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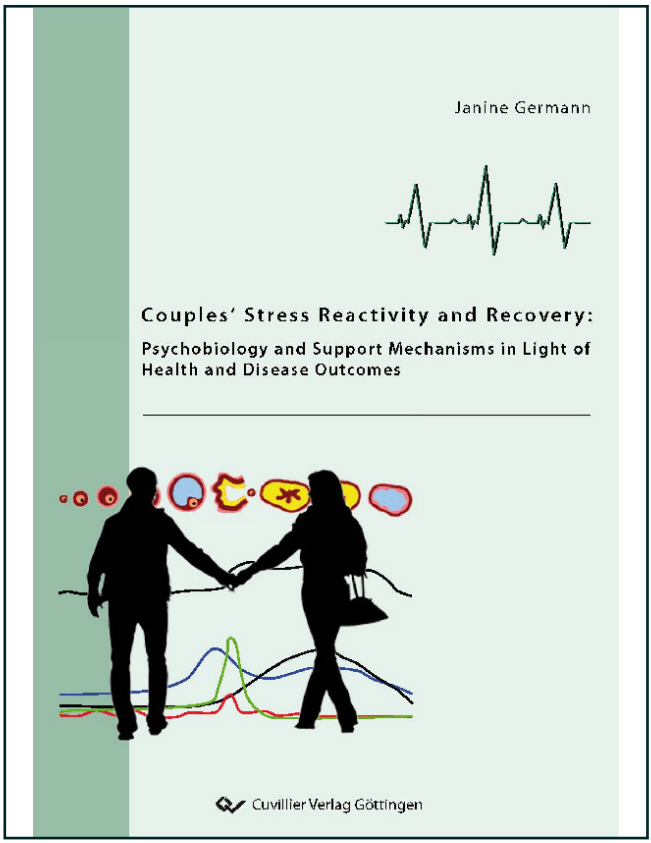
Couples‘ stress reactivity and recovery

Psychobiology and support mechanisms in light of health and disease outcomes



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1 Stress and health

Mental health disorders are one of the most frequent and serious diseases and are one of the reasons for a restricted quality of life. An increasing prevalence of these disorders in European countries is well documented. The World Health Organization (WHO) reported in 2003 that about 30% of all consultations with general medical practitioners in Europe were related to mental health problems. This growth has been associated, among other reasons, with the increase in workplace stress (Lademann et al., 2006).

The increase in mental health and other stress–related disorders is a significant cost factor in industrialized countries. An overview of the total costs in different European countries is provided by Andlin-Sobocki and colleagues (Andlin-Sobocki, Jonsson, Wittchen, & Olesen, 2005). According to a rather conservative estimate, the total economic costs of mental health problems are about 386 billion Euros in the European Union, Norway, Iceland, and Switzerland. In Switzerland, the costs alone due to stress–related disorders was estimated at 7.8 billion Euros ten years ago (Ramaciotti & Perriard, 2003).

Consequently, research on the prevention and intervention of stress–related disorders has grown over the last several decades. The gap between research and clinical application, however, is still enormous. Nevertheless, strategies and approaches to overcome this gap already exist (for an overview see Hellhammer & Hellhammer, 2008). The challenge in investigating the impact of stress on human well–being is the subjectivity in perceiving it since „(…) our concept of stress is very subjective and does not take into account the enormous individual differences that exist in coping with the environment" (McEwen, 1998b, p. 33). Thus, the objective of this chapter is to present the concept of stress, to describe the body's defense systems that react to stress, and to link them to different diseases and health indicators.

1.1 The concept of stress

In describing the body's fight–and–flight response, Walter Cannon (1914) was one of the first contributor to the concept of stress. Instead of *stress*, he used the words "painful or strong experiences" and assumed that "under these circumstances the increase of adrenalin and sugar in the blood is useful" (p. 362). Furthermore, he introduced the term *homeostasis* as a description of the physiological reactions to maintain stable internal states of the body (Cannon, 1929, p. 400). In referring to stress situations that challenge homeostasis, he extensively described the stress reactions of the body's different organs (heart, lungs, kidneys, brain, nerves, etc.).

In 1950, Hans Selye defined the concept of the *general adaptation syndrome* (GAS) as "all living organisms can respond to stress" and that "the basic reaction pattern is always the same, irrespective of the agent used to produce stress" (p. 4667). The GAS implies that there is a chronological development of the body's response to prolonged stressors: from the alarm stage to the resistance stage leading to the exhaustion stage (Selye, 1976). He already named the *hypothalamus–hypophyseal–adrenocortical axis* (also known as the hypothalamus–pituitary–adrenal axis discussed in chapter 1.2.2) and the *catecholaminergic system* (also known as the sympathetic–adrenomedullary system discussed in chapter 1.2.1) as systems regulating output to stress. In addition, he pointed out the importance of psychological stressors and emotional arousal as causes of the human response to stress. Figure 1-1 depicts his ideas in describing how *specific* stressors, emerging from everyday situations, can lead to *unspecific* outcomes through the body's pathways that experience stress (Selye, 1976).

Figure 1-1 Selye's description of specific stressors leading to unspecific outcomes because of the same pathway (modified from Selye, 1976, p. 55).

In the early–1960s, stress was considered a *transactional phenomenon* with the perceiver's personal meaning of the stimulus as the central element (Lazarus, 1966). A widely accepted and established model of stress and coping was then presented in the 1980s by Lazarus and Folkman (1984). The model was formulated in order to understand individual variances in stress response and coping and to point out the important notion of subjective appraisal (see also Coyne & Lazarus, 1980). According to their model, cognitive appraisal processes are made. For example, a person facing a stressful event evaluates, in a *primary appraisal*, the threat or challenge posed by the event (irrelevant, benign–positive, or stressful). If the person rates the event as stressful (threat or challenge), resources available are checked to deal with the situation (*secondary appraisal*). This is an evaluative process that is crucial for the outcome (can the person deal with the situation or not). Based on new information, *reappraisal* follows usually accompanied by emotions (person's reaction; stress or no stress) (Lazarus & Folkman, 1984).

McEwen (1998a; 1998b) combined the psychological with the biological components of the stress response. He stated that individual perception and physical health determine individual stress reaction and outcome. Adaptive processes to a potentially stressful event, referred to as *allostasis* (Sterling & Eyer, 1988), incorporate physiological responses of the hypothalamus–pituitary–adrenal axis, the autonomic nervous system, and the immune system to maintain homeostasis. Long–term consequences of allostasis leading to an imbalance of the involved systems are called *allostatic load.*

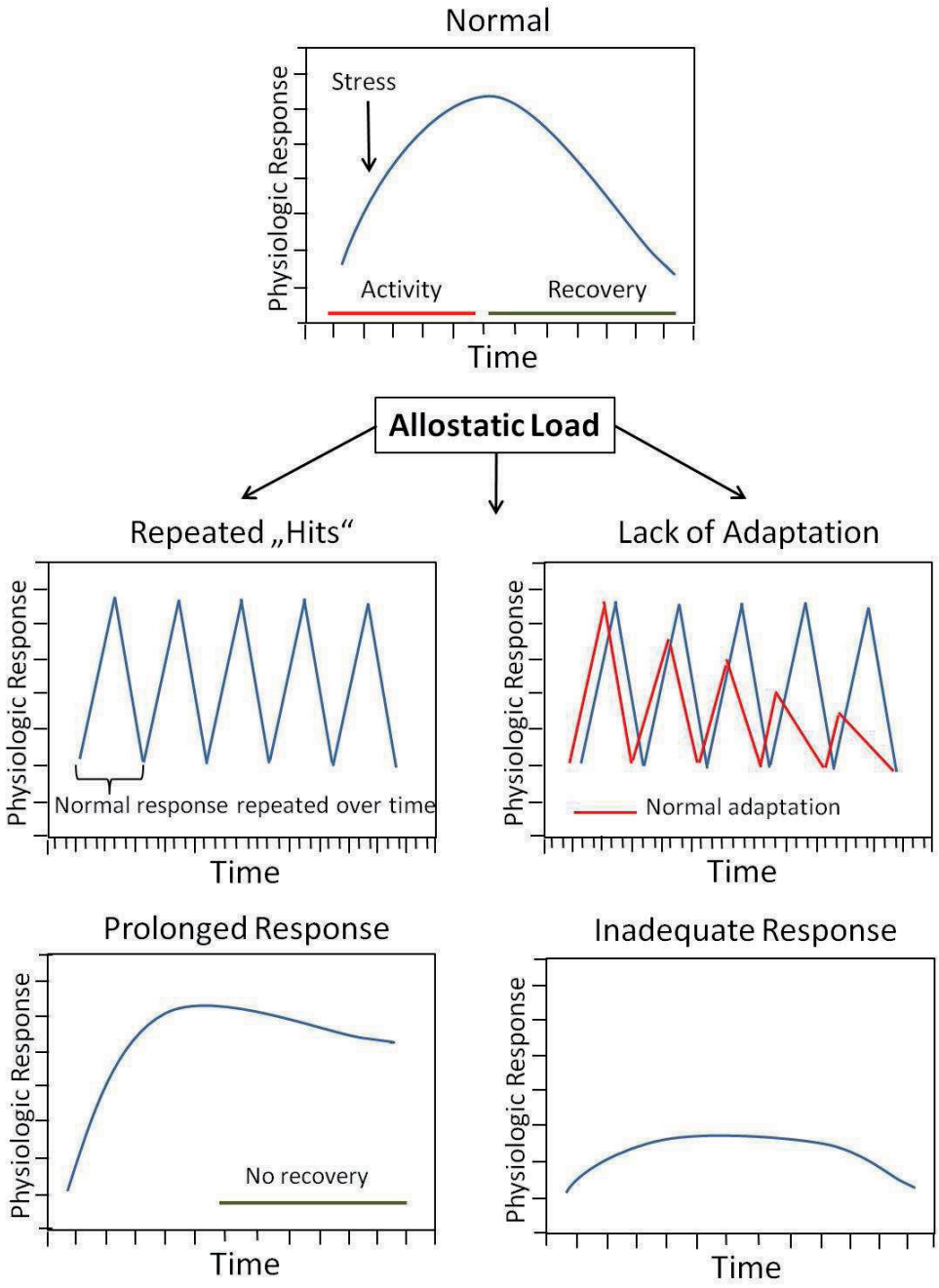

Figure 1-2 Four types of physiological responses leading to allostatic load (modified from McEwen, 1998a, p. 174).

Four types of physiological responses leading to allostatic load were named (see Figure 1-2): repeated “hits” (frequent stress); lack of adaptation (no habituation following the same stressor); prolonged response (failed shut–off, no recovery); and inadequate response (non–response; failed turn–on) (McEwen, 1998a).
In addition to an individual’s disposition (e.g. genes) and environmental stressors (work, home, etc.), characteristics of the individually behavioral response (personal behavior, fight–or–flight) in relation to allostasis determine whether physiological responses lead to adaptation or to allostatic load. Furthermore, health–promoting behaviors such as exercise, or health–damaging behaviors such as smoking and drinking clearly impact allostasis and allostatic load (McEwen, 1998a; 1998b).

In summary, McEwen’s *Allostasis Model* incorporates characteristics of the physiological response (from Cannon to Selye) and individual, cognitive appraisals (Lazarus and Folkman). McEwen claimed that:

> “(…) humans are more at risk for allostatic load than animals, because of the enormous individual differences in stress responsiveness and aging among humans, which relate to life experiences, personality, and physiologic phenotype” (1998a, p. 177).

Focusing on individual survival and pathology (especially in animals), Romero, Dickens, and Cyr (2009) recently presented their *Reactive Scope Model*. Compared to the Allostasis Model, this model provides direction on how mild stressors can have a beneficial effect on an animal’s development, resulting in lower vulnerability through phenotypic change. Nevertheless, the above–described physiological systems reacting to stress situations remain the same and, consequently, have to be described.

1.2 Biology of stress

In humans and other mammals, two distinct but connected systems are responsible for the stress response. These systems are the sympathetic–adrenomedullary system (SAM) (Vollmer, 1996) and the hypothalamic–pituitary–adrenocortical (HPA) system (Charmandari, Tsigos, & Chrousos, 2005) that produce different reactions to stress. In the following two chapters, both systems are described in more detail and related diseases and disorders are presented.

1.2.1 The sympathetic–adrenomedullary system and cardiovascular diseases

The SAM is a component of the autonomous nervous system (ANS), which consists of the sympathetic and the parasympathetic branches. These two branches are involved in almost every regulatory process of the body and primarily serve internal organs. Actors are the

catecholamines epinephrine (EPI) and norepinephrine (NE) released by the medulla or center of the adrenal gland (Vollmer, 1996).

Concentrations of those peripheral catecholamines indicate the activity of the SAM. EPI reflects general arousal of the acute mental state and NE reflects physical strain. The release of EPI and NE provides rapid mobilization of metabolic resources: increase in heart rate (HR), higher breathing rates, greater levels of glucose in blood, and vasodilatation in muscles (Tsapatsaris & Breslin, 1989). Preparing various target organs for fight–and–flight reaction, the SAM is the stress system that reacts first (for an overview see also Gunnar & Quevedo, 2007).

On the other hand, the parasympathetic system has a more regenerative function on the body organs. Parasympathetic innervation of the heart occurs via the vagus nerve, where the activity is indexed by heart rate variability (discussed in chapter 2). In contrast to sympathetic dominance, HR and blood pressure decrease under parasympathetic innervation. When the sympathetic branch is activated, parasympathetic mediated systems usually shut down (e.g. the digestive system). Thus, the sympathetic and parasympathetic systems act mostly in an antagonistic way on the body's organs (for an overview see Thayer & Sternberg, 2006).

Perception of stress causes the hypothalamus via nervous connections to activate sympathetic fibers, which in turn activate the cardiovascular system. Factors of this activation (reviewed by Szczepanska-Sadowska, Cudnoch-Jedrzejewska, Ufnal, & Zera, 2010) are as follows:

- neurotransmitters (acetylcholine, dopamine, serotonin, histamine, NE, and EPI)
- neuropeptides (vasopressin, oxytocin, CRH)
- steroids (estrogens and testosterone)

Dysfunction or imbalance of the autonomic nervous system can lead to a variety of diseases or disorders, related to cardiovascular disease (CVD) including coronary artery disease, arrhythmia, hypertension, heart failure, shock, and arteriosclerosis (Brook & Julius, 2000). In addition, decreased vagal activity has been linked to depression, generalized anxiety disorder, and post–traumatic stress disorder (see Thayer & Sternberg, 2006).
Figure 1-3 displays the factors involved in the autonomic imbalance of the sympathetic and the parasympathetic branch, in which sympathetic activity typically is predominant (Brook & Julius, 2000; Thayer & Lane, 2007). However, over–activation of the vagus nerve has been linked to sudden infant death syndrome for example (Lucet, de Bethmann, & Denjoy, 2000).

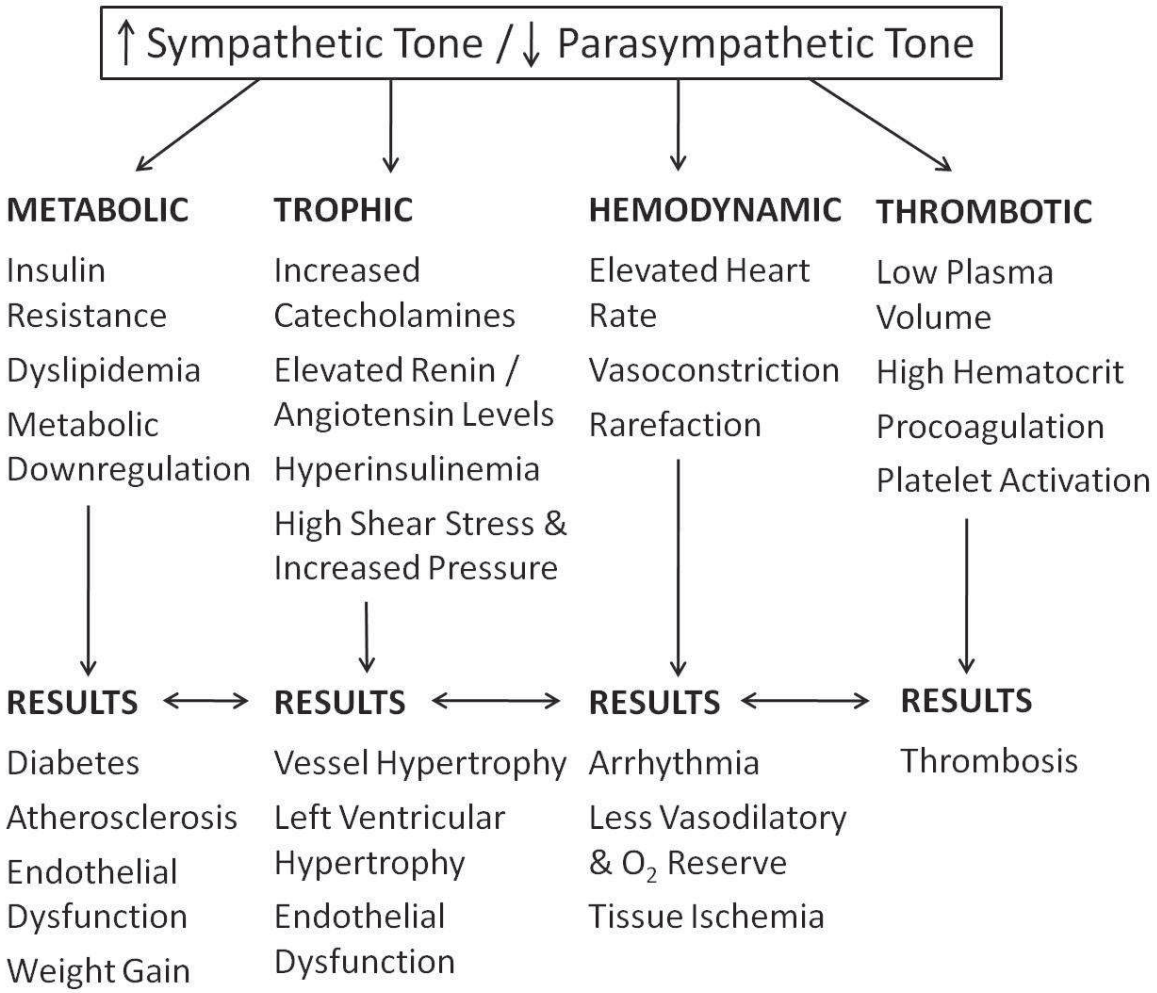

Figure 1-3 Autonomic imbalance (modified from Brook & Julius, 2000, p. 113).

In Western countries, CVD is the leading cause of death and its risk is higher in men than in women (Roger, Jacobsen, Pellikka, Miller et al., 1998). After menopause, however, risk of CVD increases in women because of hormonal changes (Kannel & Wilson, 1995). The steroid estrogen is suggested to be cardio–protective, consequently, estrogen replacement therapy after menopause reduces cardiovascular mortality by about 50% (for an overview see Sullivan & Fowlkes, 1996). However, it is unlikely that the protective effect of estrogen alone is responsible for this difference. Lawlor and colleagues assumed that, besides geographical trends, fat consumption or the response to dietary fat differs between men and women (Lawlor, Ebrahim, & Davey Smith, 2001; 2002). There is some evidence for this assumption; women's concentration of high density lipid profiles seems to react more appropriately in response to diets than men's concentration (see also Miller, 1994; Turgeon, Carr, Maki, Mendelsohn, & Wise, 2006). Table 1-1 in chapter 1.4.1 lists additional diseases linked to the influence of exogenous estrogens.