Blood Lipid and Hormonal Changes From Jogging and Weight Training of Middle-Aged Men

Danny Blessing¹, Mike Stone¹, Ronald Byrd¹, Dennis Wilson¹, Ralph Rozenek¹, Dhanarajan Pushparani², and Harry Lipner²

¹National Strength Research Center Auburn University Auburn, Alabama 36849

²Department of Biology Physiology Group Florida State University Tallahassee, Florida 32302

Abstract

Resistive training effects on cardiovascular function, serum lipids, and hormone levels are not well understood, thus the results of 12 weeks of weight training or jogging on body composition, time to target heart rate (THR), strength, power, total cholesterol, HDL-C, TC/HDL-C, testosterone, dihydrostestosterone, estradiol, LH, and FSH were studied. Male volunteers (X=44 years) were assigned to weight training (N=9), jogging (N=11), or control (N=13) groups. The THR was determined during progressive loading on a cycle ergometer. Body composition was estimated hydrostatically. Performance variables measured were the half-squat, bench press, and vertical jump. Blood lipids were measured using standard techniques, and hormones were assayed by RIA. Both experimental groups increased lean body mass and decreased fat weight with no change in body weight. Weight trainers and joggers increased time to THR at 6 weeks compared to controls, with joggers being superior to both other groups at 12 weeks. Strength and power measures increased in weight trainers compared to the other groups. Serum HDL-C increased and TC/HDL-C decreased in both experimental groups compared to controls. Estradiol decreased in weight trainers and FSH decreased in joggers. These results suggest that both jogging and weight training may produce desirable changes in physical fitness and health.

Key Words: Weight training, physical fitness, hormones, lipids

Introduction

Longitudinal studies show that longshoremen (23, 24) in jobs with the greatest energy demands had fewer incidents of cardiovascular disease (CVD) and a lower mortality rate. Most longshoreman work is clearly more similar to weight training tasks than to aerobic training. Yet, while jogging and other aerobic activities positively affect various factors associated with physical fitness and CVD risk (8, 13, 34), it is generally believed that weight training is of little or no value in modifying the risk for CVD. One reason for this belief is that research shows weight training does little or

nothing to increase aerobic power. Equating aerobic power to CVD risk, though, is not without problems. Risk is a multifactorial matter and may be associated with factors other than aerobic power.

One obstacle to understanding the relationship between weight training and CVD risk is that relatively little is known about the contribution of resistive training to changes in serum lipids, hormone levels - especially hormones possibly affecting lipids or other CVD risk factors - or other physiologic measures that might affect risk. Because a large segment of the population engages regularly in weight training, it would appear that extensive research is needed on the effects of this type of exercise on a broader sample of measures that might relate to CVD risk. This study examined the effects of 12 weeks of weight training or jogging with respect to changes in body weight, body composition, strength, power, heart rate response to submaximal work, serum cholesterol, HDL cholesterol, testosterone, dihydrostestosterone, estradiol, luteinizing hormone, and follicle stimulating hormone. The purpose of this study was to compare the pattern of changes in these variables across time among weight training, jogging, and control groups.

Methods

Subjects

Thirty-three males volunteered as subjects. All had lived relatively sedentary lives for several years prior to the study. They were divided into three groups: (1) weight training, n=9; x age=45.2±4.8 years; (2) joggers, n=11; x age=44.0±7.5 years, and (3) controls, n=13; x=44.7±6.7 years. All signed informed consent for the experiment, which was in accord with guidelines of the American College of Sports Medicine.

Training

Both exercise programs were conducted on Mondays, Wednesdays, and Fridays. A 10-minute warm-up consisting of general flexibility and calisthenic exercises preceded workouts for both groups. The noncircuit resistive training program is shown in Table 1. Each subject progressed at his own rate (within the confines of proper technique), depending on speed of individual adaptation. All training sessions were monitored to insure that proper technique and safety precautions were observed. Each training session lasted approximately 45 minutes.

The jogging program was conducted on an indoor track. The first three weeks involved walking/jogging at 70 percent to 75 percent of the subject's predicted maximum heart rate. This level was raised to 75 percent to 80 percent during the second 3 weeks and to 80 percent to 85 percent during the final 6 weeks. The duration of training was 20 minutes from 0 to 3 weeks, 25 minutes from 4 to 6 weeks, and 30 minutes during the last 6 weeks. All sessions were monitored, with experimenters regularly checking heart rates to control training intensity.

Cycle Test

Prior to exercising, each subject's resting heart rate (HR), blood pressure (BP), and resting electrocardiogram (ECG) were obtained and screened for acceptability. Following these preliminary measurements, subjects performed a progressive submaximal bout at 60 RPM on a cycle ergometer to 85 percent of predicted maximum HR. The initial minute of work was at 0 W, with increases of 25 W each minute thereafter. An ECG CM5 lead placement was used to obtain HR during the last 10 seconds of each minute. As the subjects neared the target HR, ECG strips were run more frequently. At target HR the time was recorded. Heart rate was determined by the average of three consecutive QRS complexes. Blood pressures were monitored by standard ausculatory procedures during the last half of each minute.

Strength and Power Tests

Leg and hip extensor strength was measured using a one repetition maximum (1 RM) half squat. The bar was placed inside a power rack and the depth of the squat (thigh at

Table 1. Weight Training Workouts

Schedule for 0-6 wks						
Monday & Friday	Sets	Reps	Wednesday	Sets Reps		
1/2 squats	3 x	10	Pulls (thigh)	3 x 10		
Pulls (knee)	3 x	10	SLDL*	1 x 10		
Leg extensions	1 x	10	Hyperextensions	3 x 10		
Leg curls	3 x	10	Lateral raises	3 x 10		
Bench press	3 x	10	Behind neck press	3 x 10		
Sit-ups	3 x	10	Sit-ups	3 x 10		
Schedule for 7-12 wks						
Monday & Friday	Sets_	Reps	Wednesday	Sets Reps		
Monday & Friday Sets Reps wednesday Sets Reps						

Monday & Friday	Sets Reps	Wednesday	Sets Reps
1/2 squats	3 x 5 (1 x 10)	Pulls (thigh)	3 x 5
Cleans	3 x 5	Power snatch	3 x 5
Leg extensions	1 x 5	SLDL	1 x 10
Leg curls	3 x 5	Hyperextensions	3 x 5
Bench press	3 x 5 (1x10)	Lateral raises	3 x 5
		Behind neck press	3 x 5
Sit-ups	3 x 5	Sit-ups .	3 x 10

*SLDL - Straight-legged dead lift--always performed with light weight. All other exercises were performed with the maximum weight possible, considering the combination of sets and repetitions. approximately 45 degrees to horizontal) was limited by a pin setting. Each individual's setting was recorded and used in subsequent trials and in training. After warm-up with lighter weights, each subject performed one repetition (by lifting the bar off the pins to full extension) with increasing weights until maximum was reached. The 1 RM bench press tested upper body strength. Maximum leg and hip extensor power was calculated using the maximum vertical jump and body weight. Subjects rested sufficiently between the strength and power tests to insure a maximum effort. The order at each test period during the experiment was: 1) vertical jump, 2) half squat, and 3) bench press.

Body Weight and Composition

Body weight was measured to the nearest tenth kilogram using a Health-O-Meter medical scale. Body composition was evaluated using a hydrostatic method in a stainless steel tank, with the subject in a prone position suspended from calibrated autopsy scales.

Residual volume was estimated using this formula: vital capacity x 0.24 (32). Body fat was estimated using the equations of Brozek et al. (4).

Blood Collection

Venous blood was collected between 7 a.m. and 9 a.m., after a 12-hour fast. The blood was centrifuged at 1,400 G for 20 minutes. Blood was drawn on two alternate days at the beginning, after 6 weeks, and after 12 weeks. Thus, duplicates were not only run on each sample; there were two blood samples for each testing period, giving a total of four analyses on each parameter for each of the three periods. Each blood drawing (T₂ and T₃) was performed at least 40 hours after the last exercise session.

Lipid Analyses

Total cholesterol (TC) was determined using the methods of Krehl et al. (17). HDL-cholesterol (HDL-C) was measured using previously described methods (3). Serum control samples were run with each assay. The coefficients of variation for the cholesterol assay were: Intra - 4.5 percent, Inter - 6.7 percent, for HDL-C: Intra - 8.6 percent, Inter - 4.5 percent. These assays conform to the Lipid Research Clinic standard.

Hormone Analyses

Serum gonadotropins were measured using kits provided by the National Institute on Arthritis, Metabolism, and Digestive Disorders. They were measured after the RIA method described by Niswender et al. (21) and modified by Rush and Lipner (26). Estradiol was measured using the method of Williams and Lipner (35). Serum testosterone concentrations were determined using the RIA methods of Gay and Kerlon (11). Dihydrotestosterone levels were determined using antisera specific to 5-- --DHT and 1, 2-3H-DHT (Radioassay Systems Laboratories).

Statistical Analysis

The statistical analysis used was a Group x Trials ANOVA. Significant differences between groups were determined from trial to trial with a single degree of freedom comparison (trend analysis). The alpha level was set at p <0.05. All variables were measured at T1 (beginning), T2 (6 weeks), and T3 (12 weeks).

Results

Both experimental groups made significant changes in body composition compared to the control group (Table 2). By week 12, the weight training group's LBM had increased by 2.2 kilograms and fat had decreased by 3 percent. At the study's end, the joggers had gained 1.6 kilogram LBM and had reduced body fat by 3.3 percent. No other significant changes in body weight or composition occurred.

As expected, the weight training group's strength gains were superior to both of the other groups at 6 weeks and 12 weeks (Table 3). The controls exhibited a gain in bench press strength superior to the joggers. The only change relative to power was shown by the weight training group, which improved in the vertical jump at 12 weeks significantly more than did the controls.

Relative to the control group, both experimental groups showed significant increases in time to target heart rate after the first 6 weeks (Table 4). The joggers continued to improve, exhibiting significantly greater changes at 12 weeks than either the control group or the weight training group.

Both experimental groups' total cholesterol, HDL-Cholesterol and TC/HDL-C showed significantly different patterns of change than did the control group (Table 5). While total cholesterol was fairly stable in the experimental groups, that of the controls increased. The HDL-C and the TC/HDL-C showed an increase and a decrease, respectively, for both training groups.

Among the hormones, only two significant differences were found (Table 6). The weight training group realized a significant drop in estradiol compared to the controls and joggers at 6 and 12 weeks. A reduction in FSH was observed in the joggers as compared to the weight trainers over 12 weeks.

Discussion

The lack of significant change in body weight over a short training period is consistent with previous findings using both middle-aged men (15) and young men (16). While the control group's body composition and body weight were relatively constant, the failure of body weight to change in the experimental groups was a function of increases in LBM and offsetting decreases in fat weight. Greatest gains in LBM occurred during the highest volume of work for each group (0 to 6 weeks for the weight group; 7 to 12 weeks for the joggers), again agreeing with previous research (15). Only the weight training group experienced significant leg and hip strength gains, suggesting some difference in the quality of change. These modifications may have included biochemical adaptations in the skeletal muscle (1, 27, 30) or possibly alterations in the central nervous system, producing different motor unit recruitment patterns (22, 31). Increases in strength provided by weight training may decrease the stress of daily work tasks, especially when coupled with positive changes in body composition.

Time to target heart rate increased in both experimental groups, with the largest change occurring in the joggers, supporting the superiority of jogging for elicting decreased HR's during cycling. Beneficial changes in myocardial dynamics, submaximal exercise HR, or in time to target HR resulting from weight training have been reported previously (16, 18) and are to an extent supported by this study. It should be noted that the statistical significance

suggesting an increased time to target HR in the weight training group may, in part, be a function of the decrease shown by the controls at T.

1)1

The exact mechanism for increases in time-to-target HR after weight training is unknown, but may be related to the fact that a given absolute load becomes a lesser relative load following gains in strength. It is also possible that weight training could have a more direct effect, producing

Table 2. Body Weight and Composition (means±SD)

Measure	Group	Initial	6 wk	12 wk
Body wt (kg)	Weight	85.1±9.4	85.7±9.6	84.8±9.7
,	Joggers	83.7±10.0	83.2±9.8	82.5±10.4
	Control	83.1±9.0	83.1±9.5	83.2±9.5
LBM (kg)	Weight	58.1±6.1	59.5±7.7	60.3±7.5*
, 0,	Joggers	57.1±6.2	57.7±6.3	58.7±6.2°
	Control	56.7±5.3	57.1±5.3	56.2±5.2
Body fat (%)	Weight	31.9±5.1	30.6±5.0	28.9±4.3°
	Joggers	31.6±4.6	30.4±4.8	28.3±5.4*
	Control	31.4±5.2	31.1±4.7	31.5±4.9

*Change significantly different from controls, p<0.05.

Table 3. Strength and Power (means±SD)

Measure	Group	Initial	6 wk	12 wk
Half squat (kg)	Weight	139±41	168±55*	197±55°
	Joggers	121±34	121±32	121±31
	Control	132±22	131±28	133±26
Bench press (kg)	Weight	55±11	63±12*	71±13*
	Joggers	53±11	52±11	52±12
	Control	63±14	65±13	67±13**
Vertical jump (cm)	Weight	37±6	39±5	41±5**
	Joggers	35±6	35±5	38±6
	Control	38±6	38±7	39±6
Power (kgm . sec ⁻¹)	Weight	115±19	118±17	120±18
,	Joggers	110±18	111±17	112±18
	Control	112±14	113±16	114±15

*Change significantly different from controls and joggers, p<0.05.

**Change significantly different from controls, p<0.05.

***Change significantly different from joggers, p<0.05.

Table 4. Min to Target Heart Rate (means±SD)

Measure	Initial	6 wk	12 wk
Weight	6.5±0.9	6.7±0.9*	6.8±0.6
Joggers	6.3±1.3	6.6±1.2*	7.1±1.1**
Controls	6.3±0.6	6.0±0.6	6.2±0.6

*Change significantly different from controls, p<0.05.

**Change significantly different from weight trainers and control, p<0.05.

a stronger, more efficient myocardium with superior cardiac dynamics (16). In any case, the potential increases in time-to-target HR suggest a health benefit.

Statistically significant alterations in total cholesterol appear to be a function of changes within the control group. Typically, short-term training does not significantly affect total cholesterol (5). Although all lipid measures, pre and post, were within the normal range, the HDL-C and TC/HDL-C changes in both experimental groups were meaningful because the alterations were significant and in directions connected to lower risk for CVD. These data are in clear agreement with a similar study (15) in which the greatest changes also took place during the highest volume of training, suggesting a connection to total metabolic turnover. In the earlier study (15), dietary analysis revealed no differences between men in weight training and sedentary controls, leading to the conclusion that changes were a result of training rather than of dietary, alcohol or cigarette consumption modification. In this study the dietary, alcohol and cigarette consumption was monitored but not controlled. [The subjects were periodically

Measure	Group	Initial	6 wk	12 wk
Total	Weight	229±40	222±47*	227±44*
Cholesterol	Joggers	231±37	238±35	232±36*
(mg . dl ⁻¹)	Control	208±28	226±38	227±40
HDL-	Weight	38±10	40±12	42±10*
Cholesterol	Joggers	38±4	38±4	44±7*
(mg %)	Control	37±6	37±4	38±6
TC/HDL-C	Weight	6.3±1.4	5.9±2.2	5.7±1.6*
	Joggers	6.1±0.8	6.3 ± 1.4	5.5±1.3*
	Control	5.8±1.5	6.1±1.2	6.1±1.1

^{*}Change significantly different from controls, p<0.05.

Measure	Group	Initial	6 wk	12 wk
Testosterone	Weight	4.8±1.6	4.7±4.9	4.7±1.9
$(ng \cdot m1^{-1})$	Joggers	6.1±4.8	6.6±3.6	6.1 ± 2.1
	Controls	8.1±3.6	8.5±4.4	7.8±3.6
Dihydrote-	Weight	594.3±575.4	648.0±355.9	548.2±405.2
stosterone	Joggers	517.6±470.9	766.3±947.3	638.3±739.1
(pg . m1 ⁻¹)	Controls	363.9±274.5	383.0±227.6	358.2±180.3
Estradiol	Weight	18.6±5.5	17.0±6.1	15.6±7.2*, **
(pg . m1 ⁻¹)	Joggers	17.3±8.5	16.7±6.3	19.1±4.8
	Controls	18.3±5.6	20.1±7.8	21.2±8.4
Estradiol/	Weight	4.1±1.5	3.9±1.7	4.7±1.6
Testosterone	Joggers	3.4 ± 2.0	2.8 ± 1.0	3.5±1.4
	Controls	2.6±1.3	3.1±2.9	3.2±1.9
LH	Weight	50.7±22.1	52.3±22.3	48.5±15.8
(ng . m1 ⁻¹)	Joggers	50.9±18.5	51.9±17.3	52.9±19.1
	Controls	47.2±11.4	46.6±11.7	47.1±11.3
FSH	Weight	242.2±66.6	245.8±67.5	249.1±74.5
(ng . m1 ⁻¹)	Joggers	254.0±57.8	240.3±57.7**	232.5±56.4**
	Controls	195.6±60.4	186.0±60.0	186.9±53.1

^{*}change significantly different from controls, p<0.05.

admonished not to make changes in their lifestyles, other than prescribed by the experimenters. Furthermore, the subjects were questioned at each testing period as to lifestyle (dietary, etc.) changes; none were noted.] Descriptive studies of young adult male weight trainers yielded data showing no differences in blood lipid profiles between the athletic groups and the control group (6, 9). However, recent descriptive studies of nonandrogen using bodybuilders (high volume weight training) showed lipid profiles similar to distance runners with high HDL-C and TC/HDL-C compared to sedentary controls (14). Comparisons are difficult because the former studies (6, 9) did not report training loads nor adequately control steroid use, which is common among strength-power athletes and leads to depression of HDL-C (33). It should be noted that the volume of training in the present study was quite high, a similarity shared by the bodybuilders (14). The existence of an age-related difference to weight training is another possibility, i.e. that young men are less susceptible to these changes. However, Goldberg et al. (12) have reported beneficial alterations in serum lipids in younger men and women after weight training, supporting the contention that this type of training is responsible for the observed lipid changes.

Androgens have been associated with protein anabolism, spermatogenesis, and a number of psychosocial and psychosomatic aspects including aggressiveness and libido (29, 33). Some relationship between androgens and CVD may exist (10, 25, 29). Recent research suggests that CVD may be associated with low testosterone and high estrogen levels (20, 25). Therefore, responses of androgens and related hormones such as the gonadotropins to training may affect a variety of physiological and psychological phenomena.

Training has been reported to produce increases in resting testosterone in middle-aged men, but not in younger more fit men (36). Recently, long duration aerobic exercise, aerobic training and exhaustion have been shown to reduce resting testosterone levels (7, 10, 28). Changes in resting concentration of testosterone may be related the type, volume, and intensity of training as well as to initial fitness and testosterone levels (7, 14, 15, 28, 36). The present study indicated that neither aerobic nor anaerobic training affected resting level of testosterone or its metabolite, dihydrostestosterone. Other serum hormones were within the normal resting values for middle-aged males (2, 19), with training having little effect. The reason for the FSH drop in the jogging group and the estradiol drop realized in the weight group is unknown.

Summary

As expected, the aerobic activity of jogging resulted in increased time-to-target HR on a standard cycle test and a healthier blood lipid profile. Also in line with expectations, weight training proved superior for development of strength. Less predictably, subjects in the weight training group experienced an increase in time-to-target HR after 6 weeks, and had HDL-C and TC/HDL-C changes of the same magnitude as the jogging group. Resting levels of serum hormones were largely unaffected by either mode of training.

Practical Application

1. It is important to understand that many people do not necessarily enjoy aerobic exercise and they use weight training as a means of improving physical fitness. This

28

^{**}change significantly different from weight trainers, p<0.05.

paper offers additional data suggesting that weight training of appropriate volume and intensity produces training effects similar to aerobic training, especially as

it affects serum lipids.

2. Health professionals, physical fitness instructors and strength coaches should be aware of the potential benefits of appropriate high-volume weight training. These benefits include the possibility of reductions in exercise HR, and beneficial alterations in body composition and serum lipids as well as the expected increases in strength and power. By understanding these potential weight training induced benefits, programs can more easily be planned and evaluated. Also, the potential for meeting trainee's goals can be increased.

References

1. Baldwin, K.M., O.M. Martinez, and W.G. Cheadle (1976) Enzymatic changes in hypertrophied fast-twitch skeletal muscle. Pflugers Arch, 364:229-234.

2. Bio-Science Handbook, 13th edition (1982) Van Nuys, California: Bio-Science Laboratories, pp. 11-12, 56-87.

- 3. Berenson, G.S., S.R. Srinivason, A., Lopez-S, B., Radhakrishnamurthy, P.S., Pargaoker, and R.H. Dupree (1972) Clinical application of an indirect method for quantitating serum lipoproteins. Clin Chim Acta, 36:175-
- 4. Brozek, J., F. Grande, J. Anderson, and A. Keys. (1963) A densiometric analysis of body composition: revision of some quantitative assumptions. Ann NY Acad Sci, 110:113-140.
- 5. Byrd, R., D.P. Smith, and C.B. Shackleford (1974) Jogging in middle-aged men: Effect on cardiovascular dynamics. Arch Phys Med Rehabil, 55:301-304.
- 6. Clarkson, P.M., R. Niwterminister, M. Fillyan, and L. Stylos (1981) High density lipoprotein cholesterol in young adult weight lifters, runners and untrained subjects. Hum Biol, 53:251-257.
- 7. Dessypris, A., K. Kuoppasalmi, and H. Adlercreutz (1976) Plasma cortisol, testosterone, and stenedione and luteinizing hormone (LH) in a non-competitive marathon run. J. Steroid Biochem, 7:33-37.
- 8. Enger, S.C., K. Hebjornsen, J. Erikssen, and A. Fretland (1977) High density lipoproteins (HDL) and physical activity: The influence of physical exercise, age, and smoking on HDL-cholesterol and the HDL-C/total cholesterol ratio. Scand J Clin Lab Invest, 3:251-255.
- 9. Farrell, P.A., M.G. Maksud, M.L. Pollock, C. Foster, J. Anholm, J. Hare, and A.S. Leon (1982) A comparison of plasma cholesterol, triglycerides and high density lipoprotein-cholesterol in speed skaters, weight lifters, and non-athletes. Eur J Appl Physiol, 48:77-82.

10. Frey, M.A.B., B.M. Doerr, L.S. Srivastava, and C.J. Glueck (1983) Exercise training, sex hormones, and lipoprotein relationships in men. J Appl Physiol, 54:757-762.

- 11. Gay, V.L., and J.T. Kerlon (1978) Serum LH and FSH following passive immunization against circulating testosterone in the intact male rat and in orchidectomized rats bearing subcutaneous implants of testosterone. Arch Androl, 1:257-266.
- 12. Goldberg, L., D.L. Elliott, R.W. Shatz, and F.E. Kloster (1984) Changes in lipid and lipoprotein levels after weight training. JAMA, 252(4):504-506.

13. Gorman, J.F (1978) Review of physical activity and serum

lipids. Am Correct Ther J, 32:183-189.

14. Hurley, B.F., D.R. Seals, J.M. Hagberg, A.C. Goldberg, S.M. Ostrove, J.O. Holloszy, W.G. Wiest, and A.P. Goldberg (1984) High density-lipoprotein cholesterol in bodybuilders versus power lifters (negative effects of androgense). JAMA, 252(4):507-513.

15. Johnson, C.C., M.H. Stone, A. Lopez-S, J.A. Hebert, L.T. Kilgore, and R.J. Byrd (1982) Diet and exercise in middleaged men. J Am Diet Assoc, 81:695-701.

16. Kanakis, C., and R.C. Hickson (1980) Left ventricular responses to a program of lower limb strength training.

Chest, 78:618-621.

17. Krehl, W.A., A. Lopez-S, and E.I. Good (1967) A rapid analytical system for determining serum lipids. Am J Clin Nutr, 20:139-148.

18. Kusinitz, K., and C.W. Keeney (1958) Effects of progressive weight training on health and physical fitness of adolescent boys. Res Q Am Assoc Health Phys Educ, 29:294-301.

19. Lewis, J.G., R. Ghanadian, and G.D. Chisholm (1976) Serum dihydrotestosterone and testosterone changes with age in man. Acta Endocrinol, 82:444-448.

20. Mendoza, S.G., A. Osuma, A Zerpa, P.S. Gartside, and J.C. Glueck. Hypertriglyceridemia and hypoalphalipoproteinemia in azoospermic and oligospermic young men: Relationships of androgenous testosterone to triglycerides and high density lipoprotein cholesterol metabolism.

21. Niswender, G.D., A.R. Midgley, S.E. Monroe, and L.E. Reichert (1968) Radioimmunoassay for rat luteinizing hormone with antiovine LH serum and ovine LH-131. Proc

Soc Exp Biol Med, 128:807-811.

22. Ono, M., M. Miyashita, and T. Asomi (1976) Inhibitory effect of long distance running training on the vertical jump and other performances among aged males. Biomechanics V-B. Baltimore: University Park Press, pp. 94-100.

23. Paffenbarger, R.S., and W.E. Hale (1975) Work activity and coronary heart mortality. New Eng J Med, 292:545-550.

- 24. Paffenbarger, R.S., M.E. Lauglin, A.S. Gima, et al (1970) Work activity of longshoremen as related to death from coronary heart disease and stroke, New Eng J Med, 282:1109-114.
- 25. Phillips, G.B. (1977) Relationship between serum sex hormones and glucose, insulin and lipid abnormalities in men with myocardial infarction. Proceedings of the National Academy of Science USA, 74:1729-1723.

 Rush, M.E., and H. Lipner (1979) Blockade of GnRH-induced secretion of pituitary FSH by inhibin-containing preparations. Endocrinology, 105:187-194.

1 (a

1) (

27. Salmons, S., and J. Henriksson (1981) The adaptive response of skeletal muscle to increased use. Muscle Nerve, 4:94-105.

28. Stone, M.H., Byrd, R., and Johnson, C. (1984) Observations on serum androgen response to short-term resistence training in middle-aged sedentary males. Natl Strength and Conditioning Assoc. J. 5(6):30-71.

29. Swain, G.T. (1969) The hormones-endocrine physiology. Boston: Little Brown Co.

- 30. Vikko, V., A. Salminen, and J. Rontamaki (1978) Oxidative and lysomal capacity in skeletal muscle of mice after endurance training of different intensities. Acta Physiol Scand, 104:74-81.
- 31. Williams, L.R.T., E.A.S. McEwen, C.D. Watkins, L. Gillespie, and H. Boyd (1979) Motor learning and performance under physical fatigue and the specificity principle. Can J Appl Sport Sci, 4:302-308.

32. Wilmore, J.H. (1969) The use of actual, predicted and constant residual volumes in the assessment of body composition by underwater weighing. Med Sci Sport, 1:87-90.

33. Wilson, J.D., and J.E. Griffin (1980) The use and misuse of androgens. Metabolism, 29:1278-1295.

34. Wood, P., W. Haskell, H. Klein, S. Lewis, M. Stern, and J. Farquhar (1976) The distribution of plasma lipoproteins in middle-aged male runners. Metabolism, 25:1249-1257.

35. Williams, A.T., and Lipner, H. (1981) Negative feedback control of gonadotropin secretion by chronically administered estradiol and porcine follicular fluid (gonodostatin) in ovarectomized rats. Endocrinology, 109:1496-1501.

36. Young, J.R., and A.H. Ismail (1978) Ability of biochemical and personality variables in discriminating between high and low physical fitness levels. J Psychsom Res, 22:193-199.