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Effects of Sprint Training on Athletic Performance Related Physiological, Cardiovascular, and Neuromuscular Parameters

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***Abstract*—**Practicing recurring resistance workout such as may cause changes in human muscle. These changes may be because combination if several factors determining physical fitness. Thus, it is important to identify these changes. Several studies were reviewed to investigate these changes. As a result, the changes included positive modifications in amplified citrate synthase (CS) maximal activity, increased capacity for pyruvate oxidation, improvement on molecular signaling on human performance, amplified resting muscle glycogen and whole GLUT4 protein content, better health outcomes such as enhancement in cardiorespiratory fitness. Sprint training also have numerous long long-term changes inhuman body such as better enzyme action, changes in muscle fiber and oxidative ability. This is important because SV is the critical factor influencing maximal cardiac output and therefore oxygen delivery and maximal aerobic power.

***Keywords*—**Sprint, training, performance, exercise.

I. Introduction

PERFORMING repetitive high-intensity exercise such as sprinting over numerous weeks or months induces great modifications in skeletal muscle. It was reported that the degree and trend of alteration in numerous variables depend on the characteristics of the working out protocol, rate of recurrence, concentration, recovery time, and length of sprint running [1] print interval training (SIT) could be defined "repetitive sessions of short, recurring bouts of extremely severe training". One of the important ways for achieving success today when undoubtedly the physical training. The basis of the physical training is to develop motoric features. The strength, among the motoric features, is the basic winning in sports has come to the forefront, is feature improving the success in many sports branch in the general sense. Today in many sports branch, it is desired to further improve the strength by implementing more strength works. The improvement of muscular strength depends on the content of well-planned and organized trainings [1]. Trainers and sports educators wish their athletes achieve success. For this purpose, they're seeking to find and implement new training methods in order to take the performance to an upper level [2]. Records regarding long term, short-term, and during exercise adaptations in muscle devotes further investigation; thus, this paper intended to review such adaptations [3].

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II. Literature Review

Many studies were carried out to investigate adaptations to sprint training. For example, [1] aimed to inspect the impact of smaller number of daily sprint training (SIT) sessions on muscle oxidative capability. It was found that a6 sessions of SIT training promoted recovery, and amplified citrate synthase (CS) maximal activity and endurance capability during cycling. On the other hand, control group's parameters did not change. Parallel to the findings of Parra et al., [4], our findings suggested that 2 weeks of (SIT) amplified citrate synthase (CS) maximal activity; however, it was indicated that training did not affect anaerobic capability probably due to persistent fatigue induced by continuous training program. Another major study findings reported that six sessions of sprint training interval performed for two weeks reduced skeletal muscle glycogenolysis and lactate accumulation during exercise [3]. No alteration in performance was reported for the control group.

While earlier studies found that there were positive adaptations after sprint training, [5] compared possible effects of low-volume SIT and usual endurance training (ET) on muscle oxidative capability. Different from previous studies, they have investigated metabolic adaptations throughout exercise and after two training methods. The proposed that similar adaptations would be after these two types of training strategies. SIT included four to six repeats of a 30 s 'all out' Wingate Test with 4.5 min recovery between repeats for 3 days per week while ET consisted of 40–60 min of continuous for 5 days per week. However, these two methods caused parallel augmentations. There were no differences between treatments. The findings of the study suggested that SIT training is a time-efficient approach to augment skeletal muscle oxidative capability and have potential to affect particular metabolic adaptations throughout exercise that are similar to traditional ET. A similar study also compared effects of SIT versus traditional endurance on human performance. Sixteen physically active subjects completed six exercise sessions completed in 14 days [6]. SIT group completed 30 seconds 'all out' cycling with 4 min recovery for six times while ET group exercised 90-120 min ongoing cycling. Training reduced the duration needed to finish trials; however, there were no differences between the treatment groups. The findings of the study showed there were

comparable augmentations in muscle oxidative capacity. It was also determined that there were similar muscle buffering capability and glycogen content between groups. Similar to the earlier study, the outcomes of the investigation suggest that SIT is a time-efficient strategy to produce since similar adaptations happened after both training modalities.

Effects of hypertrophic strength exercise on acute and long-term hormonal and neuromuscular adaptations were investigated in thirteen recreationally strength-trained men. A six-months of hypertrophic strength-training interval consisted of two-split three-month working out periods, a working out protocol of two minutes' rest as compared with a five-minute rest among the sets. This investigation showed that, within typical hypertrophic strength-training protocols used in the present study, the duration of the recovery times between the sets did not comprise an induce on the extent of acute hormonal and neuromuscular responses or long-term training adaptations in muscle strength and mass in previously strength-trained men [7].

While it was suggested to elucidate the impact of different acute exercise 'impulses' on molecular signaling on human performance, different from earlier studies, [8] reviewed molecular responses to high-intensity interval work out. It was proposed that from a cell-signaling view, short-time intense exercise is usually related with resistance exercise and connected to pathways that stimulate growth whereas short frequent sessions of high-intensity interval exercise training (HIT) stimulate rapid phenotypic alterations that being similar to habitual endurance exercise. The review reported that little amount of very intense interval work out, equal to only 2 min of all-out cycling, was adequate to augment PGC-1alpha mRNA during recovery. HIT raining was reported to rapidly raise the activity of signaling pathways connected to PGC-1alpha and mitochondrial biogenesis and HIT was reported to expand mitochondrial biogenesis and an augmented capacity for glucose and fatty acid oxidation. Cardiorespiratory Fitness such as HIT has been reported to increase stroke volume in cardiac failure patients and obese women, and improve cardiac output in middle-aged adults. This is important because SV is the critical factor influencing maximal cardiac output and therefore oxygen delivery and maximal aerobic power (i.e., cardiorespiratory fitness). Maximal stroke volume explains the difference between females and males, between trained and untrained individuals, between world-class endurance athletes and the average person. VO2 max and stroke volume may vary by a factor of three while HR and a-vO2 difference remain essentially the same between high fit and low fit subjects. The increase in SV has to do with the increased end diastolic volume (EDV) -or filling due to increased chamber size and greater ejection fraction due to increased hypertrophy and contractility of the muscle. Regarding myocardial hypertrophy is also an interesting effect. Aerobic exercise causes an elevated preload (EDV) whereas weight training elicits an increased afterload causing the heart to work against a greater pressure or resistance causing a thicker myocardium (concentric hypertrophy) than endurance trained (eccentric hypertrophy which elicits greater cavity dimensions). The extent of exercise induced adaptation of VO2max and cardiomyocyte function/ structure depends on exercise intensity [9]. High intensity exercise (at 85-90% VO2max) induces a hypertrophic response that is seen after a few weeks of training and reaches a plateau after ~ 2 months. This response is significantly greater than that induced by moderate intensity exercise at 60-70% VO2max) 2-3x greater. HIT training also improves the contractile capacity of the cardiomyocyte by increasing the extent and rates at which it shortens during systole and relaxes during diastole and by improving its ability to generate force. The effects of high intensity training are twice those of moderate intensity. Some other positive adaptations to HIT include an increase in endothelial function in obese adults and adolescents and adults with metabolic syndrome. Endothelial function is dependent on nitric oxide bioavailability. This response may be from the increased nitric oxide availability in response to HIT.

Potential mechanism to that low-volume high-intensity interval training induced mitochondrial biogenesis in human skeletal muscle was investigated [10]. The findings of the study indicated that training amplified exercise capability and maximal activity of CS and COX. Training also amplified resting muscle glycogen and total GLUT4protein content.

Another major study aimed to verify the efficiency of short intense interval exercise on health outcomes [11]. A 36 physically active subjects accomplished 12 weeks of intense interval running. Training modalities included either a 40 minute of total running per week, or about 150 min of prolonged running per week, and approximately 150 minutes of strength exercise per week while control group sustained their usual daily life without involvement in physical exercise. The enhancement in cardiorespiratory fitness was greater in the intense training group than two training groups. The blood glucose concentration was lower for both training groups after training. On the other hand, prolonged running led to better improvement for lowering the participants' resting heart rate, fat proportion, and dropping the ratio between total and HDL plasma cholesterol. Besides, intense training groups' total bone mass and lean body mass were not changed while the strength-training program led to an increase in these parameters.

While earlier studies presented in this paper investigated short-term effects of sprint training, [12] reviewed long-term adaptations of high intensity exercise. Enzyme adaptations represent a major metabolic adaptation to sprint training; however, detraining did not change enzyme levels. Glycolytic enzyme activity has been shown to augment after exercise. Large portion of studies recommends that these enzymes restore to pre-training levels after from 7 weeks to 6 months of detraining. Mitochondrial enzyme activity as well boost subsequent to sprint exercise, mostly when long sprints or short recovery. In addition to metabolic changes, morphological adaptations to sprint exercise consist of alterations in muscle fibre type, sarcoplasmic reticulum, and fibre cross-sectional area. A suitable sprint training could induce a modification to type II a muscle, augment muscle cross-sectional area and assist make available of Ca (2+). It

was also reported that training volume and/or frequency of sprint training is important criteria for these modifications. Detraining seems to cause change fiber type towards to type II b, even though muscle atrophy is also possible to take place. Adaptations from a sprint-training training and these modifications take a substantial amount of time to go back to baseline following a stage of detraining. Nevertheless, the complicatedness of the interface between different variables and with personal differences is obviously troublesome to the shift of knowledge and recommendation from laboratory to coach and athlete.

III. Discussion

Various studies that included high intensity spring training varying from 15 minutes to 60 minutes were investigated. All the studies reviewed in this paper revealed that sprint training caused less or more metabolic and morphological changes. Positive modifications in amplified citrate synthase (CS) maximal activity [1]. Amplified resting muscle glycogen and whole GLUT4 protein content, better health outcomes such as enhancement in cardiorespiratory fitness has been shown, Furthermore, resistance training that includes spring workout could have effect on adaptations in human muscle such as fiber type [12] and muscle oxidative potential and have potential to affect particular metabolic adaptations throughout exercise that are similar to traditional ET [5].

MCT proteins are considered to be important lactate transporters. Lactate transfer is augmented when muscle action is increased and persistent stimulus and lactate transfer declines with muscle inactivity. Lactate travels the cell membranes of various tissues via a facilitated MCT transport system. MCT are in all places circulated among many tissues, and the transcripts of numerous MCT exist within numerous of the same tissues. This complicates the detection of their metabolic task. MCT1 expression is greatly linked with the oxidative capability of skeletal muscles and with their ability to take up lactate from the circulation. MCT1 is also current in heart and is positioned on the plasma membrane, T-tubules, and in caveolae. With training, MCT1 is amplified in rat and human muscle, and in rat hearts, resultant in an augmented uptake of lactate from the buffers perfused through these tissues and an augment in lactate efflux out of purified vesicles. In humans, the training-induced increases in MCT1 are connected with an augmented lactate efflux out of muscle. MCT3-M/MCT4 is not associated with the muscles' oxidative capacities but is evenly rich in Type II a and II b muscles, while it is noticeably lesser in slow-twitch muscles [13]. Another important component of fitness is anaerobic performance. A study aimed to find out the association between anaerobic work ability (AWC) and anaerobic capability (AC). Twenty-five females performed a 'Wingate' test and two Critical Power tests all on separate days. Anaerobic capacity was defined as the overall work performed during the 30-second 'Wingate' test. Anaerobic capability and AWC were considerably associated. Also, there were no major differences between the test-retest means for CP or AWC. Test-retest correlations for CP and AWC were noteworthy. When considered in combination with earlier studies, the outcomes of this study indicated that the Critical Power test was an applicable and reliable method in exploring anaerobic capability as well as the maximal pace of fatigueless work [14].

"Oxygen debt" is one of the important subjects in fitness and "oxygen debt" hypothesis was a challenge to connect the metabolism of lactic acid with the O2 expenditure in surplus of resting that occurs after work out. The O2 debt was hypothesized to characterize the oxidation of a small portion (1/5) of the lactate created during exercise, to supply the energy to reconvert the remainder (4/5) of the lactate to glycogen during recovery. This hypothesis was modified to distinguish between initial, fast ("alactacid"), and second, slow ("lactacid"), O2-debt curve components. It was hypothesized that the rapid period of the post-exercise O2 utilization curve was due to the reinstatement of phosphagen (ATP + CP). O2 debt seemed to be too simplistic. Many studies on numerous species have provided proof indicating dissociation between the lactate removal and the slow section of the post-training VO2. The elevated concentration of lactate current at the conclusion of work out may be viewed as a "reservoir of carbon," which may operate as a source of oxidative ATP creation. As no absolute clarification of the post-training metabolism exists, it is suggested that the term "O2 debt" be used to express a set of phenomena during recovery from training. The terms "alactacid debt" and "lactacid debt," which propose a mechanism, are unacceptable. Use of alternative terms, e.g., "excess post-exercise oxygen consumption" (EPOC) and "recovery O2," will get out of consequence of causality in telling the rise in metabolic rate above resting levels subsequent to training [15].

IV. Conclusion

In summary, high-intensity exercise such as sprinting over several weeks or months bring about great metabolic and morphological improvements in human muscle; yet, the degree and trend of alteration in numerous variables depend on the characteristics of the working out protocol, rate of recurrence, concentration, recovery time, and length of sprint running.

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