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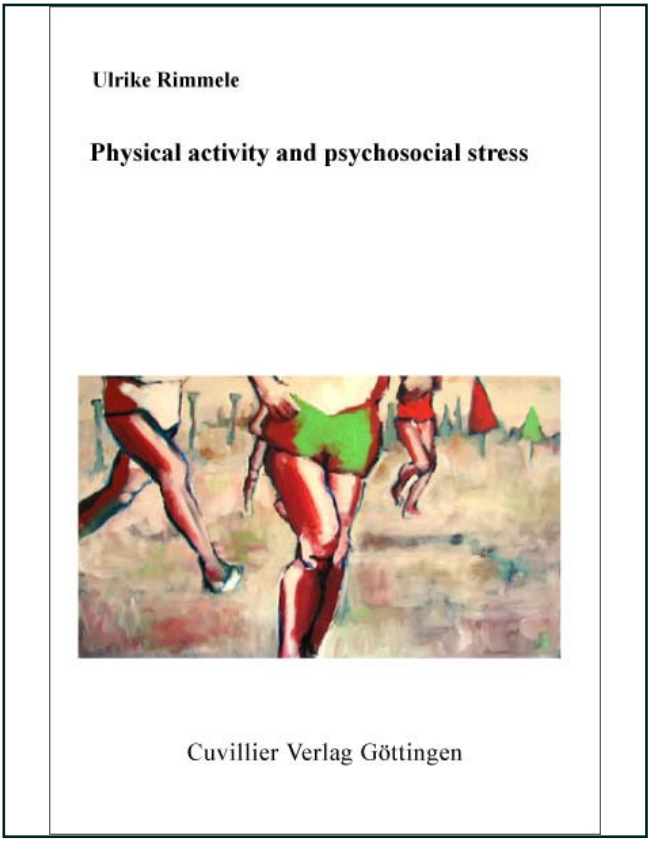
Physical activity and psychosocial stress



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1. Introduction

“Mens sana in corpore sano”. “In a healthy body is a healthy mind“. This world famous quotation by Decimus Iunius Iuvenalis, a Roman orator and poet, shows that 2000 years ago people had already recognized a connection between the body and the mind. Today, the disciplines of psychology and neurosciences are examining the nature of this connection. There is growing scientific evidence that physical activity not only keeps the body healthy, but also the mind. For example, research findings show that physical activity is beneficial for the cardiovascular system, in that it positively influences for instance hypertension or coronary morbidity (Steptoe et al., 1993; Talbot et al., 2002; Ketelhut et al., 2004; Barlow et al., 2006). In addition, it has consistently been shown that regular exercisers show lower anxiety and depression levels (Ross and Hayes, 1988) and that physical activity improves mental health disorders such as depression (Blumenthal et al., 1999; Babyak et al., 2000; Nabkasorn et al., 2006). Furthermore, several studies have documented reduced susceptibility to the adverse influences of life stress in physically active people (Tucker et al., 1986; Steptoe et al., 1989; Throne et al., 2000).

Stress is a risk factor for the development of cardiovascular diseases such as hypertension, and for the development of psychiatric disorders such as depression (McEwen, 2000b; , 2002; Vanitallie, 2002). Since stress-related diseases cost a considerable amount to economy and healthcare systems (Kalia, 2002; Miller and O'Callaghan, 2002), potential protective factors against the development of stress-related disorders are sought. Physical activity, with its beneficial effects on physiological stress systems, has long been proposed as such a protective factor against stress and stress-related disorders. For example, it has been suggested that by moderating stress-related hemodynamic reactions, physical activity influences hypertension (Perkins et

al., 1986). However, cross-sectional as well as longitudinal studies, which sought to determine the influence of physical activity on the cardiovascular, hormonal and psychological response to psychological stress, reported inconsistent findings. For example, some studies reported alleviated reactivity or a more rapid recovery of heart rate following psychosocial stress in trained men (Sinyor et al., 1983; Holmes and Roth, 1985; Sinyor et al., 1986; Crews and Landers, 1987; Moya-Albiol et al., 2001; Spalding et al., 2004), while others found no such effects, or even reported higher reactivity in physically trained groups (de Geus et al., 1993). Furthermore, some studies reported lower levels of fitness to be associated with an augmented norepinephrine response to stress (Sothmann et al., 1991; Moyna et al., 1999), while others reported no significant effect of physical activity on norepinephrine and epinephrine response to stress (Brooke and Long, 1987; Claytor et al., 1988; de Geus et al., 1993). Still others showed higher norepinephrine levels in trained subjects early on in the stress period (Sinyor et al., 1983). Considering hypothalamic-pituitary-adrenal (HPA) axis reactivity to psychosocial stress, studies reported no significant effects of physical activity on cortisol levels (Sinyor et al., 1983; Moyna et al., 1999).

It is possible that the heterogeneous findings between studies are due to differences in subjects' physical activity levels, and differing methodologies between studies. Because of this, it has not been possible to make conclusive statements about the relationship between stress reactivity and physical activity.

The intended goal of this dissertation was to resolve some of the open debates in the literature regarding the association between stress reactivity and physical activity. We set out to determine whether physical activity has positive effects on psychological and physiological stress responses to a

psychosocial stressor. Therefore, we compared subjects at the opposite ends of the physical activity spectrum, i.e. elite sportsmen relative to untrained men. We further expanded the measurements of stress reactivity beyond previous studies, by including cardiovascular and endocrine, as well as psychological parameters. The findings of the first study showed that elite sportsmen exhibit lower stress reactivity compared to untrained men.

Based on these findings, a second study was conducted to determine potentially influencing factors. We were interested in the influence of various physical activity levels on stress reactivity and a possible dose-dependency of physical activity on psychosocial stress reactivity. Thus, we included subjects of different physical activity levels (i.e. elite sportsmen, amateur sportsmen, and untrained men). Furthermore, the second study aimed at investigating the influences of personality traits or appraisal and coping strategies on stress reactivity. Several studies have shown that personality traits, appraisal and coping strategies influence reactivity to psychological stressors (Harrison et al., 2001; Gaab et al., 2005). Sportsmen have been found to exhibit different levels of personality traits (e.g., competitiveness), which have been found to influence stress reactivity (Jones and Swain, 1992; Houston et al., 1997; Frederick, 2000). We therefore tested the influence of determinants of stress reactivity, such as personality traits and appraisal and coping strategies on stress reactivity.

The first section of this dissertation will discuss current theories on the biological and the psychological underpinnings of stress. Furthermore it will describe the state of research regarding the relationship between physical activity and psychological stress. The second section will then describe the two studies in detail. The last section of this dissertation discusses the findings of these studies and provides an outlook for prospective studies.

PART I Theoretical Background

2. Stress

Walter B. Cannon originally developed the concept of stress (Cannon, 1914) as a term for the physiological reaction due to a stressor (i.e., an aversive or threatening situation). In the middle of the 20th century, the term stress was again described by Hans Selye (Selye, 1933; Selye and Fortier, 1949; Selye, 1985). Both Cannon and Selye developed a biological stress concept, which focuses on a person's reaction to a stress-provoking stimulus. In contrast, later-developed psychological concepts of stress focus on the subjective experience between the pressure to react and the options for adaptive reactions (Mason, 1975; Lazarus, 1984; Folkman, 1997). Thus, a stress-provoking situation might be stressful for someone, while the same situation is experienced as controllable by someone else.

2.1. Biological and psychological stress concepts

According to Hans Seyle's pioneering work in stress research, stressors lead to a rise in hypothalamic-pituitary-adrenal (HPA) axis activity resulting in heightened levels of glucocorticoids (GCs) (Selye, 1950). This finding was complemented by Walter Cannon's idea of an "emergency activation" of the sympathetic nervous system (SNS) leading to a fight or flight reaction (Cannon, 1914). Both Selye, as well as Cannon, postulate that the reaction is independent of the nature of the stressor. No matter whether the stressor is a noxious agent, an injury, social threat or other, it always induces the same reaction (i.e. activation of the sympathetic nervous system causing heightened secretion of epinephrine through the sympathoadrenergic system (SAM) (Cannon, 1914) and a rise in glucocorticoids mediated through HPA axis

activity (Selye, 1950). Thus, Cannon and Selye concluded that the stress reaction is unspecific (Selye, 1933).

According to Miller et al. (2002) this stress-induced activation of the SNS and the HPA axis results in a series of neural and endocrine adaptations known as the "stress response" or "stress cascade" (Miller and O'Callaghan, 2002). Sapolsky describes the endocrine stress reaction occurring in two waves (Sapolsky et al., 2000). The first wave happens within seconds and includes (1) activation of the sympathetic nervous system, resulting in increased epinephrine and norepinephrine secretion, (2) hypothalamic release of corticotrophin releasing hormone (CRH), and (3) seconds later, an increased secretion of adrenocorticotropic hormone (ACTH). The second wave has a time course of minutes and involves changes in steroid hormonal levels, such as increased secretion of glucocorticoids (Sapolsky et al., 2000). An overview of these stress systems is given in Fig.1. Stress reactivity of the SNS and of the HPA axis will be described in more detail in the following chapters.

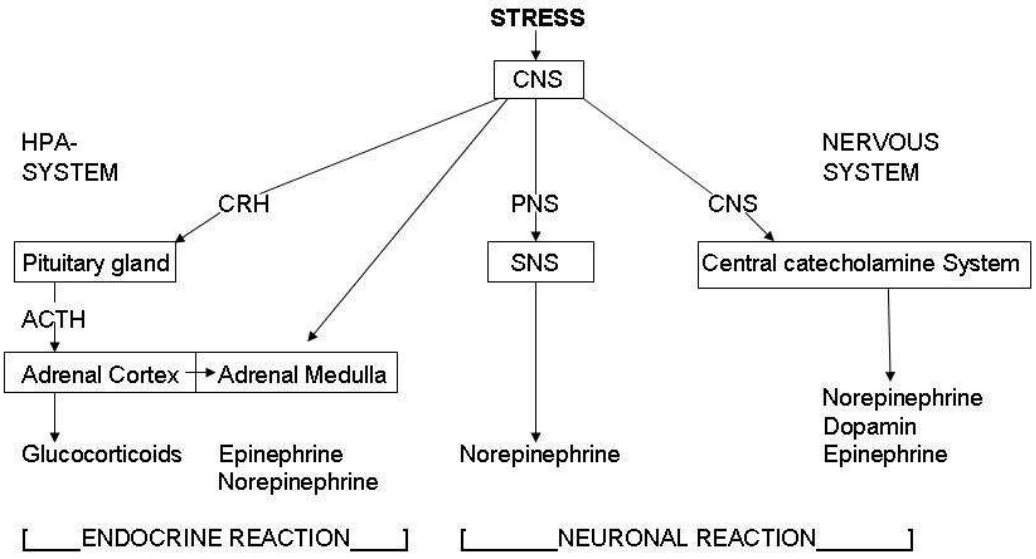

Figure 1 shows the endocrine and neuronal stress reactions (Kirschbaum and Hellhammer, 1999)

The stress response described above provides the body with the opportunity to optimally adapt and cope with challenges by inducing physiological and metabolic changes. In the long-term course, the body reacts to stress in three phases, which Selye termed the general-adaptation-syndrome (Selye, 1950): (1) the alarm reaction, (2) the stage of resistance, (3) the stage of exhaustion. During the first phase, *the alarm reaction*, the stressor causes an activation of the autonomic nervous system and an acute rise in glucocorticoid levels. During the second phase, the stage of *resistance*, an organism is still in a state of heightened arousal and uses its capacities to regain its inner balance (homeostasis). When the stressor continues, homeostasis can no longer be maintained resulting in the third stage of *exhaustion*. Thus, if the stress reaction is enduring, the long-ranging biochemical changes, like the secretion of glucocorticoids, damages the organism, leading to prominent structural changes of the body (e.g. enlargement of the adrenal glands, development of ulcers, shrinkage of the thymus, and finally to death) (Selye and Fortier, 1950; Selye, 1955).

Adaptation to stress has also been described by Bruce McEwen's concept of allostasis (McEwen, 1998b). While homeostasis refers to the regulation of the body to a balance by single point tuning, allostasis is the process of achieving stability, or homeostasis, through physiological or behavioural change. This process of "maintaining stability through change" involves the SNS and the HPA axis. For these systems, activation leads to catecholamine and cortisol release. Inactivation returns the systems to base-line levels of cortisol and catecholamine secretion, which usually happens, when the stressor is over. Thus these systems generally promote adaptation and coping, at least in the short term (McEwen, 1998b). Chronic overactivity or underactivity of allostatic systems lead to allostatic load, which refers to the

physiological costs of chronic exposure to the neural or neuroendocrine stress response (McEwen and Seeman, 1999). Allostatic overload leads to disease (McEwen, 1998b; McEwen and Seeman, 1999; McEwen, 2005).

Selye's hypothesis of a non-specific stress reaction has been revised in recent years. In particular, Mason (1975) suggested that the non-specificity concept has focussed on lower level physiological mechanisms, while neglecting higher level psychological processes. He argued that psychological factors influence the physiological stress response (Mason, 1968). Accordingly, the emotional reaction to a stressor (e.g. anxiety), but not the stressor itself triggers the stress response. Thus, differences between the individual neuroendocrine stress responses are due to psychological differences (Mason, 1975).

According to Lazarus's Transaction Theory of Stress (Figure 2), individual differences in stress response to the same stressor are caused by variations in the way individuals *appraise* stress (Lazarus, 1993). In his model, Lazarus and colleagues (1984) define stress as "a particular relationship between the person and the environment that is appraised by the person as taxing or exceeding his or her resources and endangering his or her well-being" indicating that stress is not solely evoked by the environment, but it is a process involving the interaction of a person with the environment.

Cognitive appraisal of stress constitutes of two processes: primary appraisal and secondary appraisal (Lazarus, 1984). During primary appraisal, a person evaluates a potential stressor either irrelevant, benign or stressful. If the situation is appraised as stressful (indicating relevance of the stressor), it will then be evaluated as a harm or loss, threat, or challenge. A harm/loss is described as something that already happened, while threat refers to a future event, for which a harm or loss is anticipated. In contrast, a stressor is

regarded as a challenge when the person feels able to effectively mobilize and develop coping resources.

During secondary appraisal, a person considers alternative approaches and evaluates his or her coping resources and the consequences of an action. Coping resources can generally be characterized in two ways: 1) as an emotional-oriented coping characterized by passive or avoidance coping strategies, or 2) as problem-focused coping, which is used to describe active efforts to find solutions to a problem.

Folkman modified Lazarus's model in order to accommodate positive psychological states, such as reappraisal, renewed problem and emotion-focused coping efforts, and the creation of positive psychological states (Folkman, 1997).

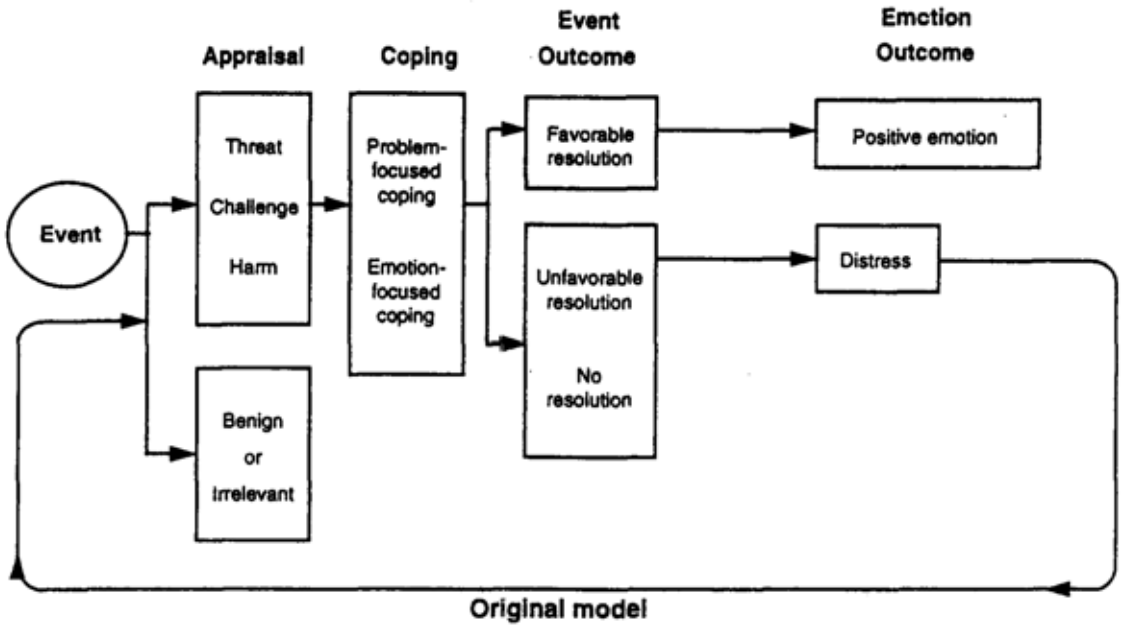

Figure 2 Lazarus model of appraisal (adapted from Folkman, 1997)