Appl. Physiol.*, **2009**, *707* (6), *18*, 64–73.
76. Hickey, M. S.; Considine, R. V.; Israel, R. G. Leptin is related to body fat content in male distance runners. *Am. J. Physiol.* **1996**, *271*, E938-E940.

77. Sihna, M. K.; Caro, J. F. Clinical aspects of leptin. *Vitamins and Hormones* **1998**, *54*, 1–30.
78. Spicer, L. J. Leptin: a possible metabolic signal affecting reproduction. *Domestic Animal Endocrinology* **2001**, *21*, 215–250.
79. Kersick, C. M.; Wismann-Bunn, J.; Fogt, D.; Thomas, A. R.; Taylor, L.; Campbell, B. I.; Wilborn, C. D.; Harvey, T.; Roberts, M. D.; La Bounty, P.; Galbreath, M.; Marcello, B.; Rasmussen, C. J.; Kreidercorresponding RB. Changes in weight loss, body composition and cardiovascular disease risk after altering macronutrient distributions during a regular exercise program in obese women. *Nutr. J.*; **2010**, *9*, 59.
80. Hammer, L. D. and Wilson, B. M. Impact of pubertal development on body fat distribution among white, *Hispanic J. Pediatr.*, **1991**, *88*, 975–980.
81. Gippini, A.; Mato, A.; Peino, R.; Lage, M.; Dieguez, C.; Casanueva, F. F. Effect of resistance exercise (body building) training on serum leptin levels in young men. Implications for relationship between body mass index and serum leptin. *J. Endocrinol. Invest.* **1999**, *22*, 824–828.
82. Thong FSL, Hudson, R.; Ross, R.; Janssen, I.; Grahan, T. E. Plasma leptin in moderately obese males: independent effects of weight loss and aerobic exercise. *Am. J. Physiol. Endocrinol. Metabol.* **2000**, *279*, E307–E313.
83. Schulze, P. C.; Kratzsch, J.; Linke, A.; Schoene, N.; Adams, V.; Gielen, S. Elevated serum levels of leptin and leptin and leptin receptor in patients with advanced chronic heart failure. *Eur. J. Heart Fail.* **2003**, *5*, 33–40.
84. Sesso, H. D.; Buring, J. E.; Rifai, N.; Blake, G. J.; Gaziano, J. M.; Ridker, P. M. C-reactive protein and the risk of developing hypertension. *J. Am. Med. Assoc.* **2003**, *200*, 2945–2951.
85. Konstantinides, S.; Schafer, K.; Koschnick, S.; Loskutoff, D. J. Leptin-dependent Platelet aggregation and arterial thrombosis suggests a mechanism for atherothrombotic disease in obesity. *J. Clin. Invest.*; **2001**, *108*, 1533–1540.
86. Kyeasiazis, G. A.; Caplan, J. D.; Lowndes, J.; Carpenter, R. L.; Dennis, K. E.; Sivo, S. A.; Angelopoulos, T. J. Moderate exercise- induced energy expenditure does not alter leptin levels in sedentary obese men, *Clin. J. Sport Med.*; **2007**, *17*, 49–51.
87. Mildred, T.; Consuelo, L. Correlates of body image satisfaction among economically depressed urban Filipino women. *Philippine J. Sci.*, **2009**, *O*: 67–74.
88. Tamer, L.; Ercan, B.; Unlu, A.; Sucu, N.; Pekdemir, H.; Eskandari, G.; Atik, U. The relationship between leptin and lipids in atherosclerosis. *Indian Heart J.* **2002**, *54*, 692–696.
89. McGill, H. C.; McMahan, A.; Hederick, E. E.; Zieske, A. W.; Malcom, G. T.; Tracy, R. E.; Strong, J. P. Obesity accelerates the progression of coronary atherosclerosis in young men. *Circulation* **2002**, *105*, 2712–2717.
90. Tam, J.; Fukumura, D.; Jain, R. K. A mathematical model of murine metabolic regulation by leptin: energy balance and defence of a stable body weight. *Cell Metab.* **2010**, *7*; *9(1)*, 52–63.
91. Corsonello, A.; Malara, A.; Ientile, R. Leptin enhances adenosine diphosphate- induced platelet aggregation in healthy subjects. *Obes. Res.* **2002**, *10*, 306–317.
92. Maughan, R. J.; J. B. Leiper, and G. E. Vist, Gastric emptying and fluid availability after ingestion of glucose and soy protein hydrolysate solutions in man. *Exp physiol*, **2004**, *89(1)*, 101–108.

93. Shamsuzzaman, A. S. M.; Winnicki, M.; Wolk, R.; Svatikova, A. A.; Phillips, B. G.; Davison, D. E.; Berger, P. B.; Somers, V. K. Independent association between plasma leptin and C-reactive protein in healthy humans. *Circulation* **2004**, *109*, 2181–2185.
94. Mc Connell, G.; Snow, R. J.; Proietto, J.; Hargreaves M. Muscle metabolism during prolonged exercise in humans: influence of carbohydrate availability. *J. Appl. Physiol.* **1999**, *87*, 1083–1086.
95. Klein, S.; Horowitz, J. F.; Landt, M.; Goodrick, S. J.; Mohamed-Ali, V.; Coppack, S. W. Leptin production during early starvation in lean and obese women. *Am. J. Endocrinol. Metabol.* **2000**, *278*, E280-E284.
96. Knudson, J. D.; Incer, U. D.; Dick, G. M.; Shibata, H.; Akahane, R.; Saito, M.; Tune, J. D. Leptin resistance extends to the coronary vasculature in prediabetic dogs and provides protective adaptation against endothelial dysfunction. *Am. J. Physiol. Heart and Circulation Physiology* **2005**, *289*, H1038-H1046.
97. Maughan R. J.; Murray R.: Sports Drinks ; Basic science and practical Aspects CRC Press, L. I. C, Boca Ranton, FL, USA, **2001**.
98. Bosch A. N.; Dennis S. C.; Noakes T. D.: influence of carbohydrate ingestion of fael substrate turnover and oxidation during prolonged exercise *J. Appl. Physiol.* **1994**, *76*, 2364–2372.
99. Rahmani-Nia, F.; Hojjati, Z. The effect of exercise training on body composition and aerobic power in sedentary college age females, *Int. J. Fitness*, India, **2005**, *1*, 24–31.

CHAPTER 8

ANTHROPOMETRIC CHARACTERISTICS OF SPORTSWOMEN

CONTENTS

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8.1 INTRODUCTION

Marathon running exercise is an endurance-promotion exercise practiced by a lot of athletes over the world. However, little is known about the effects of 100 m training habit on hour recovery after exercise and cardiac (ANS) modulation. Time and frequency domain analysis of heart rate variability (HRV) exercise was showed which may contribute to the reduction in mortality. The (HRV) has been proven to be a noninvasive technique capable of providing information on autonomic modulation of the sinus node [1–5].

In 1856, workers found in a limestone quarry the skull of a **NEANDERTHAL** man. The discovery was jointly announced in 1857, giving rise to the discipline of **PALEOANTHROPOLOGY**. A multitude of factors effects physical performance. The effects of both physiological and anthropometric factors vary, depending on the intensity, duration, mode and some other peculiarities of physical exercise. Some researchers found a significant correlation between height and performance in 45 m sprint test. It seems in healthy matured athletes there may be a correlation between height (stretch stature), blood pressure and Running Time. Some researchers and coaches can use a regression line by these variables for their selection and predict exercise blood pressures of their athletes from height or sprint Running Time.

Potentially even more important, historical anthropometrics broadens the understanding of “well-being” beyond the one-dimensional “ruler” of income, providing another lens through which the quality of historical life can be viewed [6–14].

Scientific curiosity in the eighteenth century also spurred development of the first textbooks on human growth, although they were more concerned with growth patterns throughout life than with stature differences across groups or over time. In the nineteenth century class differences in height were easily observable in England. The moral outrage generated by the “tiny children” (Charles Dickens’ “*Oliver Twists*”) along with the view that medicine had a preventive as well as a curative function, meant that anthropometry was directed primarily at the poor, especially children toiling in the factories of English and French industrial cities. Later, fear in Britain over the “degeneration” of its men

and their potential as an effective fighting force provided motivation for large-scale anthropometric surveys, as did efforts evolving out of the child-welfare movement. The early-twentieth century saw the establishment of a series of longitudinal population surveys (which follow individuals as they age) in North America and in Europe. In some cases this work was directed toward the generation of growth standards, while other efforts evaluated social-class differences among children. Such studies can be seen as transitional steps between contemporary and historical anthropometrics. Since the 1950s, anthropometry has been utilized for a variety of purposes in both the developed and underdeveloped world. Population groups have been measured in order to refine growth standards, to monitor the nutritional status of individuals and populations during famines and political disturbances, and to evaluate the effectiveness of economic development programs [15–22].

8.2 MATERIALS AND METHODS

This book presents an improved modeling based on measurements of anthropometric data including blood pressure of runner related to Runner Velocity or Running Time and runner height. Blood pressure of runner was measured in two cases: first at the running state and second at the before of running. Twenty healthy and young sprint runner females participate in this study. Consequently, the results of this work will help to reduce the risk of sportswomen activities.

In this work, dateline for field tests data collection was: at 11:00 a.m., 9/08/2012 until 19/08/2012. Locaton of work field tests model was at Rasht city in the north of Iran. In this work the following are considerations was made when using and applying anthropometric data for blood pressure of runner related to Runner Velocity (Running Time) and Runner Stature. Blood pressure of runner was measured in two cases: first at the running state and second at the before of running. Twenty healthy and young sprint runner females (Table 1) participate in this study. They signed a written/informed consent [23–29].

TABLE 1 Field tests model for blood pressure.

No.	Age	Height (cm)	Weight (kg)	BMI	100 m Running Time (sec)	Static Blood pressure (mm-Hg)	Dynamic Blood pres- sure (mm-Hg)	WHR
1	19	165	50	18.38	13.45	108	111	.71
2	16	159	52	20.63	13.66	96	120	.75
3	18	168	47	16.66	15.03	94	114	.73
4	16	158	50	20.08	13.67	96	100	.74
5	14	164	52	19.44	14.30	90	118	.71
6	14	166	44	16	14.73	96	109	.76
7	14	161	54	20.84	17.10	117	126	.70
8	20	161	46	17.76	17.55	102	142	.73
9	21	159	48	19.04	16.28	97	120	.75
10	14	167	52	18.7	16.03	112	134	.76
11	18	168	56	19.85	16.27	111	148	.74
12	15	164	55	20.52	16.77	125	134	.68
13	23	154	45	18.98	17.35	102	139	.77
14	22	170	56	19.37	15.22	96	120	.71
15	21	174	59	19.53	14.98	106	110	.72
16	21	151	55	24.12	14.90	103	116	.74
17	22	170	62	21.45	14.51	120	126	.73
18	28	158	55	22.08	13.98	96	113	.73
19	22	165	56	20.58	14.45	102	113	.71
20	24	166	62	22.54	16.98	86	119	.80

All subjects completed a medical questionnaire to ensure that they were not taking any medication, and were free cardiac, respiratory, renal or metabolic diseases. They didn't have any orthopaedic injury that would

inhibit physical activity. The subjects were tested in the follicular phase of the menstrual cycle based on their previous menses. All participants consumed their last meal at least 2 hours before the test and refrained from intensive exercise in the 24 hours period preceding testing. All anthropometric measures were recorded in the morning by the same experienced anthropometrist. Stretch stature was measured using a wall-mounted stadiometer (± 0.1 cm). The exercise systolic and diastolic blood pressures were measured on left arm of each participant using cuffs of appropriate size (Table 1). The measurements were recorded just after 100 m intense running in standing position. In present work, for professional runner at fast running, blood pressure of runner, Runner Velocity and stature have been recorded. Curve estimation procedure has used these data, which have been detected field tests (Table 1). These data have been compared by blood pressure of runner and Runner Velocity and stature data, which have been collected from actual systems (field tests). The model was calibrated using one set of data, without changing parameter values. It was used to match a different set of results [30–33]. Regression model (Table 1) has been built based on field tests data. Scatter diagram, Correlation (Figs. 1–7) and Autocorrelations procedure (Fig. 4, Table 2) for blood pressure was formed by estimating regression statistics and producing related plots for field tests model (Table 1).

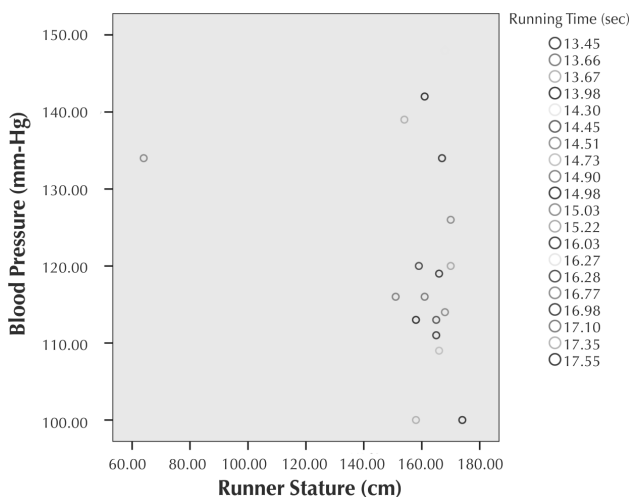


FIGURE 1 Scatter diagram for dynamic blood.

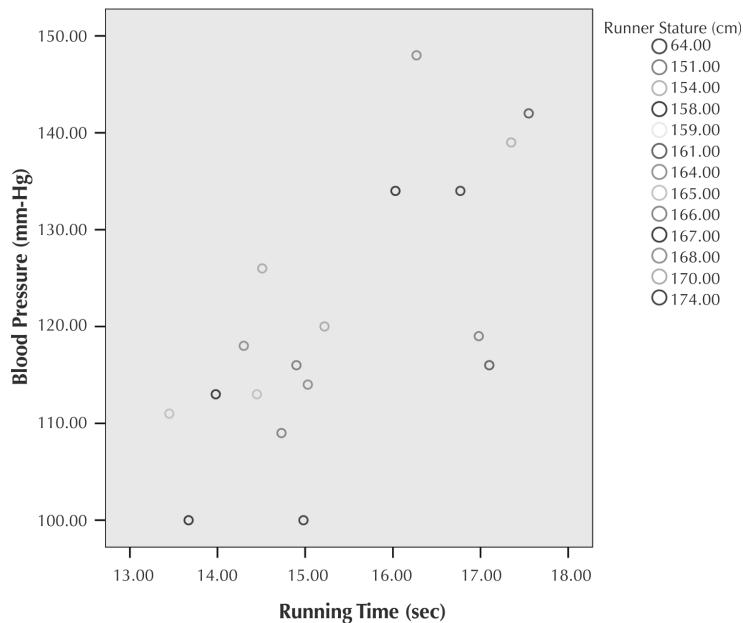


FIGURE 2 Scatter diagram for dynamic blood pressure against Running Time.

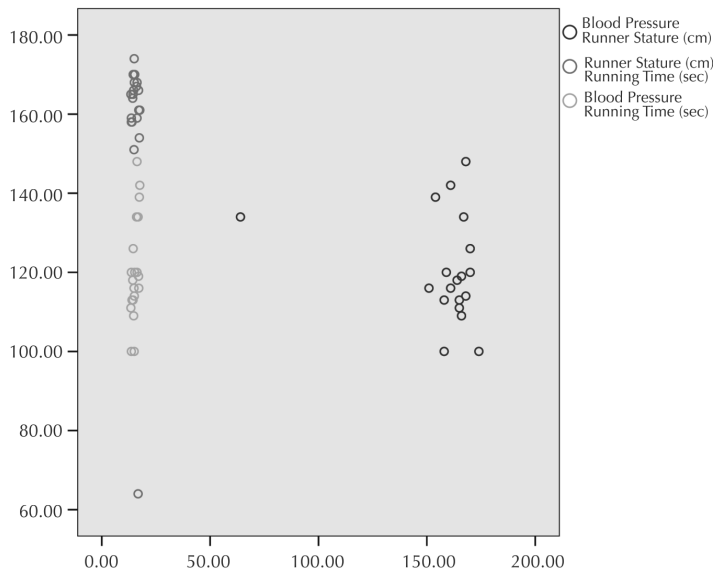


FIGURE 3 Correlation for dynamic blood pressure against Runner Stature and Running Time.

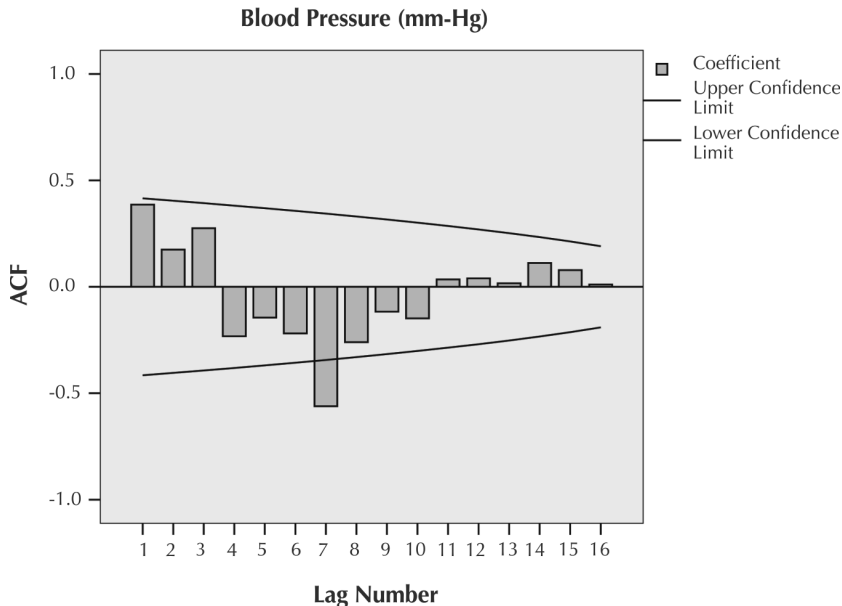


FIGURE 4 Autocorrelations for dynamic blood pressure.

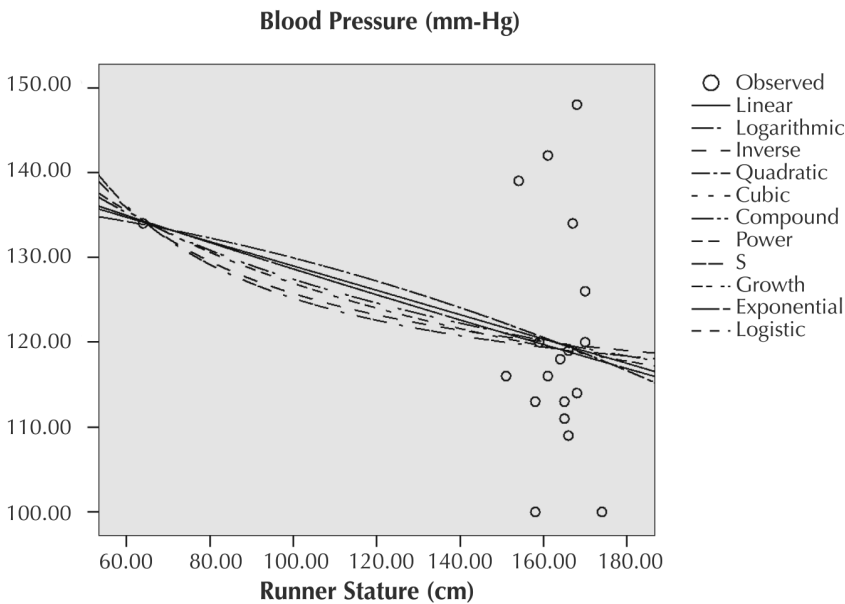


FIGURE 5 Curve fit for runner dynamic blood pressure against Runner Stature.

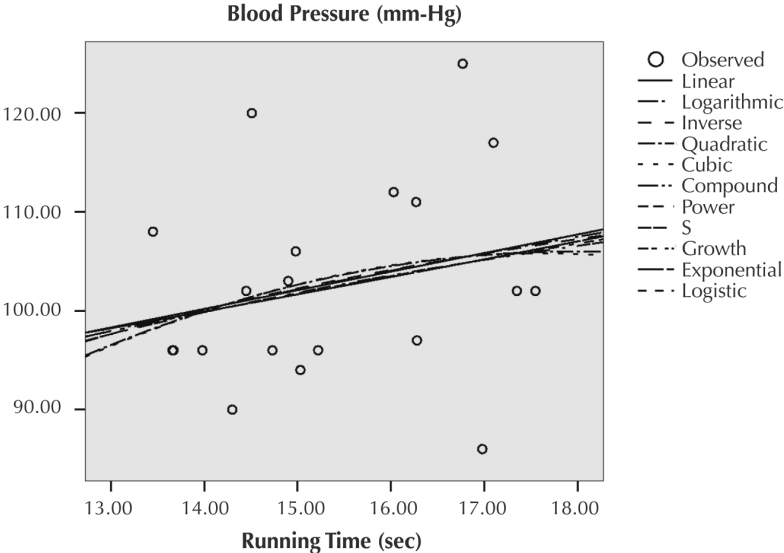


FIGURE 6 Regression and curve fit for runner static blood pressure against Running Time

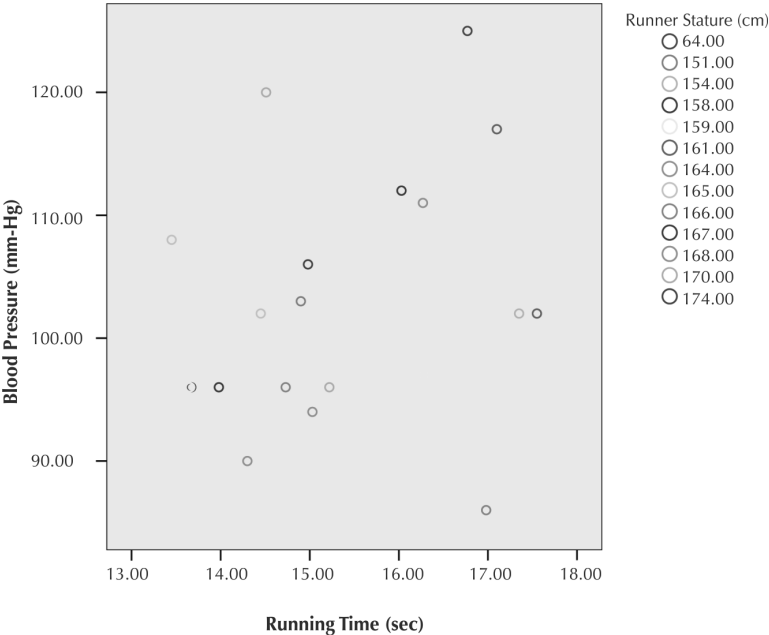


FIGURE 7 Scatter diagram for static blood pressure against Running Time.

TABLE 2 Autocorrelations for blood pressure.

Lag	Autocorrelation	Std.Error (a)	Box-Ljung Statistic		
			Value	df	Sig. (b)
1	.386	.208	3.453	1	.063
2	.175	.202	4.199	2	.123
3	.275	.197	6.162	3	.104
4	-.232	.191	7.648	4	.105
5	-.145	.185	8.264	5	.142
6	-.219	.178	9.776	6	.134
7	-.561	.172	20.437	7	.005
8	-.260	.165	22.919	8	.003
9	-.117	.158	23.464	9	.005
10	-.148	.151	24.431	10	.007
11	.035	.143	24.489	11	.011
12	.040	.135	24.577	12	.017
13	.016	.126	24.594	13	.026
14	.112	.117	25.508	14	.030
15	.078	.107	26.045	15	.038
16	.011	.095	26.059	16	.053

^aThe underlying process assumed is independence (white noise).

^bBased on the asymptotic chi-square approximation.

8.3 RESULTS AND DISCUSSION

In this work conclusions were drawn on the basis of experiments and calculations for independent variables (Running Time and Runner Stature) based on the two cases:

Case 1:

$$p = f(S) \quad (1)$$

Case 2:

$$p = f(V) \quad (2)$$

Dependent variable: P – Blood pressure ($mm-Hg$) with nomenclature “ Y ” for first: at the running state and the second: before of running condition.

Independent variable with nomenclature “ X ” such as:

V – Runner Velocity ($m.s^{-1}$) is the most important variable, S – Runner Stature (cm).

Case 1:

$$p = f(S) \quad (1)$$

The independent variable is Runner Stature (cm).

The most important effects that were observed based on regression for model summary (Tables 3–5) are as follows (Figs. 1, 3, 5).

Present work compared the anthropometric data for blood pressure of runner related to Runner Velocity (Running Time) and Runner Stature in two cases. The dependent and independent variables of runner were measured in the following conditions :

First: at the running state at dynamic condition (Figs. 1–5).

Second: at the before of running at static condition (Figs. 6–7).

TABLE 3 Model summary.

R	R-square	Adjusted R-square	Std. Error of the Estimate
.651	.424	.392	10.157

The independent variable is Running Time (sec).

TABLE 4 Model summary for curve fit.

	Sum of Squares	Df	Mean Square	F	Sig.
Regression	1365.876	1	1365.876	13.240	.002
Residual	1856.924	18	103.162		
Total	3222.800	19			

The independent variable is Running Time (sec).

TABLE 5 Coefficients for curve fit.

	Unstandardized Coefficients		Standardized Coefficients	T	Sig.
	B	Std. Error	Beta		
ln(Running Time (sec))	98.790	27.150	.651	3.639	.002
(Constant)	-148.929	74.108		-2.010	.060

Case 2:

The independent variable is Running Time (sec).

$$p = f(V) \quad (2)$$

Estimating regression statistics (Tables 6–11), and producing related plots for different models. In second assumption for static blood pressure against Running Time and for runner dynamic blood pressure against Runner Stature, the curve estimation procedure (Figs. 2, 3, 6, and 7) allows quick Hence the auto-regression procedure by regression software “SPSS 10.0.5” was selected for the curve estimation procedure in the present work [30–36]. The regression model has been built based on field test data. Regression software “SPSS” has fitted the function curve and provided regression analysis. So, the regression model has been found in the final procedure.

TABLE 6 Model Summary for Curve Fit for Std. Error.

R	R-square	Adjusted R-square	Std. Error of the Estimate
.251	.063	.011	12.953

The independent variable is Runner Stature (cm)

TABLE 7 Model summary for curve fit for Sum of Squares.

	Sum of Squares	df	Mean Square	F	Sig.
Regression	202.600	1	202.600	1.207	.286
Residual	3020.200	18	167.789		
Total	3222.800	19			

The independent variable is Runner Stature (cm).

TABLE 8 Coefficients for curve fit Unstandardized and Standardized Coefficients.

	Unstandardized Coefficients		Standardized Coefficients	t	Sig.
	B	Std. Error	Beta		
ln (Runner Stature) (cm)	-15.376	13.993	-.251	-1.099	.286
(Constant)	198.229	70.705		2.804	.012

Table 9. Coefficients for curve fit logarithmic-model summary R-square

R	R-square	Adjusted R-square	Std. Error of the Estimate
.246	.061	.008	10.139

The independent variable is Running Time (sec).

TABLE 10 Coefficients for curve fit logarithmic-model summary sum of squares.

	Sum of Squares	df	Mean Square	F	Sig.
Regression	119.270	1	119.270	1.160	.296
Residual	1850.480	18	102.804		
Total	1969.750	19			

The independent variable is Running Time (sec).

TABLE 11 Coefficients for curve fit logarithmic-model summary.

	Unstandardized Coefficients		Standardized Coefficients	t	Sig.
	B	Std. Error	Beta		
ln(Running Time (sec))	29.193	27.103	.246	1.077	.296
(Constant)	23.104	73.979		.312	.758

This model has compared field test results using Lab. model results. Although related plots for the field test model were produced. Regression software “SPSS 10.0.5” performs multidimensional scaling of

proximity data to find least-squares representation of the objects in a low-dimensional space [37–38]. The curve estimation procedure allows quickly estimating regression statistics and producing related plots for different models. Curve estimation is most appropriate when the relationship between the dependent variable(s) and the independent variable is not necessarily linear. Linear regression is used to model the value of a dependent scale variable based on its linear relationship to one or more predictors. Nonlinear regression is appropriate when the relationship between the dependent and independent variables is not intrinsically linear. Binary logistic regression is most useful in modeling of the event probability for a categorical response variable with two outcomes. The auto regression procedure is an extension of ordinary least-squares regression analysis specifically designed for time series. One of the cases underlying ordinary least-squares regression is the absence of autocorrelation in the model residuals. Time series, however, often exhibit first-order autocorrelation of the residuals.

In the presence of auto-correlated residuals, the linear regression procedure gives inaccurate estimates of how much of the series variability is accounted for by the chosen predictors. This can adversely affect choice of predictors and hence the validity of the model. The auto regression procedure accounts for first-order auto correlated residuals. It provides reliable estimates of both goodness-of-fit measures and significance levels of chosen predictor variables. The auto regression procedure by regression software “SPSS 10.0.5” has been selected for the curve estimation procedure in present work. The regression model includes:

Case 1:

$$p = f(S) \quad (1)$$

Case 2:

$$p = f(V) \quad (2)$$

It has been built based on field tests data and in the final procedure they were compared by them [39–85].

8.3.1 COMPARISON WITH OTHER WORKS

Rahmani-Nia and Hojjati obtained the effect of exercise training on body composition and aerobic power of females. Comparison showed similarity in present work and the results of that works [86].

8.4 CONCLUSIONS

This work showed the effects of the Runner Velocity as the most important variable and Runner Stature on runner blood pressure. Present work emphasized that “regression model” is accurate model for modeling of anthropometric and body motion. Hence in order to presentation for importance of Runner Velocity on blood pressure phenomenon, it was compared the models for laboratory; computational and field tests experiments. Based on these procedures, it was showed that the regression based model for anthropometric of body motion.

Therefore, the dynamic or running state was compared by static or before of running condition. On the other hand, this idea were included the proper analysis to provide a dynamic response to the shortcomings of the body motion. Because increased vagal activity has been associated with a reduction in the risk of sudden cardiac death, this work has demonstrated that a delayed decrease in hours after exercise would be a powerful and independent predictor of all cause mortality in patients or in general population. Consequently, the results of this work will help to reduce the risk of anthropometric of female athletes sport women activities.

NOMENCLATURES

P	Runner Blood Pressure (mm-Hg)
t	Running Time (sec)
V	Velocity (m/s)
S	Runner Stature (cm)

KEYWORDS

- **anthropometric**
- **blood pressure**
- **body motion**
- **computational modeling**
- **regression**

REFERENCES

1. Hurst Thomas, D. Skull Wars Kennewick Man, Archaeology and the Battle for Native American Identity, Washington University: School of Law - Anheuser-Busch Hall, 2001; 38–41.
2. Nabieh, A.; Mohamed, I. Anthropometric Measurements as a Significant for Choosing Juniors in Both Volleyball and Handball Sports (Factorial Analysis Study), *World J. Sport Sci.* **2010**, *3*(4), 277–289.
3. Rogana, S.; Hilfikerd, R.; Clarysb, P.; Clijsenc, R.; Taeymansac, J. Position-specific and Team-ranking-related Morphological Characteristics in German Amateur Soccer Players – a Descriptive Study, *Int. J. Appl. Sports Sci.*, **2011**, *23*(1), 168–182.
4. Luigi P. W.; Bercades, T. somatotypes of national elite combative sport athletes, *Braz. J. Biometricity*, **2009**, *3*(1), 21–30.
5. Dacres-Mannings, S.; Rochester, S.; Frail, H. Anthropometric profiles of Australian Rugby Institute, club and state level Rugby Union players. **2010**.
6. Gaurav, V.; Singh, M.; Singh, S. Anthropometric characteristics, somatotyping and body composition of volleyball and basketball players, *J. Phys. Educ. Sports Manage.* **2010**, *1*(3), 28–32, Available online <http://www.acadjourn.org/jpesm>
7. Beat K.; Kohler, G. Influence of anthropometry on race performance in ultra-endurance triathletes in the longest triathlon in North America, *Int. SportMed J.*, **2007**, *8*(2), 87–96, <http://www.ismj.com>
8. Joseph, J.; Greene, M. S.; Timothy, A.; McGuine, A. T. C.; Leverson, G.; Thomas M. Anthropometric and Performance Measures for High School Basketball Players, *J. Athl. Training*, **1998**, *33*(3), 229–232
9. Amatya, D. L. Comparative Study of Somatotype of Nepalese Sportsmen, National Association for Sports Health and Fitness. **1999**.
10. Sang Hong, K. Kinanthropometric Study of Korean and Nepalese Marathon Runners, keimyung university Diwakar Lal Amatya, MSC-Athletics and Sports Science-India,

- Sports Kinanthropometrist-Australia, Sports Expert-National Sports Council, January. **2008**.
11. Gaunt, B. W. Anthropometric and Demographic Factors Affecting Distance Hopped-aid Limb Symmetry Index for the Crossover Hop-for distance test in High School athletes, *J. Orthop. Sport Phys.*, **2001**, 31(3), 145–151
 12. Gross, M.; Dailey, E. S.; Melissa, D.; Dalton, A.; lee, K.; Wendy, I.; Ashley, C. Relationship Between Lifting Capacity and Anthropometric Measures, *J. Orthop. Sport Phys.*, **2000**, 30(5), 237–247, 258–262.
 13. Roberto, C, Jose, A.; Perez, J.; Cortell, m. Juan, J.; Rivas, J. correlations among anthropometric parameters, jump power, and position in professional basketball players, **2008**.
 14. Pui, W. K.; Hendrik H. Anthropometric, gait and strength characteristics of Kenyan distance runners, *J. Sports Sci. Med.* **2008**, 7, 499–504, <http://www.jssm.org>
 15. Jawis, M. N.; Singh, R.; Singh, H. J.; Yassin, M. N. Anthropometric and physiological profiles of sepak takraw players, *Br. J. Sports Med.* **2005**, 39, 825–829. doi: 10.1136/bjbm.2004.016915.
 16. Lephart, S. M.; Abt, J. P.; Ferris, C. M.; Sell, T. C.; Nagai, T.; Myers, J. B.; Irrgang, J. Neuromuscular and biomechanical characteristic changes in high school athletes: a plyometric versus basic resistance program, *Br. J. Sports Med.* **2005**, 39, 932–938. doi: 17.1136/bjbm.2005.019083.
 17. Darr, K. C.; Bassett, D. R.; Morgan, B. J.; Thomas, D. P. Effects of age and training status on heart rate recovery after peak exercise. *Am. J. Physiol.* **1988**, 254, H340-H343.
 18. Engerman, S. ‘The Height of U. S. Slaves’, *Local Population Studies*, **1976**, 16, 1, 45–50.
 19. Sunder, M. The Making of Giants in a Welfare State: The Norwegian Experience in the Twentieth Century’, *Economics and Biology*, **2004**, 75–86.
 20. Woitek, U. Height Cycles in the Eighteenth and Nineteenth Centuries’, *Econ. Hum. Biol.*, **2003**, 243–258.
 21. Fogel, R. ‘Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the making of Economic Policy’, *Am. Econ. Rev.*, **1994**, 369–394.
 22. Goonetilleke, R. S.; Ho, Cheuk Fan E.; So, R. H. Y. Foot Anthropometry in Hong Kong. (pp. 81–88) *Proceedings of the ASEAN 97 Conference*, Kuala Lumpur, Malaysia. **1997**.
 23. Baten, J. “Economic Development and the Distribution of Nutritional Resources in Bavaria, 1797–1839, in *J. Income Distrib.* **2000**, 9, 89–106.
 24. Hariri Asli K. *Water Hammer Research; Advances in Nonlinear Dynamics Modeling*, (2012, pp. 88–121). Canada, USA: Published by Apple Academic Press, Inc., Exclusive worldwide distribution by CRC Press, a Taylor and Francis Group, Print ISBN: 9781926895314, eBook: 978-1-46-656887-7.

25. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. "Some Aspects of Physical and Numerical Modeling of Water Hammer in Pipelines," *Nonlinear Dynamics An International Journal of Nonlinear Dynamics and Chaos in Engineering Systems*, ISSN: 1573–269X (electronic version) Journal /1071 Springer, Netherlands, **2009**, ISSN: 0924–090X (print version), Heidelberg, Germany, Springer, Volume 60, Number 4 / June, **2010**, 677–701.
26. Heather, E. Collins-Schramm and others, "Markers that Discriminate Between European and African Ancestry Show Limited Variation Within Africa," *Human Genetics* **2002**, 111, 566–9.
27. Komlos, J.; Baur, M. "From the Tallest to One of the Fattest: The Enigmatic Fate of the Size of the American Population in the Twentieth Century," *Econ. Hum. Biol.* **2004**, 2(1), 57–74.
28. Komlos, J.; Kriwy, P. "The Biological Standard of Living in the Two Germanies," *German Econ. Rev.* **2003**, 4, 493–507.
29. Levy, W. C.; Cerqueira, M. D.; Harp, G. D.; Johannessen, K. A.; Abrass, I. B.; Schwartz, R. S.; Stratton, J. R. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. *Am. J. Cardiol.*, **1998**, 82, 1236–1241.
30. Margo, R. The Heights of American Slaves. *Social Science History*, **1982**, 6, 516–538.
31. Rahmani-Nia, F.; Hojjati, Z. The effect of exercise training on body composition and aerobic power in sedentary college age females, *Int. J. Fitness*, India, **2005**, 1, 24–31.
32. Rahmani-Nia, F.; Rahnama, N.; Hojjati, Z.; And Soltani, B. **2008**, Acute effects of aerobic and resistance exercise on serum leptin and some risk factors of coronary heart disease in obese females. *Sport sciences for health*, 2(3), 118–124.
33. Rahmani-Nia, F.; Hojjati, Z.; Rahnama, N.; Soltani, B." Leptin, heart disease and exercise. *World J. Sport Sci.*, Iran, **2009**, 2(1), 13–20.
34. Rushton, J. P. Race, Brain Size, and Intelligence: A Rejoinder to Cain and Vanderwolf, *Personality and Individual Differences*, **2004**, 11, 785–794.
35. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. A computational approach to study fluid movement, *Nanomaterials Yearbook – 2009*, From Nanostructures, Nanomaterials and Nanotechnologies to Nanoindustry, (2010, pp. 181–196). USA: Nova Science Publications. ISBN: 978-1-60876-451-8.16
36. Young, J. W. Anthropometric and Mass Distribution Characteristics of the Adult Female FAA Civil Aeromedical Institute **2008**, FAA 9–83.
37. Bartlett, R. Introduction to Sports Biomechanics, 2nd Edition. Taylor and Francise- Library. **2007**.
38. Hariri Asli, K.; Haghi, A. K. A Numerical Study on Fluid Flow and Pressure drop in Microtubes, *J. Balkan Tribological Association*, Tribotechnics and tribomechanics, Sofia, Bulgaria, **2010**, 16(3), 382–392.
39. Knudson, J. D.; Incer, U. D.; Dick, G. M.; Shibata, H.; Akahane, R.; Saito, M.; Tune, J. D. Leptin resistance extends to the coronary vasculature in prediabetic dogs and provides protective adaptation against endothelial dysfunction. *Am. J. Physiol. Heart and Circulation Physiology* **2005**, 289, H1038-H1046.

40. Unal, M.; Unal, D. D. O.; Baltaci, A. K.; Mgulkoc, R. Investigation of serum leptin levels and Vo_{2max} value in trained young male athletes and healthy males. *Acta Physiol. Hungary* **2005**, *92*, 173–179.
41. McMahon, M.; Skaggs, B. J.; Sahakian, L.; Grossman, J.; FitzGerald, J.; Ragavendra, N.; Charles-Schoeman, C.; Chernishof, M.; Gorn, A.; Witztum, J. L.; Wong, W. K.; Weisman, M.; Wallace, D. J.; La Cava, A.; Hahn, B. H. High plasma leptin levels confer increased risk of atherosclerosis in women with systemic lupus erythematosus, and are associated with inflammatory oxidised lipids. *Ann. Rheum. Dis.* **2011**, *70*(9), 1619–1624.
42. Delavar, A. Research methods in psychology and educational sciences; 1st edition; Tehran; Payame Noor University Press. **2000**.
43. McHugh, P. R.; Moran, T. H. Calories and gastric emptying: a regulatory capacity with implications for feeding. *Am. J. Physiol.* **1979**, *236*(5), R 254–60.
44. Shirreff, S. M.; P. Watson, and R. J. Maughan, Milk and effective post – exercise rehydration drink. *Br. J. Nutr.*, **2007**, *98*(11), 173–80.
45. Merson S. J.; Shirreff S. M.; Leiper J. B.; Maughan R. J: Changes in blood plasma and red cell volume after ingestion of hypotonic and hypertonic solutions. *Proc. Nutr. Soc.* (In the Press), **2013**.
46. Melov, S. et al., Resistance exercise reverses aging in human skeletal muscle. *PLOS One*. **2007**, *2*(5), E 465.
47. Miller, S. L.; et al., Metabolic response to provision of mixed protein – carbohydrate supplementation during endurance exercise. *Int. J. Sport Nutr. Exerc. Metab.*, **2002**, *12*(4), 384–97.
48. Miller S. L.; et al., The effects of nutritional supplementation throughout an endurance run on leucine kinetics during recovery. *Int J Sport Nutr Metab*, **2007**, *17*(5), 456–67.
49. Cooke, J. P.; Oka, R. K. Does leptin cause vascular disease? *Circulation* **2002**, *106*, 1904–1905.
50. Canavan, B.; Salem, R. O.; Schurgin, P. K.; Lipinska, I.; Laposata, M.; Grinspoon, S. Effect of physiological leptin administration on markers of inflammation, platelet activation, and platelet aggregation during caloric deprivation. *The J. Clin. Endocrinol. Metabol.* **2005**, *90*(10), 5779–5785.
51. Cooke, J. P.; Oka, R. K. Does leptin cause vascular disease? *Circulation* **2002**, *106*, 1904–1905.
52. Ahima, A. S.; Flier, J. S. Adipose tissue as an endocrine organ. *Trends Endocrinol. Metabol.* **2000**, *11*, 327–331.
53. Altman, R. Risk factors in coronary atherosclerosis athero-inflammation: the meeting point. *Thrombosis J.* **2003**, *1*, 4–7.
54. Konstantinides, S.; Schafer, K.; Koschnick, S.; Loskutoff, D. J. Leptin-dependent Platelet aggregation and arterial thrombosis suggests a mechanism for atherothrombotic disease in obesity. *J. Clin. Invest.* **2001**, *108*, 1533–1540.

55. Kaye, S. A.; Fdosom, A. R.; Jacobs, D. R. Psychosocial correlates of body fat distribution in Black and white young adult. *Int. J. Obes.*, **1995**, *17*, 271–277.
56. Montain S. J.; Coyle E. F.: Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J. Appl. Physiol.* **1992a**, *73*, 1340–1350.
57. Meier, U.; Gressner, A. M. Endocrine regulation of energy metabolism: review of pathobiology and clinical chemical aspects of leptin, ghrelin and adiponectin, and resistin. *Cli. Chem.* **2004**, *50*, 1511–1525.
58. Mokdad, A. H.; Ford, E. S.; Bowman, B. A.; Dietz, W. H.; Vinicor, F.; Bales, V. S.; Marks, J. S. Prevalence of obesity, *Diabetes* and obesity-related health risk factors, 2001. *J. Am. Med. Assoc.* **2003**, *289*, 76–79.
59. Ostlund, R. E.; Jr. Yang, S.; Gingerich, R. Relation between plasma leptin concentration and body fat, gender, diet, age, and metabolic covariates. *J. Clin. Endocrinol. Metabol.* **1996**, *81*, 3909–3913.
60. Baratta, M. Leptin from a signal of adiposity to a hormone mediator in peripheral tissues. *Med. Sci. Monit.* **2002**, *8*, RA282-RA292.
61. Gomez-Merino, D, Chennaoui, M, Drogou, C, Bonneau, D, Guezennec, CY, Decrease in serum leptin after prolonged physical activity in men, *Med. Sci. Sports Exerc.*; **2002**, *34*, 1594–1599.
62. Boden, G.; Chen, X.; Mozzoli, M.; Ryan, I. Effect of fasting on serum leptin in normal human subjects. *J. Clin. Endocrinol. Metabol.* **1996**, *81*, 3419–3423.
63. Brabeau, P.; Gutin, B.; Litaker, M. S.; Ramsey, L. T.; Cannady, W. E.; Allison, J.; Lemmon, C. R.; Owens, S. Influence of physical training on plasma leptin in obese youths. *Canadian J. Appl. Physiol.* **2003**, *28*, 382–396.
64. Houmard, J. A.; Cox, J. H.; Maclean, P. S.; Barakat, H. A. Effect of short-term exercise training on leptin and insulin action. *Metabolism* **2000**, *49*, 858–861.
65. Kukuljan, S.; et al., Effects of resistance exercise and fortified milk on skeletal muscle mass, muscle size, and functional performance in middle -aged and older men: an 18-mo randomized controlled trial. *J. Appl. Physiol.*, **2009**, *707(6)*, 18 64–73.
66. Hickey, M. S.; Considine, R. V.; Israel, R. G. Leptin is related to body fat content in male distance runners. *Am. J. Physiol.* **1996**, *271*, E938-E940.
67. Sihna, M. K.; Caro, J. F. Clinical aspects of leptin. *Vitamins and Hormones* **1998**, *54*, 1–30.
68. Spicer, L. J. Leptin: a possible metabolic signal affecting reproduction. *Domestic Animal Endocrinology* **2001**, *21*, 215–250.
69. Kerssick, C. M.; Wismann-Bunn, J.; Fogt, D.; Thomas, A. R.; Taylor, L.; Campbell, B. I.; Wilborn, C. D.; Harvey, T.; Roberts, M. D.; La Bounty, P.; Galbreath, M.; Marcelllo, B.; Rasmussen, C. J.; Kreidercorresponding, R. B. Changes in weight loss, body composition and cardiovascular disease risk after altering macronutrient distributions during a regular exercise program in obese women, *Nutr. J.*; **2010**, *9*, 59.

70. Hammer, L. D. and Wilson, B. M. Impact of pubertal development on body fat distribution among white, *Hispanic J. Pediatr.*, **1991**, 88, 975–980.
71. Gippini, A.; Mato, A.; Peino, R.; Lage, M.; Dieguez, C.; Casanueva, F. F. Effect of resistance exercise (body building) training on serum leptin levels in young men. Implications for relationship between body mass index and serum leptin. *J. Endocrinol. Invest.* **1999**, 22, 824–828.
72. Thong FSL, Hudson, R.; Ross, R.; Janssen, I.; Grahan, T. E. Plasma leptin in moderately obese males: independent effects of weight loss and aerobic exercise. *Am. J. Physiol. Endocrinol. Metabol.* **2000**, 279, E307–E313.
73. Schulze, P. C.; Kratzsch, J.; Linke, A.; Schoene, N.; Adams, V.; Gielen, S. Elevated serum levels of leptin and leptin and leptin receptor in patients with advanced chronic heart failure. *Eur. J. Heart Fail.* **2003**, 5, 33–40.
74. Sesso, H. D.; Buring, J. E.; Rifai, N.; Blake, G. J.; Gaziano, J. M.; Ridker, P. M. C-reactive protein and the risk of developing hypertension. *J. Am. Med. Assoc.* **2003**, 290, 2945–2951.
75. Konstantinides, S.; Schafer, K.; Koschnick, S.; Loskutoff, D. J. Leptin-dependent Platelet aggregation and arterial thrombosis suggests a mechanism for atherothrombotic disease in obesity. *J. Clin. Invest.*; **2001**, 108, 1533–1540.
76. Kyeasiazis, G. A.; Caplan, J. D.; Lowndes, J.; Carpenter, R. L.; Dennis, K. E.; Sivo, S. A.; Angelopoulos T. J. Moderate exercise- induced energy expenditure does not alter leptin levels in sedentary obese men, *Clin. J. Sport Med.*; **2007**, 17, 49–51.
77. Mildred, T.; Consuelo L. Correlates of body image satisfaction among economically depressed urban Filipino women. *Philippine J. Sci.*, **2009**, O: 67–74.
78. Tamer, L.; Ercan, B.; Unlu, A.; Sucu, N.; Pekdemir, H.; Eskandari, G.; Atik, U. The relationship between leptin and lipids in atherosclerosis. *Indian Heart J.* **2002**, 54, 692–696.
79. McGill, H. C.; McMahan, A.; Hederick, E. E.; Zieske, A. W.; Malcom, G. T.; Tracy, R. E.; Strong, J. P. Obesity accelerates the progression of coronary atherosclerosis in young men. *Circulation* **2002**, 105, 2712–2717.
80. Tam, J.; Fukumura, D.; Jainl RK. A mathematical model of murine metabolic regulation by leptin: energy balance and defence of a stable body weight. *Cell Metab.* **2010**, 7; 9(1), 52–63.
81. Corsonello, A.; Malara, A.; Ientile, R. Leptin enhances adenosine diphosphate- induced platelet aggregation in healthy subjects. *Obes. Res.* **2002**, 10, 306–317.
82. Maughan, R. J.; J. B. Leiper, and G. E. Vist, Gastric emptying and fluid availability after ingestion of glucose and soy protein hydrolysate solutions in man. *Exp physiol*, **2004**, 89(1), 101–108.
83. Shamsuzzaman, A. S. M; Winnicki, M.; Wolk, R.; Svatikova, A. A.; Phillips, B. G.; Davison, D. E.; Berger, P. B.; Somers, V. K. Independent association between plasma leptin and C- reactive protein in healthy humans. *Circulation* **2004**, 109, 2181–2185.

84. Mc Connell, G.; Snow, R. J.; Proietto, J.; Hargreaves M. Muscle metabolism during prolonged exercise in humans: influence of carbohydrate availability. *J. Appl. Physio.* **1999**, *87*, 1083–1086.
85. Klein, S.; Horowitz, J. F.; Landt, M.; Goodrick, S. J.; Mohamed-Ali, V.; Coppack, S. W. Leptin production during early starvation in lean and obese women. *Am. J. Endocrinol. Metabol.* **2000**, *278*, E280-E284.
86. Rahmani-Nia, F.; Hojjati, Z. Effect of selected training on body composition and aerobic power of females college. *J. Harkat, Iran*, **2004**, *1*, 18–21.

CHAPTER 9

REGRESSION METHOD IN ENGINEERING AND SPORT

CONTENTS

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9.1 INTRODUCTION

It seems in healthy matured athletes there may be a correlation between Runner Velocity (Running Time), weight and heart rate. Some researchers and coaches can use a regression line by these variables for their selection and predict exercise and Running Time of their athletes from weight or heart rate [1–5].

Jump power and the anthropometric characteristics of professional basketball players were studied to assess the relationship between these attributes and a players' position.

The analysis can provide objective data for individualizing training. During the 2004 and 2005 preseasons, the anthropometric characteristics (body composition) and jump power (squat jump and countermovement jump) of members of an ACB (Spanish Basketball League) team were assessed. The centers showed significantly higher height, total weight, arm span, and fat and muscle weight when compared to the forwards and point guards. With regards to the jump test, the forwards showed significantly higher values for the countermovement jump than the point guards and centers, though there were no significant differences when they performed the squat jump. When the results were correlated, there were negative relationships between higher fat weight and body mass index with countermovement jump power. Centers should focus their training on achieving greater jump values, not losing body weight, which would make them lose position and physical presence in their area of play, but by gaining muscle tissue through loss of fat [6–10].

Anthropometry, jump power, and position 4 Jump power has been described as a factor affecting performance in basketball [11–15].

It varies according to a player's position, as it is affected by physical characteristics, which are adapted to the performance requirements of each position on the court. Determining the relationship of jump power with the anthropometric variables of the players, differentiated by the play position, provides objective data allowing strength and conditioning coaches to individualize players' training, adapting to their characteristics and the demands of the game.

Kenyan runners are well recognized for their success in distance running. As of today, male Kenyan (and East African) runners are in the top

of the International Association of Athletics Federation (IAAF) world lists in all events from 800 m to the marathon (Table 1). About three quarters of the top Kenyan runners come from the Kalenjin tribe, which makes up about 12% of Kenya's population and approximately 1/2,000 of the world's population. Yet, they have won about three-eighths of international men's distance running prizes and three times more Olympic medals in distance running than any other whole nation. These performances have been described as the greatest geographical concentration of achievement in the annals of sport [16–20].

TABLE 1 Field tests and regression anthropometric model for velocity (Running Time).

No.	Age	Weight (kg)	Dynamic Heart Rate	Static Heart Rate	100 m Running Time (sec)
1	19	50	176	74	13.45
2	16	52	173	72	13.66
3	18	47	174	70	15.03
4	16	50	111	70	13.67
5	14	52	173	79	14.3
6	14	44	178	77	14.73
7	14	54	191	111	17.1
8	20	46	133	83	17.55
9	21	48	160	82	16.28
10	14	52	150	79	16.03
11	18	56	153	75	16.27
12	15	55	166	81	16.77
13	23	45	140	94	17.35

TABLE 1 *(Continued)*

No.	Age	Weight (kg)	Dynamic Heart Rate	Static Heart Rate	100 m Running Time (sec)
14	22	56	154	73	15.22
15	21	59	164	79	14.98
16	21	55	177	77	14.9
17	22	62	93	75	14.51
18	28	55	170	68	13.98
19	22	56	166	70	14.45
20	24	62	168	83	16.98

Researchers have attempted to gain insight into contributing factors behind the remarkable performance of these Kalenjin athletes. Their success has been hypothesized to be related to the fact that Kenyans generally live and train at high altitude (around 2,000 m above sea level) influencing their oxygen capacity. However, research has not supported this hypothesis as no difference was found in maximal oxygen uptake (VO_{2max}) between elite Kenyan and Scandinavian runners or between untrained Kenyan and Danish adolescents [21–24].

Another hypothesis states that the athletes' nutrition may have contributed to success in running. While Kenyan runners' diet matched the recommendations by the Food and Agriculture Organization/World Health Organization/United Nations University (FAO/WHO/UNU) for endurance athletes for macronutrients intake, it was far from adequate for vitamin and mineral intake. During intense training periods prior to competition, Kenyan runners are in negative energy balance leading to body mass reduction that may potentially contribute to short-term success by reducing the energy cost of running. However, no association was found between nutrition and marathon performance in African runners.

Endurance running performance is positively related to type I fibers in skeletal muscles but this does not explain the success of Kenyan runners as muscle fiber size and composition are similar between elite Kenyan and Scandinavian runners. Recent research has evaluated the genetic expression in elite Kenyan runners but no association between their genetic makeup and performance has been confirmed. Another popular presumption Research article Biomechanical characteristics of Kenyan runners 500 is that Kenyan children run long distances to school.

Compared to the general Kenyan population, elite Kenyan runners travelled further to school, mostly by running [25–26]. While there was no difference in aerobic trainability between Kenyan village and town boys, it has been speculated that the physical activity in childhood, combined with intense training as teenagers, were related to the high aerobic capacity in Kenyan runners. Other factors that may be positively associated with African distance runners' success include their slender body shape, good running economy, and higher fractional utilization of $\text{VO}_{2\text{max}}$.

In running, biomechanical factors can contribute to success in performance in terms of improving running economy and preventing injury. Running economy has been shown to correlate with certain gait characteristics such as stride length, ground contact time, vertical oscillation and lower extremity angles. Lower extremity injuries are related to altered running mechanics and imbalances in muscle strength.

It is possible that Kenyan runners run in a form that positively contributes to their superior performance and keeps them free from injury. To our knowledge, the only biomechanical study on Kenyan runners available in English. This study reported kinematic differences between elite Kenyan and Japanese runners and concluded that the Kenyan runners were able to swing their leg forward faster and through a greater range. Although limited data were presented in this abstract, their findings highlight that biomechanical factors may play a significant role in the success of Kenyan distance runners.

This study aimed to take a biomechanical approach to contribute to the understanding of the success of Kenyan runners. Anthropometric, gait and lower extremity strength characteristics of six elite Kenyan distance runners were analyzed [27–29].

9.2 MATERIALS AND METHODS

Present book shows a computational modeling for anthropometric of athletes. According to this technique, in this work an improved modeling based on measurements was classified and compared with a special type of weights of sportswomen with other anthropometric classes of sportswomen. On the other hand present work compares the anthropometric data for Runner Velocity (Running Time) related to heart rate and runner weight in two cases. Consequently, the results of this work will help to reduce the risk of sportswomen activities.

In this work, dateline for field tests data collection was: at 11:00 a.m., 10/09/2012 until 20/09/2012. Locaton of work field tests model was at Rasht city in the north of Iran. In this work the following are considerations was made when using and applying anthropometric data for heart rate of runner related to Runner Velocity (Running Time) and runner weight. Heart rate of runner was measured in two cases: first at the running state (dynamic heart rate) and second at the before of running (static heart rate). Twenty healthy and young sprint runner females (Table 1) participate in this study. They signed a written/informed consent [30–33].

All subjects completed a medical questionnaire to ensure that they were not taking any medication, and were free cardiac, respiratory, renal or metabolic diseases. They didn't have any orthopaedic injury that would inhibit physical activity. The subjects were tested in the follicular phase of the menstrual cycle based on their previous menses. All participants consumed their last meal at least 2 hours before the test and refrained from intensive exercise in the 24 hours period preceding testing. All anthropometric measures were recorded in the morning by the same experienced anthropometrist. The weight was measured using a wall-mounted stadiometer (± 0.1 cm). The exercise systolic and diastolic heart rates were measured on left arm of each participant using cuffs of appropriate size (Table 1). The measurements were recorded just after 100 m intense running in standing position. In present work, for professional runner at fast running, heart rate of runner, Runner Velocity and weight were recorded. Curve estimation procedure has used these data, which have been detected field tests (Table 1). These data have been compared by heart rate of runner and runner velocity, which have been collected from actual systems (field

tests). The model was calibrated using one set of data, without changing parameter values [34–37].

It was used to match a different set of results. Regression model (Table 1) has been built based on field tests data. Scatter diagram, correlation (Figs. 1–5) and autocorrelations procedure for blood pressure was formed by estimating regression statistics and producing related plots for field tests model (Table 1).

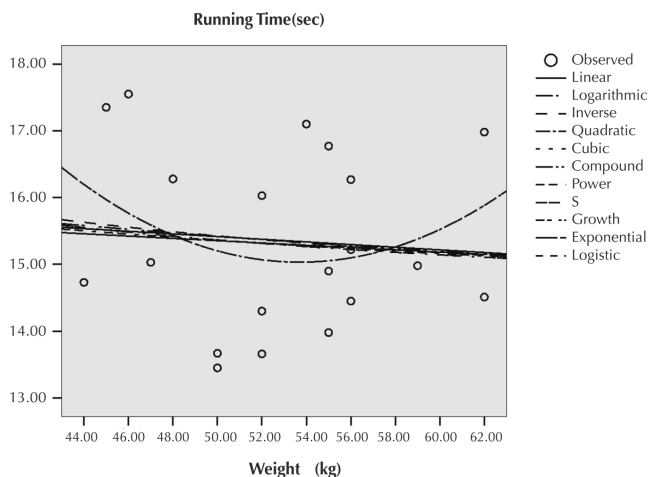


FIGURE 1 Curve fit for Running Time against weight.

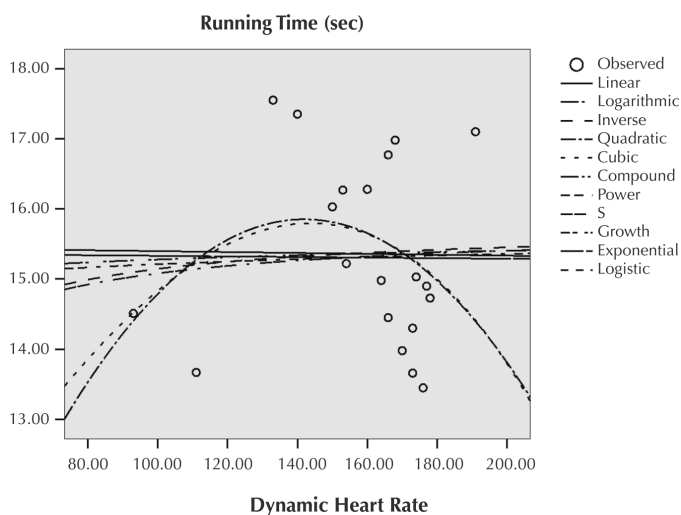


FIGURE 2 Curve fit for Running Time against dynamic heart rate.

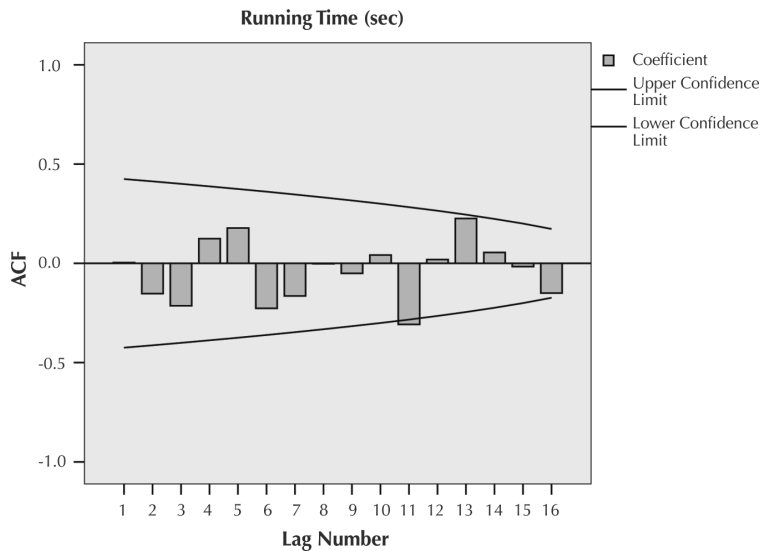


FIGURE 3 Autocorrelations for Running Time against dynamic heart rate.

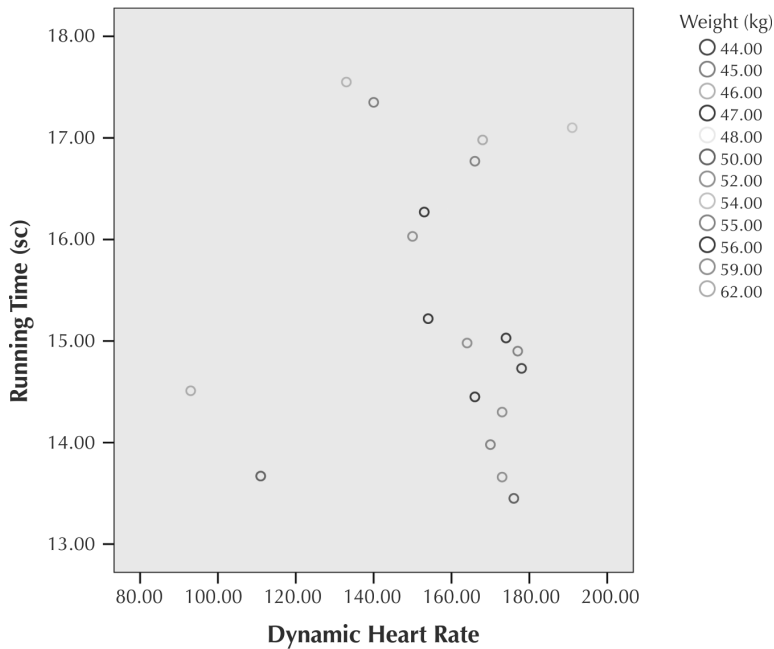


FIGURE 4 Scatter diagram for Running Time against dynamic heart rate.

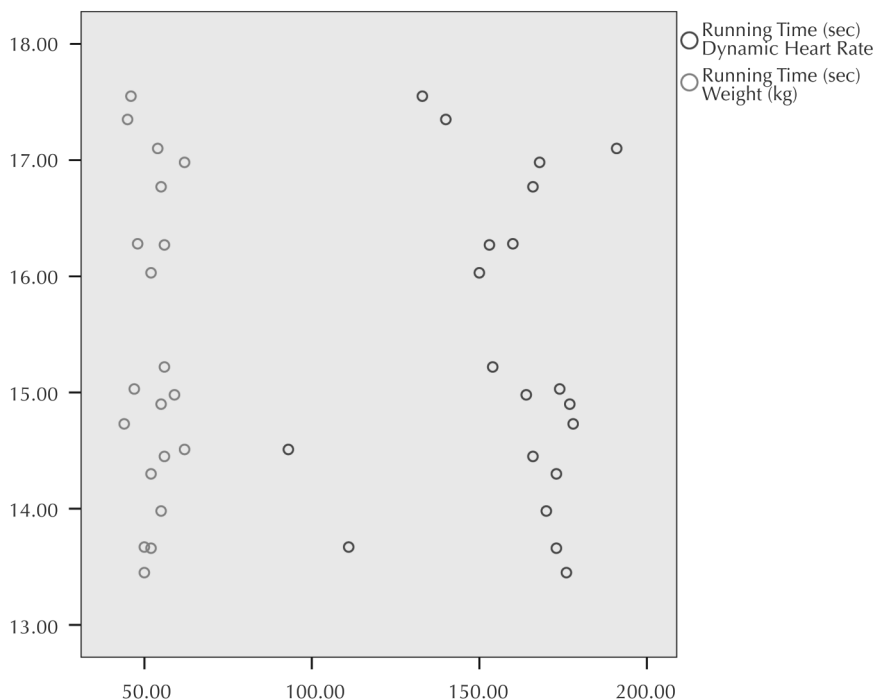


FIGURE 5 Correlation for Running Time against dynamic heart rate.

9.3 RESULTS AND DISCUSSION

In this work conclusions were drawn on the basis of experiments and calculations for independent variables (running weight and runner heart rate) based on the two cases:

Case 1:

$$V = f(W) \quad (1)$$

Case 2:

$$V = f(H) \quad (2)$$

Dependent variable: V – Runner Velocity (Running Time), with nomenclature “ Y ” for first: at the running state and the second: before of running condition.

Independent variable with nomenclature “ X ” such as:

W – runner weight (kg) is the most important variable, H – runner heart rate.

Case 1:

$$V = f(W) \tag{1}$$

The most important effects that were observed based on regression for model summary (Table 2) are as follows (Fig. 1).

TABLE 2 Model summary and parameter estimates for running time.

Dependent Variable: Running Time (sec).									
Equation	Model Summary					Parameter Estimates			
	R-square	F	df1	df2	Sig.	Constant	b1	b2	b3
Linear	.006	.110	1	18	.744	16.410	-.020		
Logarithmic	.009	.156	1	18	.697	20.281	-1.242		
Inverse	.012	.210	1	18	.652	13.933	74.681		
Quadratic	.082	.761	2	17	.482	50.686	-1.328	.012	
Cubic	.082	.761	2	17	.482	50.686	-1.328	.012	.000
Compound	.005	.083	1	18	.776	16.240	.999		
Power	.007	.125	1	18	.728	20.344	-.072		
S	.010	.173	1	18	.682	2.644	4.396		
Growth	.005	.083	1	18	.776	2.787	-.001		
Exponential	.005	.083	1	18	.776	16.240	-.001		
Logistic	.005	.083	1	18	.776	.062	1.001		

The independent variable is Weight (kg).

Present work compared the anthropometric data for blood pressure of runner related to Runner Velocity (Running Time) and runner heart rate in two cases. The dependent and independent variables of runner were measured in the following conditions [38]:

First: at the running state at dynamic condition (Figs. 2–5).

Second: at the before of running (Table 5) at static condition (Fig. 6).

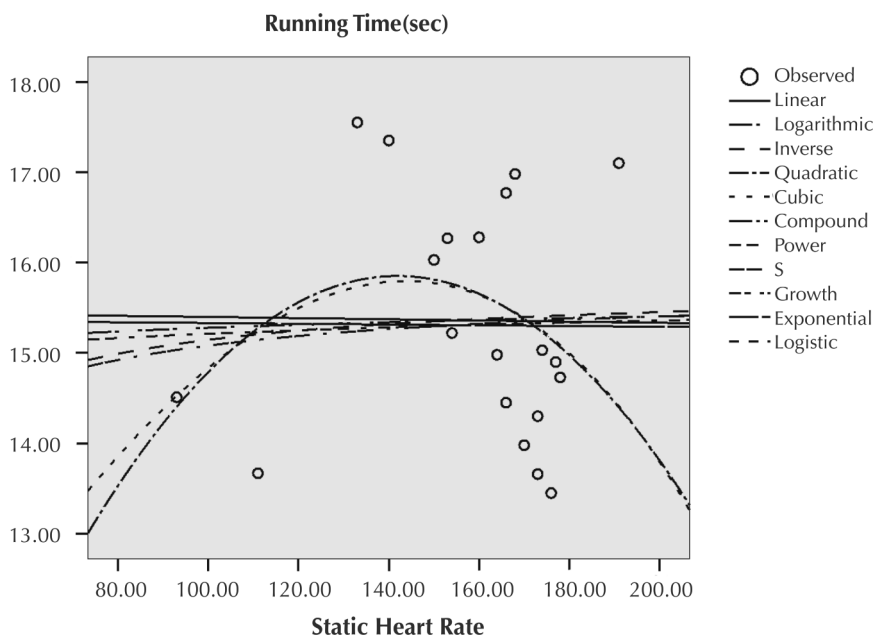


FIGURE 6 Correlation for Running Time against static heart rate.

Case 2:

The independent variable is heart rate.

$$V = f(H) \quad (2)$$

In second assumption for static heart rate against Running Time and for runner dynamic heart rate against runner weight, the curve estimation procedure (Figs. 1–5) allows quick estimating regression statistics (Tables 2–5), and producing related plots for different models. Hence the auto-regression procedure by regression software “SPSS 10.0.5” was selected for the curve estimation procedure in the present work. The regression model

TABLE 3 Parameter estimates for running time.
Dependent Variable: Running Time (sec).

Equation	Model Summary					Parameter Estimates			
	R-square	F	df1	df2	Sig.	Constant	b1	b2	b3
Linear	.000	.002	1	18	.961	15.464	-.001		
Logarithmic	.001	.010	1	18	.921	14.437	.183		
Inverse	.004	.067	1	18	.799	15.760	-61.517		
Quadratic	.097	.910	2	17	.421	3.672	.172	-.001	
Cubic	.082	.762	2	17	.482	8.095	.080	.000	-1.30E-006
Compound	.000	.001	1	18	.975	15.373	1.000		
Power	.001	.014	1	18	.907	14.273	.014		
S	.004	.075	1	18	.787	2.756	-4.226		
Growth	.000	.001	1	18	.975	2.733	-2.74E-005		
Exponential	.000	.001	1	18	.975	15.373	-2.74E-005		
Logistic	.000	.001	1	18	.975	.065	1.000		

The independent variable is Dynamic Heart Rate.

has been built based on field test data. Regression software “SPSS” has fitted the function curve and provided regression analysis. So, the regression model has been found in the final procedure. Although related plots for the field test model were produced. Regression software “SPSS 10.0.5” performs multidimensional scaling of proximity data to find least-squares representation of the objects in a low-dimensional space. The curve estimation procedure allows quickly estimating regression statistics and producing related plots for different models.

TABLE 4 Autocorrelations for Running Time.

Series: Running Time (sec).

Lag	Autocorrelation	Std.Error (a)	Box-Ljung Statistic		
			Value	df	Sig. (b)
1	.004	.212	.000	1	.987
2	-.152	.206	.545	2	.762
3	-.214	.200	1.687	3	.640
4	.124	.194	2.098	4	.718
5	.178	.187	3.004	5	.699
6	-.227	.181	4.580	6	.599
7	-.164	.173	5.478	7	.602
8	-.002	.166	5.478	8	.705
9	-.050	.158	5.578	9	.781
10	.042	.150	5.656	10	.843
11	-.308	.142	10.385	11	.496
12	.019	.132	10.406	12	.580
13	.226	.123	13.806	13	.388
14	.055	.112	14.047	14	.446
15	-.016	.100	14.074	15	.520
16	-.150	.087	17.053	16	.382

^a The underlying process assumed is independence (white noise).

^b Based on the asymptotic chi-square approximation.

TABLE 5 Parameter estimates for Running Time against static heart rate.

Equation	Model Summary					Parameter Estimates			
	R-square	F	df1	df2	Sig.	Constant	b1	b2	b3
Linear	.000	.002	1	18	.961	15.464	-.001		
Logarithmic	.001	.010	1	18	.921	14.437	.183		
Inverse	.004	.067	1	18	.799	15.760	-61.517		
Quadratic	.097	.910	2	17	.421	3.672	.172	-.001	
Cubic	.082	.762	2	17	.482	8.095	.080	.000	-1.30E-006
Compound	.000	.001	1	18	.975	15.373	1.000		
Power	.001	.014	1	18	.907	14.273	.014		
S	.004	.075	1	18	.787	2.756	-4.226		
Growth	.000	.001	1	18	.975	2.733	-2.74E-005		
Exponential	.000	.001	1	18	.975	15.373	-2.74E-005		
Logistic	.000	.001	1	18	.975	.065	1.000		

The independent variable is Static Heart Rate / Dependent Variable: Running Time (sec).

Curve estimation is most appropriate when the relationship between the dependent variable(s) and the independent variable is not necessarily linear. Linear regression is used to model the value of a dependent scale variable based on its linear relationship to one or more predictors. Nonlinear regression is appropriate when the relationship between the dependent and independent variables is not intrinsically linear. Binary logistic regression is most useful in modeling of the event probability for a categorical response variable with two outcomes. The auto regression procedure is an extension of ordinary least-squares regression analysis specifically designed for time series.

One of the cases underlying ordinary least-squares regression is the absence of autocorrelation in the model residuals. Time series, however, often exhibit first-order autocorrelation of the residuals. In the presence of auto-correlated residuals, the linear regression procedure gives inaccurate estimates of how much of the series variability is accounted for by the chosen predictors. This can adversely affect choice of predictors and hence the validity of the model. The auto regression procedure accounts for first-order auto correlated residuals. It provides reliable estimates of both goodness-of-fit measures and significance levels of chosen predictor variables [39–90]. The auto regression procedure by regression software “SPSS 10.0.5” has been selected for the curve estimation procedure in present work. The regression model includes:

Case 1:

$$V = f(W) \quad (1)$$

Case 2:

$$V = f(H) \quad (2)$$

It has been built based on field tests data and in the final procedure they were compared by them.

9.3.1 COMPARISON WITH OTHER WORKS

Rahmani-Nia and Hojjati obtained the effect of exercise training on body composition and aerobic power of females. Comparison showed similarity in present work and the results of that works [82].

9.4 CONCLUSIONS

This work showed the effects of the Runner Velocity as the most important variable and runner weight against runner heart rate. Present work emphasized that “regression model” is accurate model for modeling of anthropometric and body motion. Hence in order to presentation for influence of runner weight on Runner Velocity, it was compared the models for computational and field tests experiments. Based on these procedures, it was showed that the regression based model for anthropometric of body motion. Therefore the dynamic or running state was compared by static or before of running condition. On the other hand, this idea were included the proper analysis to provide a dynamic response to the shortcomings of the body motion. Because increased vagal activity has been associated with a reduction in the risk of sudden cardiac death, this work has demonstrated that a delayed decrease in HR after exercise would be a powerful and independent predictor of all cause mortality in patients or in general population. Consequently, the results of this work will help to reduce the risk of anthropometric of female athletes sport women activities.

NOMENCLATURES

P	Runner Blood Pressure (mm-Hg)
t	Running Time (sec)
V	Velocity (m/s)
S	Runner Stature (cm)

KEYWORDS

- anthropometric
- blood pressure
- body motion
- computational model
- regression

REFERENCES

1. Hurst Thomas, D. Skull Wars Kennewick Man, Archaeology and the Battle for Native American Identity, Washington University: School of Law - Anheuser-Busch Hall, 2001; 38–41.
2. Nabieh, A.; Mohamed, I. Anthropometric Measurements as a Significant for Choosing Juniors in Both Volleyball and Handball Sports (Factorial Analysis Study), *World J. Sport Sci.* **2010**, 3(4), 277–289.
3. Rogana, S.; Hilfiker, R.; Clarys, P.; Clijsen, R.; Taeymans, J. Position-specific and Team-ranking-related Morphological Characteristics in German Amateur Soccer Players – a Descriptive Study, *Int. J. Appl. Sports Sci.*, **2011**, 23(1), 168–182.
4. Luigi P. W.; Bercades, T. somatotypes of national elite combative sport athletes, *Braz. J. Biomotricity*, **2009**, 3(1), 21–30.
5. Dacres-Mannings, S.; Rochester, S.; Frail, H. Anthropometric profiles of Australian Rugby Institute, club and state level Rugby Union players. **2010**.
6. Gaurav, V.; Singh, M.; Singh, S. Anthropometric characteristics, somatotyping and body composition of volleyball and basketball players, *J. Phys. Educ. Sports Manage.* **2010**, 1(3), 28–32, Available online <http://www.acadjourn.org/jpesm>
7. Beat K.; Kohler, G. Influence of anthropometry on race performance in ultra-endurance triathletes in the longest triathlon in North America, *Int. SportMed J.*, **2007**, 8(2), 87–96, <http://www.ismj.com>
8. Joseph, J.; Greene, M. S.; Timothy, A.; McGuine, A. T. C.; Leverson, G.; Thomas M. Anthropometric and Performance Measures for High School Basketball Players, *J. Athl. Training*, **1998**, 33(3), 229–232
9. Amatya, D. L. Comparative Study of Somatotype of Nepalese Sportsmen, National Association for Sports Health and Fitness. **1999**.
10. Sang Hong, K. Kinanthropometric Study of Korean and Nepalese Marathon Runners, keimyung university Diwakar Lal Amatya, MSC-Athletics and Sports Science-India, Sports Kinanthropometrist-Australia, Sports Expert-National Sports Council, January. **2008**.
11. Gaunt, B. W. Anthropometric and Demographic Factors Affecting Distance Hopped-aid Limb Symmetry Index for the Crossover Hop-for distance test in High School athletes, *J. Orthop. Sport Phys.*, **2001**, 31(3), 145–151
12. Gross, M.; Dailey, E. S.; Melissa, D.; Dalton, A.; Lee, K.; Wendy, I.; Ashley, C. Relationship Between Lifting Capacity and Anthropometric Measures, *J. Orthop. Sport Phys.*, **2000**, 30(5), 237–247, 258–262.
13. Roberto, C.; Jose, A.; Perez, J.; Cortell, m. Juan, J.; Rivas, J. Correlations among anthropometric parameters, jump power, and position in professional basketball players, **2008**.
14. Pui, W. K.; Hendrik H. Anthropometric, gait and strength characteristics of Kenyan distance runners, *J. Sports Sci. Med.* **2008**, 7, 499–504, <http://www.jssm.org>
15. Jawis, M. N.; Singh, R.; Singh, H. J.; Yassin, M. N. Anthropometric and physiological profiles of sepak takraw players, *Br. J. Sports Med.* **2005**, 39, 825–829. doi: 10.1136/bjism.2004.016915.

16. Lephart, S. M.; Abt, J. P.; Ferris, C. M.; Sell, T. C.; Nagai, T.; Myers, J. B.; Irrgang, J. Neuromuscular and biomechanical characteristic changes in high school athletes: a plyometric versus basic resistance program, *Br. J. Sports Med.* **2005**, *39*, 932–938. doi: 17.1136/bjsm.2005.019083.
17. Darr, K. C.; Bassett, D. R.; Morgan, B. J.; Thomas, D. P. Effects of age and training status on heart rate recovery after peak exercise. *Am. J. Physiol.* **1988**, *254*, H340–H343.
18. Engerman, S. ‘The Height of U. S. Slaves’, *Local Population Studies*, **1976**, *16*(1), 45–50.
19. Sunder, M. The Making of Giants in a Welfare State: The Norwegian Experience in the Twentieth Century’, *Economics and Biology*, **2004**, 75–86.
20. Woitek, U. Height Cycles in the Eighteenth and Nineteenth Centuries’, *Econ. Hum. Biol.*, **2003**, 243–258.
21. Fogel, R. ‘Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the making of Economic Policy’, *Am. Econ. Rev.*, **1994**, 369–394.
22. Goonetilleke, R. S.; Ho, Cheuk Fan E.; So, R. H. Y. Foot Anthropometry in Hong Kong. (1997, pp. 81–88) Proceedings of the ASEAN 97 Conference, Kuala Lumpur, Malaysia.
23. Baten, J. “Economic Development and the Distribution of Nutritional Resources in Bavaria, 1797–1839, in *J. Income Distrib.* **2000**, *9*, 89–106.
24. Hariri Asli K. Water Hammer Research; Advances in Nonlinear Dynamics Modeling, (2012, pp. 88–121). Canada, USA: Published by Apple Academic Press, Inc., Exclusive worldwide distribution by CRC Press, a Taylor and Francis Group, Print ISBN: 9781926895314, eBook: 978-1-46-656887-7.
25. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. “Some Aspects of Physical and Numerical Modeling of Water Hammer in Pipelines,” *Nonlinear Dynamics An International Journal of Nonlinear Dynamics and Chaos in Engineering Systems*, ISSN: 1573–269X (electronic version) Journal /1071 Springer, Netherlands, **2009**, ISSN: 0924–090X (print version), Heidelberg, Germany, Springer, Volume 60, Number 4 / June, **2010**, 677–701.
26. Heather, E. Collins-Schramm and others, “Markers that Discriminate Between European and African Ancestry Show Limited Variation Within Africa,” *Human Genetics* **2002**, *111*, 566–9.
27. Komlos, J.; Baur, M. “From the Tallest to One of the Fattest: The Enigmatic Fate of the Size of the American Population in the Twentieth Century,” *Econ. Hum. Biol.* **2004**, *2*(1), 57–74.
28. Komlos, J.; Kriwy, P. “The Biological Standard of Living in the Two Germanies,” *German Econ. Rev.* **2003**, *4*, 493–507.
29. Levy, W. C.; Cerqueira, M. D.; Harp, G. D.; Johannessen, K. A.; Abrass, I. B.; Schwartz, R. S.; Stratton, J. R. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. *Am. J. Cardiol.*, **1998**, *82*, 1236–1241.
30. Margo, R. The Heights of American Slaves. *Social Science History*, **1982**, *6*, 516–538.
31. Rahmani-Nia, F.; Hojjati, Z. The effect of exercise training on body composition and aerobic power in sedentary college age females, *Int. J. Fitness*, India, **2005**, *1*, 24–31.

32. Rahmani-Nia, F.; Rahnama, N.; Hojjati, Z.; And Soltani, B. Acute effects of aerobic and resistance exercise on serum leptin and some risk factors of coronary heart disease in obese females. *Sport sciences for health*, **2008**, 2(3), 118–124.
33. Rahmani-Nia, F.; Hojjati, Z.; Rahnama, N.; Soltani, B. Leptin, heart disease and exercise. *World J. Sport Sci.*, Iran, **2009**, 2(1), 13–20.
34. Rushton, J. P. Race, Brain Size, and Intelligence: A Rejoinder to Cain and Vanderwolf, Personality and Individual Differences, **2004**, 11, 785–794.
35. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. A computational approach to study fluid movement, *Nanomaterials Yearbook – 2009*, From Nanostructures, Nanomaterials and Nanotechnologies to Nanoindustry, (2010, pp. 181–196). USA: Nova Science Publications. ISBN: 978-1-60876-451-8.16
36. Young, J. W. Anthropometric and Mass Distribution Characteristics of the Adult Female FAA Civil Aeromedical Institute **2008**, FAA 9–83.
37. Bartlett, R. Introduction to Sports Biomechanics, 2nd Edition. Taylor andFrancise- Library. **2007**.
38. Hariri Asli, K.; Haghi, A. K. A Numerical Study on Fluid Flow and Pressure drop in Microtubes, *J. Balkan Tribological Association*, Tribotechnics and tribomechanics, Sofia, Bulgaria, **2010**, 16(3), 382–392.
39. Schulze, P. C.; Kratzsch, J.; Linke, A.; Schoene, N.; Adams, V.; Gielen, S. Elevated serum levels of leptin and leptin and leptin receptor in patients with advanced chronic heart failure. *Eur. J. Heart Fail.* **2003**, 5, 33–40.
40. Unal, M.; Unal, D. D. O; Baltaci, A. K.; Mguilkoc, R. Investigation of serum leptin levels and Vo_{2max} value in trained young male athletes and healthy males. *Acta Physiol. Hungary* **2005**, 92, 173–179
41. Askari, H.; Tykodi, G.; Liu, J.; Dagogo-Jack S. Fasting plasma leptin level is a surrogate measure of insulin sensitivity. *J. Clin. Endocrinol. Metab.*; **2010**, 95(8), 3836–3843.
42. Reseland, J. E.; Andessen, S. A.; Solvoll, K.; Hjermann, I.; Urdal, P.; Holme, I.; Drevon, C. A. Effect of long-term change in diet and exercise on plasma leptin concentration. *Clin. Nutr.* **2001**, 73, 240–245.
43. Mokdad, A. H.; Ford, E. S.; Bowman, B. A.; Dietz, W. H.; Vinicor, F.; Bales, V. S.; Marks, J. S. Prevalence of obesity, *Diabetes* and obesity-related health risk factors, 2001. *JAMA* **2003**, 289, 76–79.
44. Mendosa-Nunez, V. M.; Garcia-Sanchez, A.; Sanchez-Rodriguez, M.; Galvan-Duart, R. E.; Fonseca-Yerena, E. F. Overweight, waist circumference, age, gender, and insulin resistance as risk factors for Hyperleptinemia. *Obes. Res.* **2002**, 10, 253–259.
45. Frank, L. L.; Sorensen, B. E.; Yasui, Y.; Tworoger, S. S.; Schwartz, R. S.; Ulrich, C. M.; Irwin, M. L.; Rudolph, R. E.; Rajan, K. B.; Stanczyk, F.; Bowen, D.; Weigle, D. S.; Potter, J. D.; McTiernan, A. Effects of exercise on metabolic risk variables in overweight postmenopausal women: a randomized clinical trial. *Obes. Res.*; **2005**, 13, 615–625.
46. Racette, S. B.; Coppack, S. W.; Landt, M.; Klein, S. Leptin production during moderate-intensity aerobic exercise. *J. Clin. Endocrinol. Metabol.* **1997**, 82, 2275–2277.
47. Altman, R.; Risk factors in coronary atherosclerosis athero-inflammation: the meeting point. *Thrombosis J.*; **2003**, 1, 4–7.

48. Stefanie, L.; Martina, E. Physical activity and endogenous sex hormones in postmenopausal women. *Cancer Causes Control*, **2011**, 22, 81–89.
49. Akbarzadeh, R. Relationship between the ratio of waist circumference to hip circumference and risk factors and increased lipids and blood pressure in patients with insulin-dependent *Diabetes* referred to the Endocrinology Institute in Tehran in 1995 and 1996. A Master's thesis, Iran University of Medical Sciences; Faculty of Nursing. **1996**.
50. Ashwell, M.; Lejeane S. Ratio of waist circumference to height maybe better indicator need for weight management. *BMI*: **1990**, 312–377.
51. McMahon, M.; Skaggs, B. J.; Sahakian, L.; Grossman, J.; FitzGerald, J.; Ragavendra, N.; Charles-Schoeman, C.; Chernishof, M.; Gorn, A.; Witztum, J. L.; Wong, W. K.; Weisman, M.; Wallace, D. J.; La Cava, A.; Hahn BH. High plasma leptin levels confer increased risk of atherosclerosis in women with systemic lupus erythematosus, and are associated with inflammatory oxidised lipids. *Ann. Rheum. Dis.* **2011**, 70(9), 1619–1624.
52. Delavar A. Research methods in psychology and educational sciences; 1st edition; Tehran; Payame Noor University Press. **2000**.
53. McHugh, P. R.; T. H. Moran, Calories and gastric emptying: a regulatory capacity with implications for feeding. *Am. J. Physiol.* **1979**, 236(5), R 254–60.
54. Shirreff, S. M.; P. Watson, and R. J. Maughan, Milk and effective post – exercise rehydration drink. *Br. J. Nutr.*, **2007**, 98(11), 173–80.
55. Merson S. J.; Shirreff S. M.; Leiper J. B.; Maughan R. J: Changes in blood plasma and red cell volume after ingestion of hypotonic and hypertonic solutions. *Proc. Nutr. Soc.* (In the Press), 2002.
56. Melov, S. et al., Resistance exercise reverses aging in human skeletal muscle. *PLOS One*. **2007**, 2(5), E 465.
57. Miller, S. L.; et al., Metabolic response to provision of mixed protein – carbohydrate supplementation during endurance exercise. *Int. J. Sport Nutr. Exerc. Metab.*, **2002**. 12(4), 384–97.
58. Miller S. L.; et al., The effects of nutritional supplementation throughout an endurance run on leucine kinetics during recovery. *Int J Sport Nutr Metab*, **2007**, 17(5), 456–67.
59. Cooke, J. P.; Oka, R. K. **2002**, Does leptin cause vascular disease? *Circulation* 106, 1904–1905.
60. Canavan, B.; Salem, R. O.; Schurgin, P. K.; Lipinska, I.; Laposata, M.; Grinspoon S. Effect of physiological leptin administration on markers of inflammation, platelet activation, and platelet aggregation during caloric deprivation. *The J. Clin. Endocrinol. Metabol.* **2005**, 90(10), 5779–5785.
61. Cooke, J. P.; Oka, R. K. Does leptin cause vascular disease? *Circulation* **2002**, 106, 1904–1905.
62. Ahima, A. S.; Flier, J. S. Adipose tissue as an endocrine organ. *Trends Endocrinol. Metabol.* **2000**, 11, 327–331.
63. Altman, R. Risk factors in coronary atherosclerosis athero-inflammation: the meeting point. *Thrombosis J.* **2003**, 1, 4–7.
64. Konstantinides, S.; Schafer, K.; Koschnick, S.; Loskutoff, D. J. Leptin-dependent Platelet aggregation and arterial thrombosis suggests a mechanism for atherothrombotic disease in obesity. *J. Clin. Invest.* **2001**, 108, 1533–1540.

65. Kaye, S. A.; Fdosom, A. R.; Jacobs, D. R. Psychosocial correlates of body fat distribution in Black and white young adult. *Int. J. Obes.*, **1995**, *17*, 271–277
66. Montain S. J.; Coyle E. F.: Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J. Appl. Physiol.* **1992a**, *73*, 1340–1350.
67. Meier, U.; Gressner, A. M. Endocrine regulation of energy metabolism: review of pathobiology and clinical chemical aspects of leptin, ghrelin and adiponectin, and resistin. *Cli. Chem.* **2004**, *50*, 1511–1525.
68. Mokdad, A. H.; Ford, E. S.; Bowman, B. A.; Dietz, W. H.; Vinicor, F.; Bales, V. S.; Marks, J. S. Prevalence of obesity, *Diabetes* and obesity-related health risk factors, 2001. *J. Am. Med. Assoc.* **2003**, *289*, 76–79.
69. Ostlund, R. E.; Jr. Yang, S.; Gingerich, R. Relation between plasma leptin concentration and body fat, gender, diet, age, and metabolic covariates. *J. Clin. Endocrinol. Metabol.* **1996**, *81*, 3909–3913.
70. Baratta, M. Leptin from a signal of adiposity to a hormone mediator in peripheral tissues. *Med. Sci. Monit.* **2002**, *8*, RA282-RA292.
71. Gomez-Merino, D, Chennaoui, M, Drogou, C, Bonneau, D, Guezennec, CY, Decrease in serum leptin after prolonged physical activity in men, *Med. Sci. Sports Exerc.*; **2002**, *34*, 1594–1599.
72. Boden, G.; Chen, X.; Mozzoli, M.; Ryan, I. Effect of fasting on serum leptin in normal human subjects. *J. Clin. Endocrinol. Metabol.* **1996**, *81*, 3419–3423.
73. Brabeau, P.; Gutin, B.; Litaker, M. S.; Ramsey, L. T.; Cannady, W. E.; Allison, J.; Lemmon, C. R.; Owens, S. Influence of physical training on plasma leptin in obese youths. *Canadian J. Appl. Physiol.* **2003**, *28*, 382–396.
74. Houmard, J. A.; Cox, J. H.; Maclean, P. S.; Barakat, H. A. Effect of short-term exercise training on leptin and insulin action. *Metabolism* **2000**, *49*, 858–861.
75. Kukuljan, S.; et al., Effects of resistance exercise and fortified milk on skeletal muscle mass, muscle size, and functional performance in middle-aged and older men: an 18-mo randomized controlled trial. *J. Appl. Physiol.*, **2009**, *707(6)*, 18 64–73.
76. Hickey, M. S.; Considine, R. V.; Israel, R. G. Leptin is related to body fat content in male distance runners. *Am. J. Physiol.* **1996**, *271*, E938-E940.
77. Sihna, M. K.; Caro, J. F. Clinical aspects of leptin. *Vitamins and Hormones* **1998**, *54*, 1–30.
78. Spicer, L. J. Leptin: a possible metabolic signal affecting reproduction. *Domestic Animal Endocrinology* **2001**, *21*, 215–250.
79. Kerkick, C. M.; Wismann-Bunn, J.; Fogt, D.; Thomas, A. R.; Taylor, L.; Campbell, B. I.; Wilborn, C. D.; Harvey, T.; Roberts, M. D.; La Bounty, P.; Galbreath, M.; Marcello, B.; Rasmussen, C. J.; Kreidercorresponding R. B. Changes in weight loss, body composition and cardiovascular disease risk after altering macronutrient distributions during a regular exercise program in obese women, *Nutr. J.*; **2010**, *9*, 59.
80. Hammer, L. D. and Wilson, B. M. Impact of pubertal development on body fat distribution among white, *Hispanic J. Pediatr.*, **1991**, *88*, 975–980.
81. Gippini, A.; Mato, A.; Peino, R.; Lage, M.; Dieguez, C.; Casanueva, F. F. Effect of resistance exercise (body building) training on serum leptin levels in young men. Implications for relationship between body mass index and serum leptin. *J. Endocrinol. Invest.* **1999**, *22*, 824–828.
82. Rahmani-Nia, F.; Hojjati, Z. Effect of selected training on body composition and aerobic power of Females College. *J. Harkat, Iran*, **2004**, *1*, 18–21.

CHAPTER 10

SPORTSWOMEN ACTIVITIES—FROM THEORY TO PRACTICE

CONTENTS

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10.1 INTRODUCTION

The literature indicates that relatively little research is available to describe the relationship between functional running tasks and characteristics of individuals who perform these tasks. Considerable research does exist, however, indicating significant relationships between the strength of specific muscle groups and anthropometric and demographic variables.

- Anthropometry is the most common technique used to assess the presence and degree of protein-energy malnutrition.
- Anthropometry is the measurement of body parameters to indicate nutritional status.
- Anthropometry can be used to measure an individual to determine if he or she needs nutrition intervention or it can be used to measure many individuals to determine if malnutrition is a problem in a population.

Some common anthropometric measurements include:

- Height or length
- Weight
- Mid-upper arm circumference (MUAC)
- Demi-span or arm span
- Knee height
- Sitting height
- Skin fold thickness
- Head circumference

Height (or length) and weight are the most common anthropometric measures used to indicate protein-energy nutritional status in emergencies.

Several studies, however, indicate the value of using multiple variables in regression analyses to predict computational modeling for anthropometric of athletes. The results of these latter studies raise the question of whether a similar approach might be useful in predicting normal functional-running Materials and Methods range on the basis of models that include multiple anthropometric and demographic predictor variables. There are several mathematical models that calculate body segment parameters from standard anthropometric measurements, such as segment lengths and circumferences. Some of these models results in large errors even in estimates of segment volumes. Others are very time-consuming,

requiring up to 200 anthropometric measurements, which take at least an hour or two to complete. All these models require density throughout the segment, or throughout large part of the segment. The greatest problem in body segment data occurs for moments of inertia. There are no simple yet accurate methods of measuring segmental moments of inertia for a living person. Many model estimations are either very inaccurate or require further validation. A relative error of 5% in segmental moments of inertia may be quite common. Norms or linear regression equations are often used, but these should be treated with caution as the errors involved in their use are rarely fully assessed. It may be necessary to allow for the nonlinear relationships between segmental dimensions and moment of inertia values. Body composition is consisting of body fat percentage and fat free mass. It can be measure with some methods like skinfold, bioelectric impedance or densitometry under water. All the methods predict body fat percentage and then the fat free mass. Anthropometric measurements affect effectively in the level of athletic achievement during the competitions, as there is a relationship between measurements and physical multi-level performance skills, the coach and those preparing for a software selection and choice of players must put into account, firstly the measures lengths and circumferences, as well as the amount of obesity of the body and then secondly research on the strength of relations between these measurements and the relative weight of impressionist in the level of skill performance of the player within the specialist focused on sports activity.

Success in sports has been associated with specific anthropometric characteristics, body composition and somatotype.

During a soccer match (90 minutes), the player's movements are characterized by high intensity, short-term actions and pauses of varying length. To be successful in such a team sport, soccer players need an optimal combination of technical, tactical, physical characteristics (e.g., somatotype) and mental motivation, among other sports characteristics. Hence, for soccer coaches, managers, sports physiotherapists, and scientists, an in-depth understanding of the determinants of success, such as the specific anthropometric characteristics of players may be important. Some studies showed evidence for position-specific anthropometric characteristics in soccer players. Goalkeepers are taller than central position players, such as defensive and central offensive players. Similar studies

on position-specific anthropometric profiles have been reported for Australian football, Gaelic soccer, rugby and American football [1–4].

10.2 MATERIALS AND METHODS

Anthropometry refers to the measurement of the human individual. An early tool of physical anthropology, it has been used for identification, for the purposes of understanding human physical variation, in paleoanthropology and in various attempts to correlate physical with racial and psychological traits. Today, anthropometry plays an important role in industrial design, clothing design, ergonomics and architecture where statistical data about the distribution of body dimensions in the population are used to optimize products. Changes in life styles, nutrition and ethnic composition of populations lead to changes in the distribution of body dimensions (e.g., the obesity epidemic), and require regular updating of anthropometric data collections. Information on anthropometric characteristics is considered very important for two reasons. Firstly, the incidence of some cancers may be related to anthropometric patterns (e.g., height, fat distribution), particularly cancers of the breast, endometrium and possibly colon. Secondly, anthropometric characteristics are linked to energy intake, energy metabolism and metabolic efficiency as well as to physical activity. Body mass index (BMI), for example, may be related to the type of energy-providing nutrients as well as to total energy intake.

Present work shows a computational modeling for anthropometric of athletes. According to this technique, in this work an improved modeling based on measurements was classified and compared with a special type of heart rate of sportswomen with other anthropometric classes of sportswomen. On the other hand present work compares the anthropometric data for Runner Velocity (Running Time) related to heart rate in two cases. Consequently, the results of this work will help to reduce the risk of sportswomen activities.

In this work, dateline for field tests data collection was: at 09:00 a.m., 10/09/2012 until 27/09/2012. Locaton of work field tests model was at Rasht city in the north of Iran. In this work the following are considerations was made when using and applying anthropometric data for heart

rate of runner related to Runner Velocity (Running Time). Heart rate of runner was measured in two cases: first at the running state (dynamic heart rate) and second at the before of running (static heart rate). Twenty healthy and young sprint runner females participate in this study. They signed a written/informed consent.

All subjects completed a medical questionnaire to ensure that they were not taking any medication, and were free cardiac, respiratory, renal or metabolic diseases. They didn't have any orthopaedic injury that would inhibit physical activity. The subjects were tested in the follicular phase of the menstrual cycle based on their previous menses. All participants consumed their last meal at least 2 hours before the test and refrained from intensive exercise in the 24 hours period preceding testing. All anthropometric measures were recorded in the morning by the same experienced anthropometrist. The exercise systolic and diastolic heart rates were measured on left arm of each participant using cuffs of appropriate size.

The measurements were recorded just after 100 m intense running in standing position. In present work, for professional runner at fast running, heart rate of runner, Runner Velocity were recorded. Curve estimation procedure has used these data, which have been detected field tests. These data have been compared by heart rate of runner and runner velocity, which have been detected from actual systems (field tests). The model was calibrated using one set of data, without changing parameter values. It was used to match a different set of results. Regression model has been built based on field tests data. Scatter diagram, correlation and autocorrelations procedure for heart rate was formed by estimating regression statistics and producing related plots for field tests model [5–11].

10.3 RESULTS AND DISCUSSION

In this work conclusions were drawn on the basis of experiments and calculations for independent variables such as runner heart rate based on the two cases:

$$V = f(H) \quad (1)$$

In first assumption for dynamic heart rate against Running Time, the curve estimation procedure allows quick estimating regression statistics (Table 1), and producing related plots for different models. Dependent variable: V – Runner Velocity (Running Time), with nomenclature “ Y ” for first: at the running state and the second: before of running condition.

Independent variable with nomenclature “ X ” such as:

H – runner heart rate is the most important variable.

The independent variable is Dynamic Heart Rate.

The most important effects that were observed based on regression for model summary are as follows. The dependent and independent variables were measured in the following conditions:

First: at the running state for dynamic condition.

Second: at the before of running for static condition.

Linear function: $A = 15.464 - .001x$ (2)

Logarithmic function: or $\log y = \log(14.437) + (.183)\log x$ $y = .065x$ (3)

Compound function: $A = 15.373e^t$ (4)

Quadratic function: $y = 3.672 + (.172)x - (.001)x^2$ (5)

Growth function: $(dA/dT) = 2.787 A$ (6)

Exponential function: $y = 15.373 (-2.74E-005)x + g$ (7)

Logistic function: $y = .065x$ (8)

Cubic function: $y = 8.095 + .08x + 0x^2 - 1.3E-006x^3$ (9)

TABLE 1 Model summary for Running Time.

Dependent Variable: Running Time (sec)									
Equation	Model Summary					Parameter Estimates			
	R-square	F	df1	df2	Sig.	Constant	b1	b2	b3
Linear $y = a_0 + a_1x$	.000	.002	1	18	.961	15.464	-.001		
Logarithmic $\log y = \log a + b \log x$	.001	.010	1	18	.921	14.437	.183		
Quadratic $y = a_0 + a_1x + a_2x^2$	.097	.910	2	17	.421	3.672	.172	-.001	
Cubic $y = a_0 + a_1x + a_2x^2 + a_3x^3$	.082	.762	2	17	.482	8.095	.080	.000	-1.30E-006
Compound $A = Ce^{kt}$	.000	.001	1	18	.975	15.373	1.000		
Growth $(dA/dT) = KA$	.000	.001	1	18	.975	2.733	-2.74E-005		
Exponential $y = ab^x + g$	.000	.001	1	18	.975	15.373	-2.74E-005		
Logistic $y = ax^b + g$	.000	.001	1	18	.975	.065	1.000		

These Eqs. (2)–(9) were built based on field tests data and in the final procedure they were compared by them. As a dynamic modeling selection, the Logarithmic function had suitable correlation on scatter diagram. Therefore it was selected as the regression model for this work. The results of present work were compared with the works of other experts.

Linear function: $y = 15.464 - .001x$ (10)

Logarithmic function: or $\log y = \log(14.437) + (.183)\log x$ $y = .065x$ (11)

Compound function: $A = 15.373e^t$ (12)

Quadratic function: $y = 3.672 - (.172)x - (.001)x^2$ (13)

Growth function: $(dA / dT) = 2.733A$ (14)

Exponential function: $y = 15.373(-2.74E - 005)^x + g$ (15)

Logistic function: $y = .065x$ (16)

Cubic function: $y = 8.095 + .08x + 0x^2 - 1.3E - 006x^3$ (17)

These Eqs. (10)–(17) were built based on field tests data and in the final procedure they were compared by them. As a dynamic modeling selection, the Logarithmic function had suitable correlation on scatter diagram.

Therefore it was selected as the regression model for this work. The results of present work were compared with the works of other experts [12–19].

Case 2:

The independent variable is static heart rate:

$$V = f(H) \quad (18)$$

In second assumption for static heart rate against Running Time, the curve estimation procedure allows quick estimating regression statistics (Table 2), and producing related plots for different models. Hence the auto-regression procedure by regression software “SPSS 10.0.5” was selected for the curve estimation procedure in the present work. The regression model has been built based on field test data. Regression software “SPSS” has fitted the function curve and provided regression analysis. So, the regression model has been found in the final procedure. Although related plots for the field test model were produced. Regression software “SPSS 10.0.5” performs multidimensional scaling of proximity data to find least-squares representation of the objects in a low-dimensional space. The curve estimation procedure allows quickly estimating regression statistics and producing related plots for different models [20–29].

Curve estimation is most appropriate when the relationship between the dependent variable(s) and the independent variable is not necessarily linear. Linear regression is used to model the value of a dependent scale variable based on its linear relationship to one or more predictors. Nonlinear regression is appropriate when the relationship between the dependent and independent variables is not intrinsically linear. Binary logistic regression is most useful in modeling of the event probability for a categorical response variable with two outcomes. The auto regression procedure is an extension of ordinary least-squares regression analysis specifically designed for time series.

One of the cases underlying ordinary least-squares regression is the absence of autocorrelation in the model residuals. Time series, however, often exhibit first-order autocorrelation of the residuals. In the presence of auto-correlated residuals, the linear regression procedure gives inaccurate estimates of how much of the series variability is accounted for by the chosen predictors. This can adversely affect choice of predictors and

TABLE 2 Parameter Estimates for Running Time against static heart rate.

Equation	Model Summary					Parameter Estimates			
	R-square	F	df1	df2	Sig.	Constant	b1	b2	b3
$y = a_0 + a_1x$ Linear	.000	.002	1	18	.961	15.464	-.001		
Logarithmic $\log y = \log a + b \log x$	.001	.010	1	18	.921	14.437	.183		
Quadratic $y = a_0 + a_1x + a_2x^2$	.097	.910	2	17	.421	3.672	.172	-.001	
Cubic $y = a_0 + a_1x + a_2x^2 + a_3x^3$	.082	.762	2	17	.482	8.095	.080	.000	-1.30E-006
Compound $A = Ce^{kt}$	.000	.001	1	18	.975	15.373	1.000		
Growth $(dA/dT) = KA$	.000	.001	1	18	.975	2.733	-2.74E-005		
Exponential $y = ab^x + g$	.000	.001	1	18	.975	15.373	-2.74E-005		
Logistic $y = ax^b + g$	.000	.001	1	18	.975	.065	1.000		

The independent variable is Static Heart Rate. / Dependent Variable: Running Time (sec)

hence the validity of the model. The auto regression procedure accounts for first-order auto correlated residuals. It provides reliable estimates of both goodness-of-fit measures and significance levels of chosen predictor variables. The auto regression procedure by regression software “SPSS 10.0.5” has been selected for the curve estimation procedure in present work [30–38].

10.3.1 COMPARISON WITH OTHER WORKS

Rahmani-Nia and Hojjati obtained the effect of exercise training on body composition and aerobic power of females. Comparison showed similarity in present work and the results of that works [39].

10.4 CONCLUSIONS

This work showed the effects of the Runner Velocity as the most important variable and runner heart rate. Present work emphasized that “regression model” is accurate model for modeling of anthropometric and body motion. Hence in order to presentation for influence of Runner Velocity, it was compared the models for computational and field tests experiments. Based on these procedures, it was showed that the regression based model for anthropometric of body motion. Therefore, the dynamic or running state was compared by static or before of running condition. On the other hand, this idea were included the proper analysis to provide a dynamic response to the shortcomings of the body motion. Because increased activity has been associated with a reduction in the risk of sudden cardiac death, this work has demonstrated that a delayed decrease in HR after exercise would be a powerful and independent predictor of all cause mortality in patients or in general population. Consequently, the results of this work will help to reduce the risk of anthropometric of female athletes sports-women activities.

NOMENCLATURES

P	Runner Blood Pressure (mm-Hg)
t	Running Time (sec)
V	Velocity (m/s)
S	Runner Stature (cm)

KEYWORDS

- **anthropometric**
- **blood pressure**
- **body motion**
- **computational modeling**
- **regression**

REFERENCES

1. Hurst Thomas, D. Skull Wars Kennewick Man, Archaeology and the Battle for Native American Identity, Washington University: School of Law - Anheuser-Busch Hall, 2001; 38–41.
2. Nabieh, A.; Mohamed, I. Anthropometric Measurements as a Significant for Choosing Juniors in Both Volleyball and Handball Sports (Factorial Analysis Study), *World J. Sport Sci.* **2010**, *3*(4), 277–289.
3. Rogana, S.; Hilfikerd, R.; Clarysb, P.; Clijsenc, R.; Taeymansac, J. Position-specific and Team-ranking-related Morphological Characteristics in German Amateur Soccer Players – a Descriptive Study, *Int. J. Appl. Sports Sci.*, **2011**, *23*(1), 168–182.
4. Luigi P. W.; Bercades, T. Somatotypes of national elite combative sport athletes, *Braz. J. Biomotricity*, **2009**, *3*(1), 21–30.
5. Dacres-Mannings, S.; Rochester, S.; Frail, H. Anthropometric profiles of Australian Rugby Institute, club and state level Rugby Union players. **2010**.
6. Gaurav, V.; Singh, M.; Singh, S. Anthropometric characteristics, somatotyping and body composition of volleyball and basketball players, *J. Phys. Educ. Sports Manage.* **2010**, *1*(3), 28–32, Available online <http://www.acadjourn.org/jpesm>

7. Beat K.; Kohler, G. Influence of anthropometry on race performance in ultra-endurance triathletes in the longest triathlon in North America, *Int. SportMed J.*, **2007**, 8(2), 87–96, <http://www.ismj.com>
8. Joseph, J.; Greene, M. S.; Timothy, A.; McGuine, A. T. C.; Leverson, G.; Thomas M. Anthropometric and Performance Measures for High School Basketball Players, *J. Athl. Training*, **1998**, 33(3), 229–232
9. Amatya, D. L. Comparative Study of Somatotype of Nepalese Sportsmen, National Association for Sports Health and Fitness. **1999**.
10. Sang Hong, K. Kinanthropometric Study of Korean and Nepalese Marathon Runners, keimyung university Diwakar Lal Amatya, MSC-Athletics and Sports Science-India, Sports Kinanthropometrist-Australia, Sports Expert-National Sports Council, January. **2008**.
11. Gaunt, B. W. Anthropometric and Demographic Factors Affecting Distance Hopped-aid Limb Symmetry Index for the Crossover Hop-for distance test in High School athletes, *J. Orthop. Sport Phys.*, **2001**, 31(3), 145–151
12. Gross, M.; Dailey, E. S.; Melissa, D.; Dalton, A.; lee, K.; Wendy, I.; Ashley, C. Relationship Between Lifting Capacity and Anthropometric Measures, *J. Orthop. Sport Phys.*, **2000**, 30(5), 237–247, 258–262.
13. Roberto, C, Jose, A.; Perez, J.; Cortell, m. Juan, J.; Rivas, J. correlations among anthropometric parameters, jump power, and position in professional basketball players, **2008**.
14. Pui, W. K.; Hendrik H. Anthropometric, gait and strength characteristics of Kenyan distance runners, *J. Sports Sci. Med.* **2008**, 7, 499–504, <http://www.jssm.org>
15. Jawis, M. N.; Singh, R.; Singh, H. J.; Yassin, M. N. Anthropometric and physiological profiles of sepak takraw players, *Br. J. Sports Med.* **2005**, 39, 825–829. doi: 10.1136/bjbm.2004.016915.
16. Lephart, S. M.; Abt, J. P.; Ferris, C. M.; Sell, T. C.; Nagai, T.; Myers, J. B.; Irrgang, J. Neuromuscular and biomechanical characteristic changes in high school athletes: a plyometric versus basic resistance program, *Br. J. Sports Med.* **2005**, 9, 932–938. doi: 17.1136/bjbm.2005.019083.
17. Darr, K. C.; Bassett, D. R.; Morgan, B. J.; Thomas, D. P. Effects of age and training status on heart rate recovery after peak exercise. *Am. J. Physiol.* **1988**, 254, H340–H343.
18. Engerman, S. ‘The Height of U. S. Slaves’, *Local Population Studies*, **1976**, 16, 1, 45–50.
19. Sunder, M. The Making of Giants in a Welfare State: The Norwegian Experience in the Twentieth Century’, *Economics and Biology*, **2004**, 75–86.
20. Woitek, U. Height Cycles in the Eighteenth and Nineteenth Centuries’, *Econ. Hum. Biol.*, **2003**, 243–258.
21. Fogel, R. ‘Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the making of Economic Policy’, *Am. Econ. Rev.*, **1994**, 369–394.

22. Goonetilleke, R. S.; Ho, Cheuk Fan E.; So, R. H. Y. Foot Anthropometry in Hong Kong. (1997, pp. 81–88) Proceedings of the ASEAN 97 Conference, Kuala Lumpur, Malaysia.
23. Baten, J. “Economic Development and the Distribution of Nutritional Resources in Bavaria, 1797–1839, in *J. Income Distrib.* **2000**, 9, 89–106.
24. Hariri Asli K. Water Hammer Research; Advances in Nonlinear Dynamics Modeling, (2012, pp. 88–121). Canada, USA: Published by Apple Academic Press, Inc., Exclusive worldwide distribution by CRC Press, a Taylor and Francis Group, Print ISBN: 9781926895314, eBook: 978-1-46-656887-7.
25. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. “Some Aspects of Physical and Numerical Modeling of Water Hammer in Pipelines,” *Nonlinear Dynamics An International Journal of Nonlinear Dynamics and Chaos in Engineering Systems*, ISSN: 1573–269X (electronic version) Journal /1071 Springer, Netherlands, **2009**, ISSN: 0924–090X (print version), Heidelberg, Germany, Springer, Volume 60, Number 4 / June, **2010**, 677–701.
26. Heather, E. Collins-Schramm and others, “Markers that Discriminate Between European and African Ancestry Show Limited Variation Within Africa,” *Human Genetics* **2002**, 111, 566–569.
27. Komlos, J.; Baur, M. “From the Tallest to One of the Fattest: The Enigmatic Fate of the Size of the American Population in the Twentieth Century,” *Econ. Hum. Biol.* **2004**, 2(1), 57–74.
28. Komlos, J.; Kriwy, P. “The Biological Standard of Living in the Two Germanies,” *German Econ. Rev.* **2003**, 4, 493–507.
29. Levy, W. C.; Cerqueira, M. D.; Harp, G. D.; Johannessen, K. A.; Abrass, I. B.; Schwartz, R. S.; Stratton, J. R. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. *Am. J. Cardiol.*, **1998**, 82, 1236–1241.
30. Margo, R. The Heights of American Slaves. *Social Science History*, **1982**, 6, 516–538.
31. Rahmani-Nia, F.; Hojjati, Z. The effect of exercise training on body composition and aerobic power in sedentary college age females, *Int. J. Fitness*, India, **2005**, 1, 24–31.
32. Hariri Asli, K.; Haghi, A. K. A Numerical Study on Fluid Flow and Pressure drop in Microtubes, *J. Balkan Tribological Association*, Tribotechnics and tribomechanics, Sofia, Bulgaria, **2010**, 16(3), 382–392.
33. Rahmani-Nia, F.; Rahnama, N.; Hojjati, Z.; And Soltani, B. Acute effects of aerobic and resistance exercise on serum leptin and some risk factors of coronary heart disease in obese females. *Sport sciences for health*, **2008**, 2(3), 118–124.
34. Rahmani-Nia, F.; Hojjati, Z.; Rahnama, N.; Soltani, B. Leptin, heart disease and exercise. *World J. Sport Sci.*, Iran, **2009**, 2(1), 13–20.
35. Rushton, J. P. Race, Brain Size, and Intelligence: A Rejoinder to Cain and Vanderwolf, *Personality and Individual Differences*, **2004**, 11, 785–794.
36. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. A computational approach to study fluid movement, *Nanomaterials Yearbook – 2009, From Nanostructures, Nanomaterials and Nanotechnologies to Nanoindustry*, (2010, pp. 181–196). USA: Nova Science Publications. ISBN: 978-1-60876-451-8.16

37. Young, J. W. Anthropometric and Mass Distribution Characteristics of the Adult Female FAA Civil Aeromedical Institute **2008**, FAA 9–83.
38. Bartlett, R. Introduction to Sports Biomechanics, 2nd Edition. Taylor and Francis- Library. **2007**.
39. Rahmani-Nia, F.; Hojjati, Z. Effect of selected training on body composition and aerobic power of Females College. *J. Harkat*, Iran, **2004**, *1*, 18–21.

CHAPTER 11

THEORETICAL AND PHYSICAL MODELING OF HUMAN MOVEMENT SCIENCE

CONTENTS

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11.1 INTRODUCTION

Many years ago some works compared the skulls of men with those of other animals. Some of them claimed that antique statues presented an angle of 90° , Europeans of 80° , Black people of 70° and the orangutan of 58° [1–3].

Swedish professor of anatomy Anders Retzius (1796–1860) first used the cephalic index in physical anthropology to classify ancient human remains found in Europe. He classed skulls in three main categories; long and thin, short and broad and intermediate length and width. Paleoanthropologists still rely upon craniofacial anthropometry to identify species in the study of fossilized hominid bones. Specimens of *Homo erectus* and athletic specimens of *Homo sapiens*, for example, are virtually identical from the neck down but their skulls can easily be told apart.

In 1856, workers found in a limestone quarry the skull of a Neanderthal man. The discovery was jointly announced in 1857, giving rise to the discipline of paleoanthropology [4–8]. Following its introduction in the 10th Asian Games in Beijing in 1990, and as a demonstration sport in the 1998 Commonwealth Games in Kuala Lumpur, sepak takraw has become one of the fastest growing games in Asia and has spread to over 20 countries including Argentina, Australia, Brazil, Canada, Korea, Germany, England, India, Japan, Puerto Rico, Spain, and the USA. The game is played on an area the size of a doubles badminton court, with three players on each side of a 1.52 m high net (Fig. 1). A team consists of three players: feeder, tekong/server, and spiker/killer. Sepak takraw combines ball skills (kicking and juggling) with the agility and acrobatic moves of gymnasts and the instinctive reflexes of competitive badminton players (Fig. 2). As in volleyball, there are passes, sets, and spikes but all without the use of hands or arms. The players are allowed to use their head, chest, feet, and thighs to propel the ball over the net. Both setting and spiking is done with the feet and the two most commonly used spikes are the “sun back” spike and the “roll” spike, which are performed aerially requiring immense agility, precision, leg strength, timing, and skill. Like badminton, squash, and tennis, the intensity of the game is intermittent, depending on the length of rallies following a serve.

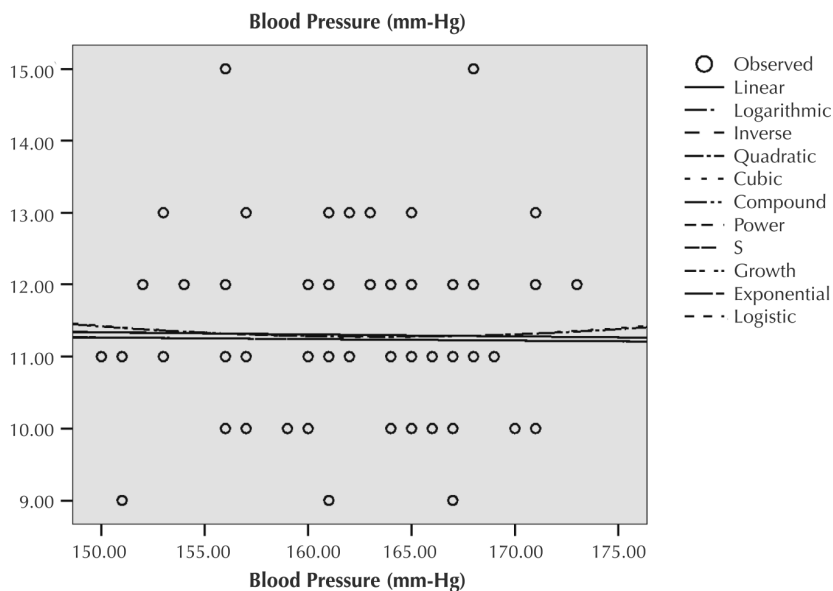


FIGURE 1 Regression and Curve Fit for runners.

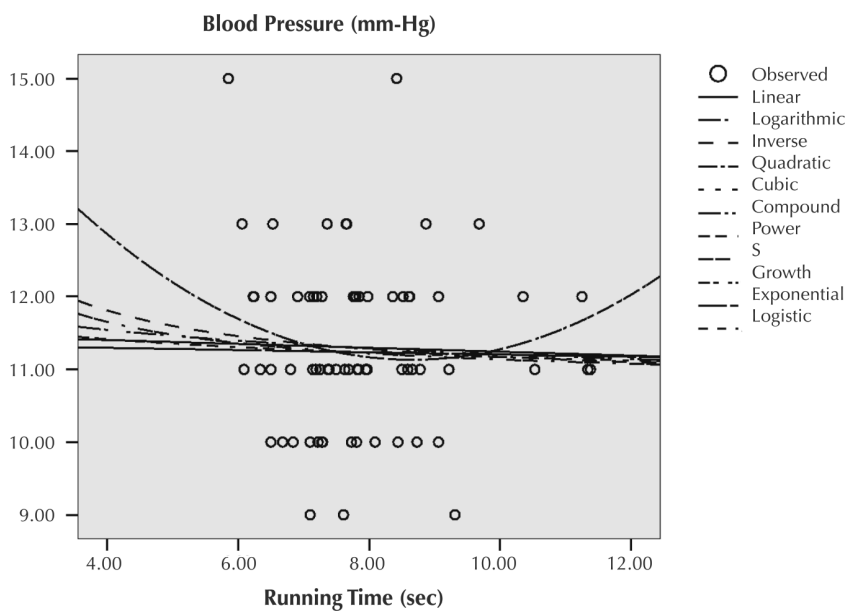


FIGURE 2 Blood pressure against Running Time Regression model.

Despite its increasing popularity, to date only one paper has been published on the physical and physiological profiles of sepak takraw players. However, player profiling has become a very important process on the road to excellence in sports. This study, therefore, presents some information on the physical and physiological profiles of the players at the National Sepak Takraw Grand Prix 2002 Championship; these are the top sepak takraw players in the country and some are in the national squad [9–16].

11.2 MATERIALS AND METHODS

Anthropometric of body motion assumed as an indicator of well-being to complement conventional indicators of living standards.

In this work the following are considerations was made when using and applying anthropometric data for pulse rate of runner heart related to Runner Velocity and Runner Stature. The dateline for field tests data collection was: at 02:00 p.m., 19/10/2012 until 29/10/2012. Locaton of work field tests model was at Rasht city in the north of Iran. In this work, at fast running with time up to 10 second, blood pressure of runner and Runner Velocity and stature have been recorded. Curve estimation procedure has used these data, which have been detected Field tests (Table 1). Also the parameters were collected by Lab. tests model (Table 2). These data have been compared by blood pressure of runner and Runner Velocity and stature data, which have been collected from actual systems (field tests). The model was calibrated using one set of data, without changing parameter values. It was used to match a different set of results. Regression model (Table 1) has been built based on field tests data. Curve estimation procedure (Figs. 1–3) was formed by estimating regression statistics and producing related plots for field tests model with the following three assumptions [17–29].

TABLE 1 Sportswomen anthropometric and body motion regression model.

Regression Model			
Blood Pressure (mm-Hg)	Runner Stature (cm)	Running Time (sec)	No.
13	163	6.53	1
11	165	7.14	2

TABLE 1 *(Continued)*

Regression Model			
Blood Pressure (mm-Hg)	Runner Stature (cm)	Running Time (sec)	No.
11	162	8.59	3
11	168	8.50	4
11	150	7.84	5
13	165	7.36	6
9	151	9.31	7
11	169	6.50	8
11	162	11.38	9
10	170	9.06	10
11	166	10.53	11
11	167	7.97	12
11	164	7.39	13
11	169	6.34	14
12	163	6.91	15
10	159	7.29	16
10	160	8.44	17
10	165	7.22	18
11	166	6.09	19
10	166	6.84	20
10	160	7.28	21
10	157	8.73	22
15	168	5.85	23
12	171	6.23	24
10	167	6.50	25
9	161	7.10	26

TABLE 1 *(Continued)*

Regression Model			
Blood Pressure (mm-Hg)	Runner Stature (cm)	Running Time (sec)	No.
12	173	6.24	27
12	161	7.85	28
12	160	8.61	29
12	161	7.98	30
10	156	6.68	31
12	165	7.28	32
11	167	7.37	33
12	167	7.15	34
12	160	8.52	35
13	157	9.68	36
12	168	7.76	37
12	171	11.25	38
9	167	7.10	39
11	161	7.63	40
12	173	6.50	41
11	161	8.78	42
11	160	8.66	43
13	161	7.66	44
11	156	7.19	45
12	165	7.20	46
12	167	7.80	47
10	167	7.81	48
11	160	7.82	49

TABLE 1 *(Continued)*

Regression Model			
Blood Pressure (mm-Hg)	Runner Stature (cm)	Running Time (sec)	No.
11	157	7.95	50
11	168	7.69	51
10	171	7.73	52
9	167	7.61	53
15	156	8.42	54
13	162	7.65	55
11	166	6.80	56
12	152	9.06	57
11	157	11.34	58
11	151	7.50	59
10	164	8.09	60
12	160	7.09	61
12	154	7.78	62
12	161	10.35	63
13	153	8.87	64
11	162	7.25	65
10	167	7.10	66
13	171	6.06	67
11	153	9.22	68
12	156	8.36	69
12	164	8.62	70

TABLE 2 Anthropometric model for elite sportswomen runners.

Equation	Model Summary					Parameter Estimates			
	R-square	F	df1	df2	Sig.	Constant	b1	b2	b3
Linear	.000	.011	1	68	.917	11.747	-.003		
Logarithmic	.000	.012	1	68	.915	13.623	-.456		
Inverse	.000	.012	1	68	.913	10.838	75.015		
Quadratic	.001	.024	2	67	.976	32.373	-.258	.001	
Cubic	.001	.028	2	67	.972	27.075	-.145	.000	1.81E-006
Compound	.000	.006	1	68	.936	11.579	1.000		
Power	.000	.007	1	68	.934	13.144	-.031		
S	.000	.007	1	68	.933	2.388	5.066		
Growth	.000	.006	1	68	.936	2.449	.000		
Exponential	.000	.006	1	68	.936	11.579	.000		
Logistic	.000	.006	1	68	.936	.086	1.000		

Dependent Variable: VAR00001
The independent variable is VAR00002.

Assumption (1):

$$p = f(S) \quad (1)$$

Assumption (2):

$$p = f(V) \quad (2)$$

Assumption (3):

$$p = f(V, S) \quad (3)$$

V – Runner Velocity is the most important variable, S – Runner Stature. Dependent variable: P – Blood pressure (mm-Hg) for starting point of running condition. The independent variable is Running Time (sec) and Runner Stature (m).

Height:

1. Remove shoes and hair ornaments.
2. Subject stands with their back to measuring rod, feet slightly apart, trunk balanced over the waist, knees straight, arms and shoulders relaxed.
3. Position head.
4. Take deep breath in and stand tall.
5. Headboard touches top of head.
6. Record height as it appears on the display.

11.3 RESULTS AND DISCUSSION

In this work conclusions were drawn on the basis of experiments and calculations for the three assumptions:

Assumption (1)

$$p = f(S) \quad (1)$$

The most important effects that were observed based on Regression for model summary (Table 1) are as follows (Fig. 3).

11.3.1 REGRESSION MODEL DUE TO FIELD TEST OF ANTHROPOMETRIC MODEL FOR BLOOD PRESSURE

The auto-regression procedure accounts for first-order auto-correlated residuals. It provides reliable estimates of both goodness of-fit measures and significant levels of chosen predictor variables [30–31].

The variables are as follows:

Dependent variable: P – blood pressure for starting point of running condition with nomenclature “ Y ,” independent variable with nomenclature “ X ” such as:

V – Runner Velocity is the most important variable, S – Runner Stature. The independent variable is Velocity (m.s^{-1}) and Runner Stature (cm). Assumption (2):

$$p = f(V) \quad (2)$$

For second assumption the curve estimation procedure (Fig. 2) allows quick estimating regression statistics, and producing related plots for different models. Hence the auto-regression procedure by regression software “SPSS 10.0.5” was selected for the curve estimation procedure in the present work. The regression model has been built based on field test data.

Regression software “SPSS” has fitted the function curve and provided regression analysis. So, the regression model has been found in the final procedure. This model has compared field test results using Lab. model results [32–36].

Assumption (3):

$$p = f(V, S) \quad (3)$$

For third assumption the curve estimation procedure is illustrated. Besides, the runner blood pressure was formed by estimating regression statistics are listed in Table 1. Although related plots for the field test model were produced. Regression software “SPSS 10.0.5” performs multidimensional scaling of proximity data to find least-squares representation of the objects in a low-dimensional space [37–68].

11.3.2 COMPARISON WITH OTHER WORKS

Goonetilleke, Ho, Edmond Cheuk Fan, and So, obtained foot anthropometry. Comparison showed similarity in present work and the results of that works [69].

11.4 CONCLUSIONS

The integrated physical performance involves anthropometric characteristics, physiological function, health and physical performance, among which physical performance is the most important athletic competence, while anthropometric characteristics, physiological function and health form a good basis for an ideal physical performance. This chapter discuss about anthropometric advantages in some sports. Consequently, the results of this work will help to reduce the risk of sport women activities.

NOMENCLATURES

P	Runner Blood Pressure (mm-Hg)
t	Running Time (sec)
V	Velocity (m/s)
S	Runner Stature (cm)

KEY WORDS

- anthropometric
- body motion
- computational model
- high technology
- regression

REFERENCES

1. Hurst Thomas, D. Skull Wars Kennewick Man, Archaeology and the Battle for Native American Identity, Washington University: School of Law - Anheuser-Busch Hall, 2001; 38–41.
2. Nabieh, A.; Mohamed, I. Anthropometric Measurements as a Significant for Choosing Juniors in Both Volleyball and Handball Sports (Factorial Analysis Study), *World J. Sport Sci.* **2010**, 3(4), 277–289.
3. Rogana, S.; Hilfiker, R.; Clarys, P.; Clijsen, R.; Taeymans, J. Position-specific and Team-ranking-related Morphological Characteristics in German Amateur Soccer Players – a Descriptive Study, *Int. J. Appl. Sports Sci.*, **2011**, 23(1), 168–182.
4. Luigi P. W.; Bercades, T. Somatotypes of national elite combative sport athletes, *Braz. J. Biomotricity*, **2009**, 3(1), 21–30.
5. Dacres-Mannings, S.; Rochester, S.; Frail, H. Anthropometric profiles of Australian Rugby Institute, club and state level Rugby Union players. **2010**.
6. Gaurav, V.; Singh, M.; Singh, S. Anthropometric characteristics, somatotyping and body composition of volleyball and basketball players, *J. Phys. Educ. Sports Manage.* **2010**, 1(3), 28–32, Available online <http://www.acadjourn.org/jpesm>
7. Beat K.; Kohler, G. Influence of anthropometry on race performance in ultra-endurance triathletes in the longest triathlon in North America, *Int. SportMed J.*, **2007**, 8(2), 87–96, <http://www.ismj.com>
8. Joseph, J.; Greene, M. S.; Timothy, A.; McGuine, A. T. C.; Leverson, G.; Thomas M. Anthropometric and Performance Measures for High School Basketball Players, *J. Athl. Training*, **1998**, 33(3), 229–232
9. Amatya, D. L. Comparative Study of Somatotype of Nepalese Sportsmen, National Association for Sports Health and Fitness. **1999**.
10. Sang Hong, K. Kinanthropometric Study of Korean and Nepalese Marathon Runners, keimyung university Diwakar Lal Amatya, MSC-Athletics and Sports Science-India, Sports Kinanthropometrist-Australia, Sports Expert-National Sports Council, January. **2008**.
11. Gaunt, B. W. Anthropometric and Demographic Factors Affecting Distance Hopped-aid Limb Symmetry Index for the Crossover Hop-for distance test in High School athletes, *J. Orthop. Sport Phys.*, **2001**, 31(3), 145–151
12. Gross, M.; Dailey, E. S.; Melissa, D.; Dalton, A.; Lee, K.; Wendy, I.; Ashley, C. Relationship Between Lifting Capacity and Anthropometric Measures, *J. Orthop. Sport Phys.*, **2000**, 30(5), 237–247, 258–262.
13. Roberto, C.; Jose, A.; Perez, J.; Cortell, m. Juan, J.; Rivas, J. correlations among anthropometric parameters, jump power, and position in professional basketball players, **2008**.
14. Pui, W. K.; Hendrik H. Anthropometric, gait and strength characteristics of Kenyan distance runners, *J. Sports Sci. Med.* **2008**, 7, 499–504, <http://www.jssm.org>
15. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. “Some Aspects of Physical and Numerical Modeling of Water Hammer in Pipelines,” *Nonlinear Dynamics An International Journal of Nonlinear Dynamics and Chaos in Engineering Systems*, ISSN: 1573–269X (electronic version) Journal /1071 Springer, Netherlands, 2009, ISSN: 0924–090X

- (print version), Heidelberg, Germany, Springer, Volume 60, Number 4 / June, **2010**, 677–701.
16. Heather, E. Collins-Schramm and others, “Markers that Discriminate Between European and African Ancestry Show Limited Variation Within Africa,” *Human Genetics* **2002**, 111, 566–569.
 17. Komlos, J.; Baur, M. “From the Tallest to One of the Fattest: The Enigmatic Fate of the Size of the American Population in the Twentieth Century,” *Econ. Hum. Biol.* **2004**, 2(1), 57–74.
 18. Komlos, J.; Kriwy, P. “The Biological Standard of Living in the Two Germanies,” *German Econ. Rev.* **2003**, 4, 493–507.
 19. Levy, W. C.; Cerqueira, M. D.; Harp, G. D.; Johannessen, K. A.; Abrass, I. B.; Schwartz, R. S.; Stratton, J. R. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. *Am. J. Cardiol.*, **1998**, 82, 1236–1241.
 20. Margo, R. The Heights of American Slaves. *Social Science History*, **1982**, 6, 516–538.
 21. Rahmani-Nia, F.; Hojjati, Z. The effect of exercise training on body composition and aerobic power in sedentary college age females, *Int. J. Fitness*, India, **2005**, 1, 24–31.
 22. Rahmani-Nia, F.; Hojjati, Z. Effect of selected training on body composition and aerobic power of Females College. *J. Harkat*, Iran, **2004**, 1, 18–21.
 23. Rahmani-Nia, F.; Rahnama, N.; Hojjati, Z.; And Soltani, B. Acute effects of aerobic and resistance exercise on serum leptin and some risk factors of coronary heart disease in obese females. *Sport sciences for health*, **2008**, 2(3), 118–124.
 24. Rahmani-Nia, F.; Hojjati, Z.; Rahnama, N.; Soltani, B. Leptin, heart disease and exercise. *World J. Sport Sci.*, Iran, **2009**, 2(1), 13–20.
 25. Rushton, J. P. Race, Brain Size, and Intelligence: A Rejoinder to Cain and Vanderwolf, *Personality and Individual Differences*, **2004**, 11, 785–794.
 26. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. A computational approach to study fluid movement, *Nanomaterials Yearbook - 2009*, From Nanostructures, Nanomaterials and Nanotechnologies to Nanoindustry, (**2010**, pp. 181–196). USA: Nova Science Publications. ISBN: 978-1-60876-451-8.16
 27. Young, J. W. Anthropometric and Mass Distribution Characteristics of the Adult Female FAA Civil Aeromedical Institute **2008**, FAA 9–83.
 28. Bartlett, R. Introduction to Sports Biomechanics, 2nd Edition. Taylor and Francis- Library. **2007**.
 29. Hariri Asli, K.; Haghi, A. K. A Numerical Study on Fluid Flow and Pressure drop in Microtubes, *J. Balkan Tribological Association*, Tribotechnics and tribomechanics, Sofia, Bulgaria, **2010**, 16(3), 382–392.
 30. Spicer, L. J. Leptin: A possible metabolic signal affecting reproduction. *Domestic Animal Endocrinology* **2001**, 21, 215–250.
 31. Unal, M.; Unal, D. D. O.; Baltaci, A. K.; Mgulkoc, R. Investigation of serum leptin levels and Vo_{2max} value in trained young male athletes and healthy males. *Acta Physiol. Hungary*. **2005**, 92, 173–179
 32. Askari, H.; Tykodi, G.; Liu, J.; Dagogo-Jack S. Fasting plasma leptin level is a surrogate measure of insulin sensitivity. *J. Clin. Endocrinol. Metab.*; **2010**, 95(8), 3836–3843.

33. Reseland, J. E.; Andessen, S. A.; Solvoll, K.; Hjermann, I.; Urdal, P.; Holme, I.; Drevon, C. A. Effect of long-term change in diet and exercise on plasma leptin concentration. *Clin. Nutr.* **2001**, *73*, 240–245.
34. Mokdad, A. H.; Ford, E. S.; Bowman, B. A.; Dietz, W. H.; Vinicor, F.; Bales, V. S.; Marks, J. S. Prevalence of obesity, *Diabetes* and obesity-related health risk factors, 2001. *JAMA* **2003**, *289*, 76–79.
35. Mendosa-Nunez, V. M.; Garcia-Sanchez, A.; Sanchez-Rodriguez, M.; Galvan-Duart, R. E.; Fonseca-Yerena, E. F. Overweight, waist circumference, age, gender, and insulin resistance as risk factors for Hyperleptinemia. *Obes. Res.* **2002**, *10*, 253–259.
36. Frank, L. L.; Sorensen, B. E.; Yasui, Y.; Tworoger, S. S.; Schwartz, R. S.; Ulrich, C. M.; Irwin, M. L.; Rudolph, R. E.; Rajan, K. B.; Stanczyk, F.; Bowen, D.; Weigle, D. S.; Potter, J. D.; McTiernan, A. Effects of exercise on metabolic risk variables in overweight postmenopausal women: a randomized clinical trial. *Obes. Res.* **2005**, *13*, 615–625.
37. Racette, S. B.; Coppack, S. W.; Landt, M.; Klein, S. Leptin production during moderate-intensity aerobic exercise. *J. Clin. Endocrinol. Metabol.* **1997**, *82*, 2275–2277.
38. Altman, R.; Risk factors in coronary atherosclerosis athero-inflammation: the meeting point. *Thrombosis J.* **2003**, *1*, 4–7.
39. Stefanie, L.; Martina, E. Physical activity and endogenous sex hormones in postmenopausal women. *Cancer Causes Control*, **2011**, *22*, 81–89.
40. Akbarzadeh, R. Relationship between the ratio of waist circumference to hip circumference and risk factors and increased lipids and blood pressure in patients with insulin-dependent *Diabetes* referred to the Endocrinology Institute in Tehran in 1995 and 1996. A Master's thesis, Iran University of Medical Sciences; Faculty of Nursing. **1996**.
41. Ashwell, M.; Lejeane, S. Ratio of waist circumference to height maybe better indicator need for weight management. *BMI*: **1990**, 312–377.
42. McMahon, M.; Skaggs, B. J.; Sahakian, L.; Grossman, J.; FitzGerald, J.; Ragavendra, N.; Charles-Schoeman, C.; Chernishof, M.; Gorn, A.; Witztum, J. L.; Wong, W. K.; Weisman, M.; Wallace, D. J.; La Cava, A.; Hahn, B. H. High plasma leptin levels confer increased risk of atherosclerosis in women with systemic lupus erythematosus, and are associated with inflammatory oxidised lipids. *Ann. Rheum. Dis.* **2011**, *70*(9), 1619–1624.
43. Delavar, A. Research methods in psychology and educational sciences; 1st edition; Tehran; Payame Noor University Press. **2000**.
44. McHugh, P. R.; T. H. Moran, Calories and gastric emptying: a regulatory capacity with implications for feeding. *Am. J. Physiol.* **1979**, *236*(5), R 254–60.
45. Shirreff, S. M.; P. Watson, and R. J. Maughan, Milk and effective post – exercise rehydration drink. *Br. J. Nutr.*, **2007**, *98*(11), 173–80.
46. Merson S. J.; Shirreff S. M.; Leiper J. B.; Maughan R. J: Changes in blood plasma and red cell volume after ingestion of hypotonic and hypertonic solutions. *Proc. Nutr. Soc.* (In the Press), 2002.
47. Melov, S. et al., Resistance exercise reverses aging in human skeletal muscle. *PLOS One*. **2007**, *2*(5), E 465.

48. Miller, S. L.; et al., Metabolic response to provision of mixed protein – carbohydrate supplementation during endurance exercise. *Int. J. Sport Nutr. Exerc. Metab.*, **2002**, 12(4): p 384–97.
49. Miller S. L.; et al., The effects of nutritional supplementation throughout an endurance run on leucine kinetics during recovery. *Int J Sport Nutr Metab*, **2007**, 17(5), 456–67.
50. Cooke, J. P.; Oka, R. K. **2002**, Does leptin cause vascular disease? *Circulation* 106, 1904–1905.
51. Canavan, B.; Salem, R. O.; Schurgin, P. K.; Lipinska, I.; Laposata, M.; Grinspoon, S. Effect of physiological leptin administration on markers of inflammation, platelet activation, and platelet aggregation during caloric deprivation. *The J. Clin. Endocrinol. Metabol.* **2005**, 90(10), 5779–5785.
52. Cooke, J. P.; Oka, R. K. Does leptin cause vascular disease? *Circulation* **2002**, 106, 1904–1905.
53. Ahima, A. S.; Flier, J. S. Adipose tissue as an endocrine organ. *Trends Endocrinol. Metabol.* **2000**, 11, 327–331.
54. Altman, R. Risk factors in coronary atherosclerosis athero-inflammation: the meeting point. *Thrombosis J.* **2003**, 1, 4–7.
55. Konstantinides, S.; Schafer, K.; Koschnick, S.; Loskutoff, D. J. Leptin-dependent Platelet aggregation and arterial thrombosis suggests a mechanism for atherothrombotic disease in obesity. *J. Clin. Invest.* **2001**, 108, 1533–1540.
56. Kaye, S. A.; Fdosom, A. R.; Jacobs, D. R. Psychosocial correlates of body fat distribution in Black and white young adult. *Int. J. Obes.*, **1995**, 17, 271–277
57. Montain S. J.; Coyle E. F. Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J. Appl. Physiol.* **1992a**, 73, 1340–1350.
58. Meier, U.; Gressner, A. M. Endocrine regulation of energy metabolism: review of pathobiology and clinical chemical aspects of leptin, ghrelin and adiponectin, and resistin. *Cli. Chem.* **2004**, 50, 1511–1525.
59. Mokdad, A. H.; Ford, E. S.; Bowman, B. A.; Dietz, W. H.; Vinicor, F.; Bales, V. S.; Marks, J. S. Prevalence of obesity, *Diabetes* and obesity-related health risk factors, 2001. *J. Am. Med. Assoc.* **2003**, 289, 76–79.
60. Ostlund, R. E.; Jr. Yang, S.; Gingerich, R. Relation between plasma leptin concentration and body fat, gender, diet, age, and metabolic covariates. *J. Clin. Endocrinol. Metabol.* **1996**, 81, 3909–3913.
61. Baratta, M. Leptin from a signal of adiposity to a hormone mediator in peripheral tissues. *Med. Sci. Monit.* **2002**, 8, RA282-RA292.
62. Gomez-Merino, D; Chennaoui, M; Drogou, C; Bonneau, D; Guezennec, C.Y. Decrease in serum leptin after prolonged physical activity in men, *Med. Sci. Sports Exerc.*; **2002**, 34, 1594–1599.
63. Boden, G.; Chen, X.; Mozzoli, M.; Ryan, I. Effect of fasting on serum leptin in normal human subjects. *J. Clin. Endocrinol. Metabol.* **1996**, 81, 3419–3423.
64. Brabeau, P.; Gutin, B.; Litaker, M. S.; Ramsey, L. T.; Cannady, W. E.; Allison, J.; Lemmon, C. R.; Owens, S. Influence of physical training on plasma leptin in obese youths. *Canadian J. Appl. Physiol.* **2003**, 28, 382–396.
65. Houmard, J. A.; Cox, J. H.; Maclean, P. S.; Barakat, H. A. Effect of short-term exercise training on leptin and insulin action. *Metabolism* **2000**, 49, 858–861.

66. kukuljan, S.; et al., Effects of resistance exercise and fortified milk on skeletal muscle mass, muscle size, and functional performance in middle-aged and older men: an 18-month randomized controlled trial. *J. Appl. Physiol.*, **2009**, 707(6), 1864–73.
67. Hickey, M. S.; Considine, R. V.; Israel, R. G. Leptin is related to body fat content in male distance runners. *Am. J. Physiol.* **1996**, 271, E938-E940.
68. Sihna, M. K.; Caro, J. F. Clinical aspects of leptin. *Vitamins and Hormones* **1998**, 54, 1–30.
69. Goonetilleke, R. S.; Ho, Cheuk Fan E.; So, R. H. Y. Foot Anthropometry in Hong Kong. (1997, pp. 81–88) Proceedings of the ASEAN 97 Conference, Kuala Lumpur, Malaysia.

CHAPTER 12

BIOMECHANICAL CAUSES OF SPORTS INJURIES

CONTENTS

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12.1 INTRODUCTION

Anthropometric measures are available for portions of many national populations as far back as the early 1700s. While these data often serve as complements to standard economic indicators, in some cases they provide the only means of assessing historical economic well-being, as “conventional” measures such as per capita GDP, wage and price indices, and income inequality measures have been notoriously spotty and problematic to develop. Anthropometric-based research findings to date have contributed to the scholarly debates over mortality trends, the nature of slavery, and the outcomes of industrialization and economic development. Height has been the primary indicator utilized to date. Other indicators include height-standardized weight indices, birth weight, and age at menarche.

12.2 MATERIALS AND METHODS

Present book shows a computational modeling for anthropometric of athletes. According to this technique, in this work an improved modeling based on measurements was classified and compared with a special type of weights of sportswomen with other anthropometric classes of sportswomen. On the other hand present work compares the anthropometric data for Runner Velocity (Running Time) related to runner weight. Consequently, the results of this work will help to reduce the risk of sportswomen activities [1–20].

In this work, dateline for field tests data collection was: at 11:00 a.m., 10/09/2012 until 20/09/2012. Locaton of work field tests model was at Rasht city in the north of Iran. In this work the following are considerations was made when using and applying anthropometric data for heart rate of runner related to Runner Velocity (Running Time) and runner weight. Seventy healthy and sprint runner females (Table 1) participate in this study. They signed a written/informed consent [21–33].

All subjects completed a medical questionnaire to ensure that they were not taking any medication, and were free cardiac, respiratory, renal or metabolic diseases. They didn't have any orthopaedic injury that would inhibit physical activity. The subjects were tested in the follicular phase

of the menstrual cycle based on their previous menses. All participants consumed their last meal at least 2 h before the test and refrained from intensive exercise in the 24 h period preceding testing. All anthropometric measures were recorded in the morning by the same experienced anthropometrist. The weight was measured using a wall-mounted stadiometer (± 0.1 cm). The exercise systolic and diastolic heart rates were measured on left arm of each participant using cuffs of appropriate size (Table 1). The measurements were recorded just after 100 m intense running in standing position. In present work, for professional runner at fast running, heart rate of runner, Runner Velocity and weight were recorded. Curve estimation procedure has used these data, which have been detected field tests (Table 1). The model was calibrated using one set of data, without changing parameter values [34–37].

It was used to match a different set of results. Regression model (Table 2) has been built based on field tests data. Scatter diagram, correlation and autocorrelations procedure for blood pressure was formed by estimating regression statistics and producing related plots for field tests model (Table 2).

TABLE 1 Sportswomen regression model.

Regression Model			
Blood Pressure (mm-Hg)	Runner Weight (kg)	Running Time (sec)	No.
13	59	6.53	1
11	57	7.14	2
11	96	8.59	3
11	83	8.50	4
11	57	7.84	5
13	91	7.36	6
9	54	9.31	7
11	73	6.50	8
11	77	11.38	9

TABLE 1 *(Continued)*

Regression Model			
Blood Pressure (mm-Hg)	Runner Weight (kg)	Running Time (sec)	No.
10	72	9.06	10
11	71	10.53	11
11	78	7.97	12
11	90	7.39	13
11	70	6.34	14
12	75	6.91	15
10	66	7.29	16
10	106	8.44	17
10	57	7.22	18
11	56	6.09	19
10	68	6.84	20
10	75	7.28	21
10	45	8.73	22
15	81	5.85	23
12	125	6.23	24
10	67	6.50	25
9	65	7.10	26
12	89	6.24	27
12	72	7.85	28
12	63	8.61	29
12	55	7.98	30
10	60	6.68	31
12	102	7.28	32

TABLE 1 *(Continued)*

Regression Model			
Blood Pressure (mm-Hg)	Runner Weight (kg)	Running Time (sec)	No.
11	86	7.37	33
12	104	7.15	34
12	68	8.52	35
13	73	9.68	36
12	66	7.76	37
12	83	11.25	38
9	75	7.10	39
11	51	7.63	40
12	53	6.50	41
11	93	8.78	42
11	80	8.66	43
13	72	7.66	44
11	66	7.19	45
12	63	7.20	46
12	80	7.80	47
10	63	7.81	48
11	77	7.82	49
11	78	7.95	50
11	79	7.69	51
10	85	7.73	52
9	69	7.61	53
15	74	8.42	54
13	64	7.65	55

TABLE 1 (Continued)

Regression Model			
Blood Pressure (mm-Hg)	Runner Weight (kg)	Running Time (sec)	No.
11	69	6.80	56
12	76	9.06	57
11	132	11.34	58
11	68	7.50	59
10	75	8.09	60
12	92	7.09	61
12	68	7.78	62
12	75	10.35	63
13	114	8.87	64
11	80	7.25	65
10	76	7.10	66
13	60	6.06	67
11	80	9.22	68
12	77	8.36	69
12	86	8.62	70

12.3 RESULTS AND DISCUSSION

In this work conclusions were drawn on the basis of experiments and calculations for independent variables (running weight). The most important effects that were observed based on regression for model summary (Table 2) are as follows.

At present work the dependent and independent variables of runner were measured in the following conditions [38]:

TABLE 2 Field test model.

No.	Age	Height (cm)	Weight (kg)	BMI	Hip circumfer- ence	Waist circum- ference	WHR	Heart rate	100 m Running Time (sec)
1	28	163	59	22.64	97	70	0.72	68	13.7
2	24	165	57	20.95	98	74	0.75	74	11.8
3	21	162	96	36.64	121	122	1	72	11.8
4	23	168	83	29.43	111	98	0.88	85	11.9
5	24	150	57	25.33	95	80	0.84	79	11.8
6	29	165	91	33.45	115	101	0.87	101	13.9
7	30	151	54	23.68	89	71	0.79	68	9.7
8	29	169	73	25.61	100	79	0.79	92	11.8

TABLE 2 (Continued)

No.	Age	Height (cm)	Weight (kg)	BMI	Hip circumfer- ence	Waist circum- ference	WHR	Heart rate	100 m Running Time (sec)
9	26	162	77	29.38	109	83	0.76	74	11.8
10	27	170	72	24.91	98	80	0.81	84	10.7
11	26	166	71	25.81	103	78	0.75	96	11.8
12	24	167	78	28.05	110	79	0.71	81	11.8
13	30	164	90	33.58	119	91	0.76	71	11.8
14	21	169	70	24.56	101	77	0.76	108	11.9
15	22	163	75	28.62	104	90	0.86	94	12.9
16	24	159	66	26.19	100	72	0.72	62	10.8
17	30	160	106	41.4	131	112	0.85	82	10.7

TABLE 2 (Continued)

No.	Age	Height (cm)	Weight (kg)	BMI	Hip circumfer- ence	Waist circum- ference	WHR	Heart rate	100 m Running Time (sec)
18	28	165	57	20.95	97	78	0.8	78	10.7
19	22	166	56	20.36	92	68	0.73	79	11.8
20	20	166	68	24.72	102	76	0.74	85	10.7
21	28	160	75	29.29	112	87	0.77	70	10.7
22	28	157	45	18.29	88	63	0.71	95	10.8
23	27	168	81	28.72	103	93	0.9	136	15.6
24	22	171	125	42.8	134	117	0.87	74	12.1
25	22	167	67	24.1	89	69	0.77	110	10.7
26	23	161	65	25.09	97	76	0.78	100	9.7

TABLE 2 (Continued)

No.	Age	Height (cm)	Weight (kg)	BMI	Hip circumfer- ence	Waist circum- ference	WHR	Heart rate	100 m Running Time (sec)
27	21	173	89	29.76	107	92	0.85	69	12.6
28	27	161	72	27.79	109	88	0.8	77	12.8
29	27	160	63	24.6	104	76	0.73	77	12.8
30	25	161	55	21.23	98	77	0.78	74	12.8
31	25	156	60	24.69	98	71	0.72	72	10.7
32	27	165	102	37.5	121	112	0.92	76	12.8
33	28	167	86	30.93	111	92	0.82	77	11.8
34	20	167	104	37.41	131	103	0.78	74	12.8

TABLE 2 (Continued)

No.	Age	Height (cm)	Weight (kg)	BMI	Hip circumfer- ence	Waist circum- ference	WHR	Heart rate	100 m Running Time (sec)
35	32	166	68	24.72	103	89	0.86	114	8.52
36	40	160	73	28.51	108	98	0.9	83	9.68
37	32	159	66	26.19	106	84	0.79	92	7.76
38	31	169	83	29.12	109	98	0.89	85	11.25
39	31	161	75	28.95	109	88	0.8	70	7.10
40	32	160	51	19.92	93	65	0.69	89	7.63
41	31	156	53	21.81	94	72	0.76	80	6.50
42	38	161	93	35.9	119	114	0.95	82	8.78
43	38	164	80	80.26	111	95	0.85	69	8.66
44	32	166	72	26.18	105	89	0.84	84	7.66
45	37	161	66	25.48	111	79	0.71	81	7.19

TABLE 2 (Continued)

No.	Age	Height (cm)	Weight (kg)	BMI	Hip circumfer- ence	Waist circum- ference	WHR	Heart rate	100 m Running Time (sec)
46	33	157	63	25.6	102	73	0.71	76	7.20
47	39	156	80	32.92	112	101	0.9	78	7.80
48	33	153	63	26.92	98	91	0.92	65	7.81
49	40	148	77	35.15	112	98	0.87	73	7.82
50	36	163	78	29.43	114	86	0.75	88	7.95
51	38	167	79	28.41	110	105	0.95	70	7.69
52	34	156	85	34.97	114	102	0.89	69	7.73
53	33	170	69	23.87	102	85	0.83	78	7.61
54	40	156	74	30.45	109	86	0.78	73	8.42
55	32	162	64	24.42	99	82	0.82	90	7.65
56	34	166	69	25.9	103	88	0.85	85	6.80
57	34	152	76	32.9	115	84	0.73	72	9.06
58	40	157	132	53.65	150	134	0.89	72	11.34
59	37	151	68	29.82	103	81	0.78	82	7.50

TABLE 2 (Continued)

No.	Age	Height (cm)	Weight (kg)	BMI	Hip circumfer- ence	Waist circum- ference	WHR	Heart rate	100 m Running Time (sec)
60	33	164	75	27.98	107	76	0.71	79	8.09
61	36	160	92	35.93	116	100	0.86	78	7.09
62	36	154	68	28.69	107	85	0.79	89	7.78
63	33	161	75	28.95	114	91	0.79	76	10.35
64	38	153	114	48.71	141	124	0.87	84	8.87
65	33	162	80	30.53	111	92	0.82	82	7.25
66	33	167	76	27.33	102	89	0.87	66	7.10
67	37	171	60	20.54	94	70	0.74	81	6.06
68	32	153	80	34.18	113	93	0.82	94	9.22
69	39	156	77	31.43	108	93	0.86	70	8.36
70	33	164	86	32.08	109	93	0.85	73	8.62

$$V = f(W) \quad (1)$$

Dependent variable: V – Runner Velocity (Running Time), with nomenclature “ Y ” for first: at the running state and the second: before of running condition.

Independent variable with nomenclature “ X ” such as:

W – runner weight (kg) is the most important variable.

Hence, the auto-regression procedure by regression software “SPSS 10.0.5” was selected for the curve estimation procedure in the present work. The regression model has been built based on field test data. Regression software “SPSS” has fitted the function curve and provided regression analysis. So, the regression model has been found in the final procedure. Although related plots for the field test model were produced. Regression software “SPSS 10.0.5” performs multidimensional scaling of proximity data to find least-squares representation of the objects in a low-dimensional space. The curve estimation procedure allows quickly estimating regression statistics and producing related plots for different models. Curve estimation is most appropriate when the relationship between the dependent variable(s) and the independent variable is not necessarily linear. Linear regression is used to model the value of a dependent scale variable based on its linear relationship to one or more predictors. Nonlinear regression is appropriate when the relationship between the dependent and independent variables is not intrinsically linear.

Binary logistic regression is most useful in modeling of the event probability for a categorical response variable with two outcomes. The auto regression procedure is an extension of ordinary least-squares regression analysis specifically designed for time series.

One of the cases underlying ordinary least-squares regression is the absence of autocorrelation in the model residuals. Time series, however, often exhibit first-order autocorrelation of the residuals. In the presence of auto-correlated residuals, the linear regression procedure gives inaccurate estimates of how much of the series variability is accounted for by the chosen predictors. This can adversely affect choice of predictors and hence the validity of the model. The auto regression procedure accounts

for first-order auto correlated residuals. It provides reliable estimates of both goodness-of-fit measures and significance levels of chosen predictor variables. The auto regression procedure by regression software “SPSS 10.0.5” has been selected for the curve estimation procedure in present work. The regression model includes:

$$V = f(W) \quad (1)$$

It has been built based on field tests data and in the final procedure they were compared by them.

Linear function: $A = 11.511 - .027x \quad (2)$

Logarithmic function: or $\log y = \log(12.05) - (.366)\log x \quad (3)$

Compound function: $A = 11.358e^{.999} \quad (4)$

Quadratic function: $y = 17.113 - (1.382)x - (.08)x^2 \quad (5)$

Growth function: $(dA / dT) = 2.43A \quad (6)$

Exponential function: $y = 11.358(-.001)^x + g \quad (7)$

Logistic function: $y = -.001x \quad (8)$

Cubic function: $y = 17.113 - 1.382x + .08x^2 - 0x^3 \quad (9)$

These Eqs. (2)–(9) were built based on field tests data and in the final procedure they were compared by them. As a dynamic modeling (Table 3) selection, the Logarithmic function had suitable correlation on scatter diagram. Therefore it was selected as the regression model for this work. The results of present work were compared with the works of other experts [39–43].

TABLE 3 Regression for anthropometric model.

Equation	Model Summary					Parameter Estimates			
	R-square	F	df1	df2	Sig.	Constant	b1	b2	b3
Linear $y = a_0 + a_1 x$	.001	.049	1	68	.825	11.511	-.027		
Logarithmic $\log y = \log a + b \log x$	.002	.134	1	68	.715	12.050	-.366		
Quadratic $y = a_0 + a_1 x + a_2 x^2$	.020	.689	2	67	.505	17.113	-1.382	.080	
Cubic $y = a_0 + a_1 x + a_2 x^2 + a_3 x^3$	.020	.689	2	67	.505	17.113	-1.382	.080	.000
Compound $A = Ce^{kt}$	.000	.017	1	68	.897	11.358	.999		
Growth $(dA/dT) = KA$	.000	.017	1	68	.897	2.430	-.001		
Exponential $y = ab^x + g$	.000	.017	1	68	.897	11.358	-.001		
Logistic $y = ax^b + g$	.000	.017	1	68	.897	.088	1.001		

Dependent Variable: VAR00001; The independent variable is VAR00003.

12.3.1 COMPARISON WITH OTHER WORKS

Rahmani-Nia and Hojjati, obtained the effect of exercise training on body composition and aerobic power of females. Comparison showed similarity in present work and the results of that works [44].

12.4 CONCLUSIONS

This work showed the effects of the Runner Velocity as the most important variable and runner weight against runner heart rate. Present work emphasized that “regression model” is accurate model for modeling of anthropometric and body motion. Hence, in order to presentation for influence of runner weight on Runner Velocity, it was compared the models for computational and field tests experiments. Based on these procedures, it was showed that the regression based model for anthropometric of body motion. Therefore, the dynamic or running state was compared by static or before of running condition. On the other hand, this idea were included the proper analysis to provide a dynamic response to the shortcomings of the body motion. Because increased activity has been associated with a reduction in the risk of sudden cardiac death, this work has demonstrated that a delayed decrease in HR after exercise would be a powerful and independent predictor of all cause mortality in patients or in general population. Consequently, the results of this work will help to reduce the risk of anthropometric of female athletes sport women activities.

NOMENCLATURES

P	Runner Blood Pressure (mm-Hg)
t	Running Time (sec)
V	Velocity (m/s)
S	Runner Stature (cm)
W	weight (kg)

KEYWORDS

- biomechanics
- injury mechanisms
- injury prevention
- risk factors
- sports injuries

REFERENCES

1. Hurst Thomas, D. Skull Wars Kennewick Man, Archaeology and the Battle for Native American Identity, Washington University: School of Law - Anheuser-Busch Hall, 2001; 38–41.
2. Bahr, Section I. **2005**, Tab 1 Page 5. (2).
3. Van Mechelen W. Am J Sports Med., Sep-Oct; **1993**, 21(5), 711–729.
4. Alderete, J.; Tupper, P.; Frisch, S. A. Arabic root cooccurrence restrictions revisited: A connectionist approach to phonological constraint induction, Proceedings of the 48th annual meeting of the Chicago Linguistics Society. **1993**.
5. Soligard T.; Myklebust G.; Steffen K. “Comprehensive warm-up programme to prevent injuries in young female footballers: cluster randomised controlled trial.” BMJ **2008**, 337, a2469. doi: 10.1136/bmj.a2469.PMC 2600961.PMID 19066253.
6. Nabieh, A.; Mohamed, I. Anthropometric Measurements as a Significant for Choosing Juniors in Both Volleyball and Handball Sports (Factorial Analysis Study), *World J. Sport Sci.* **2010**, 3(4), 277–289.
7. Rogana, S.; Hilfikerd, R.; Clarysb, P.; Clijsenc, R.; Taeymansac, J. Position-specific and Team-ranking-related Morphological Characteristics in German Amateur Soccer Players – a Descriptive Study, *Int. J. Appl. Sports Sci.*, **2011**, 23(1), 168–182.
8. Luigi P. W.; Bercades, T. somatotypes of national elite combative sport athletes, *Braz. J. Biometricity*, **2009**, 3(1), 21–30.
9. Dacres-Mannings, S.; Rochester, S.; Frail, H. Anthropometric profiles of Australian Rugby Institute, club and state level Rugby Union players. **2010**.
10. Gaurav, V.; Singh, M.; Singh, S. Anthropometric characteristics, somatotyping and body composition of volleyball and basketball players, *J. Phys. Educ. Sports Manage.* **2010**, 1(3), 28–32, Available online <http://www.acadjourn.org/jpesm>
11. Beat K.; Kohler, G. Influence of anthropometry on race performance in ultra-endurance triathletes in the longest triathlon in North America, *Int. SportMed J.*, **2007**, 8(2), 87–96, <http://www.ismj.com>

12. Joseph, J.; Greene, M. S.; Timothy, A.; McGuine, A. T. C.; Levenson, G.; Thomas M. Anthropometric and Performance Measures for High School Basketball Players, *J. Athl. Training*, **1998**, 33(3), 229–232
13. Amatya, D. L. Comparative Study of Somatotype of Nepalese Sportsmen, National Association for Sports Health and Fitness. **1999**.
14. Sang Hong, K. Kinanthropometric Study of Korean and Nepalese Marathon Runners, keimyung university Diwakar Lal Amatya, MSC-Athletics and Sports Science-India, Sports Kinanthropometrist-Australia, Sports Expert-National Sports Council, January. **2008**.
15. Gaunt, B. W. Anthropometric and Demographic Factors Affecting Distance Hopped-aid Limb Symmetry Index for the Crossover Hop-for distance test in High School athletes, *J. Orthop. Sport Phys.*, **2001**, 31(3), 145–151
16. Gross, M.; Dailey, E. S.; Melissa, D.; Dalton, A.; lee, K.; Wendy, I.; Ashley, C. Relationship Between Lifting Capacity and Anthropometric Measures, *J. Orthop. Sport Phys.*, **2000**, 30(5), 237–247, 258–262.
17. Roberto, C, Jose, A.; Perez, J.; Cortell, m. Juan, J.; Rivas, J. correlations among anthropometric parameters, jump power, and position in professional basketball players, **2008**.
18. Pui, W. K.; Hendrik H. Anthropometric, gait and strength characteristics of Kenyan distance runners, *J. Sports Sci. Med.* **2008**, 7, 499–504, <http://www.jssm.org>
19. Jawis, M. N.; Singh, R.; Singh, H. J.; Yassin, M. N. Anthropometric and physiological profiles of sepak takraw players, *Br. J. Sports Med.* **2005**, 39, 825–829. doi: 20.1136/bjism.2004.016915.
20. Lephart, S. M.; Abt, J. P.; Ferris, C. M.; Sell, T. C.; Nagai, T.; Myers, J. B.; Irrgang, J. Neuromuscular and biomechanical characteristic changes in high school athletes: a plyometric versus basic resistance program, *Br. J. Sports Med.* **2005**, 39, 932–938. doi: 17.1136/bjism.2005.019083.
21. Darr, K. C.; Bassett, D. R.; Morgan, B. J.; Thomas, D. P. Effects of age and training status on heart rate recovery after peak exercise. *Am. J. Physiol.* **1988**, 254, H340-H343.
22. Engerman, S. 'The Height of U. S. Slaves', *Local Population Studies*, **1976**, 16(1), 45–50.
23. Sunder, M. The Making of Giants in a Welfare State: The Norwegian Experience in the Twentieth Century', *Economics and Biology*, **2004**, 75–86.
24. Woitek, U. Height Cycles in the Eighteenth and Nineteenth Centuries', *Econ. Hum. Biol.*, **2003**, 243–258.
25. Fogel, R. 'Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the making of Economic Policy', *Am. Econ. Rev.*, **1994**, 369–394.
26. Baten, J. "Economic Development and the Distribution of Nutritional Resources in Bavaria, 1797–1839, in *J. Income Distrib.* **2000**, 9, 89–106.

28. Hariri Asli K. Water Hammer Research; Advances in Nonlinear Dynamics Modeling, (2012, pp. 88–121). Canada, USA: Published by Apple Academic Press, Inc., Exclusive worldwide distribution by CRC Press, a Taylor and Francis Group, Print ISBN: 9781926895314, eBook: 978-1-46-656887-7.
29. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. "Some Aspects of Physical and Numerical Modeling of Water Hammer in Pipelines," Nonlinear Dynamics An International Journal of Nonlinear Dynamics and Chaos in Engineering Systems, ISSN: 1573–269X (electronic version) Journal /1071 Springer, Netherlands, 2009, ISSN: 0924–090X (print version), Heidelberg, Germany, Springer, Volume 60, Number 4 / June, 2010, 677–701.
30. Heather, E. Collins-Schramm and others, "Markers that Discriminate Between European and African Ancestry Show Limited Variation Within Africa," Human Genetics 2002, 111, 566–569.
31. Komlos, J.; Baur, M. "From the Tallest to One of the Fattest: The Enigmatic Fate of the Size of the American Population in the Twentieth Century," Econ. Hum. Biol. 2004, 2(1), 57–74.
32. Komlos, J.; Kriwy, P. "The Biological Standard of Living in the Two Germanies," German Econ. Rev. 2003, 4, 493–507.
33. Levy, W. C.; Cerqueira, M. D.; Harp, G. D.; Johannessen, K. A.; Abrass, I. B.; Schwartz, R. S.; Stratton, J. R. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. Am. J. Cardiol., 1998, 82, 1236–1241.
34. Margo, R. The Heights of American Slaves. Social Science History, 1982, 6, 516–538.
35. Rahmani-Nia, F.; Hojjati, Z. The effect of exercise training on body composition and aerobic power in sedentary college age females, Int. J. Fitness, India, 2005, V1, 24–31.
36. Rahmani-Nia, F.; Hojjati, Z. Effect of selected training on body composition and aerobic power of Females College. J. Harkat, Iran, 2004, V1, 18–21.
37. Rahmani-Nia, F.; Rahnema, N.; Hojjati, Z.; Soltani, B. Acute effects of aerobic and resistance exercise on serum leptin and some risk factors of coronary heart disease in obese females. Sport sciences for health, 2008, 2(3), 118–124.
38. Rushton, J. P. Race, Brain Size, and Intelligence: A Rejoinder to Cain and Vanderwolf, Personality and Individual Differences, 2004, 11, 785–794.
39. Hariri Asli, K.; Nagiyev, F. B.; Haghi, A. K. A computational approach to study fluid movement, Nanomaterials Yearbook - 2009, From Nanostructures, Nanomaterials and Nanotechnologies to Nanoindustry, (2010, pp. 181–196). USA: Nova Science Publications. ISBN: 978-1-60876-451-8.16
40. Young, J. W. Anthropometric and Mass Distribution Characteristics of the Adult Female FAA Civil Aeromedical Institute, 2008, FAA 9–83.
41. Bartlett, R. Introduction to Sports Biomechanics, 2nd Edition. Taylor and Francis- Library. 2007.

42. Hariri Asli, K.; Haghi, A. K. A Numerical Study on Fluid Flow and Pressure drop in Microtubes, *J. Balkan Tribological Association*, Tribotechnics and tribomechanics, Sofia, Bulgaria, **2010**, *16*(3), 382–392.
43. Goonetilleke, R. S.; Ho, Cheuk Fan E.; So, R. H. Y. Foot Anthropometry in Hong Kong. (pp. 81–88) Proceedings of the ASEAN 97 Conference, Kuala Lumpur, Malaysia. **1997**.
44. Rahmani-Nia, F.; Hojjati, Z.; Rahnama, N.; Soltani, B. Leptin, heart disease and exercise. *World J. Sport Sci.*, Iran, **2009**, *2*(1), 13–20.

CHAPTER 13

SOME ASPECT AND PRACTICAL HINTS OF PHYSICAL ACTIVITY

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13.1 INTRODUCTION

The analysis of data shows that the norms of WHR, BMI, and WC among female employees are the predictor indices in epidemiological and metabolism studies and also for the outbreak of diseases related to obesity. These differences are probably due to a series of cultural, social, and economic differences, nutrition disorders, lack of regular exercise, and lack of awareness of risks resulting from obesity and sedentary lifestyle.

13.2 MATERIALS AND METHODS

This article focuses on the benefits of physical activity for human health. As mentioned before, the objective of the present study is to evaluate the relationship between body mass index and waist hip ratio and also the level of physical activity of the respondents. All female employees of the Islamic Azad University of Lahijan ($n = 60$) aging from 20 to 60 years old were studied in terms of physical activity, height, weight, BMI, and WHR. The questionnaire of rapid assessment of physical activity was used in this study to collect data. This questionnaire consists of two parts; the first part is related to aerobic activities, and the second part deals with the strength trainings and stretching activities (Yoga). It also includes nine questions, seven of them are related to aerobic activities which are rated by the type of activity (inactive = 1, under active = 2 and 3, regularly under active = 4 and 5, and active = 6 and 7), the next two questions are related to strength trainings with the rating of 1, and the last question is about stretching activities (Yoga) that is rated 2. The rating for the use of any type of physical activity is 3 [1–18].

After measuring the height and weight of the respondents using a digital scales made in Germany by Seca company, the obtained values were put in the formula, $\text{Weight (kg)}/\text{Height (m}^2\text{)}$, in order to determine the body mass index (BMI). Waist to hip ratio was determined by measuring the maximum size of the hips from slightly above the navel and using the equation $\text{waist circumference}/\text{pelvis circumference}$. It is noteworthy to say that personal information including education, being athlete or not, being single or married, and so on was also collected. Physical activity level and

personal information of the respondents were reported using descriptive statistics. After performing the necessary measurements and data collection, Spearman and Pearson correlation coefficients were used to evaluate the relationships of above-mentioned factors. Chi-2 test was also used to show the relationship between qualitative variables. All statistical analyses were done by the SPSS software [19–21].

13.3 RESULTS AND DISCUSSION

The average of age, height, and weight of the respondents were 39.75 ± 9.05 years, 160.63 ± 4.74 cm, and 65.57 ± 10.63 kg, respectively. Mean values of BMI, WHR, and WC were also 25.43 ± 4.72 kg/m², 0.89 ± 0.06 cm, and 95.05 ± 11.5 cm, respectively (Table 1).

TABLE 1 Anthropometric characteristics of the participants ($\bar{X} \pm SD$).

Height (cm)	Weight (kg)	WC (cm)	BMI (kg/m ²)	WHR
160.63 ± 4.74	65.57 ± 10.63	95.05 ± 11.5	25.43 ± 4.72	0.89 ± 0.06

Among the respondents, 1.66% (1 person) had a less than normal weight, 51% (31 people) had a normal weight, and 46.66% (28 people) were overweight (25–30) or obese (30–35) (Table 2).

TABLE 2 Information about body mass index (BMI) of the female subject.

Percentage	Range	BMI
1.66	<18.5	Lightweight
51.68	18.5–25	Normal
46.66	>25	Overweight or Obese

According to the final standardization (Table 3), about half of the female employees were in the range of 18.5–25 (kg/m²) of BMI and about 47% were in the range of obese. Obtaining a average BMI of 28.30 kg/m² in the age group above 51 years indicates that these people have a more in-

consistent body composition than the other age groups [22–26]. In this age group, which is the onset of elderly, the highest percentage of malnutrition in terms of obesity was observed among the respondents and the prevalence of obesity was increased in higher ages moreover, the highest BMI was observed among them (Table 3).

TABLE 3 Descriptive information about the respondents at different age groups.

Age group	Number	Mean weight (kg)	Mean height (cm)	Mean BMI (kg/m ²)	Mean WHR
20–30	5	66.18	157.8	25.26	0.87
30–40	24	65.19	161.95	24.79	0.89
40–50	22	65.04	160.40	25.46	0.89
50–60	9	66.55	159.22	28.30	0.9

The results show that mean value of WHR in the participants was 0.89 ± 0.06 (Table 1). Among them, 1.66% (1 person) was less than normal rate, 81.68% (49 people) had high WHR, and 16.66% (10 people) had a normal WHR, who are susceptible to imaginary diseases such as heart disease (Table 4).

TABLE 4 Information about WHR index (waist to hip ratio) of the participants.

WHR	Range	Percentage
Under normal	<0.75	1.66
Normal	0.75–0.85	16.66
Obese	>0.85	81.68

Although this ratio is more or less equal in different age groups of the study population, what that has been obtained from the results is that the average WHR was increased with aging in age groups, as the minimum average of WHR (0.87) was observed in the age group 20–30, and its maximum (0.9) belonged to the age group above 51 (Table 1).

Another finding of the present study is an average positive significant relationship between BMI and WHR and WC ($r = 0.29$ and $r = 0.69$,

respectively) ($p \leq 0.05$). The relationship between BMI and physical activity levels of females was also examined and a negative significant relationship was observed between them, as an increase in the physical activity of these women was followed by a reduction in this index ($p \leq 0.01$).

The participants were also divided in two groups in term of education. These two groups included below the bachelor and above the bachelor employees, but there was no significant difference between them in terms of BMI and WHR [27–33] (Table 5).

TABLE 5 The correlation of anthropometric indices and physical activity in female employees.

	Activity level	p-Value
WHR	−0.3	0.8
BMI	−0.43*	0.01

*significant correlation.

The values of waist circumference (WC) showed that 83.33% of the females are exposed to high risk of diseases related to health. The World Health Organization (WHO) has recommended the WHR to be 0.8 for women. Higher values can lead to abdominal obesity and increased risk of cardiovascular diseases in women. Therefore, 86.66% of the participants in the present study are susceptible to cardiovascular diseases (Table 6).

TABLE 6 The relationship between anthropometric indices and risk of cardiovascular diseases.

Obesity index	N	%
WC (cm)		
No Risk (>88 cm)	10	16.66
At Risk (>88 cm)	50	83.33
WHR (cm)		
No Risk (<0.85)	8	13.33
At Risk (>0.85)	52	86.66

As previously mentioned, the value of BMI in 60 female employees with an average age of 39 years old was $25.43 \pm 4.72 \text{ kg/m}^2$ (Table 1), while BMI was obtained $26.6 \pm 4.2 \text{ kg/m}^2$ for the faculty members of Tarbiat Moaalem University of Sabzevar in a study conducted by Hamed-Nia.

Moreover, previous studies have shown that a BMI more than 25 is the overweight or obesity indicator for women that put them at the risk of diseases. So, 46.6% of the participants in the present study are in this range (Table 3) and it can be inferred that about the half of them are overweight or obese. The average BMI and WHR in the age groups 20–30, 30–40, 40–50, and 50–60 were determined as 25.26 and 0.87, 24.79 and 0.89, 25.46 and 0.89, and 28.30 and 0.9, respectively. These results are consistent, to some extent, with findings of Gaeni and Lameyi [1], in which the average BMI and WHR for women in Tehran in the age groups 36–40 years and above 51 years were obtained as 26.38 ± 0.9 and 25.68 ± 0.9 , respectively. In a study entitled “Evaluation of obesity and underweight of the elderly using BMI and WHR indices in Isfahan” by Naeini et al., [23] the highest prevalence of obesity (according to BMI) was observed in the age group 60–69.

The results show that the average WHR of the respondents is 0.89 (Table 1). Although this ratio is more or less equal in different age groups of the study population, what that has been obtained from the results is that the average WHR was increased with aging in age groups, as the minimum average of WHR (0.87) was observed in the age group 20–30, and its maximum (0.9) belonged to the age group above 51 (Table 2). According to another classification of obesity in women based on WHR index, women with a WHR more than 0.8 are classified as obese individuals [34–39].

The findings of the present study also indicate that 86.66% of the participants are at the risk of cardiovascular diseases. Josephine [38] in a study on factors threatening the health of new students entering the University of Santo Tomas, showed that 4.47% of students were at risk and this low figure was mainly due to the fact that the studied students were young and had a suitable BMI and body composition.

One of the findings of the present study was the positive relationship between BMI and WHR in female employees of the Islamic Azad University of Lahijan ($r = 0.29$), which is in argue with the findings of most researchers. This correlation was reported as 0.374 in the study of Gaeni

and Lameyi. [1] and Josephine [38] also obtained a correlation of 0.14 in a study on anthropometric characteristics (BMI and WHR) of first-year students. In the present study, the BMI of those who had a physical activity score above 5 was 24.06 ± 2.52 , while in the study of Stephanie on physical activity and endogenous sex hormones changes in postmenopausal women, BMI was obtained 25.8 in those who had a physical activity score above 5 (Table 7).

TABLE 7 Selected characteristics of participants, categorized by physical activity levels.

	Level of sport activity			
	1(0.0)	2(0.1–2.9)	3(3.00–6.9)	4(7.0–9.9)
	$\bar{x} \pm SD$	$\bar{x} \pm SD$	$\bar{x} \pm SD$	$\bar{x} \pm SD$
BMI(kg/m ²)	–	28.17 ± 5.93	24.06 ± 2.52	23.29 ± 0.73
WHR	–	0.92 ± 0.3	0.98 ± 0.56	0.85 ± 0.04

In a study by Gupta [37] on body mass index (BMI), the ratio of waist circumference to pelvis circumference (WHR), and cardiovascular risk factors, a negative correlation was observed between physical activity and BMI ($r = -0.22$). As expected, these findings are in line with the results of the present study.

In the present study, the relationship between anthropometric indices, which are known markers for cardiovascular diseases, was also evaluated.

The correlation between BMI and WHR in female employees of the Islamic Azad University of Lahijan was obtained as 0.65. This correlation was reported 0.84 in a study by Mildred [39] on correlates of body image satisfaction among economically depressed urban Filipino women.

Similar studies by Janssen et al., [30] have shown that the combination of BMI and WHR can be the best predictor of metabolic risk factors rather than WC alone. In the present study, 83.33% of women whose WC was above 88 cm were at the risk of heart diseases, and they mostly had big and abnormal abdomens. Low levels of physical activity and poor nutrition are the possible reasons for the high WC in the studied female employees here.

In the study of Josephine [38], about 59% of people whose WC was above 88 cm were at the risk of heart diseases. This is, to some extent, less than WC in female employees in the present study, which can be attributed to lack of regular physical activity in their daily schedule because of their businesses and the economic condition of society [40].

13.4 CONCLUSION

Given the inverse relationship between physical activity and body mass index (BMI), effective weight control should be one of the components of interceptive programs of university staff. Since the results of the present study show a gradual increase in overweight and obesity and given their many risks such as cardiovascular diseases, strokes, diabetes, and even death, specific interventions aimed at regulating body mass index (BMI) should be taken into account more than before by the university officials. Thus, the role of physical education instructors and observational education in relation to physical activity seem to be important and should be considered.

KEYWORDS

- **body fat**
- **body mass index**
- **cardiovascular diseases**
- **physical activity**

REFERENCES

1. Gaeeni, A.; Lameyi, T. **2003**, The relationship between percent body fat (%BF) and Body mass index (BMI), and the ratio of waist to hip circumference (WHR) women over fifteen years in Tehran. *Harkat Journal*, 17, 95–105. (In Persian with English abstract).

2. Caspersen, C. J.; Powell, K. E.; Christenson, G. M. Physical activity, exercise, and physical fitness: definitions and distinctions for health-related research [abstract]. *Public Health Report*. 1985; *100*(2), 126–131. Accessed July 2, 2009. PMID: 1424733 <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1424733>
3. World Health Organization Regional Office for Europe Website. A Physically Active Life Through Everyday Transport. Accessed September 28, 2010. Download the pdf document here or log on at <http://www.euro.who.int/en/what-we-do/health-topics/environmental-health/Transport-and-health/publications/pre-2009/a-physically-active-life-through-everyday-transport-2002>
4. Caspersen, C. J.; Powell, K. E.; Christenson, G. M. Physical activity, exercise, and physical fitness: definitions and distinctions for health-related research. *Public Health Reports*. 1985; *100*(2), 126–131. Accessed July 2, 2009. <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1424733>
5. Cornil, A.; De Coster, A.; Copinschi, G.; Franckson, J. R. M. “Effect of muscular exercise on the plasma level of cortisol in man.” *European J. Endocrinol.* **1965**.
6. Cohen S.; Williamson, G. M. “Stress and infectious disease in humans.” *Psychological Bulletin* **1991**, *109*, 5–24. doi: 10.1037/0033-2909.109.1.5. PMID 2006229.
7. Jason Menoutis, Ed. D. “Physical Activity and Health”(Abstract). *Nasm Pro*. Retrieved 2008–08–25. **2008**.
8. Borer, K. T.; Wuorineen, E. C.; Lukos, J. R.; Denver, J. W.; Porges, S. W.; Burant F (August 2009). “Two bouts of exercise before meals but not after meals, lower fasting blood glucose.” *Medicine in Science and Sports and Exercise* *41* (8), 1606–14. doi: 10.1249/MSS.0b013e31819dfe14. PMID 19568199.
9. Silberman, Joanne. “100 years Ago, Exercise Was Blended Into Daily Life.” *npr.org*. Retrieved 23 November 2010. **June 7, 2010**.
10. Wislett, Ulrik; Ellingsen, O.; Kemi O. “High-Intensity Interval Training to Maximize Cardiac Benefit of Exercise Training?” *Exercise and Sports Sciences Reviews* **July 2009**, *37*(3), 139–146. doi: 10.1097/JES.0b013e3181aa65fc. PMID 19550205.
11. Hanc, J. 1987. Your Health Behind the Runner’s Euphoria. *Newsday*, April 21, **1987**, 11. Retrieved October 5, 2006, from ProQuest database
12. Sparling, P. B.; Giuffrida, A.; Piomelli, D.; Roskopf, L.; Dietrich A (December 2003). “Exercise activates the endocannabinoid system.” *NeuroReport* *14*(17), 2209–2213. doi: 10.1097/01.wnr.0000097048.56589.47. PMID 14625449.
14. Burfoot, Amby. “Runner’s High.” *Runner’s World*. Retrieved 2009–10–15. **June 2004**.
16. Bouchard, Claude; Ping An, Treva Rice, James S. Skinner, Jack H. Wilmore, Jacques Gagnon, Louis Perusse, Arthus S. Leon, D. C.; Rao. “Familial aggregation of $\dot{V}O_{2\max}$ response to exercise training: results from the HERITAGE Family Study.” *J. Appl. Physiol.* *87*(3), 1003–1008. PMID 10484570. Retrieved 2007–07–17. **September 1, 1999**.
17. Kolata, Gina (February 12, 2002). “Why Some People Won’t Be Fit Despite Exercise.” *The New York Times*. Retrieved 2007–07–17.
18. Hubal, MJ; Gordish-Dressman, H.; Thompson, P. D.; Price, T. B.; Hoffman, E. P.; Angelopoulos, T. J.; Gordon, P. M.; Moyna, N. M.; Pescatello, L. S.; Visich, P. S.; Zoeller, R. F.; Seip, R. L.; Clarkson, P. M. “Variability in muscle size and strength gain after

- unilateral resistance training.” *Med. Sci. Sports Exerc.* **June 2005**, 37 (6), 964–972. PMID 15947721.
19. Brutsaert, Tom D.; Esteban J. Parra “What makes a champion? Explaining variation in human athletic performance” (PDF). *Respiratory Physiol. Neurobiol.* **2006**, 151 (2–3): 109–123. doi: 10. 1016/j. resp. 2005. 12. 013. PMID 16448865. Archived from the original on 2007–08–10. Retrieved 2007–07–17.
 20. Geddes, Linda (2007–07–28). “Superhuman.” *New Scientist*. 35–41.
 21. Stefanie, L.; Martina E Physical activity and endogenous sex hormones in postmenopausal women. *Cancer Causes Control*, **2011**, 22, 81–89.
 22. Enas, E. A. Coronary artery disease epidemic in Indians: a cause for alarm and far action. *J. Indian Med. Assoc.*, **2000**, 98, 694–702.
 23. Naeini, M.; Dorostimotla, A.; Aghdak, P. Checking of obesity, low weight, elderly and factors associated with BMI, WHR, PBF the use of city indexes. *Journal of mazandaran University of Medical Sciences*, **2006**, 52(50), 117–123.
 24. Delavar, A. Research methods in psychology and educational sciences; 1st edition; Tehran; Payame Noor University Press. **2000**.
 25. Akbarzadeh R **1996**, Relationship between the ratio of waist circumference to hip circumference and risk factors and increased lipids and blood pressure in patients with insulin-dependent *Diabetes* referred to the Endocrinology Institute in Tehran in 1995 and 1996. A Master’s thesis, Iran University of Medical Sciences; Faculty of Nursing.
 26. Kaye, S. A.; Fdosom, A. R.; Jacobs, D. R. **1995**, Psychosocial correlates of body fat distribution in Black and white young adult. *Int. J. Obes.*, 17, 271–277
 27. Folsom, A. R.; Kaye, S. A.; Sellers, T. A.; Hong, C. P.; Cerhan, J. R.; Potter, J. D.; Pclineass RJC, Body fat distribution and 5-year risk of death in older women. *JAMA*, **1993**, 269, 483–487.
 28. Dalton, M.; Cameron, A. S.; Zimmet, P. Z.; Shaw, J. E.; Jolley, D.; Dunstan, D. W.; Welbon, T. A. Waist circumference, waist-hip ratio and body mass indices and their correlation with cardiovascular disease risk factors in Australian adults. *J Intern Med*, **2003**, 254, 255–563.
 29. Hammer, L. D. and Wilson, B. M. Impact of pubertal development on body fat distribution among white, *Hispanic J. Pediatr.*, **1991**, 88, 975–980.
 30. Janssen, I.; Heymsfield, S.; Allison, D.; Kotler, D.; Ross, R. Body mass and waist circumference independently contribute to the prediction of non-abdominal subcutaneous and visceral fat. *Am. J. Clin. Nutr.*, **2002**, 75, 683–8.
 31. Ashwell, M.; Lejeane, S. Ratio of waist circumference to height maybe better indicator need for weight management. *BMI*: **1990**, 312–377.
 32. Folsom ARJ, Stevens, P. J.; Schreiner, P. A.; McGovern, Body mass index, waist hip ratio and coronary heart disease incidence in African Americans and whites. *Am. J. Epidmeiol.*, **1998**, 148, 1187–94.
 33. Gharakhanloo, R.; Gaeyni, A.; Peyghoon, A. Standardization waist -to-hip ratio for men over 40years to the city of Ahvaz and its association with cardiovascular risk factor-*Diabetes* and cardiovascular desease. *Olympic J. Iran*, **2002**, 3-4, 59–73. (In Persian with English abstract).
 34. Pollock, M. L.; Wilmore, Y. N. **1990**, Exercise in health and disease (2th Ed). Philadelphia: W. B. Sundials.

35. World Health Organization. Obesity: preventing and managing the global epidemic. Report of WHO consultation on obesity, Geneva, June3–5. **1998**.
36. Hamed-Nia, M.; Rezaei, S. Some risk factors associated with physical activity and body fat percentage cardio-vascular university faculty of Medical sciences and Health Services Blog, The Eleventh: **2007**, 34–40. (In Persian with English abstract).
37. Gupta, R. P.; Rastogi et al. Body mass index, waist-size, waist-hip ratio and cardiovascular Risk factors in urban subjects. *JAPI*, **2007**, 55, 621–627.
38. Josephine J B, Lorenz, M. et al. Health risks determinants among freshmen students of the University of Santo Tomas. *International Journal of sport science and Engineering*, **2011**, 1, 58–64.
39. Mildred, T.; Consuelo, L. Correlates of body image satisfaction among economically depressed urban Filipino women. *Philippine J. Sci.*, **2009**, O: 67–74.
40. Vansant, G. B.; Beaten, C.; Weststrate, J.; Deurenberg, P. Body fat distribution and the prognosis for weight reduction preliminary observation. *Int. J. Obes.*, **1988**, 12, 1330–1340.

CHAPTER 14

HEALTH AND TOTAL AMOUNT OF BODY FAT

CONTENTS

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14.1 INTRODUCTION

Resistance training (RT) is a behaviourally feasible and efficacious alternative to endurance exercise for weight control. RT can reduce total body fat mass in men and women, independent of dietary caloric restriction. However, regional distribution of fat may be more important to health than the total amount of body fat. Excessive central obesity and especially visceral adipose tissue have been linked with the development of hyperlipidemia, hypertension, insulin resistance and glucose intolerance, diabetes, and heart disease. Fat distributed in the arms and legs, however, appears to impose little or no risk. Although there may be a genetic predisposition for visceral adipose tissue, increasing age, high fat diets, and a sedentary lifestyle are also important determinants.

14.2 MATERIALS AND METHODS

Several studies have demonstrated decreases in visceral adipose tissue after RT programs. Treuth and co-workers assessed body composition in older men by using dual-energy x-ray absorptiometry and in older women by using computed tomography and observed significant decreases in visceral fat after 16 weeks of RT. Ross et al., used magnetic resonance imaging to measure regional fat losses after exercise combined with diet interventions. In their first study both diet plus aerobic exercise and diet plus RT elicited similar losses of visceral fat that were greater than losses of whole-body subcutaneous fat. In a follow-up study, they isolated the effects of endurance exercise training and RT by comparing the responses to diet alone and diet combined with each training modality in middle-aged obese men. All three groups lost significant amounts of total body fat, and all three groups experienced a significantly greater visceral fat loss compared with whole-body subcutaneous fat loss. The changes amounted to a 40% reduction in visceral fat in the RT and diet group, 39% in the endurance training and diet group, and a 32% reduction in the diet-only group [1–22].

14.3 RESULTS AND DISCUSSION

Studies of the efficacy of RT in the context of total body weight loss have had mixed results. Studies that use more severe caloric intake restriction have not shown gains in muscle mass, whereas RT studies with less severe caloric restriction have shown muscle mass gains with only modest losses in body weight. RT studies that attempt to maintain caloric balance during the intervention typically do not observe major changes in body weight in either gender, despite significant reductions in fat mass and percent body fat. In essence, body weight does not change much because loss of fat mass is generally offset by the gain in muscle mass. Conversely, endurance training-induced decreases in fat mass are more likely to be associated with reductions in body weight because there is no offsetting gain in muscle mass [23–27].

14.4 CONCLUSION

A summary of RT guidelines is presented in. RT of all major muscle groups can be accomplished through the use of expedient programs. Indeed, adherence rates in RT interventional studies are high and due, in part, to the minimal time requirement for full participation. Most studies report that RT 3 days per week elicits superior strength gains when compared with training regimens of lower frequency. However, if training intensity remains high (7 to 10 repetitions performed to momentary muscular failure), RT only 2 days per week produces approximately 80% of the strength benefits reported by studies using traditional 3 days-per-week routines. Using scientific evidence and expert opinion, the American Heart Association, with endorsement of the American College of Sports Medicine, has promulgated RT guidelines for individuals with and without CV disease. The guidelines for those without CV disease are summarized briefly herein. RT is recommended a minimum of 2 days per week, with progression to 3 days per week. A typical workout should consist of 8 to 10 exercises to cover the major muscle groups, which includes the chest, shoulders, arms, back, abdomen, thighs, and lower legs. The resistance or weight lifted should be moderate, which is defined as 30% to 40% of 1 RM for upper

body exercises and 50% to 60% of 1 RM for lower body exercises. If maximal strength testing is not available, the individual can, through trial and error, use a weight that can be lifted for a minimum of 8 to 10 repetitions. When 12 to 15 repetitions can be accomplished with little difficulty, the weight is increased. This progressive resistance strategy meets the requirements of the overload principle, which is the basis for improvement in strength. Furthermore, by using moderate weight and gradually increasing the workload in stages, there is less risk of musculoskeletal injury while maintaining effectiveness of the workout.

KEYWORDS

- **aerobic activities**
- **body exercises**
- **maximal strength**
- **resistance training**
- **training regimens**

REFERENCES

1. Hurst Thomas, D. Skull Wars Kennewick Man, Archaeology and the Battle for Native American Identity, Washington University: School of Law - Anheuser-Busch Hall, **2001**, 38–41.
2. Bahr, Section I. 2005, TAB 1 Page 5. (2).
3. Van Mechelen W. Am J Sports Med., Sep-Oct; **1993**, 21(5), 711–9.
4. Alderete, J.; Tupper, P.; Frisch, S. A. Arabic root cooccurrence restrictions revisited: A connectionist approach to phonological constraint induction, Proceedings of the 48th annual meeting of the Chicago Linguistics Society. **1993**,
5. Soligard T.; Myklebust G.; Steffen K. “Comprehensive warm-up programme to prevent injuries in young female footballers: cluster randomised controlled trial.” BMJ **2008**, 337, a2469. doi: 10.1136/bmj.a2469.PMC 2600961.PMID 19066253.
6. Nabieh, A.; Mohamed, I. Anthropometric Measurements as a Significant for Choosing Juniors in Both Volleyball and Handball Sports (Factorial Analysis Study), *World J. Sport Sci.* **2010**, 3(4), 277–289.
7. Rogana, S.; Hilfigerd, R.; Clarysb, P.; Clijsenc, R.; Taeymansac, J. Position-specific and Team-ranking-related Morphological Characteristics in German Amateur Soccer Players – a Descriptive Study, *Int. J. Appl. Sports Sci.*, **2011**, 23(1), 168–182.

8. Luigi P. W.; Bercades, T. Somatotypes of national elite combative sport athletes, *Braz. J. Biomotricity*, **2009**, 3(1), 21–30.
9. Dacres-Mannings, S.; Rochester, S.; Frail, H. Anthropometric profiles of Australian Rugby Institute, club and state level Rugby Union players. **2010**.
10. Gaurav, V.; Singh, M.; Singh, S. Anthropometric characteristics, somatotyping and body composition of volleyball and basketball players, *J. Phys. Educ. Sports Manage.* **2010**, 1 (3), 28–32, Available online <http://www.acadjourn.org/jpesm>
11. Beat K.; Kohler, G. Influence of anthropometry on race performance in ultra-endurance triathletes in the longest triathlon in North America, *Int. SportMed J.*, **2007**, 8(2), 87–96, <http://www.ismj.com>
12. Joseph, J.; Greene, M. S.; Timothy, A.; McGuine, A. T. C.; Leversson, G.; Thomas M. Anthropometric and Performance Measures for High School Basketball Players, *J. Athl. Training*, **1998**, 33(3), 229–232
13. Amatya, D. L. Comparative Study of Somatotype of Nepalese Sportsmen, National Association for Sports Health and Fitness. **1999**.
14. Sang Hong, K. Kinanthropometric Study of Korean and Nepalese Marathon Runners, keimyung university Diwakar Lal Amatya, MSC-Athletics and Sports Science-India, Sports Kinanthropometrist-Australia, Sports Expert-National Sports Council, January. **2008**.
15. Gaunt, B. W. Anthropometric and Demographic Factors Affecting Distance Hopped-aid Limb Symmetry Index for the Crossover Hop-for distance test in High School athletes, *J. Orthop. Sport Phys.*, **2001**, 31(3), 145–151
16. Gross, M.; Dailey, E. S.; Melissa, D.; Dalton, A.; lee, K.; Wendy, I.; Ashley, C. Relationship Between Lifting Capacity and Anthropometric Measures, *J. Orthop. Sport Phys.*, **2000**, 30(5), 237–247, 258–262.
17. Roberto, C. Jose, A.; Perez, J.; Cortell, m. Juan, J.; Rivas, J. Correlations among anthropometric parameters, jump power, and position in professional basketball players, **2008**.
18. Pui, W. K.; Hendrik, H. Anthropometric, gait and strength characteristics of Kenyan distance runners, *J. Sports Sci. Med.* **2008**, 7, 499–504, <http://www.jssm.org>
19. Jawis, M. N.; Singh, R.; Singh, H. J.; Yassin, M. N. Anthropometric and physiological profiles of sepak takraw players, *Br. J. Sports Med.* **2005**, 39, 825–829. doi: 20.1136/bjism.2004.016915.
21. Lephart, S. M.; Abt, J. P.; Ferris, C. M.; Sell, T. C.; Nagai, T.; Myers, J. B.; Irrgang, J. Neuromuscular and biomechanical characteristic changes in high school athletes: a plyometric versus basic resistance program, *Br. J. Sports Med.* **2005**, 39, 932–938. doi: 17.1136/bjism.2005.019083.
22. Darr, K. C.; Bassett, D. R.; Morgan, B. J.; Thomas, D. P. Effects of age and training status on heart rate recovery after peak exercise. *Am. J. Physiol.* **1988**, 254, H340–H343.
23. Engerman, S. ‘The Height of U. S. Slaves’, *Local Population Studies*, **1976**, 16, 1, 45–50.
24. Sunder, M. The Making of Giants in a Welfare State: The Norwegian Experience in the Twentieth Century’, *Economics and Biology*, **2004**, 75–86.
25. Woitek, U. Height Cycles in the Eighteenth and Nineteenth Centuries’, *Econ. Hum. Biol.*, **2003**, 243–258.

26. Fogel, R. 'Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the making of Economic Policy', *Am. Econ. Rev.*, **1994**, 369–394.
27. Baten, J. "Economic Development and the Distribution of Nutritional Resources in Bavaria, 1797–1839, *J. Income Distrib.* **2000**, 9, 89–106.

CHAPTER 15

PRACTICE HINTS FOR REGULATING BODY WEIGHT

CONTENTS

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15.1 INTRODUCTION

The discovery of obese gene over 12 years ago provided new insight into the mechanisms through which body weight and composition are regulated. Evidence has suggested that leptin plays a specific role in the intricate cascade of cardiovascular events. Although many studies have been published on the effect of exercise on leptin, numerous questions remain to be answered. There is need for more investigations to study the effects of resistance exercise on leptin. The objective of this study was to study leptin and some coronary heart disease risk factors responses in young females after resistance exercise training. Eighteen inactive obese girls (age = 21.1 ± 1.4 years, BMI = 32.1 ± 1.5 kg/m²) volunteered to participate in the study. They were divided to two equal resistance exercise (RT) and control (Co) groups. Fast blood samples were collected at baseline, 4 weeks and 8 weeks after RT. The exercise program was for 8 weeks and 3 sessions (12 exercises, 4 sets, 15 repetitions, 60% of 1RM, with 3 minutes rest) per week. Body composition (BI method) and $\text{VO}_{2\text{max}}$ (standard exercise test and gas analyzer) were assessed at the same time of blood sampling. All examinations were done in follicular phase of menstrual cycle. RT group subjects were undertaken an isocaloric diet. Data were compared with a 2 (group) $\times$ 3 (before, 4 weeks and 8 weeks of exercise training) repeated measures ANOVA. One-way ANOVA was used for determining between group differences. LSD post hoc was used to determine specific differences when a significant interaction or group effect ($p < 0.05$) was obtained. No significant differences ($p < 0.05$) were observed between the exercise and control groups on serum leptin, insulin, LDL-C, HDL-C, total cholesterol (TC), triglyceride (TG) and glucose (Glue) at any trails ($p < 0.05$). Waist to hip ratio (WHR), BMI and body fat percent (BF) significantly decreased with RT ($p < 0.05$). $\text{VO}_{2\text{max}}$ increased after 4 weeks and 8 weeks trails compare to baseline ($p < 0.05$). Main finding of this study is that 8 weeks resistance training was not sufficient to product favorite changes on serum leptin and some dependent factors to coronary heart disease. Obesity gene discovery about 12 years ago on how the new horizons opened up mechanisms regulating body weight and body composition. Leptin is an obesity gene product, the results of research will play a special role in heart problems. Brnan

effect of resistance training on serum leptin, eighteen girls were obese, sedentary lifestyle and diet in the last year or did not participate in any training program, volunteered to participate in the study. Of these, nine patients were randomized to resistance training (RT) (5.1 ± 1.21 years, $\text{BMI} = 27.1 \pm 9.31 \text{ kg/m}^2$) and nine controls (Co) (3.1 ± 2.21 years, $\text{BMI} = 3.1 \pm 2.32 \text{ kg/m}^2$) were used. Experimental group 8 weeks of resistance training three sessions per week for about an hour (12 stations, four courses, each of 15 repetitions at 60% one repetition maximum 3-minute break) came into force. Subjects Biochemical factors, body composition and a $\text{Vo}_{2\text{max}}$ at baseline, 4 and 8 weeks after training in both groups were measured. Measurements were performed during the follicular menstrual cycle (based on Importantly, three months before starting the study). Biochemical factors include leptin, insulin (ELISA) and some risk markers for coronary heart disease. Data obtained in this study, analysis of variance with repeated measures and LSD post hoc test was used to determine the location and orientation differences. The results of this study showed no significant changes in leptin after 4 and 8 weeks of starting the program, the experimental group and the control ($p < .05$). At the same time, changes in insulin three times the size of the two groups showed no significant difference ($p < .05$). The total cholesterol (TC), LDL-C, HDL-C, triglycerides (TG) and glucose (Glue) levels were measured at intervals between any two groups showed no significant change ($p < .05$). However, the ratio of waist to hip circumference (WHR) and body mass index (BMI) had decreased significantly in the RT group ($p < .05$). Reduce fat in the first group of measurements to 4 weeks was significant, but not significant from 4 to 8 weeks. course, the difference between the percentages of fat was significant pretest to 8 weeks ($p < .05$). Values in the RT group increased $\text{Vo}_{2\text{max}}$ between 4 and 8 weeks after training, and it demonstrated the increasing trend was observed between 4 and 8 weeks ($p < .05$). It seems to be an 8-week resistance training program that does not limit calories for desired changes in leptin in obese girls, not enough. Ahtmalachnanchh time program over 12 weeks increased or dietary restrictions were also imposed, favorable results were obtained in leptin changes. Leptin is a hormone mainly secreted by adipose tissue, is now the subject of many investigations. These hormones as peripheral feedback, informs the hypothalamus on energy re-

sources and may play a key role in understanding the causes of obesity are reasonable. The main function of leptin is to decrease NPY (appetite stimulating factor) may have an inhibiting effect on appetite and increase the performance of the sympathetic nervous system, energy consumption also increased. In fact, normal people do not have the complication of obesity, anti-obesity hormone leptin as is, which means that after eating, the amount of this substance in the blood increases, and consequently, the recipient of the hypothalamic satiety center, stimulated and the person will lose the appetite to eat [1–13]. Changes in serum leptin levels and impaired dynamic alert mechanism that can correct the destructive impact of body weight on the left. Leptin receptor deficiency or lack of appropriate practice for the genetic roots of obesity seem adequate. However, the majority of human obesity, type II (trouble maker), but it actually shows the onset of obesity is not. High long-term leptin increases arterial pressure and heart rate via central and peripheral mechanisms are high levels of leptin in adolescent smoking is a sign of weakness. In obese people, leptin increases blood loss and cardiac function, simultaneous reactions occur, the problem is not found in normal individuals. The leptin damage, abnormal proliferation and accelerate the replacement of calcium in the blood vessels. Some studies focus on the effects of these hormones are produced and accelerated clot shows. In research on human subjects have shown that high levels of leptin in blood platelet aggregation in vitro speeds. In an epidemiological study, increased leptin as an independent risk factor for development of cardiovascular disease has been reported. Any kind of action to reduce the levels of leptin, may play an important role in preventing cardiovascular disease. In many studies in recent decades, various types of exercise and diet regime as an effective method of treatment in this field has been emphasized. The method of training, resistance-training (RT) applications in a recent research works, has appeared. Ryan and colleagues 16 weeks of resistance training on plasma leptin and insulin action were examined. Some of the subjects lost weight, and the rest of their body weight did not change significantly. Leptin weight of 36% in the group that had come down. Changes in leptin levels were not associated with changes in resting metabolic rate or plasma catecholamine. Researchers stated that weight loss (in weight) may increase insulin action has been reported in this study.

Short-term effects of resistance training and channel partners as well as changes resulting from resistance exercise in men and women with type 2 diabetes were studied. The results showed a 30% decrease in leptin levels at rest and after 24 hours of the first session of resistance training in people with diabetes. However, these effects were not seen in non-diabetic subjects. But the researchers, the long-term effects of resistance training on leptin levels in both groups rejected. In an interesting study in 2002, and his Asymsh on young girls, two types of resistance training (three weeks) and endurance (three weeks) to run left. Between training (rest) and after exercise (second break) there was a week break. Blood samples were collected before resistance training, then, after a week of rest, after exercise and after a week of rest was secondary. The results shows a decrease of leptin resistance training and even after a week of rest, the decrease was maintained. BMI and body fat remained unchanged during the study period and hormone levels had no correlation. Energy consumption during intensive training of researchers Dashtd likely resistance, decreased leptin and hypothalamic thyroid axis function is reduced, and this reduction is independent of BMI or body fat. Finally, they reported lower levels of leptin are associated with exercise intensity. His cane after effects of resistance training on serum leptin in young men studied. They stated that despite a characteristic high BMI in athletes (athletes with resistance training), no correlation between leptin and BMI in this group does not exist. The authors also stated that research work; experience independent of the effect of resistance training on body composition does, the effect is particularly leptin. But recent researches and colleagues on the effects of resistance training in older men were studied. The results of this study showed that leptin response to resistance training in older men change, which is related to exercise intensity. Such changes of leptin resistance exercise is greater when exercise intensity is above 80% of maximum heart rate. The authors of this study showed that changes in leptin expression is correlated with changes metric. In reviewing research about weight lifting routine can be said that the short-term effects of this practice, in terms of energy consumption has been emphasized. Training intensity and volume in some important research and other changes without affecting leptin has been noted. Actuate exercise reduced leptin delay, which was also observed in stud-

ies related to exercise, the training method also can be seen. A considerable decrease in leptin levels with no change in BMI in some studies, sometimes with and sometimes without the connection has been consistent hormonal changes. The study of the effects of training on risk factors for cardiovascular disease has been associated with lower leptin levels [14–22]. Obesity, with its much well-known co-morbidity, has become so prevalent that it is often described as a global epidemic. The contribution from lifestyle factors such as diet and satiety may be predominantly responsible for the recent dramatic increase in the prevalence of obesity and secondary health risks. Obesity is associated with an array of health problems in adult and pediatric populations. Obesity accelerates atherothrombosis and increase incidence of cardiovascular morbidity and mortality. Numerous observations suggest that it is the interplay of the metabolic disorders that frequently accompany excess body weight (e.g., insulin resistance, hyperinsulinemia, hypertension, hypertriglyceridemia, and decreased HDL cholesterol), that promote the progression of atherosclerotic lesions in obese individuals. Young obese but otherwise healthy subjects are characterized by reduced coronary vasoreactivity. In atherosclerosis, signs of inflammation are accompanied by incipient lipid accumulation in the artery wall. The initial phase of inflammation is usually silent and the atherosclerosis preclinical window is fairly long. LDL oxidation is a main cause of endothelial injury and induces the expression of proinflammatory molecules in endothelial cells. Leptin is produced primarily by adiposities and play a key role in the regulation of appetite and body weight. Since its identification in 1994, leptin has attracted much attention as one of the most important signals for the regulation of food intake and energy homeostasis. Leptin regulates energy homeostasis and reproductive, neuroendocrine, immune, and metabolic functions. Leptin concentration rise exponentially with increasing percentage body fat and obese individuals have markedly increased leptin production probably as a consequence of resistance to its action. Leptin may be a nutrient sensing signal of adipose and muscle tissue, it is thought to be the missing link between adipose tissue and certain system regulating body mass and energy expenditure. The effect of obesity on vascular function may be mediated by leptin. Leptin may contribute to cardiac cachexia and to obesity-related cardiovascular dis-

ease by a variety of mechanisms. Hyperleptinaemia in the general population is also associated with atherosclerosis, hypertension and metabolic syndrome. Fasting serum leptin levels were independently associated with arterial distensibility. Leptin has proinflammatory proliferative and calcification promoting effects in vasculature. Leptin may also produce a pro-thrombotic state. The evidence for these effects as well as their pathophysiological significance in obesity hypertension, heart failure, atherosclerosis and thrombosis are discussed. Excess body weight, is associated with several other risk factor. For example increased blood levels of high-density lipoprotein cholesterol and low levels of high-density lipoprotein cholesterol are some kind of disorders. Some studies have linked leptin resistance to cardiovascular disease much more strongly than cholesterol and they are in fact at least partially responsible for cholesterol abnormalities. There are large variations in leptin concentration. Potential modifiers of leptin concentrations are energy-yielding nutrients such as fatty acids, carbohydrates, proteins, and alcohol. None pharmacological treatments for obesity include behavior therapy, exercise, and caloric-restricted diets. Exercise is a potent stimulus for secretion of many hormones, and exercise mediated negative energy balance may contribute to the regulation of plasma leptin concentration. Weight reduction after physical exercise is correlated with reductions in plasma concentrations in obese women. Emerging research also suggests that leptin plays a more important role in acute (e.g., fasting) and chronic energy-deficient states (e.g., diet- or exercise-induced hypothalamic amenorrhea and lipoatrophy) than in energy-replete states (e.g., obesity). The rapid decrease in serum leptin levels during fasting indicated that leptin release was regulated by factors other than changes in body fat mass. But Thong found that exercise in the absence of weight loss did not alter leptin levels and changes in leptin correlated with changes in total adipose tissue. They concluded that reduction in adipose tissue after weight loss resulted in a collateral decrease in circulating leptin and exercise independent of its effects on weight loss, had no profound influence on leptin secretion. Longitudinal exercise training studies have reported conflicting results. Kraemer et al., did not find any changes in leptin in obese women after a nine-week aerobic class. Dirlwanger et al., found no changes in plasma leptin in response to moderate exercise

performed during three-day period. Exercise with calorie restriction was the only treatment that produced a decrease in plasma leptin concentrations 24 hours after exercise. If plasma leptin concentrations are to be altered, an undefined threshold for total energy deficit as result of either training or reduced calorie intake probably exists. The number of hours of exercise training was significantly correlated with changes in leptin levels, during the 16-mo. period. There is the potential that individuals with larger fat masses respond differently to exercise than leaner subjects in terms of leptin. Resistance training and detraining may alter leptin and adiponectin response in an intensity-dependent manner. Skin-fold sum and BMI were reduced by resistance training with high intensity being more effective than moderate-intensity/low-intensity training. Strength maximal oxygen consumption, RMR, and exercise energy cost increased after training in an intensity-dependent manner. Leptin changes were strongly associated with anthropometric changes. Resistance exercise induces marked metabolic and endocrine changes, significant energy flux perturbations, acidosis, and altered carbohydrate metabolism. Leptin response patterns are a direct result of the intensity and duration of high-energy expenditure and the subsequent excess post-exercise oxygen consumption of the acute resistance exercise protocols. Leptin is sensitive to relatively short term intense exercise when all major muscles are involved. Exercise is a potent stimulus for secretion of many hormones, and exercise mediated negative energy balance may contribute to the regulation of plasma leptin concentrations. Plasma leptin decreases markedly during short-term total fasting not in proportion to the loss in fat mass and returns to baseline concentrations with refeeding. insulin-mediated glucose availability may play a role in the control of leptin synthesis by adipocytes. The rapid decrease in serum leptin levels during fasting indicated that leptin release was regulated by factors other than changes in body fat mass. Potential modifiers of leptin concentrations are energy-yielding nutrient such as fatty acids, carbohydrates, Proteins, and alcohol. Most published so far indicate that fasting and refeeding may change plasma leptin concentrations whereas little is known about the effect of specific nutrients in humans. Thong et al., found somewhat similar results by inducing weight loss through diet restriction only, exercise only or exercise without weight loss. The diet

only and exercise only protocols both resulted in significant reductions in body weight and in plasma leptin, yet the exercise without weight loss intervention produced no change in plasma leptin concentration.

15.2 MATERIALS AND METHODS

Eighteen girls were obese, sedentary lifestyle and diet in the last year or did not participate in any training program, volunteered to participate in the study. Of these nine patients were randomized to resistance training (RT) (5.1 ± 1.21 years, $\text{BMI} = 7.1 \pm 9.31 \text{ kg/m}^2$) and nine controls (Co) (3.1 ± 2.21 years, $\text{BMI} = 3.1 \pm 2.32 \text{ kg/m}^2$) were used. Before the study, written consent was obtained from participants. Experimental group 8 weeks of resistance training three sessions per week for about an hour (12 stations, four courses, each of 15 repetitions at 60% one repetition maximum of three minutes rest) were performed. Control group continued their usual lifestyle during the study and did not participate in a particular diet or exercise. Blood tests, body composition and a $\text{Vo}_{2\text{max}}$ at baseline, 4 and 8 weeks after training in both groups was performed. Measurements were performed during the follicular menstrual cycle (based on three months prior to the study). Resistance training three times a week for almost an hour was done. Training includes four courses with 15 repetitions at 60% of one repetition maximum 1 RM run was. Necessary to mention that exercise at 12 stations were carried out and the rest of the course for 3 minutes. Indeed stations the next phase was to run exercise. A 10 minutes warm up and 10 minutes cool-down at the end of the exercise, with a variety of stretching exercises were performed. The exercises performed were: Scott from Runic Huck pull forward, leg press, chest press, open knees, traction bars, T in the sitting position, legs bent, biceps, triceps, long stretching sessions and Runic from behind. In the first weeks of resistance training, the subjects were familiar with the method of exercises and proper breathing. Used to resist this week, the estimate was calculated (Table 1) and the subjects more or less than estimated, the practice took off running, the weights can be changed. It explained that most subjects in most movements, the applicable amount of weight, was lower than the estimated value. Training program with three sessions per week for 8 weeks. In fact, the exercise

was conducted during the period of study plus a load was applied in this case [23–29].

TABLE 1 Estimation of rehabilitation estimated at RM 1 (17).

Body Weight Motion
Runic traction front and rear, traction bars, and log T Krl 40%
Bench press 50%
Leg press 75%
Hawke ESCO 70%
Triceps and biceps 30%
Extension lag of 45%

After the first week for a more accurate estimate (RM 1), subjects estimated 50% of the weight for one repetition maximum (RM 1) were performed in each movement. The weights were then added, so that only one could do it. Sixty percent of the weight was chosen as weight training. Then once in every 15 days, estimated 1RM was performed. The subjects that will feel comfortable doing the exercise, measurements were performed at less intervals.

1. Biochemical factors including leptin, insulin, glucose, LDL-C, HDL-C, total cholesterol (TC), and triglyceride (TG), respectively.
2. Body composition and anthropometric measurements including height (cm), weight (kg), percent body fat (BF), fat-free weight (FFW) and the ratio of waist to hip kg, respectively.
3. Oxygen consumption (VO_{2max}).

Blood samples (10 ml) from the vein was collected in a 12-h fasting. The subjects had not consumed caffeine 14 hours before blood sampling. Blood samples were collected at least 8 hours of sleep in the night. Three days before the blood were measured. This form of a written recommendation to consume a certain amount of food was given to each person. The subjects were measured three times, 72 minutes before exercise were not measured. This work is to minimize the acute effects of exercise and diet will do. Blood samples three times between the hours of 8 to 10 in the

morning was to be observed circadian rhythm of leptin secretion [30–36]. To keep the sample tubes to clot to form was fixed. The serum was separated and then centrifuged at room temperature at 70-degrees, were stored for biochemical measurements. To measure leptin ELISA kit dbc Canada (No: CAN-L-4260 Version: 6.0) was used. This kit measures the sensitivity at 0.50 ng/ml. Percent standard error within 7.3%, and the percentage error in the outer 8.5%. Insulin was measured by ELISA kit Bayvsrvs Company (Europo SA) detection sensitivity 15.0 U μ /ml and a standard deviation of outer and inner, respectively 3.5% and the 5.9%, were evaluated. Enzymatic method for the measurement of total cholesterol, calorimeter Photo meters were used in single point mode. Measuring range of the assay sensitivity of 5 to 500 mg dl and cholesterol levels measured at least 5 mg dl. Percent respectively external and internal SE 62.1% and 14.1%, respectively. Rndvks method for direct measurement of HDL-C, LDL-C was used. Triglycerides and glucose were measured by enzymatic methods, photovoltaic energy meter single point mode m was used. TG measurement range between 5 to 700 mg per deciliter and standard deviation, respectively, the outer and the inner 53.1% and 60.1%, respectively. Measurements range between 5 to 400 mg per deciliter and standard deviation, respectively, the outer and the inner 74.1% and 19.1%, respectively. Height was measured with a tape that was mounted to the wall in the morning, between the hours of 8 to 10 in the morning without shoes and was calculated in centimeters. These measurements were performed only in the initial test. The subjects' weight and body composition measured on the device shows the body composition were evaluated. The trade name of the company Inbody 3.0 Biospace (the butter). Devices, many of the specifications including weight, body fat percentage, lean body weight, and BMI was calculated. Measured by waist-to-hip ratio (WHR) waist circumference between the waist and hip circumference at the narrowest part in the broadband was measured by tape. Divided by the number of waist circumference to hip circumference were obtained. [37–46]. It should be noted that all measurements except height, three times the pre-test was performed 4 and 8 weeks. All subjects underwent assessment of maximal oxygen uptake in three steps using a gas analyzer (Part 2001 N) were used. To do this test on a treadmill using a modified Bruce was measured. The program was defined as a modified Bruce test was easily elected. In

this study, subjects previously covered sports clothing and proper footwear used to running on a treadmill. After a number of motion exercises, 5 minutes on the treadmill at a speed 44.0 meters per second and walked out to warm up the slope. Note that subject, special belt, heart rate and respiratory gas collection mask for the start of the warm up on treadmill was closed. After warm up, tilt the device to the 10% rate to 75.0 meters per second as well as the slope and the speed was increased for every 3 minutes. Test, due to exhaustion and lack of desire, to continue to rise to reach maximum heart rate for your age, unbearable pain in the legs and chest area, observing any unusual condition, such as bruising, imbalance subjects discontinued was. More information about software Quark 2 respiratory gases during the test was recorded and the $Vo_{2\max}$ test subjects was estimated. Measured by $Vo_{2\max}$ time between the morning hours at a temperature between 20–26°C and 9–12°C. Collected blood samples and measurements were done in a day. The subjects after blood sampling (FBS) came to the physiology laboratory and the measurement of body composition and waist-to-hip ratio, ate a small breakfast. Period of rest, and then to estimate the $Vo_{2\max}$ test on the gas analyzer Bruce attended. Diet in this study were polymorphic. for 72 hours before each blood test was one of them. For this purpose, the approximate value of the resting metabolic rate formula $[496 + (BW \times 7.14)]$ was obtained. The value of the coefficient 5.1 (activity factor) was calculated by multiplying the amount of calories everyday subjects [47–56]. Because of the number of the Benedict equation subjects were sedentary. The approximate amount of the diet containing 55% carbohydrate, 30% fat and 15% protein, the subjects were told to perform in their writing. The resistance training group, which estimated their energy consumption (up to meet the needs of) the following formula to approximate rates of 400 to 500 kcal diet, energy agenda, regardless of the level of resistance was considered. This means that the energy consumption resulting from the exercise, the energy intake of the subjects was not added. Energy consumption resistance training using the following formula was calculated (1):

$$\begin{aligned} &(\text{Duration of activity in minutes}) \times (\text{body weight in kg}) \times \\ & (86\ 0/0) = \text{kcal} \end{aligned} \quad (1)$$

Nutritional counseling sessions were held each week for group exercise. In counseling sessions, information about food calories, behavioral techniques for controlling food intake and energy expenditure and physical activity of the subjects was considered. During the study period, the control group were asked not to alter their diet or lifestyle. Diet groups at 72 hours before the test was administered. Diet and exercise groups at this time (72 h) taking exercise was controlled. Statistical analysis was performed with the software SPSS.13. To determine the significance of different training methods in time, a way ANOVA with repeated measures was used. Factors, exercise, and control (two levels) and time (three levels) to determine the effects of different methods on blood samples, body composition and aerobic power during the interaction with leptin, insulin, glucose, total cholesterol, LDL-C, HDL-C, triglycerides, body weight, body fat, lean body weight, waist-to-hip ratio was used. LSD post hoc tests to determine significant differences between the exercise and control groups (ANOVA one-way), and between test sessions at each of the values was used. To determine the correlation between changes in leptin and each of the other variables, the Pearson correlation coefficient was used. Excel software was used to draw tables [57–62].

15.3 RESULTS AND DISCUSSION

Personal characteristics of the study subjects in both groups is shown in Table 2.

TABLE 2 Individual characteristics of subjects in each group ($\pm$ SD Mia Pearl).

	Experimental group	Control group
Age	5.1 $\pm$ 1.21	3.1 $\pm$ 2.21
Weight (kg)	4.4 $\pm$ 2.824.4	2.4 $\pm$ 2.85
BMI (kg/m ²)	7.1 $\pm$ 9.31	4.1 $\pm$ 7.32
WHR	01.0 $\pm$ 97.0	02.0 $\pm$ 98.0
Leptin	9.40 $\pm$ 99.0%	5.42 $\pm$ 6.1%
Vo _{2 max} (ml/kg/min)	1.1 $\pm$ 8.23	2.1 $\pm$ 8.24

The results showed no significant difference in test scores between serum leptin (1.1– and 6.3+ ng/ml), pre-test and final test (1+ and 7.3+ ng/ml) and test-intermediate final test (9.1+ 1.0+ ng/ml), respectively, the experimental and the control group ($p<.05$) (Fig. 1).

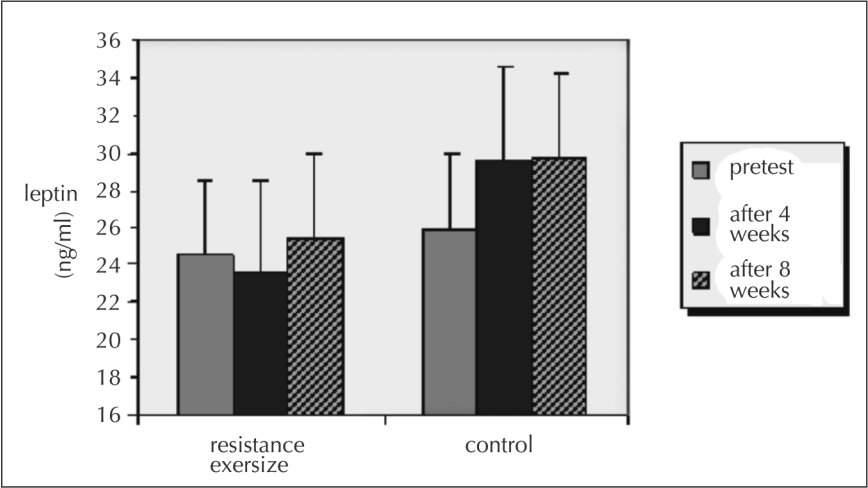


FIGURE 1 Changes in three-measured leptin resistance training and control groups.

In contrast to leptin changes between the two groups in the time between the pre-test to mid-test, mid-test and pre-test to final test to final test was not significant ($p<.05$).

Despite the decline in insulin during the study period in the RT group, the difference between pre-test scores (–17), pre-test and final test (–2/38) and the final exam intermediate test (–2/21) means was not significant ($p<.05$) (Fig. 2). The control group differences in changes in serum insulin levels in the pre-test to mid-test, pre-test to mid-test and final test to final test was not significant ($p<.05$). However, none of the measured intervals, the difference between groups was not significant for changes in insulin levels ($p<.05$) (Figs. 2 and 3).

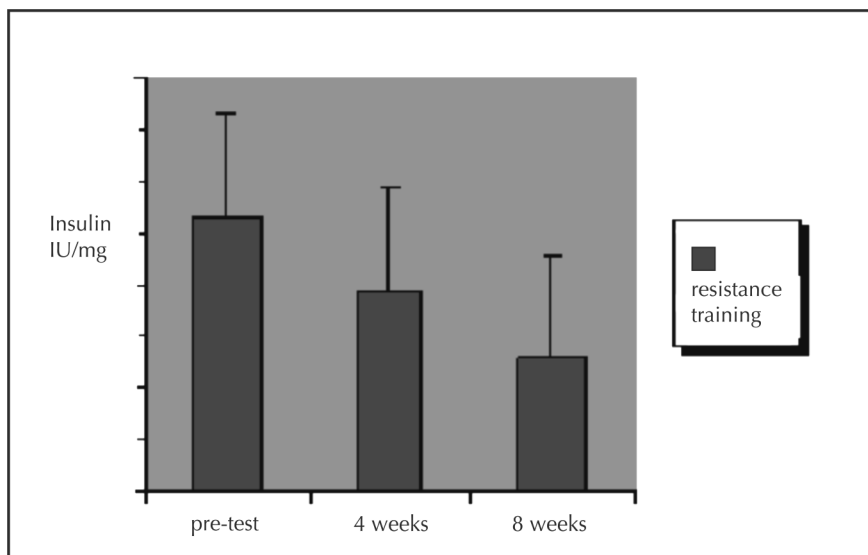


FIGURE 2 Insulin levels were measured three times in the RT group.

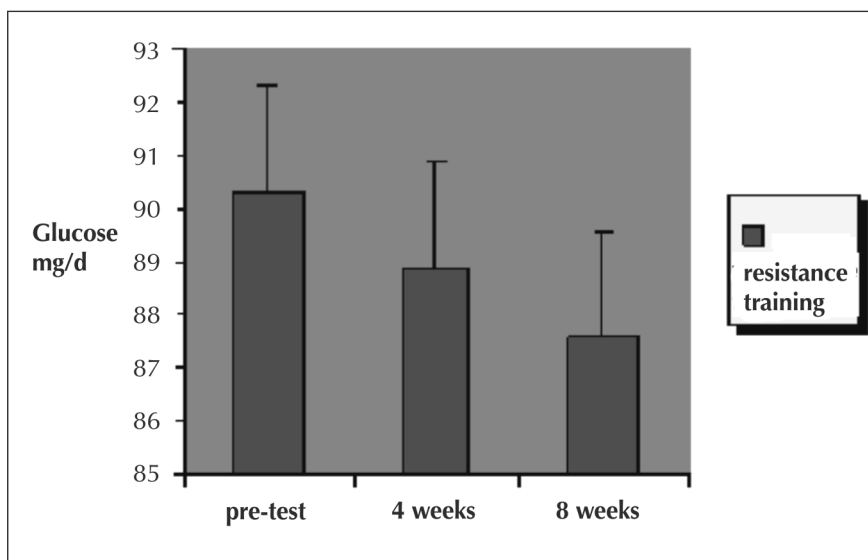


FIGURE 3 Glucose levels were measured three times in the RT group.

In both study groups, significant differences in LDL-C between pre-test and mid-test, pre-test and final test between test and final test was observed ($p < .05$). Difference between the groups in any of the intervals observed No ($p < .05$) (Table 3). However, the average decrease in triglycerides of up to 4 weeks after the first measurement of the resistance training group (77/20 ng/ml) concentration in the drop was not significant ($p < .05$). Between 4 to 8 weeks in both groups, significant changes were observed in serum triglyceride levels ($p < .05$). But it changes the resistance-training group and a control group measured within the first 4 weeks after starting the program, was significant ($p < .05$) (Table 3). The first measure to reduce fat in groups of 4 to 8 weeks, 4 weeks, but no significant difference was not significant. The difference between the percentages of body fat to 8 weeks before the test was also significant ($p < .05$). Fat changes between pre-test and final exam ($79.0 \pm 96/1\%$) were statistically significant. Fat-free weight (FFW) intra-group and inter-group differences significant at any measurement interval revealed ($p < .05$). Vo_{2max} values in the RT group increased significantly after 4 and 8 weeks demonstrated increased between 4 and 8 weeks and it continued ($P < .05$) (Figs. 4 and 5).

TABLE 3 p-Values measured variables in three groups Variable group pretest 4 weeks and 8 weeks.

Variable	Group	Pretest	4 Weeks	8 Weeks
TG	RT	77.111 $\pm$ 33.19	11. 91 $\pm$ 77.14 *	22.04 $\pm$ 1.18
(mg/dl)	Co	109 $\pm$ 46.18	11.124 $\pm$ 05.21	11.116 $\pm$ 03.22
TC	RT	4.181 $\pm$ 01.6	66.179 $\pm$ 71.4	44.187 $\pm$ 81.10
(mg/dl)	Co	8.176 $\pm$ 98.8	11.176 $\pm$ 45.9	33.174 $\pm$ 16.8
LDL-C	RT	121 $\pm$ 33.4	5.123 $\pm$ 25.5	136. 88 $\pm$ 6.10
(mg/dl)	Co	108.66 $\pm$ 33.6	112 $\pm$ 44.7	108.55 $\pm$ 12.9
HDL-C	RT	66.38 $\pm$ 66. 1	11.30 $\pm$ 33.5	22.38 $\pm$ 20.2
(mg/dl)	Co	55.46 $\pm$ 65.3	34.32 $\pm$ 66.3	44.42 $\pm$ 65.3

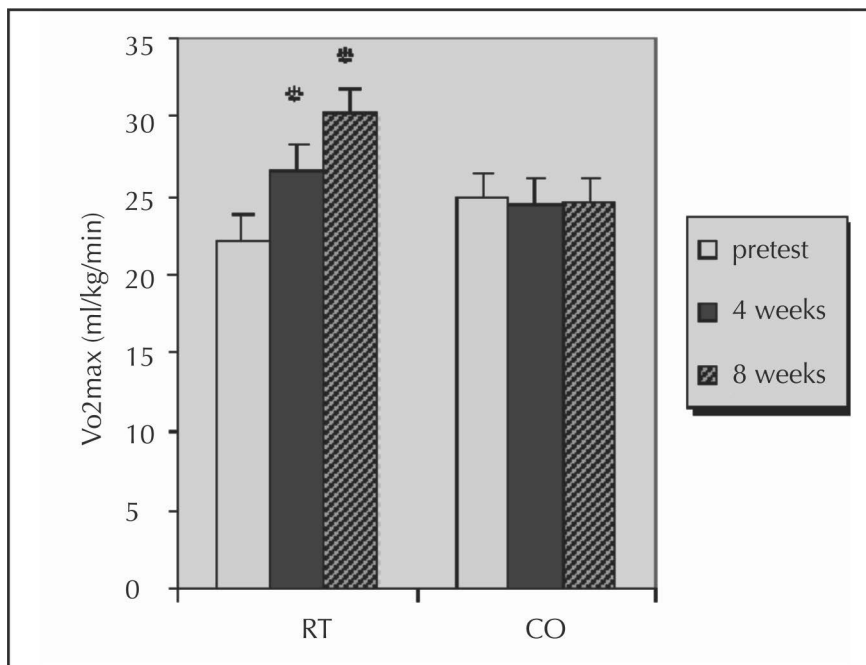


FIGURE 4 Changes in $\text{Vo}_{2\text{max}}$ groups compared with the test group ($p > .05$).

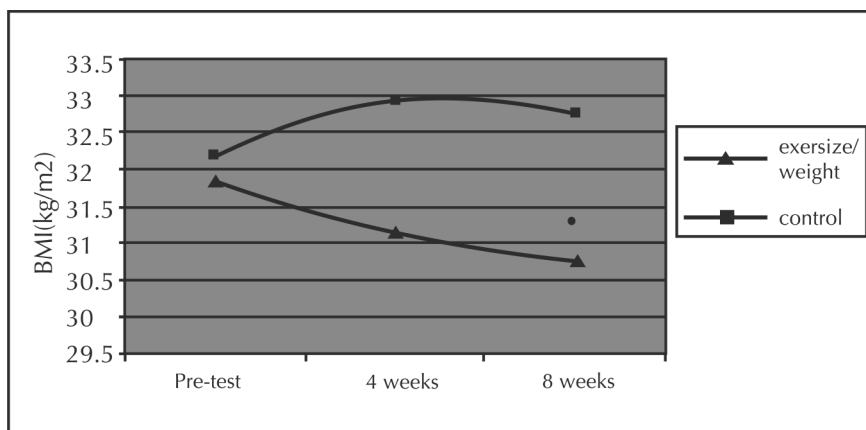


FIGURE 5 Changes in body mass index (BMI) measured in three groups ($p < .05$), compared with the pre-test between the two groups.

In a duration of 0 to 4 weeks and 0 to 8 weeks, BMI showed a significant decrease in the RT group ($p<.05$).

BMI reduction in the interval from 0 to 8 weeks of weight training in the control group (15.1–) showed significant differences ($p<.05$). Kmrh hip circumference ratio (WHR) in the RT group pre–test to mid-test, pre-test and final test Taazmvn middle to the end of the test, showed a significant difference ($p<.05$). In the control group no significant interval change in the size measure waist–hip ratio was observed ($p<.05$). Between the first and last measure changes in leptin and BMI change over the same period showed significant correlation ($r=.42$) there. This change in leptin correlated with WHR ($r=.42$) also applies to ($p<.05$). Leptin changes in any of the measured distances with other variables, showed a significant correlation (Table 4).

TABLE 4 F-values and significance levels of all variables in the two study groups (repeated measures).

Significance levels	F variable	Groups	Variables
.71	.34	resistance training	Leptin
.4	.96	Control	
.38	1.01	resistance training	Insulin
.32	1.21	Control	
.5	.71	resistance training	Glucose
.12	2.39	Control	
.68	.39	resistance training	Witness
.75	.28	Control	
.75	.29	resistance training	LDL-C
.85	.16	Control	
.12	2.34	resistance training	HDL-C

TABLE 4 (Continued)

Significance levels	<i>F</i> variable	Groups	Variables
.12	2.34	Control	
.21	1.72	resistance training	RT
.1	2.64	Control	
* 0	26.36	resistance training	VO _{2 max}
.52	.68	Control	
*.001	11.03	resistance training	WHR
.52	.68	Control	
* 0	20.32	resistance training	BMI
.41	.93	Control	
*.01	6.24	resistance training	Fat-percent
.75	.29	Control	
.53	.65	resistance training	F a t - f r e e weight
.76	.26	Control	

15.4 CONCLUSION

As seen in this study, leptin was identified as an independent risk factor in heart disease in obese adolescents show high levels (34.2 ± 7.30 ng/ml). Total cholesterol, LDL-C, and TG levels in many subjects at risk than it was at or above that age would not normally be seen. Thus it can be said that the young participants of the Chronic obesity is somewhat glaring at them. The present results contradict some earlier research. Ryan and his colleagues' say their findings indicate that weight loss, leptin levels also significantly reduced. However, in this study there was a significant

reduction in BMI and WHR, a significant decrease in serum leptin levels occurred. It is also a significant decrease was seen in most subjects. The reason for this difference in results can be traced to several causes. Among the reasons for the application, which in most previous research programs for more than 12 weeks of resistance could make the appropriate changes. On the other hand, some studies with a shorter program to achieve the desired results. The reason for this difference must be sought in intensity. There, the highest value was less than 500 kcal. Exercises done every other day, so we had a significant amount of calorie restriction. Leptin levels are very sensitive to diet, it is important to study the changes of leptin. In this study, a complete diet-to-diet recommendations for any specific person will be considered. Weekly meetings and reports as well as a way of keeping track of nutritive diet will continue to run. But the possibility still exists that subjects accurately reported void. Even small changes in diet recommended, especially in the 72 hours before collecting blood samples, may affect the results. If the diet seems to be more accurate (clinical), control was achieved favorable results in changes in leptin. Another significant point to be made here is that the lower weight, BMI and body fat in such a program, fat-free weight was not significantly changed. The same thing that causes an increase in both measured $\text{Vo}_{2\text{max}}$ (4 and 8 weeks) before the test is. In fact, weight loss and body fat than lean tissue to total body weight increases and the increase of oxygen consumed per kilogram of body weight. Probably other reasons, such as changes in muscle cells and capillary networks were not ineffective in this regard.

As was seen in triglyceride changes before the final test to test the difference between groups was significant. The decrease in triglycerides in the training groups compared with the control group was significant. This represents a reduction of triglycerides from start to end and the positive metabolic effects of resistance exercise exposes changes in body fat, but this would not apply. It seems that hormonal and metabolic factors play an important role in this regard. On the other hand, since the trend of changes in BMI and WHR RT group, has been reduced, it can be concluded that the leptin (in most subjects) also applies, although on the leptin level was not significant. Field application of these findings is that the style of training in individuals with lower WHR (which is easy to measure) came into existence, we can expect that leptin levels may also be reduced.

Increasing the duration of the program, or conditions in a way that higher intensity workouts and longer runs in each session was also attended metabolic expect more changes. Perhaps employing a low-calorie diet for quick results in this field have yielded more and more clear.

Comparison of the effects of these types of programs, leptin in obese and lean men and women at different ages of the major research areas are considered.

KEYWORDS

- **chronic obesity**
- **heart disease**
- **insulin**
- **leptin**
- **obesity**

REFERENCES

1. Canavan, B.; Salem, R. O.; Schurgin, P. K.; Lipinska, I.; Laposata, M.; Grinspoon, S. Effect of physiological leptin administration on markers of inflammation, platelet activation, and platelet aggregation during caloric deprivation. *J. Clin. Endocrinol. Metabol.* **2005**, *90*(10), 5779–5785.
2. Kraemer, K. K.; Chu, H.; Castracane, V. D. Leptin and exercise. *Exp. Biol. Med.* **2002**, *227*, 701–708.
3. Paquot, N.; Tappy, L. Adipocytokines: link between obesity, type 2 *Diabetes* and atherosclerosis. *Rev. Med. Liege.* **2005**, *60*, 369–373.
4. Barbato, K. B.; Martins, Rde. C. Rodrigues, Mde. L. Braga, J. U.; Francischetti, E. A.; Genelhu, V. Effects of greater-than5% weight reduction on hemodynamic, metabolic and neuroendocrine profiles of grade 1 obese subjects. *Cardiology* **2006**, *87*, 12–21.
5. Ostlund, R. E.; Jr. Yang, S.; Gingerich, R. Relation between plasma leptin concentration and body fat, gender, diet, age, and metabolic covariates. *J. Clin. Endocrinol. Metabol.* **1996**, *81*, 3909–3913.
6. Baratta, M. Leptin from a signal of adiposity to a hormone mediator in peripheral tissues. *Med. Sci. Monit.* **2002**, *8*, RA282-RA292.
7. Jequier, E.; Tappy, L. Regulation of body weight in humans. *Physiological Review* **1999**, *79*, 451–480.

8. Ren, J. Leptin and hyperleptinemia-from friend to foe for cardiovascular function. *J. Endocrinol.* **2004**, *181*, 1–10.
9. Cooke, J. P.; Oka, R. K. Does leptin cause vascular disease? *Circulation* **2002**, *106*, 1904–1905.
10. Nammi, S.; Koka, S.; Chinnala, K. M.; Boini, K. M. Obesity: an overview on its current perspectives and treatment options. *Nutrition J.* **2004**, *3*, 3–10.
11. Franklin, S. S. Arterial stiffness and hypertension: a two way street? *Hypertension* **2005**, *45*, 349–355.
12. Livshits, G.; Pantsulaia, I.; Gerber, L. M. Association of leptin levels with obesity and blood pressure: possible common genetic variation. *Int. J. Obes.* **2005**, *29*, 85–92.
13. Brabeau, P.; Gutin, B.; Litaker, M. S.; Ramsey, L. T.; Cannady, W. E.; Allison, J.; Lemmon, C. R.; Owens, S. Influence of physical training on plasma leptin in obese youths. *Canadian J. Appl. Physiol.* **2003**, *28*, 382–396.
14. Rahmouni, K.; Haynes, W. G. Leptin and the cardiovascular system. Resent Progress in Hormone Research **2004**, *59*, 224–244.
15. Singhal, A.; Farooqi, I. S.; Cole, T. J. O’Rahilly, S.; Fewtrell, M.; Kattenhorn, M.; Lucas, A.; Deanfield, J. Influence of leptin on arterial distensibility: a novel link between obesity and cardiovascular disease? *Circulation* **2002**, *106*, 1919–1926.
16. Wallace, A. M.; McMahon, A. D.; Pakard, C. J.; Mibioli, A. K.; Shephard, J.; Gaw, A.; Sattar, N. Plasma leptin and the risk of cardiovascular disease in the west of Scotland coronary prevention study. *Circulation* **2001**, *104*, 3052–3060.
17. Fenkci, S.; Sarsan, A.; Rota, S.; Ardic, F. Effects of resistance or aerobic exercises on metabolic parameters in obese women who are not on a diet. *Adv. Therapeutic* **2006**, *23*, 404–413.
18. Ryan, A. S.; Praley, R. E.; Goldberg, A. P. Changes in plasma leptin and insulin action with resistive training in postmenopausal females. *Int. J. Obes. and Relation Metabolic Disorders* **2000**, *24*, 27–32.
19. Kanaley, J. A.; Fenicchia, L. M.; Miller, C. S Ploutz Synder, L. L.; Weinstock, R. S.; Carhart, R.; Azevedo, J. L. Resting leptin responses to acute and chronic resistance training in type 2 diabetic males and females. *Int. J. Obes.* **2001**, *25*, 1474–1480.
20. Smisch, C.; Lormes, W.; Petersen, K. G.; Baur, S.; Liu, Y.; Hackney, A. C.; Lemann, M.; Stenacker, J. M. Training intensity influences leptin and thyeasoid hormones in highly trained rowers. *Int. J. Sports Med.* **2002**, *23*, 422–427.
21. Gippini, A.; Mato, A.; Peino, R.; Lage, M.; Dieguez, C.; Casanueva, F. F. Effect of resistance exercise (body building) training on serum leptin levels in young men. Implications for relationship between body mass index and serum leptin. *J. Endocrinol. Invest.* **1999**, *22*, 824–828.
22. Fatouros, I. G.; Tournis, S.; Leontsini, D.; Jamurtas, A. Z.; Sxina, M.; Thomakos, P.; Manousaki, M.; Douroudos, I.; Taxildaris, K.; Mitrakou, A. Leptin and adiponectin responses in overweight elderly following resistance training and detraining are intensity related. *The J. Clin. Endocrinol. Metabol.* **2005**, *90*, 5970–5977.
23. Zafeiridis, A.; Smilios, I.; Conisidine, V.; Tokmakidis, S. P. Serum responses after acute resistance exercise protocols. *J. Appl. Physiol.* **2003**, *94*, 591–597.
24. Nindl, B. C.; Kreamer, W. J.; Arciero, P. J.; Samatalle, N. Leone, C. D.; Mayo, M. F.; Hafeman, D. L. Leptin concentrations experience a delayed reduction after resistance exercise in men. *Medicine and Science in Sport and Exercise* **2002**, *34*, 608–613.

25. Frank, L. L.; Sorensen, B. E.; Yasui, Y.; Tworoger, S. S.; Schwartz, R. S.; Ulrich, C. M.; Irwin, M. L.; Rudolph, R. E.; Rajan, K. B.; Stanczyk, F.; Bowen, D.; Weigle, D. S.; Potter, J. D.; McTiernan, A. Effects of exercise on metabolic risk variables in overweight postmenopausal women: a randomized clinical trial. *Obes. Res.* **2005**, *13*, 615–625.
26. Reseland, J. E.; Andessen, S. A.; Solvoll, K.; Hjermann, I.; Urdal, P.; Holme, I.; Drevon, C. A. Effect of long-term change in diet and exercise on plasma leptin concentration. *Clin. Nutr.* **2001**, *73*, 240–245.
27. Ryan, A. S.; Praley, R. E.; Goldberg, A. P. Changes in plasma leptin and insulin action with resistive training in postmenopausal females. *Int. J. Obes. and Relation Metabolic Disorders* **2000**, *24*, 27–32.
28. Racette, S. B.; Coppack, S. W.; Landt, M.; Klein, S. Leptin production during moderate-intensity aerobic exercise. *J. Clin. Endocrinol. Metabol.* **1997**, *82*, 2275–2277.
29. Weltman, A.; Pritzlaff, C. J.; Wideman, L.; Considine, V.; Fryburg, A.; Gutgesell, M. E.; Hartman, M. L.; Veldhuis, J. D. Intensity of acute exercise does not affect serum leptin concentrations in young males. *Med. Sci. Sports Exerc.* **2000**, *32*, 1556–1561.
30. Ishii, T.; Yamakita, T.; Yamagami, K.; Yamamoto, M.; Miyamoto, M.; Kawasaki, K.; Hosoi, M.; Yashioka, K.; Sato, T.; Tanaka, S.; Fujii, S. Effects of exercise training on serum leptin levels in type 2 diabetic patients. *Metabolism* **2001**, *50*, 1136–1140.
31. Nindl, B. C.; Kreamer, W. J.; Arciero, P. J.; Samatalla, N.; Leone, C. D.; Mayo, M. F.; Hafeman, D. L. Leptin concentrations experience a delayed reduction after resistance exercise in men. *Medicine and Science in Sport and Exercise* **2002**, *34*, 608–613.
32. Jurimae, J.; Jurimae, T. Leptin responses to short term exercise in college level male rowers. *British J. Sports Med.* **2005**, *39*, 6–9.
33. Leal-Cerro, A.; Garcia-Luna, P. P.; Astorga, R. Serum leptin levels in male marathon athletes before and after the marathon run. *J. Clin. Endocrinol. Metabol.* **1998**, *83*, 2376–2379.
34. Olive, J. L.; Miller, G. D. Differential effects of maximal- and moderate-intensity run on plasma leptin in healthy trained subjects. *Nutrition* **2001**, *17*, 365–369.
35. Reseland, J. E.; Andessen, S. A.; Solvoll, K.; Hjermann, I.; Urdal, P.; Holme, I.; Drevon, C. A. Effect of long-term change in diet and exercise on plasma leptin concentration. *Clin. Nutr.* **2001**, *73*, 240–245.
36. Fatouros, I. G.; Tournis, S.; Leontsini, D.; Jamurtas, A. Z.; Sxina, M.; Thomakos, P.; Manousaki, M.; Douroudos, I.; Taxildaris, K.; Mitrakou, A. Leptin and adiponectin responses in overweight inactive elderly following resistance training and detraining are intensity related. *The J. Clin. Endocrinol. Metabol.* **2005**, *90*, 5970–5977.
37. Kanaley, J. A.; Fenicchia, L. M.; Miller, C. S.; Ploutz Synder, L. L.; Weinstock, R. S.; Carhart, R.; Azevedo, J. L. Resting leptin responses to acute and chronic resistance training in type 2 diabetic males and females. *Int. J. Obes.* **2001**, *25*, 1474–1480.
38. Essig, D. A.; Alderson, N. L.; Ferguson, M. A.; Bartolli, W. P.; Durstine, J. L. Delayed effects of exercise on the plasma leptin concentration. *Metabolism* **2000**, *49*, 395–399.
39. Hurst Thomas, D. Skull Wars Kennewick Man, Archaeology and the Battle for Native American Identity, Washington University: School of Law - Anheuser-Busch Hall, 38–41.
40. Van Mechelen W. **1993**, *Am J Sports Med.*, Sep-Oct; **2001**, *21(5)*, 711–9.
41. Alderete, J.; Tupper, P.; Frisch, S. A. Arabic root cooccurrence restrictions revis-

- ited: A connectionist approach to phonological constraint induction, Proceedings of the 48th annual meeting of the Chicago Linguistics Society. **1993**.
42. Soligard T.; Myklebust G.; Steffen, K. "Comprehensive warm-up programme to prevent injuries in young female footballers: cluster randomised controlled trial." *BMJ* **2008**, 337, a2469. doi: 10.1136/bmj.a2469. MC 2600961.PMID 19066253.
 43. Nabieh, A.; Mohamed, I. Anthropometric Measurements as a Significant for Choosing Juniors in Both Volleyball and Handball Sports (Factorial Analysis Study), *World J. Sport Sci.* **2010**, 3(4), 277–289.
 44. Rogana, S.; Hilfikerd, R.; Clarysb, P.; Clijsenc, R.; Taeymansac, J. Position-specific and Team-ranking-related Morphological Characteristics in German Amateur Soccer Players – a Descriptive Study, *Int. J. Appl. Sports Sci.*, **2011**, 23(1), 168–182.
 45. Luigi P. W.; Bercades, T. Somatotypes of national elite combative sport athletes, *Braz. J. Biomotricity*, **2009**, 3(1), 21–30.
 46. Dacres-Mannings, S.; Rochester, S.; Frail, H. Anthropometric profiles of Australian Rugby Institute, club and state level Rugby Union players. **2010**.
 47. Gaurav, V.; Singh, M.; Singh, S. Anthropometric characteristics, somatotyping and body composition of volleyball and basketball players, *J. Phys. Educ. Sports Manage.* **2010**, 1(3), 28–32, Available online <http://www.acadjourn.org/jpesm>
 48. Beat K.; Kohler, G. Influence of anthropometry on race performance in ultra-endurance triathletes in the longest triathlon in North America, *Int. SportMed J.*, **2007**, 8(2), 87–96, <http://www.ismj.com>
 49. Joseph, J.; Greene, M. S.; Timothy, A.; McGuine, A. T. C.; Leverson, G.; Thomas, M. Anthropometric and Performance Measures for High School Basketball Players, *J. Athl. Training*, **1998**, 33(3), 229–232
 50. Amatya, D. L. Comparative Study of Somatotype of Nepalese Sportsmen, National Association for Sports Health and Fitness. **1999**.
 51. Sang Hong, K. Kinanthropometric Study of Korean and Nepalese Marathon Runners, keimyung university Diwakar Lal Amatya, MSC-Athletics and Sports Science-India, Sports Kinanthropometrist-Australia, Sports Expert-National Sports Council, January. **2008**.
 52. Gaunt, B. W. Anthropometric and Demographic Factors Affecting Distance Hopped-aid Limb Symmetry Index for the Crossover Hop-for distance test in High School athletes, *J. Orthop. Sport Phys.*, **2001**, 31(3), 145–151
 53. Gross, M.; Dailey, E. S.; Melissa, D.; Dalton, A.; lee, K.; Wendy, I.; Ashley, C. Relationship Between Lifting Capacity and Anthropometric Measures, *J. Orthop. Sport Phys.*, **2000**, 30(5), 237–247, 258–262.
 54. Roberto, C, José, a., Pérez, J.; Cortell, m. Juan, J.; Rivas, J. Correlations among anthropometric parameters, jump power, and position in professional basketball players, **2008**.
 55. Pui, W. K.; Hendrik H. Anthropometric, gait and strength characteristics of Kenyan distance runners, *J. Sports Sci. Med.* **2008**, 7, 499–504, <http://www.jssm.org>
 56. Jawis, M. N.; Singh, R.; Singh, H. J.; Yassin, M. N. Anthropometric and physiological profiles of sepak takraw players, *Br. J. Sports Med.* **2005**, 39, 825–829. doi: 20.1136/bjism.2004.016915.
 57. Lephart, S. M.; Abt, J. P.; Ferris, C. M.; Sell, T. C.; Nagai, T.; Myers, J. B.; Irrgang, J. Neuromuscular and biomechanical characteristic changes in high school athletes: a

- plyometric versus basic resistance program, *Br. J. Sports Med.* **2005**, *39*, 932–938. doi: 58.1136/bjism.2005.019083.
59. Darr, K. C.; Bassett, D. R.; Morgan, B. J.; Thomas, D. P. Effects of age and training status on heart rate recovery after peak exercise. *Am. J. Physiol.* **1988**, *254*, H340-H343.
 60. Engerman, S. 'The Height of U.S. Slaves', *Local Population Studies*, **1976**, *16*, *1*, 45–50.
 61. Sunder, M. The Making of Giants in a Welfare State: The Norwegian Experience in the Twentieth Century', *Economics and Biology*, **2004**, 75–86.
 62. Woitek, U. Height Cycles in the Eighteenth and Nineteenth Centuries', *Econ. Hum. Biol.*, **2003**, 243–258.

Computational Modeling for Anthropometry

This book provides a broad understanding of the main computational techniques used for anthropometric data, focusing specifically on data for female athletes. A number of data analysis techniques are introduced along with the application of such in a sports setting. These techniques will have potential for application in several disciplines that cover orthopedic injury. Chapters range from new methods to novel applications of existing methods to give readers a better understanding of the topic.

The book's authors also performed the technology and high speed detector equipment to determine correct operational procedures to avoid hazard to human health. The authors believe the information in the book will help to reduce the risk of sports activities.

The book also includes the latest coverage of sports databases and the development of new computational methods and efficient algorithms for sports and engineering software.

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