THE EFFECTS OF FITNESS TRAINING ON PHYSIOLOGICAL STRESS-REACTIVITY

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Summary

The main goal of this thesis was to show that fitness training counters the detrimental effects of stress on cardiovascular health. More specifically, fitness training was thought to improve psychological make-up, reduce the acute physiological response to stress, and counteract the effects of chronic stress on the cardiovascular risk factors.

In chapter 2, evidence is reviewed for a preventive effect of regular exercise on the development and progression of cardiovascular disease. It is noted that regular exercise is most effective when it causes an increase in aerobic fitness, and the relative risk for subjects in the least fit quartile is more than 4.8 times higher than that of subjects in the most fit quartile. Since smoking, blood pressure and cholesterol do not entirely explain the beneficial effects of regular exercise, it is hypothesized that exercising may partly protect against cardiovascular disease by preventing the detrimental effects of stress.

Chapter 3 elaborates on the possible role of physiological stress-reactivity in cardiovascular disease. Current psychosomatic theory regards the repeated and prolonged occurrence of autonomic nervous system activity (fight-flight reaction) as the main cause for stress-induced disease. During psychosocial stress-situations the sometimes fierce cardiovascular and metabolic responses are not "used up" by the muscles and this may result in increased blood pressure and atherosclerosis. Research has concentrated on the striking individual differences in the physiological stress-reactivity that occur during a variety of effortful mental stress tasks performed in the laboratory. Exaggerated reactivity to such stressors is thought to predispose the individual to cardiovascular disease.

Chapter 4 reviews 30 studies that have examined aerobic fitness as a possible mediator of the physiological stress-reactivity. Virtual all studies found that high fit subjects had lower heart rate and diastolic blood pressure levels during exposure to the stressors than low fit subjects. The lower stress levels were mainly based on lower basal levels in the high fit, but lower stress-reactivity was also seen to contribute in about half of the studies. In agreement with popular belief, this suggested that aerobic fitness training could be used to prevent the effects of stress on cardiovascular disease. However, some concerns remained. Most studies had been cross-sectional, comparing sedentary subjects with subjects who had been exercising for years. The favorable psychological and physiological profile found in regular exercisers (o.a. reflected in high aerobic fitness) does not necessarily follow from their exercise behavior. Alternatively, it depends on endowment and/or self-selection.

Chapters 5 and 6 present the results of two longitudinal studies that aimed to directly examine the effect of fitness training programs on psychological make-up and stress-reactivity. Aerobic fitness of these subjects was measured as the maximal oxygen consumption per kg body weight during an exhaustive exercise test (Vo_{2max}). Although all subjects were non-exercisers before the start of the study, substantial variation existed in their Vo_{2max}, probably reflecting hereditary influences. In the first study (N=26), these pre-existing differences in aerobic fitness were found to be related to cardiovascular reactivity during two stressful reaction time tasks. More fit subjects were found to have diminished vagal withdrawal during stress, and smaller diastolic blood pressure reactivity. However, these effects were not replicated in a second study (N=62), when the stress tasks were made far more challenging by team competition. In fact, more fit subjects showed higher systolic and diastolic blood pressure reactivity to the stressors. Both studies failed to show a relationship between fitness and important indices of sympathetic

nervous system reactivity like the pre-ejection period, cardiac output, or urinary catecholamine excretion. In chapter 5, seven weeks of training were not found to change either reactivity or recovery of any of the variables, although Vo_{2max} was seen to increase significantly. Chapter 6 replicated this lack of training effects in a larger group of subjects, using a training program of longer duration (4 to 8 months). In addition, no effect was found of fitness training on a host of psychological variables (e.g. neuroticism, anger expression, coping style, self-esteem and mood). The beneficial effects of training were limited to a reduction in the overall levels of heart rate and diastolic blood pressure.

Chapter 7 deals with the effects of exercise training on a cluster of CHD risk variables that may have a common background in insulin-insensitivity, i.e. body composition, cholesterol, triglycerides, and plasminogen activator inhibitor (PAI) activity, a risk indicator for disturbances in the coagulation/fibrinolysis balance. Total cholesterol and percentage body fat significantly decreased in response to training, and increased again during de-training. PAI activity always decreased in response to training, particularly in subjects with high initial PAI levels, but definite causality could not be established because of substantial seasonal shifts in the non-training control groups.

Chapters 8.1 and 8.2 of the general discussion summarize other recent studies on the effects of fitness training on psychological functioning and acute physiological stress-reactivity. It is hypothesized that the popular belief in stress-alleviating effects of exercise largely reflects its short-term neurophysiological effects on mood, blood pressure, and even reactivity. Lasting psychological benefits of exercise may be limited to subjects who experience severe psychological disturbances at the start of training. The psychological differences found between exercisers and non-exercisers in the population at large must predominantly reflect self-selection, i.e. emotionally well-adjusted, self-confident and self-disciplined persons may be most attracted to sports and exercise. None of the studies provided convincing evidence for an effect of training on stress-reactivity, even if initially low fit and high stress-reactive subjects were used. It is concluded therefore, that exercise does not increase the resistance to stress by influencing psychological make-up or acute psychophysiologic reactivity.

Chapter 8.3 re-evaluates the idea that a reduction in stress-reactivity is crucial to a beneficial effect of training. Reliability, intertask consistency, construct, predictive and ecological validity of cardiovascular reactivity to laboratory stress are reviewed, and it is concluded that reactivity may not be suited as a risk factor for cardiovascular disease. In fact, two-stage models of stress suggest that high sympathetic reactivity during acute exposure to stress may be a positive, rather than a negative trait, as long as it is paired to faster recovery and lower overall levels of heart rate, blood pressure, and metabolic risk factors during chronic stress.

Chapter 8.4 attempts to explore new ways to direct our future research on stress and exercise. It is noted that long-term exposure to psychosocial stress and long-term exercise habits show completely opposite effects on most known or suspected pathogenic processes involved in cardiovascular disease (coronary blood flow, triglycerides, LDL/HDL cholesterol ratio, plasma insulin, hypofibrinolysis, and hypercoagulability). Several possible sites for a direct interaction between chronic stress and regular exercise were explored, i.e. cardiac autonomic balance, adrenoceptor sensitivity, vascularization of skeletal muscle, and insulin-sensitivity. Although the exact mechanism remains to be found, it is concluded that fitness training should be seen as an excellent way to compensate or prevent stress-related deterioration of cardiovascular health, even if psychological make-up or the acute reactivity to stress are unchanged after training.