CHAPTER- II

REVIEW OF RELATED LITERATURE

A study of relevant literature is an essential step to get a full picture of what has been done and said with regard to the problem under study. It is an answer to the question why the hypothesis was selected for of the present problem. It is a key to the thinking of the investigator. Collection of relevant literature provides the basic understanding of the problem and the depth. Such a review brings about a deep insight and a clear perspective of the overall field.

The research scholar has gone through all the relevant literatures, which were available to the present study. The review of literature is instrumental in selection of the topic, transaction of hypothesis and deductive reasoning leading to the problem. It helps to get a clear idea and supports the finding with regard to the problem under study.

The related studies pertaining to this research are presented in the following heads:

2.1. STUDIES ON THE EFFECT OF CYCLING EXERCISE ON LIPID PROFILE

Kang, et.al. (2005) compared metabolic and perceptual responses between exercise performed at constant intensity (CON) and with a Spinning protocol of variable intensity (VAR). Fifteen subjects, including

seven males and eight females (23 +/- 5 yr, 72 +/- 17 kg, and 171 +/- 10 cm), underwent two experimental trials. During each trial, subjects performed a 30-min cycle exercise protocol that was followed by a 30-min recovery period. Exercise was performed at 67 +/- 3% (means +/- SD) of HR(max) in CON. In VAR, the similar intensity (68 +/- 4% HR(max)) was also achieved, although the protocol entailed alternating phases of both higher and lower intensity arranged similarly to what is designed for a typical Spinning workout. Oxygen uptake (VO2) and HR were measured at rest and throughout both exercise and recovery, whereas RPE were recorded during exercise only. Plasma lactate concentrations [La] were determined at rest, the end of exercise, and the end of recovery. No differences in average VO2, HR, and RPE were found during exercise between CON and VAR. However, average VO2 and HR were higher ($P < 0.05$) in VAR than CON (0.33 +/- 0.03 vs 0.26 +/- 0.02 L x min(-1) and 91 +/- 3 vs 80 +/- 2 beats x min(-1), respectively). [La] was higher ($P < 0.05$) at the end of exercise in VAR than CON (7.2 +/- 0.8 vs 2.7 +/- 0.3 mmol x L(-1)), but became similar at the end of recovery. An exercise regimen in which intensity varies exerts no added effect on metabolic and perceptual responses during exercise as long as the average intensity is kept the same. However, VAR resulted in a greater [latin capital V with dot above]O2 after exercise, and this augmented postexercise oxygen consumption may be mediated in part by elevated plasma [La].

Bassett. (1989) reported that the rotating flywheel of a cycle ergometer possesses kinetic energy (KE) by virtue of its rotation about the center of mass. The energy released as the flywheel velocity (FV) decreases during the course of a Wingate test is used to accomplish mechanical work. The subject should not be "credited" with this work since the energy storage occurred prior to the start of the 30-s test. The total KE (KE-total) in the flywheel of a Monark ergometer was determined using the formula KEtotal = 1/2 I omega 2. The KE available to do work (KEwork) was measured by loading the ergometer with 1 Kp (9.8 N), spinning it at predetermined rates, and observing the number of revolutions completed as it coasted to a stop. The difference between KEtotal and KE-work was attributable to friction. The mechanical power supplied by the flywheel in any 5-s period of the Wingate test was found to be: Flywheel power (W) = .00185 (FVstart2-FVend2)/5s where FV is expressed in rpm. This indicates that Wingate test scores should be corrected by subtracting the flywheel power output from the total power output. The correction lowers peak power (PP), mean power (MP), and fatigue index (% fatigue) by 6.2%, 3.0%, and 6.6% in active male subjects (P less than 0.05).

Adamo, et.al. (2010) examined the efficacy of interactive video game stationary cycling (GameBike) in comparison with stationary cycling to music on adherence, energy expenditure measures, submaximal aerobic fitness, body composition, and cardiovascular disease risk markers in overweight

and obese adolescents, using a randomized controlled trial design. Thirty overweight (with at least 1 metabolic complication) or obese adolescents aged 12-17 years were stratified by gender and randomized to video game or music condition, with 4 participants (2 per group) failing to complete the twice weekly 60 min sessions of the 10-week trial. The music group had a higher rate of attendance compared with the video game group (92% vs. 86%, $p < 0.05$). Time spent in minutes per session at vigorous intensity (80%-100% of predicted peak heart rate) (24.9 ± 20 min vs. 13.7 ± 12.8 min, $p < 0.05$) and average distance (km) pedaled per session (12.5 ± 2.8 km vs. 10.2 ± 2.2 km, $p < 0.05$) also favoured the music group. However, both interventions produced significant improvements in submaximal indicators of aerobic fitness as measured by a graded cycle ergometer protocol. Also, when collapsed, the exercise modalities reduced body fat percentage and total cholesterol. The present study indicates that cycling to music was just as effective as stationary cycling while playing video games at improving fitness, body composition, and cholesterol profiles in overweight and obese teens, and resulted in increased attendance, vigorous intensity of physical activity, and distance pedaled. Therefore, our data support the superiority of cycling to music and indicate investing in the more expensive GameBike may not be worth the cost.

Poole, et.al. (2011) evaluated the combined effect of a meal replacement and an alleged weight loss supplement (WLS) on body composition, fitness

parameters, and clinical health in moderately overweight college-aged men and women. Body mass, bench press 1 repetition maximum (1RM), leg press 1RM, body composition, V(O2)max, fasting glucose (GLU), and lipid panels were evaluated before (T1) and after (T2) 8 weeks of combined resistance training (RT) and cardiovascular training (CVT). After T1, subjects were randomly assigned in a double-blind fashion to either the WLS (6 men, 7 women; 21 ± 5 years, 168 ± 8 cm, 75.4 ± 12.7 kg, 31.6 ± 7.7%BFAT) or placebo (PLA: 6 men, 6 women; 22 ± 4 years, 174 ± 9 cm, 84.1 ± 8.8 kg, 30.2 ± 5.6%BFAT) group. Both groups performed 3 d · wk(-1) of combined progressive RT (2 × 12 reps of 8 exercises at 75-80% 1RM) and CVT (30 minutes on a cycle ergometer at 70-85% heart rate reserve). Subjects consumed 4 capsules per day and a once-daily meal replacement throughout the protocol. Percent body fat, bench press 1RM, and leg press 1RM significantly improved ($p < 0.05$) in both groups. Blood GLU (G × T; $p = 0.048$) improved in WLS and systolic blood pressure (SBP) approached significance (G × T; $p = 0.06$) in the WLS group. Follow-up analysis of SBP revealed a significant within-group decrease in the WLS group, whereas no within-group changes were found for either group for GLU. Practically speaking, daily supplementation with a meal replacement and a thrice weekly exercise program can increase fitness levels and improve body composition, whereas adding a thermogenic substance provides no additional benefit over

fitness or body composition changes but may favorably alter serum markers of clinical health.

Tantiwong et.al. (2010) measured NF-κB DNA-binding activity and the mRNA level of putative NF-κB-regulated myokines interleukin (IL)-6 and monocyte chemotactic protein-1 (MCP-1) in muscle samples from T2DM, obese, and lean subjects immediately before, during (40 min), and after (210 min) a bout of moderate-intensity cycle exercise. At baseline, NF-κB activity was elevated 2.1- and 2.7-fold in obese nondiabetic and T2DM subjects, respectively. NF-κB activity was increased significantly at 210 min following exercise in lean (1.9-fold) and obese (2.6-fold) subjects, but NF-κB activity did not change in T2DM. Exercise increased MCP-1 mRNA levels significantly in the three groups, whereas IL-6 gene expression increased significantly only in lean and obese subjects. MCP-1 and IL-6 gene expression peaked at the 40-min exercise time point. We conclude that insulin-resistant subjects have increased basal NF-κB activity in muscle. Acute exercise stimulates NF-κB in muscle from non diabetic subjects. In T2DM subjects, exercise had no effect on NF-κB activity, which could be explained by the already elevated NF-κB activity at baseline. Exercise-induced MCP-1 and IL-6 gene expression precedes increases in NF-κB activity, suggesting that other factors promote gene expression of these cytokines during exercise.

Koppo, (2010) evaluated the relative contributions of various hormones involved in the regulation of lipid mobilization in subcutaneous adipose tissue (SCAT) during exercise and to assess the impact of obesity on this regulation. Eight lean and eight obese men performed a 60-min cycle exercise bout at 50% of their peak oxygen uptake on two occasions: during intravenous infusion of octreotide (a somatostatin analog) or physiological saline (control condition). Lipolysis in SCAT was evaluated using in situ microdialysis. One microdialysis probe was perfused with the adrenergic blockers phentolamine and propranolol while another probe was perfused with the phosphodiesterase and adenosine receptor inhibitor aminophylline. Compared with the control condition, infusion of octreotide reduced plasma insulin levels in lean (from approximately 3.5 to 0.5 microU/ml) and in obese (from approximately 9 to 2 microU/ml), blunted theexercise-induced rise in plasma GH and epinephrine levels in both groups, and enhanced the exercise-induced natriuretic peptide (NP) levels in lean but not in obese subjects. In both groups, octreotide infusion resulted in higher exercise-induced increases in dialysate glycerol concentrations in the phentolamine-containing probe while no difference in lipolytic response was found in the aminophylline-containing probe. The results suggest that insulin antilipolytic action plays a role in the regulation of lipolysis during exercise in lean as well as in obese subjects. The octreotide-induced enhancement of exercise lipolysis in lean subjects was associated with an increased exercise-induced plasma NP

response. Adenosine may contribute to the inhibition of basal lipolysis in both subject groups.

Numao, (2011) investigated the influence of aerobic exercise intensity on changes in the concentrations of total adiponectin and adiponectin oligomers (high-molecular weight [HMW] and middle- plus low-molecular weight [MLMW] adiponectin), and the endocrine mechanisms involved in exercise-induced changes in adiponectin oligomer profiles in middle-aged abdominally obese men. Using a crossover design, 9 middle-aged abdominally obese men (age, 54.1 ± 2.4 years; body mass index, 27.9 ± 0.6 kg/m²) underwent 2 trials that consisted of 60 minutes of stationary cycle exercise at either moderate-intensity (ME) or high-intensity (HE) aerobic exercise (50% or 70% of peak oxygen uptake, respectively). Blood samples were collected to measure the concentrations of adiponectin oligomers, hormones (catecholamines, insulin, and growth hormone), metabolites (free fatty acid, glycerol, triglyceride, and glucose), and cytokines (interleukin-6 and tumor necrosis factor-α). After exercise, plasma catecholamine concentrations were higher during HE than during ME ($P < .05$). Total adiponectin concentration decreased at the end of HE ($P < .05$), but remained unchanged after ME. The HMW adiponectin concentration did not change at either intensity, whereas the MLMW concentration decreased at the end of HE ($P < .05$). The ratio of HMW to total adiponectin concentration increased significantly ($P < .05$), whereas the ratio

of MLMW to total adiponectin concentration decreased significantly ($P < .05$), at the end of HE. The percentage changes in epinephrine concentration from baseline to the end of exercise were correlated with the percentage changes in total adiponectin concentration ($r = -0.67$, $P < .05$) and MLMW adiponectin concentration ($r = -0.82$, $P < .05$) from baseline to the end of HE. Our results indicate that the change in total adiponectin was mainly due to a change in MLMW adiponectin concentration during high-intensity exercise in middle-aged abdominally obese men. Epinephrine may partially regulate the decrease in total and MLMW adiponectin concentrations during high-intensity exercise.

Haufe, et.al. (2010) reported that Endurance training at an intensity eliciting maximal fat oxidation may have a beneficial effect on body weight and glucose metabolism in obese patients. However, the exercise intensity at which maximal fat oxidation occurs and the factors limiting fat oxidation are not well studied in this population. Obese, otherwise healthy men (n=38) and women (n=91) performed an incremental exercise test up to exhaustion on a cycle ergometer. Substrate oxidation was estimated using indirect calorimetry. Magnetic resonance tomography and spectroscopy were conducted to assess body fat distribution and intramyocellular fat content. We determined the exercise intensity at which maximal body fat oxidation occurs and assessed whether body composition, body fat distribution, intramyocellular fat content, or oxidative capacity predict exercise-induced fat oxidation. Maximal exercise-

induced fat oxidation was 0.30+/-0.02 g/min in men and 0.23+/-0.01 g/min in women ($p<0.05$). Exercise intensity at the maximum fat oxidation was 42+/-2.2% VO (2 max) in men and 43+/-1.7% VO (2 max) in women. With multivariate analysis, exercise-induced fat oxidation was related to fat-free mass, percent fat mass, and oxidative capacity, but not to absolute fat mass, visceral fat, or intramyocellular fat content. We conclude that in obese subjects the capacity to oxidize fat during exercise appears to be limited by skeletal muscle mass and oxidative capacity rather than the availability of visceral or intramyocellular fat.

Ben Ounis, **et.al. (2009)** examined if, in young obese patients, an individualized training programme in association with a caloric restriction programme which had an effect on whole-body lipid oxidation, was able to induce changes on plasma adipocytokine concentrations. Twenty-seven obese female adolescents participated in the study. Whole-body lipid oxidation during exercise was assessed by indirect calorimetry during a graded cycle ergometer test. Body mass (BM), body mass index (BMI), percentage of body fat (%BF), insulin homeostasis model assessment (HOMA-IR) and fasting levels of circulating adipocytokines were assessed prior and after a two-month diet programme, individualized training programme targeted at Lipox(max) corresponded to the power at which the highest rate of lipids was oxidized and combined diet/training programme. The diet/training programme induced both a shift to a higher-power intensity of Lipox(max) (+27.8 + or - 5.1

W; p<0.01) and an increase of lipid oxidation at Lipox(max) (+96.8 + or - 16.2mg/min; p<0.01). The enhancement in lipidoxidation was significantly (p<0.01) correlated with the diet/training-induced improvement in %BF (r = -0.47), HOMA-IR (r = -0.66), leptin (r = -0.41), TNF-alpha (r = -0.48), IL-6 (r = -0.38), adiponectin (r = 0.43) and resistin (r = 0.51). This study showed that in obese female adolescents a moderate training protocol targeted at Lipox(max) and combined with a diet programme improved their ability to oxidize lipids during exercise, and that this improvement was associated with changes in plasma adipocytokine concentrations.

Holtz, et.al. (2008) determined if varying carbohydrate availability, with energy intake held constant, mediates post-exercise insulin action. Ten young (21 +/- 2 y, overweight (body fat 37% +/- 3%) men and women completed 3 conditions in random order: (i) no-exercise (BASE), (ii) exercise with energy balance but carbohydrate deficit (C-DEF), and (iii) exercise with energy and carbohydrate balance (C-BAL). In the exercise conditions, subjects expended 30% of total daily energy expenditure on acycle ergometer at 70% VO2 peak. Following exercise, subjects consumed a meal that replaced expended energy (~3000 kJ) and was either balanced (intake = expenditure) or deficient (-100 g) in carbohydrate. Twelve hours later, insulin action was measured by continuous infusion of glucose with stable isotope tracer (CIG-SIT). Changes in insulin action were evaluated using a one-way ANOVA with repeated measures. During

CIG-SIT, non-oxidative glucose disposal (i.e., glucose storage) was higher in C-DEF than in BASE (27.2 +/- 3.2 vs. 16.9 +/- 3.5 micromol.L-1.kg-1.min-1, $p < 0.05$). Conversely, glucose oxidation was lower in C-DEF (8.6 +/- 1.3 micromol.L-1.kg-1.min-1) compared with C-BAL (12.2 +/- 1.2 micromol.L-1.kg-1.min-1), and BASE (17.1 +/- 2.2 micromol.L-1.kg-1.min-1), $p < 0.05$). Fasting fat oxidation was higher in C-DEF than in BASE (109.8 +/- 10.5 vs. 80.7 +/- 9.6 mg.min-1, $p < 0.05$). In C-DEF, enhanced insulin action was correlated with the magnitude of the carbohydrate deficit ($r = 0.82$, $p < 0.01$). Following exercise, re-feeding expended energy, but not carbohydrate, increased fasting fat oxidation, and shifted insulin-mediated glucose disposal toward increased storage and away from oxidation.

2.2 STUDIES ON CYCLING EXERCISES ON TESTOTERONE

Hough, et.al. (2011) compared plasma and salivary cortisol and testosterone responses to 4 exercise trials; these were (a) continuous cycle to fatigue at 75% peak power output (Wmax) (FAT); (b) 30-minute cycle alternating 1-minute 60% and 1 minute 90% Wmax (60/90); (c) 30-minute cycle alternating 1-minute 55% and 4-minute 80% Wmax (55/80); and (d) Squatting 8 sets of 10 repetitions at 10 repetition maximum (RESIST). Blood and saliva samples were collected pre-exercise and at 0, 10, 20, 30, 40, 50, and 60 minute postexercise. Pre- to postexercise plasma cortisol increased in allexercise trials, except 60/90. Increases in 55/80 remained above pre-exercise levels for the entire postexercise

period. Salivary cortisol increased from pre- to postexercise in FAT and 55/80 trials only. Once elevated after 55/80, it remained so for the postexercise period. Plasma testosterone increased from pre- to postexercise in all trials except 55/80. Salivatestosterone increased from pre- to postexercise in all trials with the longest elevation occurring after 55/80. Area under the curve analysis indicated that the exercise response of salivary hormones was greater in all cycle trials (cortisol) and in the 60/90 and 55/80 trials (testosterone) compared with the other trials. This study indicates that the 55/80 cycle protocol induces a prolonged salivary and plasma cortisol and salivary testosterone response compared with the other trials and so may be a useful diagnostic tool of overreaching.

Koch, et.al. (2011) investigated the association of total serum testosterone and sex hormone-binding globulin (SHBG) levels on exercise capacity and maximal power output in men using a cross-sectional, population-based adult cohort. From the Study of Health in Pomerania (SHIP), 624 men age 25 to 85 years who underwent a standardized progressive incremental exercise protocol on a cycle ergometer were included in the analyses. Exercise capacity was characterized by oxygen uptake at anaerobic threshold (V'O(2) at L) and peak exercise (V'O(2 peak)) as well as maximal power output at peak exertion. Multivariable linear regression analyses adjusted for age, sex, body mass index, physical activity, and smoking were performed. Further, linear regression analyses with cubic splines and sensitivity analyses were undertaken.

At peak exercise performance, testosterone and SHBG levels showed no associations with V'O(2 peak), V'O(2) at L as well as maximal power output, even after controlling for confounding factors including age, body mass index, physical activity, and smoking. An adverse association between the free testosterone index and V'O(2) at L was found. Linear regression analyses with cubic splines did not change the main results. In conclusion, this is the first study focusing on the association of total serum testosterone and SHBG on exercise capacity and physical performance in healthy volunteers based on a large-scale population-based study. After adjustment for relevant influencing factors, neither total serum testosterone nor SHBG levels had any interference with peak exercise capacity, aerobic exercise capacity, or maximal power output in men.

Cadore, et.al. (2010) investigated the effects of concurrent strength and endurance training on neuromuscular and hormonal parameters in elderly men. 23 healthy men (65±4 years) were randomly assigned to 1 of 3 groups: concurrent (CG, n=8), strength (SG, n=8) or endurance group (EG, n=7). The programs consisted, of strength training, endurance training on a cycle ergometer or a combination of both in the same session 3 times per week over a duration of 12 weeks. Subjects were evaluated on parameters related to muscle strength, muscle activation and serum hormones. There were significant increases in lower-body strength in all groups ($P<0.05$), with higher increases in SG (67%) than CG (41%) and both were higher than EG (25%) ($p<0.01$). Only SG and CG increased upper-

body strength ($p<0.01$), with no significant difference between the 2 groups. Furthermore, there were significant decreases in free testosterone in EG after training. Significant increases in isometric strength and maximal muscle activation ($p<0.05$) as well as decreases in the submaximal muscle activation to the same load, were only seen in SG ($p<0.05$). The present results suggest that the interference effect observed due to concurrent strength and endurance training could be related to impairment of neural adaptations.

Beaven, et.al. (2010) identified episodic steroid secretion via frequent salivary sampling and investigate any interaction between ultradian rhythmicity and induced changes in testosterone. Salivary testosterone and cortisol concentrations of seven males (age 20-40 years) were measured every 10 min between 0800 and 1600 h on three consecutive days. On either the second or third day, three interventions designed to elicit a hormonal response were randomly assigned: sprint exercise (two 30-s maximal efforts on a cycle ergometer); boxing (two 30-s maximal punching efforts); and a violent video game (10 min of player vs. player combat). On the other days subjects were inactive. Testosterone data on non-intervention days suggested pulsatile secretion with a pulse interval of 47 +/- 9 min (mean +/- SD). The sprint intervention substantially affected hormones: it elicited a small transient elevation intestosterone (by a factor of 1.21; factor 90% confidence limits x/ divided by 1.21) 10 min after exercise, and a moderate elevation in cortisol peaking 50 min post-exercise (factor 2.3; x/ divided by 2.6).

The testosterone response correlated with the change in testosterone concentration in the 10 min prior to the sprint (r = 0.78; 90% CL 0.22-0.95) and with a measure of randomness in testosterone fluctuations (r = 0.83; 0.35-0.96). Thus, the salivary testosterone response to exercise may be dependent on the underlying ultradian rhythm and aspects of its regulation. This interaction may have important implications for adaptation to exercise

Crewther, (2010) validated the testosterone (T) and cortisol (C) concentration measures in saliva in response to short high-intensity exercise. Nine healthy males provided matching saliva and plasma samples before and after a 30-second Wingate cycle test. Saliva was assayed for T (Sal-T) and C (Sal-C) concentrations, and plasma for total T and total C, sex hormone-binding globulin, corticosteroid-binding globulin (CBG) and albumin concentrations. The plasma free and bioavailable hormones were calculated. The Sal-T and plasma T correlations were weak to moderate (r=0.57-0.61) when examined between individuals (pooled data for all participants), but these relationships improved (r = 0.71-0.73) within individuals (data for each participant on average). The Sal-C and plasma C correlations were strong both between individuals (r=0.81-0.84) and within individuals (r=0.83-0.84). The peak relative increases in Sal-T (35+/-9%) and Sal-C (63+/-29%) concentrations exceeded the plasma total and/or free hormones, but not the bioavailable hormones. Albumin (10+/-3%) and CBG (16+/-4%) also increased with exercise, along with blood lactate (943+/-119%).

The Sal-T and Sal-C concentration measures were validated in response to short high-intensity exercise, especially for individuals. The hormonal changes in saliva were also more sensitive to exercise (i.e. greater relative responses) than the plasma total and/or free hormones, potentially arising from changes in the binding proteins and blood lactate. These findings support the use of saliva as a medium for steroid determination in sport.

Derbré, et.al. (2010) investigated the effects of a 6-month sprint training program on plasma androgens and catecholamine (CA) concentrations in response to a 6 s sprint in adolescent boys [training group (TG), n=6; control group (CG), n=6]. A 6 s-sprint test was performed on a cycle ergometer before and after training (Pre-T and Post-T, respectively). Plasma total testosterone (TT), bioavailable testosterone (BT), and CA concentrations were measured at rest, after a warm-up, immediately after a 6 s-sprint, and during the recovery (i. e. 5 and 20 min). After training period, plasma TT concentrations increased significantly at the end of the sprint and during the recovery in the TG. No effects for sampling times and period were observed in BT levels. Plasma TT concentrations after 5 min of recovery were positively correlated with the corresponding values of plasma lactate (La) concentrations and with post-6 s-sprint plasma adrenaline (A) concentrations ($r=0.52$; $p<0.01$ and $r=0.61$; $p<0.01$, respectively). These results suggest that sprint training increases plasma TT concentrations in response to sprint exercise in adolescent boys. Plasma A and

plasma La concentrations increases in response to sprint exercise could be involved in this elevation of plasma TT concentrations.

de Sousa, et.al. (2010) evaluated the effects of a micro cycle of overload training (1st-8th day) on metabolic and hormonal responses in male runners with or without carbohydrate supplementation and investigated the cumulative effects of this period on a session of intermittent high-intensity running and maximum-performance-test (9th day). The participants were 24 male runners divided into two groups, receiving 61% of their energy intake as CHO (carbohydrate-group) and 54% in the control-group (CON). The testosterone was higher for the CHO than the CON group after the overload training (694.0 +/- 54.6 vs. CON 610.8 +/- 47.9 pmol/l). On the ninth day participants performed 10 x 800 m at mean 3 km velocity. An all-out 1000 m running was performed before and after the 10 x 800 m. Before, during, and after this protocol, the runners received solution containing CHO or the CON equivalent. The performance on 800 m series did not differ in either group between the first and last series of 800 m, but for the all-out 1000 m test the performance decrement was lower for CHO group (5.3 +/- 1.0 vs. 10.6 +/- 1.3%). The cortisol concentrations were lower in the CHO group in relation to CON group (22.4 +/- 0.9 vs. 27.6 +/- 1.4 pmol/l) and the IGF1/IGFBP3 ratio increased 12.7% in the CHO group. During recovery, blood glucose concentrations remained higher in the CHO group in comparison with the CON

group. It was concluded that CHO supplementation possibly attenuated the suppression of the hypothalamic-pituitary-gonadal axis and resulted in less catabolic stress, and thus improved running performance.

Tishova, and Kalinchenko. (2009) reported that the metabolic syndrome (MS) is associated with low serum testosterone levels. Conversely, low testosterone levels induce MS. These operational mechanisms reinforce one another and induce a vicious cycle. This is a report on a morbid obesity 42 year-old man with the MS and serum testosterone of 5.0 nmol/L (N: 12.0-33.0), who was resistant to treatment with diet and exercise. He was treated with testosterone undecanoate for 16 months. Anthropological and laboratory variables were measured before and during testosterone administration. Also the aging Male Symptom Scale (AMS), the International Index of Erectile Function (IIEF) and Beck's Depression Inventory were assessed. After 16 months, there was a weight loss of 50 kg and a decrease in waist circumference of 36.5 cm. Blood pressure normalized and laboratory variables returned to the normal range. The patient did not meet the criteria for the MS anymore. There were improvements on the AMS, the IIEF and Beck's Depression Inventory. Normalizing testosterone in men with morbid obesity in combination with diet and exercise, with the MS and low testosterone levels, may rescue them from the MS, improving their mood and their stamina to follow a diet and to exercise.

Crewther, et.al. (2011) examined the effects of short-cycle sprints on power, strength, and salivary hormones in elite rugby players. Thirty male rugby players performed an upper-body power and lower-body strength (UPLS) and/or a lower-body power and upper-body strength (LPUS) workout using a crossover

design (sprint vs. control). A 40-second upper-body or lower-bodycycle sprint was performed before the UPLS and LPUS workouts, respectively, with the control sessions performed without the sprints. Bench throw (BT) power and box squat (BS) 1 repetition maximum (1RM) strength were assessed in the UPLS workout, and squat jump (SJ) power and bench press (BP) 1RM strength were assessed in the LPUS workout. Saliva was collected across each workout and assayed for testosterone (Sal-T) and cortisol (Sal-C). The cycle sprints improved BS (2.6 ± 1.2%) and BP (2.8 ± 1.0%) 1RM but did not affect BT and SJ power. The lower-body cycle sprint produced a favorable environment for the BS by elevating Sal-T concentrations. The upper-body cycle sprint had no hormonal effect, but the workout differences (%) in Sal-T (r = -0.59) and Sal-C (r = 0.42) concentrations correlated to the BP, along with the Sal-T/C ratio (r = -0.49 to -0.66). In conclusion, the cycle sprints improved the BP and BS 1RM strength of elite rugby players but not power output in the current format. The improvements noted may be explained, in part, by the changes in absolute or relative hormone concentrations. These findings have practical implications for prescribing warm-up and training exercises.

Lane, et.al. (2010) examined the effect of dietary carbohydrate (CHO) consumption on the free testosterone to cortisol (fTC) ratio during a short-term intense micro-cycle of exercise training. The fTC ratio is a proposed biomarker for overreaching-overtraining (i.e., training stress or imbalance) in athletes. The

ratio was studied in two groups, control-CHO (approximately 60% of daily intake, n = 12) and low-CHO (approximately 30% of daily intake, n = 8), of male subjects who performed three consecutive days of intensive training (approximately 70-75% maximal oxygen consumption, 60 min per day) with a dietary intervention (on the day before and during training). Resting, pre-exercise blood samples were collected under standardized-controlled conditions before each day of training (Pre 1, 2, 3) and on a fourth day after the micro-cycle (Rest). Bloods were analyzed for free testosterone and cortisol via radioimmunoassay procedures. Subjects performed no additional physical activity other than prescribed training. Statistical analysis (ANCOVA) revealed the fTC ratio decreased significantly ($p < 0.01$) from pre-study resting measurement (Pre 1) to the final post-study resting measurement (Rest) in the low-CHO group (-43%), but no change occurred ($p > 0.05$) in the control-CHO group (-3%). Findings suggest if the fTC ratio is utilized as a marker of training stress or imbalance it is necessary for a moderately high diet of CHO to be consumed to maintain validity of any observed changes in the ratio value.

2.3 STUDIES ON PROTEIN SUPPLEMENTATION ON LIPIDS

Brinkworth, et.al. reported that dietary restriction on and increased physical activity are recommended for obesity treatment. Very low carbohydrate diets are used to promote weight loss, but their effects on physical function and exercise tolerance in overweight and obese individuals are largely unknown. The

aim of this study was to compare the effects of a very low carbohydrate, high fat (LC) diet with a conventional high carbohydrate, low fat (HC) diet on aerobic capacity, fuel utilization during submaximal exercise, perceived exercise effort (RPE) and muscle strength. Sixty subjects (age: 49.2 +/- 1.2 years; BMI: 33.6 +/- 0.5 kg/m(2)) were randomly assigned to an energy restricted (~6-7 MJ, 30% deficit), planned isocaloric LC or HC for 8 weeks. At baseline and week 8, subjects performed incremental treadmill exercise to exhaustion and handgrip and isometric knee extensor strength were assessed. Weight loss was greater in LC compared with HC (8.4 +/- 0.4% and 6.7 +/- 0.5%, respectively; P = 0.01 time x diet). Peak oxygen uptake and heart rate were unchanged in both groups (P > 0.17). Fat oxidation increased during submaximal exercise in LC but not HC (P < 0.001 time x diet effect). On both diets, perception of effort during submaximal exercise and handgrip strength decreased (P </= 0.03 for time), but knee extensor strength remained unchanged (P > 0.25). An LC weight loss diet shifted fuel utilization toward greater fat oxidation during exercise, but had no detrimental effect on maximal or submaximal markers of aerobic exercise performance or muscle strength compared with an HC diet. Further studies are required to determine the interaction of LC diets with regular exercise training and the long-term health effects.

Parente EB, et.al. reported that diet and exercise help improve obese adults' lipid profile. However, their effect on obese children, the aim of the

present study, is poorly known. Fifty obese children were studied into 2 paired groups: Group D (1,500 - 1,800 kcal diet: 55% carbohydrate, 30% fat, 15% protein), and Group DE (same diet + aerobic physical activity 1 hour/day 3 times a week). After 5 months BMI, triglycerides, total cholesterol (TC) and fractions were assessed. No change in triglycerides, TC and low-density lipoprotein cholesterol (LDL-C) levels were reported in both groups. However, high-density lipoprotein cholesterol (HDL-C) increased (+10.3%; p< 0.01) only in DE Group. Screening patients with TC > 170 mg/dL, LDL-C > 110 mg/dL and HDL-C < 35 mg/dL we had: similar reduction for TC in both groups (-6.0% x -6.0%; p= ns), LDL-C reduction in both groups (-14.2% x -13.5%; p= ns), and HDL-C increase only in DE Group (+10.0%; p< 0.05). CONCLUSIONS: 1) Hypocaloric diet (HD) + exercise, rather than diet only, increase obese children's HDL-C levels irrespective of baseline levels; 2) HD only and HD + exercise lead to TC and LDL-C reduction in obese children with TC and LDL-C above normal values.

Woo.(2011) reported that with increasing life expectancy in developed and developing countries, maintaining health and function in old age has become an important goal, including avoidance or optimal control of chronic diseases; maintenance or retarding the decline of physical and cognitive function; optimizing psychological health; and maintaining independent functioning in tasks related to self-care and societal interaction. This article discusses all of

those, as well as other components of successful aging such as social network and socioeconomic status.

Ponnampalam, et.al. (2011) investigated the effect of positional distribution of palmitic acid (Sn-1, 2 & 3) of palm oil on cardiovascular health and development of obesity, using weaner pigs as a model for young children. Male and female weaner piglets were randomly allocated to 4 dietary treatment groups: 1) pork lard (LRD); 2) natural palm olein (NPO); 3) chemically inter-esterified PO (CPO) and 4) enzymatically inter-esterified PO (EnPO) as the fat source. Diets were formulated with 11% lard or with palm olein in order to provide 31% of digestible energy from fat in the diet and were balanced for cholesterol, protein and energy across treatments. From 8 weeks onwards, pigs on EnPO diet gained ($P < 0.05$) more weight than all other groups. Feed conversion efficiency (feed to gain) over the 12 week experimental period did not vary between treatment groups. Plasma LDL-C content and LDL-C/HDL-C ratio in pigs fed natural PO tended to be lower compared to all other diets. The natural PO lowered ($P < 0.02$) the plasma triglyceride (TG) content relative to the lard or EnPO diets, but was not different from the CPO diet. The natural PO diet was associated with lower ($P < 0.05$) saturated fat levels in subcutaneous adipose tissue than the CPO and EnPO diets that had lower saturated fat levels than the lard diet. Female pigs had lower lean and higher fat and fat:lean ratio in the body compared with male pigs. No difference in weight gain or blood lipid parameters

was observed between sexes. The observations on plasma TG, muscle and adipose tissue saturated fatty acid contents and back fat (subcutaneous) thickness suggest that natural palm oil may reduce deposition of body fat. In addition, dietary supplementation with natural palm oil containing palmitic acid at different positions in meat producing animals may lead to the production of meat and meat products with lower saturated fats. An increase in fat content and a decrease in lean content in female pigs resulted in an increased body fat : lean ratio but gender had no effect on blood lipid parameters or insulin concentrations.

Lee, et.al. (2011) investigated the effects of coenzyme Q(10) supplementation on metabolic parameters, inflammatory markers, arterial stiffness, and fatigue in obese subjects. We performed a randomized, double-blind, placebo-controlled, single-center study on 51 obese subjects with a body mass index (BMI) of ≥25kg/m(2). We collected anthropometric measurements, blood for laboratory testing, brachial-ankle pulse wave velocity (baPWV) as an indicator of arterial stiffness, and responses to a fatigue severity scale (FSS) questionnaire at the initial (0 week) and final (12 weeks) visits. A total of 36 subjects successfully completed the study protocol. Serum coenzyme Q(10) levels increased significantly from 0.65±0.27μg/mL to 1.20±0.38μg/mL in the coenzyme Q(10) group (P<.001). Oral administration of coenzyme Q(10) did not significantly affectlipid profiles, oxidative and inflammatory markers [including lipoprotein (a), oxidized low-density lipoprotein level, C-reactive protein, and

white blood cell count], or baPWV. The mean FSS score decreased significantly from 40.1 to 33.1 in the coenzyme Q(10) group (P=.017), but no significant change was seen in the placebo group (P□=□.464). However, the extents of the change in mean FSS score between the placebo and coenzyme Q(10) groups were not significantly different (P=.287). In conclusion, we found no evidence that coenzyme Q(10) affects fatigue index, arterial stiffness, metabolic parameters, or inflammatory markers.

Lira, et.al. (2011) examined the effects of vitamin D3 and vitamin E supplementation on levels of IL-6 and IL-10 (as a marker of anti-inflammatory cytokines since, a balance between pro- and anti-inflammatory cytokines is maintained) protein expression in adipose tissue of mice provided with an HFD. Additionally, we measured the effects of vitamin E and vitamin D3 treatment on LPS-stimulated 3T3-L1 adipocytes IL-6 and IL-10 secretion. IL-6 protein levels and the IL-6/IL-10 ratio were decreased in epididymal white adipose tissue in groups receiving vitamins E and D3 supplementation compared to the HFD group. A 24-hour treatment of vitamin D3 and vitamin E significantly reduced the IL-6 levels in the adipocytes culture medium without affecting IL-10 levels. Vitamin D3 and vitamin E supplementation in an HFD had an anti-inflammatory effect by decreasing IL-6 production in epididymal adipose tissue in mice and in 3T3-L1 adipocytes stimulated with LPS. Our results suggest that vitamin E and

D3 supplementation can be used as an adjunctive therapy to reduce the proinflammatory cytokines present in obese patients.

Neff, et.al. (2011) examined the effects of DHA on plasma lipid and lipoprotein concentrations and other biomarkers of cardiovascular risk in the absence of weight loss. In this randomized, controlled, double-blind trial, 36 overweight or obese adults were treated with 2 g/d of algal DHA or placebo for 4.5 mo. Markers of cardiovascular risk were assessed before and after treatment. In the DHA-supplemented group, the decrease in mean VLDL particle size ($P \leq 0.001$) and increases in mean LDL ($P \leq 0.001$) and HDL ($P \leq 0.001$) particle sizes were significantly greater than changes in the placebo group. DHA supplementation also increased the concentrations of large LDL ($P \leq 0.001$) and large HDL particles ($P = 0.001$) and decreased the concentrations of small LDL ($P = 0.009$) and medium HDL particles ($P = 0.001$). As calculated using NMR-derived data, DHA supplementation reduced VLDL TG ($P = 0.009$) and total TG concentrations ($P = 0.006$). Plasma IL-10 increased with DHA supplementation to a greater extent than placebo ($P = 0.021$), but no other significant changes were observed in glucose metabolism, insulin sensitivity, blood pressure, or markers of inflammation with DHA. In summary, DHA supplementation resulted in potentially beneficial changes in some markers of cardiometabolic risk, whereas other markers were unchanged.

Kelishadi, et.al. (2010) evaluated the effects of zinc sulfate in comparison with placebo on markers of insulin resistance, oxidative stress, and inflammation in a sample of obese prepubescent children. This triple-masked, randomized, placebo-controlled, crossover trial was conducted among 60 obese Iranian children in 2008. Participants were randomly assigned to two groups of equal number; one group received 20 mg of elemental zinc and the other group received placebo on a regular daily basis for 8 weeks. After a 4-week washout period, the groups were crossed over. In addition to anthropometric measures and blood pressure, fasting plasma glucose, lipidprofile, insulin, apolipoproteins A-1 (ApoA-I) and B, high-sensitivity C-reactive protein (hs-CRP), leptin, oxidized low-density lipoprotein (ox-LDL), and malondialdehyde were determined at all four stages of the study. Irrespective of the order of receiving zinc and placebo, in both groups, significant decrease was documented for Apo B/ApoA-I ratio, ox-LDL, leptin and malondialdehyde, total and LDL-cholesterol after receiving zinc without significant change after receiving placebo. In groups, hs-CRP and markers of insulin resistance decreased significantly after receiving zinc, but increased after receiving placebo. In both groups, the mean body mass index (BMI) Z-score remained high, after receiving zinc, the mean weight, BMI, BMI Z-score decreased significantly, whereas these values increased after receiving placebo. These results are particularly important in light of the deleterious consequences of childhood obesity and early changes in

markers of inflammatory and oxidative stress. We suggest exploring the direct clinical application of zincsupplementation in childhood obesity in future studies.

Aronsson, et.al. (2010) determined the mechanism by which the probiotic bacteria Lactobacillus paracasei ssp paracasei F19 (F19) could alter fat storage. Angiopoietin-like 4 (ANGPTL4) is a circulating lipoprotein lipase (LPL) inhibitor that controls triglyceride deposition into adipocytes and has been reported to be regulated by gut microbes. A diet intervention study of mice fed high-fat chow supplemented with F19 was carried out to study potential mechanistic effects on fat storage. Mice given F19 displayed significantly less body fat, as assessed by magnetic resonance imaging, and a changed lipoprotein profile. Given that previous studies on fat storage have identified ANGPTL4 as an effector, we also investigated circulating levels of ANGPTL4, which proved to be higher in the F19-treated group. This increase, together with total body fat and triglyceride levels told a story of inhibited LPL action through ANGPTL4 leading to decreased fat storage. Co-culture experiments of colonic cell lines and F19 were set up in order to monitor any ensuing alterations in ANGPTL4 expression by qPCR. We observed that potentially secreted factors from F19 can induce ANGPTL4 gene expression, acting in part through the peroxisome proliferator activated receptors alpha and gamma. To prove validity of in vitro findings, germ-free mice were monocolonized with F19. Here we again found changes in serum triglycerides as well as ANGPTL4 in response to F19. Our

results provide an interesting mechanism whereby modifying ANGPTL4, a central player in fat storage regulation, through manipulating gut flora could be an important gateway upon which intervention trials of weight management can be based.

Bloomer, et.al. (2010) compared blood epinephrine (EPI), norepinephrine (NE), free fatty acids (FFA) and glycerol concentrations in response to a capsaicinoid supplement or placebo in healthy adults before and after acute exercise. Twenty subjects ingested a placebo or supplement (Capsimax, OmniActive Health Technologies; 2 mg capsaicinoids in a microencapsulated matrix) with one week separating conditions. Fasting blood samples were collected during each visit; 30 minutes following a rest period and before placebo or supplement intake (Pre); 2 hours post intake (2 hr); one minute following the cessation of 30 minutes of exercise performed at 65% of maximal heart rate reserve (2.5 hr); 90 minutes following the cessation of exercise (4 hr). Heart rate (HR), systolic (SBP) and diastolic (DBP) blood pressure were recorded at all times. A time effect was noted for HR, SBP, and DBP ($p < 0.05$), with HR and SBP higher at 2.5 hr compared to Pre (due to exercise) and DBP lower at 2.5 hr compared to Pre. No interaction or condition effects were noted for EPI, NE, FFA, or glycerol ($p > 0.05$). However, a time effect was noted for all variables ($p < 0.0001$), with values higher than Pre at 2.5 hr for EPI and glycerol, at 2 hr and 2.5 hours for FFA, and at 2 hr, 2.5 hr, and 4 hr for NE ($p <$

0.05). In terms of percent change from Pre, glycerol was higher with Capsimax than for placebo at 4 hr ($p = 0.011$) and FFA was higher with Capsimax than for placebo at 2 hr ($p = 0.025$) and at 2.5 hr ($p = 0.015$). Ingestion of low dose (2 mg) Capsimax was associated with an increase in blood FFA and glycerol at selected times post ingestion, as compared to placebo. However, Capsimax had no differing effect on EPI or NE compared to placebo. Lastly, no difference was noted in HR, SBP, or DBP between placebo and Capsimax.

Ramel, et.al. (2010) investigated the effects of weight loss and seafood consumption on inflammation parameters during energy restriction. In this 8-week intervention trial, 324 subjects (aged 20-40 years, body mass index 27.5-32.5 kg/m(2) from Iceland, Spain and Ireland) were randomized to one of four energy-restricted diets (-30% relative to estimated requirements): salmon (3 x 150 g/week, 2.1 g LC n-3 PUFA per day); cod (3 x 150 g/week, 0.3 g LC n-3 PUFA per day); fish oil capsules (1.3 g LC n-3 PUFA per day); and control (sunflower oil capsules, no seafood). Body weight, high-sensitivity C-reactive protein (CRP), interleukin-6 (IL-6), glutathione reductase and prostaglandin F2 alpha (PGEF2alpha) were measured at baseline and end point. Subjects experienced weight loss (-5.2+/-3.2 kg, $P<0.001$). Taken together for all subjects, there were significant decreases in all inflammation parameters. On a group level, salmon consumption was most effective, three of the four inflammation parameters decreased in the salmon group (high-sensitivity CRP=-

32.0%; IL-6=-18.4%; PGEF2alpha=-18.5%; all P<0.05). Cod consumption decreased high-sensitivity CRP and IL-6 (-21.5 and -10.8%, respectively, both P<0.05). Changes in the other two groups were not significant, which can be partly explained by the large s.d. The mean concentrations of inflammation parameters decreased during a period of weight loss anddietary intervention. In our study, salmon consumption was most effective, three of the four measured inflammation parameters decreased significantly in the salmon group.

Codoñer-Franch, et.al. (2010) evaluated the effect of supplementing a hypocaloric diet with mandarin juice, a food with a high content of antioxidants (vitamin C, flavonoids and carotenoids), on biomarkers of oxidant/antioxidant status of severe obese children. Forty obese children were randomized into two groups pair-wise in a 4-week controlled intervention study. Both groups followed a hypocaloric diet. One group received additionally a supplementation of 500mL of 100% mandarin juice daily. Clinical data, anthropometry, dietary intake and fasting blood samples were collected at baseline and after the intervention. Lipid peroxidation was assessed by circulating levels of malondialdehyde, and protein oxidation was determined by the concentration of plasma carbonyl groups. The antioxidant defence was evaluated by red cell-reduced glutathione and plasma levels of α-tocopherol and vitamin C. The supplemented group experienced a de =0.006) and an increase in antioxidants (α-tocopherol +16.1%, p=0.006, glutathione +36.1%, p =0.014)

and carbonyl groups (-36.1%, p crease in the levels of malondialdehyde (-9.6%, p< 94.6%, p + 0.0001, and vitamin C < The mandarin juice consumption with a reduced calorie diet positively affects the antioxidant defence and produces a decrease in biomarkers of oxidative stress in 0.0001).

Pal, et.al. (2010) documented that the health benefits currently associated with increased dairy intake may be attributable to the whey component of dairy proteins. The present study evaluated the effects of whey protein supplementation on body composition, lipids, insulin and glucose in comparison to casein and glucose (control) supplementation in overweight/obese individuals for 12 weeks. The subjects were randomised to whey protein, casein or glucose supplementation for 12 weeks according to a parallel design. Fasting blood samples and dual-energy X-ray absorptiometry measurements were taken. Seventy men and women with a mean age of 48.4 (SEM 0.86) years and a mean BMI of 31.3 (SEM 0.8) kg/m2 completed the study. Subjects supplemented with whey protein had no significant change in body composition or serum glucose at 12 weeks compared with the control or casein group. Fasting TAG levels were significantly lowered in the whey group compared with the control group at 6 weeks (P = 0.025) and 12 weeks (P = 0.035). There was a significant decrease in total cholesterol and LDL cholesterol at week 12 in the whey group compared with the casein (P = 0.026 and 0.045, respectively) and control groups (P < 0.001 and 0.003, respectively). Fasting

insulin levels and homeostasis model assessment of insulin resistance scores were also significantly decreased in the whey group compared with the control group (P = 0.049 and P = 0.034, respectively). The present study demonstrated that supplementation with whey proteins improves fasting lipids and insulin levels in overweight and obese individuals.

2.4 STUDIES ON PROTEIN SUPPLEMENTATION TESTOSTERONE

Banerjee, et.al. (2011) found that with the progressive aging of the human population, there is an inexorable decline in muscle mass, strength and function. Anabolic supplementation with testosterone has been shown to effectively restore muscle mass in both young and elderly men. In this study, we were interested in identifying serum factors that change with age in two distinct age groups of healthy men, and whether these factors were affected by testosterone supplementation. We measured the protein levels of a number of serum biomarkers using a combination of banked serum samples from older men (60 to 75 years) and younger men (ages 18 to 35), as well as new serum specimens obtained through collaboration. We compared baseline levels of all biomarkers between young and older men. In addition, we evaluated potential changes in these biomarker levels in association with testosterone dose (low dose defined as 125 mg per week or below compared to high dose defined as 300 mg per week or above) in our banked specimens. We identified nine serum biomarkers that differed between the young and older subjects. These age-

associated biomarkers included: insulin-like growth factor (IGF1), N-terminal propeptide of type III collagen (PIIINP), monokine induced by gamma interferon (MIG), epithelial-derived neutrophil-activating peptide 78 (ENA78), interleukin 7 (IL-7), p40 subunit of interleukin 12 (IL-12p40), macrophage inflammatory protein 1β (MIP-1β), platelet derived growth factor β (PDGFβ) and interferon-inducible protein 10 (IP-10). We further observed testosterone dose-associated changes in some but not all age related markers: IGF1, PIIINP, leptin, MIG and ENA78. Gains in lean mass were confirmed by dual energy X-ray absorptiometry (DEXA). Results from this study suggest that there are potential phenotypic biomarkers in serum that can be associated with healthy aging and that some but not all of these biomarkers reflect gains in muscle mass upontestosterone administration.

Naghii, et.al. (2011) determined whether acute (hourly or daily), and weekly supplementation could have any significant biological effects on the steroid hormones and further on some inflammatory biomarkers. Eight healthy male volunteers attended the laboratory on three occasions (days 0, 1 and 7). On the first day (day 0), a blood sample collection at 8.00 A.M was followed by ingestion of placebo with the breakfast. On the next day (supplementation-day 1), similar procedure was followed by ingestion of a capsule containing 10mg of boron. On both occasions blood was collected every 2h for the next 6h. Subjects were requested to consume a capsule of 10mg boron every day with their

breakfast, and on the day 7, the blood collection was carried out at 8.00 A.M, again. Boron in plasma increased significantly following hours and weekly consumption. Six hours supplementation showed a significant decrease on sex hormone binding globulin (SHBG), high sensitive CRP (hsCRP) and TNF-α level. After one week (in samples taken at 8.00 A.M, only), the mean plasma free testosterone increased and the mean plasma estradiol decreased significantly. Dihydrotestosterone, cortisol and vitamin D was elevated. Also, concentrations of all three inflammatory biomarkers decreased after supplementation. Of note, despite decreased proinflammatory cytokines, based on recent clinical data, this must be the first human study report to show an increase level of free testosterone after boron consumption.

Allan, and McLachlan (2010) documented that as testosterone levels are frequently reduced in obesity, an understanding of the relationship between serum testosterone and adiposity is necessary in the clinical evaluation of these men, in particular when considering testosterone therapy. Population and interventional data suggest a bi-directional relationship exists between testosterone and obesity in men, with lower total testosterone and sex hormone binding globulin (SHBG) (and to a lesser extent freetestosterone) levels than their non obese peers; obesity having an impact at least as important as ageing. Abnormalities in the hypothalamo-pituitary-testicular axis are seen with increasing obesity. Weight loss in massive obesity increases testosterone levels

but its role in mild-moderate obesity is unclear. Testosterone supplementation reduces total body fat in hypogonadal and ageing men although the effects on regional fat distribution are less well described. Favourable changes in total body fat and regional fat distribution suggest a potential role for testosterone in obesity. However, lifestyle advice to achieve sustained weight loss should be the mainstay of management. Obese men with confirmed androgen deficiency can be offered treatment, whereas in those with low-normal testosterone levels more research is needed.

Duschek , et.al. (2005) made a study on effects of raloxifene on IGF-1 levels and the associated increase in serum testosterone were compared to the effects of oral testosterone supplementation. Thirty healthy elderly men between 60 and 70 years received raloxifene 120 mg/day or placebo in a randomised double blind fashion for 3 months. Secondly, seven female to male (F to M) transsexuals undergoing hormonal sex reassignment received testosterone undecanoate 160 mg/day. Measurement: At baseline and after three months serum levels of testosterone, IGF-1 and its most important binding protein, IFGBP-3 was measured. In the group transsexuals also serum gonadotrophins and 17beta-oestradiol was measured. Compared to placebo raloxifene increased serum testosterone by 20% but it decreased serum IGF-1 levels by 24.5% (95% confidence interval (CI): -13.0 to -36.1%). No significant change in serum IGFBP-3 levels was found. The effect of raloxifene on serum

IGF-1 has been observed with other oral oestrogens, and, therefore, is likely to be ascribed to the partial oestrogen agonist activity of raloxifene. In the F to M transsexuals, serum testosterone levels increased from median <1.0 nmol/l to 6.2 nmol/l, without significant changes in serum gonadotrophins and 17beta-oestradiol levels. Serum IGF-1 levels increased by 12.1% (95% CI: 1.9-22.3%) versus baseline. No effect was observed on serum IGFBP-3 levels. Both raloxifene and oral testosterone increased serum testosterone, but raloxifene significantly decreased serum IGF-1 levels without affecting IGFBP-3. By contrast, oral testosterone supplementation in F to M transsexuals increased IGF-1 levels. In both treatment groups no significant change in serum IGFBP-3 was found.

Vermeulen, et.al. (1996) reported that several aspects of the regulation of androgen secretion and plasma levels in males remain controversial. Among these, we cite the problem of whether the age-related decrease in testosterone (T) levels is an intrinsic aging phenomenon or is a sequel of previous illness, the mechanisms underlying the increase in sex hormone-binding globulin (SHBG)-binding capacity in aging men and the supranormal capacity observed immediately after a weight-reducing diet, and the role of insulin in the age-associated decrease in dehydroepiandrosterone (sulfate) [DHEA (DHEAS)] levels. To gain further insight into these issues, we investigated the influence of age, smoking, body mass index (BMI), serum albumin, insulin, GH, and insulin-

like growth factor I (IGF-I) levels, respectively, on androgen levels and SHBG-binding capacity in a nonobese healthy population (n = 250) as well as in an obese population (n = 50) before and after weight loss. The influence of GHsupplementation on SHBG, DHEAS, DHEA, and insulin levels was studied in a small group of men (n = 8) with isolated GH deficiency. In nonobese healthy men, age was inversely correlated with serum levels of all androgens studied (although total T levels stayed relatively stable until age 55 yr) as well as with albumin, GH, and IGF-I levels and positively correlated with BMI, insulin levels, and SHBG-binding capacity. Nevertheless, SHBG levels were significantly negatively correlated with insulin levels ($P < 0.001$) as well as with mean 24-h GH and IGF-I levels. Among possible confounding factors affecting (free) T [(FT)] levels in healthy men, smoking appeared to be accompanied by higher (F)T levels than those in nonsmokers. BMI increased with age, but although BMI was negatively correlated with T, FT, and SHBG, respectively, the age-dependent decrease in T levels persisted after correction for BMI. Data not corrected for BMI may, nevertheless, overestimate the age-associated decrease in T levels. The albumin concentration decreased with age, and if FT is the feedback regulator of plasma T levels, albumin concentration might be a codeterminant (although, evidently, less important than SHBG) of T levels and contribute to the age-associated decrease in T levels. In any case, albumin concentration is a codeterminant of DHEAS concentration. T, DHEA, and DHEAS levels were

significantly correlated, but this correlation disappeared after controlling for age; hence, there is no evidence for an adrenal-gonadal interaction in men. In obese men, T, FT, and SHBG levels were significantly lower than those in the nonobese men and inversely correlated with BMI; DHEAS levels were slightly lower than those in the nonobese controls, but no significant correlation between DHEA or DHEAS, and insulin levels was observed. After a weight-reducing, protein-rich diet, resulting in a mean weight loss of +/- 15 kg, SHBG-binding capacity increased to normal values notwithstanding the fact that the subjects were still obese and that the insulin levels remained higher than those in the nonobese controls. Considering that after weight loss, GH and IGF-I levels remained lower than those in the nonobese controls, that adult men with isolated GH deficiency presented with higher SHBG levels than normal controls, which decreased to normal levels during GH substitution, and that elderly men have elevated SHBG levels notwithstanding high insulin levels, we suggest that the low GH and/or IGF-I levels might play a role in the elevated SHBG levels observed in both elderly males and obese men after a weight-reducing diet. As weight loss did not influence DHEAS levels notwithstanding an important decrease in insulin levels, our data do not support a role of insulin in the regulation of plasma DHEAS levels.

Mårin, et.al. (1993) reported that mdle-aged men with abdominal obesity were treated in a double-blind study with moderate doses of

transdermal preparations of testosterone (T), dihydrotestosterone (DHT), or placebo. This resulted in moderately elevated T concentrations and marked decreases in follicle stimulating and luteinizing hormones in the group treated with T, while the DHT group showed elevated DHT, markedly lower T values, and less diminution of gonadotropin concentrations. In the group treated with T visceral fat mass decreased (measured by computerized tomography) without significant changes in other depot fat regions. Lean body mass did not change. In the group treated with T, glucose disposal rate, measured with the euglycemic hyperinsulinemic clamp method, was markedly augmented. Plasma triglycerides, cholesterol, and fasting blood glucose concentrations as well as diastolic blood pressure decreased. There were no such changes in the DHT or placebo treatment groups. The men treated with T reported increased well-being and energy. In none of the groups did prostate volume, specific prostate antigen concentration, genito-urinary history, or urinary flow measurement change. It is suggested that supplementation of abdominal obese men with moderate doses of T might have several beneficial effects.

Kyung, et.al. (1985) demonstrated that during a 10-day fast in mildly obese men, urinary gonadotropin excretion significantly increased, and serum testosterone concentrations significantly decreased. The mechanisms by which these changes occur are unknown. We postulated that the mechanism of the gonadotropinuria might involve decreased proximal renal tubular reabsorption of

gonadotropins during fasting and might be related to renal tubular reabsorption of ketones during fasting, a process that is enhanced by carbohydrate (CHO) administration. We studied the effects of CHO supplementationon ketosis, ketonuria, and reproductive hormone secretion and excretion in 14 mildly obese men, 24-54 yr old, who were 14-69% above ideal body weight. Group I (n = 6) received no CHO supplementation, group II (n = 4) received 15 g CHO, and group III (n = 4) received 45 g CHO daily during the 10-day fast (F). During the control (C) and refeeding (R) periods, all subjects received a 1500-cal diet. Daily 24-h urine collections were made for the measurement of total ketones (millimolar concentrations) and LH and FSH (expressed as international units of the Second International Reference Preparation of human menopausal gonadotropin). Values (mean +/- SE) for 3 representative days (control day 3, fasting day 8, and refeeding day 3) for all subjects are shown below: (table; see text) We also studied the effects of CHO supplementation on serum levels of pituitary gonadotropins, LH and FSH responses to exogenous LHRH stimulation, biological activity of LH, and circulating total and free testosterone levels. Neither dose of CHO prevented the decline in total and free testosteronelevels. Serum LH concentrations, as measured by both the RIA and in vitro bioassay did not change significantly with fasting. Serum FSH concentrations in daily samples did not change significantly. The previously reported decline in the FSH response to LHRH stimulation with fasting was not prevented by CHO. We conclude that

CHO supplementationprevents the gonadotropinuria of fasting in men. The effect appears to occur in the kidney. The mechanisms may be related to that by which CHO promotes the renal tubular reabsorption of ketones. The reduced serum testosterone level cannot be explained by a lack of biologically active LH. It appears that fasting has a direct effect on the testis, possibly by reducing its responsiveness to gonadotropic stimulation or by inhibiting steroidogenesis.

2.5 SUMMARY OF RELATED STUDIES

In this chapter the researcher reviewed related studies pertaining to effects of cycling exercises on lipid profiles and testosterone and effects of protein supplementation on lipid profiles and testosterone of different groups. The reviews proved that there was further scope for research in finding out the isolated and combined effect of cycling exercises and protein supplementation on selected lipid profiles and testosterone among software professionals. Based on the experience gained, the investigator formulated suitable methodology to be adopted in this research, which is presented in Chapter III.