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Overview of Psychophysiological Stress and the Implications for Junior Athletes

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Abstract Psychophysiological stress appears to be a significant parameter in youth competitive sport participation. Primarily because adolescent athletes are faced with the extreme pressurized scenarios, youth competitive sport offers. Numerous youth sport performers have not learned adaptive coping skills to ameliorate the effects of an inherently stressful environment. This oftentimes results in the elicitation of stress responses, which when prolonged cause an overproduction of hormones which can have severe negative psychological and physiological implications. The implications include a disruption of metabolism and cognitive functioning, as well as cell production in the immune system which may influence the course of chronic diseases and disorders. A detailed understanding of the molecular and genetic events underlying the association of stressors with the psychophysiological pathways is crucial for the design of psychological interventions tailored specifically for athletes so as to improve well-being and performance.

Keywords: *psychophysiological stress, youth competitive sports, athletes, stress responses, hormones*

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1. Introduction

For many, sport participation represents leisure activities which promote social, mental and physical well-being. Youth' competitive sport participation, especially among adolescents who participate in elite sports however can offer an entirely different set of circumstances. For them it is a great challenge, dealing with extensive training schedules and large number of competitions, replete with stressors and constraints. Stress is inherent and endemic in youth competitive sport participation. Primarily, due to the high demands and extremely pressurized scenarios adolescent athletes face, from the colossal mental and physical investments they put into sport preparation and competition.

Studies have shown that much emphasis has been placed on competitive success in youth sport participation [1,2,3,4]. This has resulted in young athletes engaging in intensified training regimes, to optimize performance. It has been observed that some athletes as young as 10 years undergo 15 - 20 hours of intensified training per week. This excessive and intensive training, oftentimes leaves athletes with little to no opportunity for recovery. Young athletes also couple sport participation with mandatory full-time academics, which can be very time-consuming. These athletes are deprived of adequate recovery periods needed for the body to rest, so as to adapt to the stress during intensified training sessions and competitions. Recovery is very important among athletes to reduce the risk of injuries and "overtraining syndrome" [5]. Even more so among young athletes who are susceptible to a variety of traumatic and overuse injuries, due to the youth period which is characterized by increased growth velocity and growth plate closure [3,6]. All these factors can lead to total overloading of the athlete, which can cause psychological and physiological consequences.

Additionally, youth athletes face specific stressors relating to competition. These include stressors experienced before, during and after competitions [7]. Stressors such as extensive time demands in preparation for competition and inadequate or arduous mental, physical and technical preparation prior to competition [8,9]. Various aspects of competing such as rivalry, meeting the expectations of people, competing for a better ranking and competing in front of spectators are also, regarded as stressors among youth sport performers [10]. Moreover, youth sport performers face an array of stressors from environmental demands within the sporting organization they operate from [11,12]. These include stressors associated with the training environments, being away from families for training/competitions and the difficulties associated with balancing sport and non-sport commitments. Behavior of coaches and styles such as the increased pressure on an athlete to win and poor teaching skills can often create a

negative impact on the youth athlete. In addition to the coaches, parents can be a stressor to child, when they set unrealistic goals and expectations for the maturing child [11]. The interaction of these multiple stressors present unique problems in youth sport participation.

An athlete' reaction to the array of stressors depends on how he/she appraises the potential threat value associated with sport participation followed by availability of coping resources. While, some adolescent athletes may appraise the threat value of sport participation and are able to cope, other adolescents have not learned adaptive coping strategies which may ameliorate the effects of an already inherently stressful environment [13]. Thus, for youths, sport participation elicits severe, prolonged stress responses, which Selye implied may cause tissue damage and diseases [14]. Ideally, the stress response systems were designated to be transient so as to maintain homeostasis. Prolonged activation of the stress response systems can have deleterious effects due to overproduction of hormones, ultimately affecting the entire metabolism, including the rate of cell growth and repair as well as cell production in the immune system [15]. These may have deleterious effects on the well-being, behavior and performance of the athlete and therefore, put the athlete at high risk for detrimental consequences.

Research on stress reactivity and health outcome assumes often, that chronic prolonged stress responses predict poor health outcome. Indeed, there are some evidence that this is true particularly in the areas of cardiovascular diseases, immunity disorders, muscle degeneration, depression, anxiety disorders and the disruption of sleep pattern. Very few studies are able to explain the effects of youth' competitive sport participation and the elicitation of stress responses. In this review, focus is on the psychophysiological pathways exerted from stressors from youth competitive sport participation and the consequences of these stressors.

2. The Stress Response Systems

Stress is defined as a psychophysiological disturbance of the body's homeostasis by physical, mental or emotional demands that often result in changes to the internal milieu of an individual [16]. This disturbance to homeostasis is counteracted by the complex interaction of physiologic and behavioral responses with intent to maintain optimality of equilibrium in the body of the threatened individual. The stress response systems depend on a highly interconnected networking of the nervous and endocrine systems along with cellular and molecular infrastructures which translate stressful stimuli into coherent biological responses. Dysregulation, however of the adaptive response system due to chronic stress can have severe negative effects and has been linked to the pathophysiology of various disorders [15,17].

Initially, exposure to stress stimulates the primary effectors in the brain which have critical, distinct roles in orchestrating stress responses. These effectors are situated mainly in the hypothalamus, amygdala and the brainstem and are highly interconnected. The paraventricular nucleus (PVN) of the hypothalamus contains neurosecretory cells that synthesize corticotropin-releasing hormones (CRH) and arginine-vasopressin (AVP). These neurosecretory cells send axons to the median eminence of the hypothalamus where the axon terminals secrete CRH and AVP into the portal circulation of the pituitary stalk. From there, the CRH are carried to the anterior pituitary gland, where the CRH regulate the cleavage of the precursor protein, proopiomelanocortin, into the opioid, beta-endorphin and the pituitary hormone adrenocorticotropin (ACTH); the action is augmented synergistically by AVP [17,18]. The ACTH is released into the systemic circulation whereby it travels to the adrenal gland to trigger an increase in the rate of production and release of cortisol into the systemic circulation. The dynamic is slow, since new cortisol needs to be synthesized after each trigger causing a delay in the final effector response [19].

There also exists an immediate stress response via activation of the autonomic nervous system (ANS) [20]. Exposure to stressors results in an immediate increase of catecholamines via the CRH neurons from the PVN that extends to the brainstem to act on the paragigantocellular and parabranchial nuclei of the medulla, as well as the locus coeruleus (LC) to initiate activation of the sympathetic nervous system as to release nor-epinephrine (NE) [21]. In addition to that, exposure to stressors result in the activation of the preganglionic sympathetic neurons in the intermediolateral cell column of the thoracolumbar spinal cord [20]. The preganglionic neurons extend to the pre- or paravertebral ganglia, which in turn projects to end organs and to the chromaffin cells of the adrenal medulla glands, which are responsible for secreting epinephrine (EPI) and nor-epinephrine (NE).

Once cortisol is released in the bloodstream, it binds to the mineralocorticoid receptors (MR) and glucocorticoid receptors (GR) found in most tissues. Cortisol permits normal metabolic and diurnal functions through MR and regulates the response to stress via the GR. It is able to exert its action due to it being water and lipid soluble hence easily crossing the blood-brain barrier and cell membranes as to bind to receptors found in the intra-cellular fluid [17]. On the other hand, EPI and NE act on target peripheral tissues with the alpha and beta-adrenergic receptors [15]. On release from the adrenal medulla, they circulate in the blood to target sites but are unable to cross the blood-brain barrier. Despite, this EPI and NE can impact the brain through their actions on sensory vagus nerve outside the blood-brain barrier carrying information transmitted into the brain via the nucleus of the solitary track.

3. Implications and Effects of the Stress Response Systems in Sport Participation

3.1. Physiological Implications

Youth sport participation is permeated with numerous psychosocial stressors, whether real, imagined, anticipated, or recalled. All working through the cognitive appraisal mechanisms to elicit a stress response [22]. Following the perception of stressful events, a cascade of changes occurs in the nervous, endocrine, cardiovascular and immune systems. These changes are constituted mainly by cortisol, NE and EPI, which are the primary end point effectors of

stress responses. Generally, the changes are adaptive in nature, when released for normal homeostasis and are transient. For example, in youth sport participation a stress response is elicited due to anticipatory effects of sport competition and stress during exercise, where the primary end point effectors are released acutely [23,24]. This occurs to prepare athletes for the competition, by shifting energy substrates from storage sites into the bloodstream. In doing so the actions of cortisol, NE and EPI inhibit glucose uptake, protein synthesis and the mobilization of fatty acids while stimulating the release of energy substrates, which include glucose, free fatty acids and amino acids, from muscles, fat tissues and liver. This leads to the allotment of crucial energy sources to tissues that become more active during stress to overcome the stressors. The end point stress effectors also stimulate pulmonary and cardiovascular function, by increasing respiration, heart rate and blood pressure [25]. Additionally, immune cells are moved to places in the athlete's body that might need them to fight potential pathogens. Less critical activities such as digestion and the production of growth and gonadal hormones are suspended during this time [26].

The aforementioned energetics and associated behaviors carried out by the end point effectors in response to a changing environment are referred to as the allostatic state [27]. In sport participation, this state tends to balance the body's energy needs, from the cumulative costs associated with the stressors. As soon as the stressors cease the stress response systems are restrained by exertion of negative feedback particularly at the pituitary, hypothalamus and the brainstem [15,17,28]. Cortisol slows the rate of proopiomelanocortin synthesis and inhibits the production of CRH. In contradiction, NE and EPI are not regulated by negative feedback but instead by the central nervous system (CNS), where NE and EPI inhibit catecholaminergic LC/NE neurons' secretion of catecholamines at the brainstem [15].

During continuous activation of the stress response systems, which happens quite often in youth competitive sport participation, athletes are not able to cope with frequent stressors and the PVN releases CRH along with AVP. This hormonal combination stimulates approximately three times the release of ACTH from the pituitary [17]. This ultimately, leads to higher release of cortisol from the adrenal cortex into the systemic circulation. This higher level of cortisol from continuous stress activation causes GR resistance by blocking cortisol from binding, similarly, to the mechanism seen in insulin-resistant diabetes [29]. In addition to the impaired binding of the GR, excess cortisol causes a diminished negative feedback sensitivity, because CRH-AVP neurons respond ten times less to cortisol feedback than CRF-only fibers [30]. This negative feedback inhibition, blocks the regulation of cortisol causing the hormone to be kept at elevated levels, causing adverse effect on major organs. Although NE and EPI are short lived, once released after stress, they can have adverse effects in continuous stress activation. The hemodynamic effects cause frequent rises in blood pressure that accelerate atherosclerosis which is a prime risk factor for myocardial infraction [31] and mal functioning of the immune system [32].

3.2. Cognitive and Behavioral Implications

Alongside the hypothalamus and the brainstem, other limbic regions such as the hippocampus, amygdala and prefrontal cortex are all interconnected to effect a stress response. The hippocampus is an essential neuronal component for memory storage processes and the encoding of context during fear conditioning [33,34]. Whereas, the amygdala is involved in the processing of threat-related stimuli and is necessary for the assessment of fear conditioning [34,35]. While the prefrontal cortex functions to regulate emotions, decision-making and memory [27,34]. These limbic structures act together during an appraisal process, to ultimately evaluate a perceived threat, then shape the decision outputs through the HPA and brainstem during stress response processes which influence endocrine end point effectors secretion [17].

Interestingly, the secreted stress endocrine end point effectors are not only necessary to meet energy demands, but also feedback to the CNS to entrain learning processes, memory consolidation and cognitive performance. Studies have explained that the effects of cortisol are due to the presence of adrenocortical receptors (MR and GR) in the hippocampus, prefrontal cortex and amygdala [36,37]. During mild stress related processes cortisol feedback to the CNS fully occupies the high-affinity MRs while, the widely dispersed low-affinity GRs are occupied only intermediately, implicating the effect of acute stress on cognition and behavior. Chronic levels of cortisol however, cause cognitive defects due to spontaneous feedback of the catabolic hormone on the forebrain region [34]. Elevated cortisol tends to overload the low-affinity GR receptors in the forebrain structure causing temporary reduction in long term potentiation, a process underpinning impaired memory [38]. Cortisol also suppresses hippocampal and amygdala neurogenesis by the shrinkage of dendrites that interact with various neurotransmitters, especially glutamate, which modulates neuroplasticity causing atrophy of dentate gyrus neurons in both hippocampus and prefrontal cortex [39,40].

4. Consequences of Chronic Stress

Elicitation of the stress response results in physiological and psychological changes, which allow individualistic homeostatic balance with the environment. Continuous or chronic activation of the stress response can exact a cost due to overexposure of the stress hormones- allostasis load which can accelerate diseases [41]. Ultimately, an individual vulnerability to the stress related trajectories is dependent on several factors which include: genetics, biobehavioral and environmental that interact to influence an individual' ability to cope [27]. An individual' coping response that proceeds stress perception and appraisal depends on the extent to which the individual has control over the given stressor and support systems in place for dealing with stressors [42,43]. As described above youth competitive sport performers can be faced with numerous and continuous stressors that cause chronic elicitation of stress hormones, which have major impact on the body physiologic and psychologic processes. The impact stressors have on these processes can lead to the

pathogenesis of diseases and disorders, if athletes are not able to cope with the potential stressors. A few of the stress related diseases/disorders are outlined below to underscore the growing evidence showing the association and impact on youth competitive sport.

Integrating studies from psychology about stressors and sport competitive participation, with cellular, molecular and biologically relevant physiologic pathways, as done in studies above, have allowed researchers to unravel the complex interplay of stress response systems in the manifestation of certain physiologic and psychologic consequences. These consequences can be extremely detrimental to athletic performance. Stress can alter physical, emotional and cognitive performance. Below, the significance of findings regarding the consequences of chronic stress, were critically examined and placed into the perspective of athleticism. Understanding these relationships can pave ways for improving the well-being of athletes as well as tailoring interventions which could improve athletic performance.

4.1. Common Mental Disorders (CMDs)

Symptoms of anxiety, depression or adverse substance use are typically referred to as common mental disorders (CMDs) [44]. The pathophysiology of the CMDs, specifically, anxiety and depression stemmed from defects or anomalies in the positive regulation and counter-regulation of the main components of the adrenocortical and adrenergic systems, respectively [28,45,46]. Nevertheless, defects or abnormalities in the regulation of adrenocortical and adrenergic systems, result in hypersecretion of CRH and central nonandrogenic hormones in individuals exposed to chronic continuous stressors [47,48]. The hypersecretion of these hormones tends to cause structural degeneration in the brain and reduced functioning of hippocampus, amygdala and the prefrontal cortex, areas with responsibilities for the assessment of threats, fear conditioning and emotions [34,49].

Although physical activity has favorable effect on mental health, surprisingly, the athletic population is frequently subjected to physical and mental stressors that can possibly cause abnormalities in stress response. Extant research has shown that the prevalence rate of CMD in the athletic population is at an alarmingly high rate. For example, cross-sectional studies among French and Australian Olympic athletes and footballers from the World Player Union, have shown the prevalence rate of CMD to be in the range of 17-45% [50,51,52]. The prevalence rate of the symptoms of the CMDs are most frequently reported in young adults (aged 16 – 34 years) than in any other age groups [51]. Youth competitive sport performers fall within this age range and are exposed to a plethora of stressors when compared to non-athletes. Yet still there are hardly any data regarding the symptoms of CMDs on young athletes. The few studies done have shown that CMDs negatively correlates with athletic performance [53,54].

4.2. Disruption of Sleep Pattern

Stressors associated with youth sport participation can lead to a disruption in the athlete' sleep pattern. This is because stress activates the neuroendocrine system, in particular the HPA and sympatho-adreno-medullary (SAM) axes which influence cortisol, ATCH, CRH and catecholamine hyperactivity [15,17]. These hormones are associated with arousal and alertness. Several studies exist linking these secretions to sleep behavior [55,56]. The neuroendocrine system is under the influence of a circadian rhythm controlled by the central clock located in the suprachiasmatic nucleus of the hypothalamus [15,57]. This causes a waveform secretion pattern of the stress end point effectors. There is a steady rise of cortisol into the waking hours, a peak after waking, and then a gradual decline throughout the day, until cortisol reaches a nadir during the first few hours of nighttime sleep [57,58]. Low levels of cortisol in the evenings and nights are associated with MR binding, while higher level activates the GRs along with NE, which preferentially increases CRH [59]. Nighttime awakening is related to the episodic release of cortisol, NE and CRH.

The onset of sleep occurs when the HPA axis activity is lowest, conversely sleep deprivation is associated with HPA activation, which is the case with stressed individuals. Since, HPA activation releases hormones associated with wakefulness, this tend to hamper a stressed athlete from adequate sleep. Sleep is important among athletes so as to facilitate fast recovery [60]. During sleep muscle growth occurs, the body responds to the micro-damage within muscle tissues and to the proteins within the muscles. This is done by sending signals to the brain that initiates an inflammatory response, through the activation of anti-inflammatory agents at the damage sites to remove damage cells and tissue and to begin the repair [61]. Also, growth factors are released during sleep, for new muscle formation and protein replacement. Thus, it is important to reduce stress among athletes especially young athletes, as to improve sleep pattern, to enhance muscle development and reduce the overtraining syndrome.

4.3. Immunity Disorders

Stress has been regarded as immunosuppressive. Immune functions can be altered by end point effectors (cortisol and catecholamines), released from, the two major stress response systems. One mechanism by which this occurs is facilitated by the presence of stress end point effector receptors on antigen presenting cells (APCs) such as lymphocytes, monocytes/macrophages, granulocytes and dendritic cells. The primary receptors of cortisol and catecholamines are GR and adrenoreceptors (α- and β-adrenergic receptors), respectively. Cortisol is lipophilic thus passes through the plasma membrane of the target cells (cells with GR) during high circulating levels [17]. While, catecholamines mediate the action on the target cells, specifically, through the intermediaries' function of β2-arenergic receptors on transmembrane, through a signaling pathway that involves G-proteins and effectors [15].

The immunosuppressive action of cortisol acts through it receptors on the APCs which are components of innate immunity so as to suppress the production of interleukin-12 (IL-12), which is the main inducer of T helper 1(Th1) response [34,62]. The IL-12 is extremely effective in

enhancing interferon (IFN-γ) and inhibiting IL-4 synthesis by T cells, representing the action by which cortisol affects the T-helper 1/ T-helper 2 (Th1/Th2) balance [63]. Cortisol downregulates the expression of IL-12 receptors on T and natural killer (NK) cells [64]. Similarly, catecholamines suppress the synthesis IL-12 and increase the production IL-10 [65]. This causes a shift in the phenotype of $CD4^+$ T helper cells, from a Th1 profile, to a Th2 profile. That is a shift from cell-mediated immune activated profile, to a profile which involves antibody production [66]. Another proposed mechanism of the immunosuppressive action of cortisol results from the inhibition of the activity of transcription factors (TF), activator protein 1 (AP-1) and nuclear factor-kappa B (NF-κB) [64]. This results in the repression of proinflammatory and immunoregulatory genes and cytokines causing inhibition of tumor necrosis factor (TNF-α) and interleukin-1 [67]. Both mechanisms result in a shift from type 1 towards type 2 immunity. The clinical implication of the shift from type 1 to type 2 immunity has been shown to increase the susceptibility to infection as well as duration of, once established [68].

Athletes from the very nature of their occupation, are exposed to immune modulation from both physical and psychological stressors. Stress induces changes in the balance of the immune system that increases the chance of injury among elite athletes, especially those that couple athleticism with academia [69]. These youths are often left with little or no opportunity to recover from excessive and long hours of training because they want to improve their physical performance. With excessive exercise training there is a possibility of high level of musculoskeletal loading which can result in tissue damage [70]. This tissue damage in turn results in local and systemic inflammation and the activation of an immune system response. This coupled with the psychological stressor cause the release of cortisol and catecholamine which shift the immune balance from type 1 towards type 2 immunity. This results in infections which prolong the duration of the injury and increases the susceptibility of mucosal infections such as upper respiratory tract infection and gut infection due to increased gut permeability [69]. Physical and psychological stressors have similar consequences on the immune system thus efforts must be made to reduce the cumulative and synergistic effects among the very vulnerable youth competitors in sport.

4.4. Muscle Degeneration

Long term exposure to stressors can cause chronic systemic levels of stress hormones, such as cortisol which has catabolic effects. Increased cortisol even within physiologic range decreases protein synthesis and increase proteolysis [71]. This happens to generate an increase in free amino acids thereby providing a rich source of carbon for hepatic gluconeogenesis which aid in maintaining blood glucose level so as to support brain function under stressful conditions. While, important as a source of metabolic energy, chronic elevation of cortisol induces rapid muscle breakdown and proximal atrophy. The mechanism of cortisol muscle atrophy involves a decrease in total protein synthesis rate, inhibition of actinin in all muscles and a down regulation of the myosin heavy chain in fast twitch muscles, these are the most important contractile protein in muscles [72,73]. This action exerted by cortisol is associated with a decrease in muscle protein synthesis leading to muscle atrophy.

There is a dearth in the literature, regarding over exposure of stress induced systemic level of cortisol on muscle thickness and weakening in the athletic population. Some studies have looked at older people or used the blood biomarker (creatinine kinase) [74] for analysis. These studies have revealed that high cortisol is related to loss of grip strength and is augmented by genetics [75]. Hitherto, a search of the literature revealed no study looked at the effect of cortisol on muscle thickness and weakening among youth competitive sport performers. Despite this group still in their growth phase, they are being exposed to the cumulative cost of both physical and psychological stressors, associated with competing in elite sport. Therefore, it is worthwhile for researchers to examine the effect of cortisol on muscle thickness and strength/weakening in the youth population. A less invasive method such as ultrasonic technology, used to track lean body mass [76] can be the employed method to study the effect of cortisol on muscle thickness and weakening.

5. Summary and Conclusion

Psychophysiological stress is a significant parameter in youth competitive sport participation. This is due to youth sport performers consistently being under the influence of the cumulative effects of both psychological and physiological stressors associated with an inherently stressful sport participation. The stressors associated with youth competitive sport participation stem from: sporting preparations, competitions and the sporting organizations, all of which are oftentimes combined with personal stressors. Depending on the availability of coping resources these stressors tend to elicit stress responses resulting in the releasing of CRH and the stress end point effectors (cortisol, EPI and NE). These end point stress effectors when released transiently and in optimality, move energy sources from storage sites to sites in the body that may need the energy during the time of stress to combat the stressors. Stress end point effectors also, play a crucial role in cognition and behavior by enabling the assessment of threat related stimuli and emotional memories necessary for coping during periods of stress. Chronic prolonged activation of the stress response systems which is quite frequent among youth sport performers without effective coping skills, can exact severe consequences leading to the pathogenesis of diseases and disorders. Some consequences of chronic stress, which might be detrimental to athletic performance are: CMDs, disruption in sleep pattern, immunity disorders, muscle degeneration and cardiovascular diseases. While the aforementioned is known, not many psychophysiological research, with consistent findings are available within the literature. Therefore, the findings outlined in this review are with the intent of providing the basis for stimulating understanding of the interactions of stressors among the psychologic, physiologic and behavioral pathways. Understanding these associations can open avenues necessary for the

improvement of the well-being of youth sport participants, which could ultimately improve athletic performance.

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