Diplomarbeit

Transcendental Meditation and Yoga

Effects on Psychological Parameters in Patients with Cardiovascular Diseases

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Ich erkläre ehrenwörtlich, dass ich die vorliegende Arbeit selbstständig und ohne fremde Hilfe verfasst habe, andere als die angegebenen Quellen nicht verwendet habe und die den benutzten Quellen wörtlich oder inhaltlich entnommenen Stellen als solche kenntlich gemacht habe.

Graz, am 21.10.2020 Sarah Wedenig eh

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Sasa, you better be reading this.

Zusammenfassung

Einleitung: Kardiovaskuläre und psychiatrische Erkrankungen zählen nicht nur für sich genommen zu den häufigsten Erkrankungen weltweit; sie treten auch sehr oft gemeinsam auf und stellen jeweils Risikofaktoren füreinander dar. Eine zugrundeliegende Ursache für diese Wechselwirkung stellt chronisches Stress-Erleben dar. Die vorliegende Arbeit untersuchte, ob stressreduzierende Maßnahmen wie Yoga oder Transzendentale Meditation (TM) das psychische Wohlbefinden und subjektiv erlebte Gesundheitsgefühl von kardiovaskulären Reha-PatientInnen positiv beeinflussen.

Methoden: Im Rahmen dieser Studie wurden 30 Personen, die nach einem kardiovaskulären Ereignis an einem vierwöchigen, stationären Rehabilitationsprogram teilnahmen, untersucht. Die PatientInnen wurden zufällig einer von drei Untersuchungsgruppen zugeteilt. Das psychische Wohlbefinden sowie das subjektiv erlebte Gesundheitsgefühl wurde vor und nach der Rehabilitation mittels acht standardisierter Fragebögen erfasst.

Ergebnisse: Personen aus der Yoga-Gruppe zeigten nach vier Wochen eine stärkere Verbesserung der Schlafqualität als die Kontrollgruppe. Personen aus der TM-Gruppe wiesen nach der Rehabilitation eine höhere Vitalität auf, als Personen der Kontrollgruppe. In allen weiteren Parametern unterschieden sich die drei Gruppen jedoch nicht signifikant voneinander.

Diskussion: Die Ergebnisse der Studie legen nahe, dass sich zusätzliche Yoga- bzw. TM-Einheiten während einer kardiovaskulären Rehabilitation, positiv auf die Schlafqualität und Vitalität der PatientInnen auswirken. Für weiterführende Studien gilt es, diese Ergebnisse mit größeren Stichproben zu überprüfen und die langfristigen Effekte von Yoga und TM auf kardiovaskuläre und psychische Gesundheit zu beleuchten.

Abstract

Introduction: Cardiovascular and psychiatric diseases are not only among the most common diseases worldwide, they also very often occur together. An underlying cause for this interaction is chronic stress. The present study investigated whether stress-reducing interventions such as yoga or Transcendental Meditation (TM) have a positive influence on the psychological well-being and subjective health of cardiovascular patients.

Methods: 30 individuals who participated in a four-week rehabilitation program following a cardiovascular event were examined. The patients were randomly assigned to one of three study groups. The psychological well-being as well as the subjectively experienced feeling of health was measured before and after rehabilitation using eight standardized questionnaires.

Results: Participants of the yoga group showed a greater improvement in sleep quality after four weeks than the control group. The TM group showed a higher vitality after rehabilitation than the control group. In all other parameters, the three groups did not differ significantly from each other.

Conclusion: Additional yoga or TM sessions during cardiovascular rehabilitation have a positive effect on sleeping quality and vitality of patients. For further studies it is important to verify these results with larger samples and to examine the long-term effects of yoga and TM on cardiovascular and mental health.

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Abbreviations

ANS	Autonomic nervous system
ASTS	Aktuelle Stimmungsskala
BDI-II	Beck's Depression Inventory II
BSI-18	Brief Symptom Inventory
CHD	Coronary heart disease
CNS	Central nervous system
CRP	C-reactive protein
CVDs	Cardiovascular diseases
EBM	Evidence-based medicine
ECG	Electrocardiogram
EEG	Electroencephalogram
ELS	Early life stressors
GAD	Generalized Anxiety Disorder
HPA	Hypothalamic–pituitary–adrenal
IL-1	Interleukin-1
IL-6	Interleukin-6
INF- α	Interferon alpha
MBSR	Mindfulness-based stress reduction
MD	Mediterranean diet
MI	Myocardial infarction
NSTEMI	Non-ST elevation myocardial infarction
NYHA	New York Heart Association Functional Classification

OPD-SQS	Operationalisierte Psychodynamische Diagnostik - Structure Questionnaire Short
PNS	Parasympathetic nervous system
PSQI	Pittsburgh Sleep Quality Index
PSQ-20	Perceived Stress Questionnaire 20 Items Version
PTSD	Post-Traumatic Stress Disorder
RAAS	Renin-angiotensin-aldosterone system
RCT	Randomized controlled trial
SD	Standard deviation
SF-36	Short Form Health Survey 36
SNS	Sympathetic nervous system
STAI-6	6-Item Short Form of the Spielberger State-Trait Anxiety Inventory
STEMI	ST-elevation myocardial infarction
TM	Transcendental meditation
TNF-α	Tumor necrosis factor alpha
UNIANOVA	Single factor analysis of variance
VNS	Vagus nerve stimulation

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Introduction

Epidemiology of Cardiovascular Diseases (CVDs)

CVDs are the most common cause of death worldwide. They include diseases of the heart, the brain vessels and the peripheral blood vessels (1). In 2004, 17.1 million people died due to a CVD. With globally increasing life expectancies, it is predicted that by 2030, CVD-related mortality will rise to about 23.4 million deaths per year. Ischemic heart diseases, such as myocardial infarction (MI), angina pectoris, or sudden cardiac death, account for the largest proportion of CVD-related deaths (2).

When looking at the impact of diseases during life, ischemic heart diseases are the second most common cause of disability in high-income countries. Here, the leading cause of disability are unipolar depressive disorders. By 2030, these two diseases are expected to be the most common causes of disability in countries of all income levels (2). However, not only are psychiatric and CVDs the most common causes of disability, they often occur together and influence each other.

Depression and CVDs

Of all psychiatric diseases, the comorbidity between depression and CVD has been studied most (3). Many studies have confirmed the epidemiological association between depression and CVDs (4). About 15% of patients who have had a severe cardiac event, such as a MI or coronary artery bypass grafting, have a major depressive disorder (5). Other studies report an incidence of up to 27% of depression in cardiac patients (4,6). In the general population, this prevalence is two to three times lower (7).

Furthermore, clinical depression is an independent risk factor for CVDs. It increases the risk of developing coronary heart disease (CHD) two to four times when compared to non-depressed populations (4,8).

Depression also has a profound impact on the prognosis of CVDs. A four-year longitudinal study that observed the influence of depression on cardiac mortality, found that the risk for cardiac mortality in patients with a CVD and a minor depression is 60% higher than that of non-depressed cardiac patients. For CVD

patients with major depression, the risk of dying from heart disease was three times higher than for CVD patients without depression. These findings were robust, even when adjusted for demographics, smoking, alcohol use, blood pressure, body mass index, and other comorbidities (9).

A previous depressive disorder is also a predictor of survival after a MI. Patients with a first-time MI and a history of depression have a significantly higher 19-year all-cause mortality than patients without previous depression (10). The finding that depressive symptoms predict future coronary events for healthy people, as well as worsen the prognosis in people with an established CVD, has been shown in a vast amount of studies (11–15).

Anxiety and CVDs

While the link between depression and CVD has been investigated intensively, less research has focused on the role of anxiety in cardiovascular patients (16). However, epidemiological studies show that anxiety disorders like generalized anxiety disorder (GAD), post-traumatic stress disorder (PTSD), and panic disorder are more common in CVD populations than in the general population (17).

Furthermore, recent studies demonstrated that anxiety disorders correlate with the onset and progression of CVD, and in many instances have been linked to adverse cardiovascular outcomes, including mortality (18).

Along with depression, anxiety disorders are the most common psychiatric comorbidities in patients with heart failure (HF). Similar to depression, these disorders are correlated with the development of HF and a worsening prognosis of the disease (19).

A meta-analysis investigating the impact of anxiety on the incidence of CHD and cardiac mortality showed that symptoms of anxiety (e.g.,, tension, worries) or an anxiety disorder were associated with a 26% increased risk of incident CHD. Anxious persons also had a 48% increased risk of cardiac mortality (20). On the other hand, a cardiac event like unstable angina or a MI can induce elevated anxiety. Grace et al. showed that in over 50% of patients who experienced elevated anxiety at a cardiac event, the anxiety persisted for one year. The authors also found that persisting anxiety (longer than six months after the cardiac event) is an independent

predictive risk factor for self-reported recurrent cardiac events at six months to one year (21).

Another study investigated the effects of GAD in patients who were admitted for MI. Patients with GAD were more likely to have a history of MI and showed an almost twofold increased risk of adverse outcomes (cardiac event, death) during the 10-year follow-up period (16).

Another anxiety disorder that is associated with the risk for CHD is PTSD. A meta-analysis showed that PTSD increases the risk for developing a CHD or cardiac-specific mortality up to 27% compared to a population without PTSD (22).

Many other studies have confirmed the link between anxiety disorders and CVDs and substantiated the influence of anxiety on the development and prognosis of heart diseases (23–26).

Stress: a potential mechanism of interaction

As demonstrated CVDs and psychiatric illnesses are not only major health problems independently, but often occur together. Bidirectionally, CVDs and psychiatric illnesses represent significant risk factors for each other and lead to a synergistic acceleration and amplification of both diseases. Yet the precise underlying interaction mechanisms remain unclear (4,27). One critical contributing factor to the bidirectional link between cardiac and mental diseases is stress (4,13,28). The physiology and pathophysiology of stress are to be explained. Furthermore, research indicating that chronic and/or severe stress can lead to diseases of the heart and the psyche will be discussed.

The Biology of Stress

Stress is life.

This quote by Hans Selye–one of the most important contributors to stress research–summarizes that stress is nothing to be rid of once and for all, but rather a substantial part of life itself. The brain and body must constantly adapt to respond to various stressors. A body that does not have to cope with stress is a dead body (29).

The term stress is defined as a state in which the body's homeostasis is perceived to be threatened by a psychological, environmental, or physiological stressor (30,31). Therefore, a stressor is any stimulus the individual perceives as demanding, challenging, and/or threatening: the death of a loved one, a broken arm, a demanding job, heat, noise, loneliness, rejection or peer pressure, to name a few of them (32). While there exist many approaches to objective stressors (e.g.,, The Social Readjustment Rating Scale (33)), it is important to bear in mind that the extent to which a certain life event is perceived as threatening and stressful is dependent upon various personal properties including genetic influences and previous experiences (28). The very same event could be stressful for some individuals but not for others (34).

On a physiological level, the body's ability to respond and adapt to such stressors to maintain its steady state (homeostasis) and protect itself, is known as the stress response. This involves changes in the central nervous system (CNS) as well as in various peripheral organs and tissues. Energy supply (increased blood glucose level) is increased, skeletal and cardiac muscles are better oxygenated, alertness is heightened, and the body's immune and inflammatory responses are inhibited. These changes enable the body to elicit the famous "fight or flight" response. In this way, the body improves the chances of the individual's survival in the face of potential harm (35).

The hypothalamic–pituitary–adrenal (HPA) axis and the autonomic nervous system (ANS) with its two major branches – the sympathetic and the parasympathetic system - represent the peripheral components of the stress system. Via these components, the CNS influences all body organs during exposure to threatening stimuli. The principal peripheral effectors are glucocorticoids (mainly cortisol in humans), which are regulated by the HPA axis and the catecholamines noradrenaline and adrenaline, which are regulated by the ANS (36).

When perceiving a stressor, the body's immediate reaction involves the activation of the sympathetic nervous system (SNS) and the secretion of catecholamines (37). The parasympathetic nervous system (PNS) may further promote sympathetic functions through its own withdrawal (36). As the SNS is activated, the secretion of adrenaline and noradrenaline leads to an increase in heart rate, blood pressure, respiratory rate, gluconeogenesis, and lipolysis.

The delayed, second stress response is controlled by the HPA axis. During acute stress, the paraventricular nuclei (PVN) of the hypothalamus increase their secretion of corticotropin-releasing hormone (CRH) into the hypophyseal portal system. Subsequently, CRH triggers the secretion of adrenocorticotropic hormone (ACTH) by the anterior pituitary gland. ACTH in turn stimulates the adrenal cortex to synthesize glucocorticoids. Cortisol helps the body to mobilize energy reserves in a stressful situation. In particular, it serves to increase blood glucose concentration. Furthermore, glucocorticoids have an immunosuppressive effect; that is, they inhibit the body's immune response.

Via a negative feedback loop, cortisol inhibits the release of CRH and ACTH from the hypothalamus and pituitary gland, resulting in reduced cortisol release. This prevents the HPA axis from overshooting. A physiological stress response is therefore one that is activated when needed, but also efficiently terminated afterward (37,38). For a graphical representation of the acute stress response, see Figure 1.

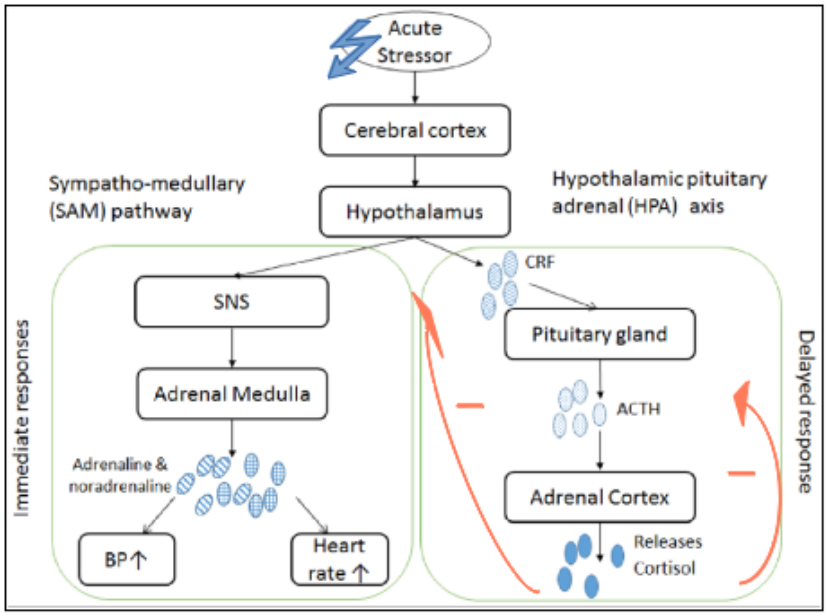

Figure 1: Acute stress response pathways. ACTH = Adrenocorticotropic hormone; BP = blood pressure; CRF = Corticotropin releasing factor; SNS = sympathetic nervous system; TPR = total peripheral resistance. Illustration adapted from: (39).

Stress and Disease

The demonstrated stress response is the vital ability of the body to maintain its physiological balance through change. This capability–often referred to as "allostasis"–is crucial for our survival. As described above, the successful allostatic response involves the immediate activation of the stress system as well as the inactivation of the system when the stressful event has passed. However, if the system is activated very frequently or fails to shut down adequately due to chronic activation, the hyperactivity of the HPA axis and the ANS results in overexposure to stress hormones. McEwen and Stellar define this *"allostatic load" phenomenon* as "*strain on the body produced by repeated ups and downs of the physiological response as well as by the elevated activity of physiological systems under challenge and the changes in metabolism and the impact of wear and tear on a number of organs and tissue"* (40). It is the allostatic load–the wear and tear of the stress system caused by severe or chronic stress–that accounts for several diseases, including cardiovascular and psychiatric disorders (see Figure 2) (28,35,41).

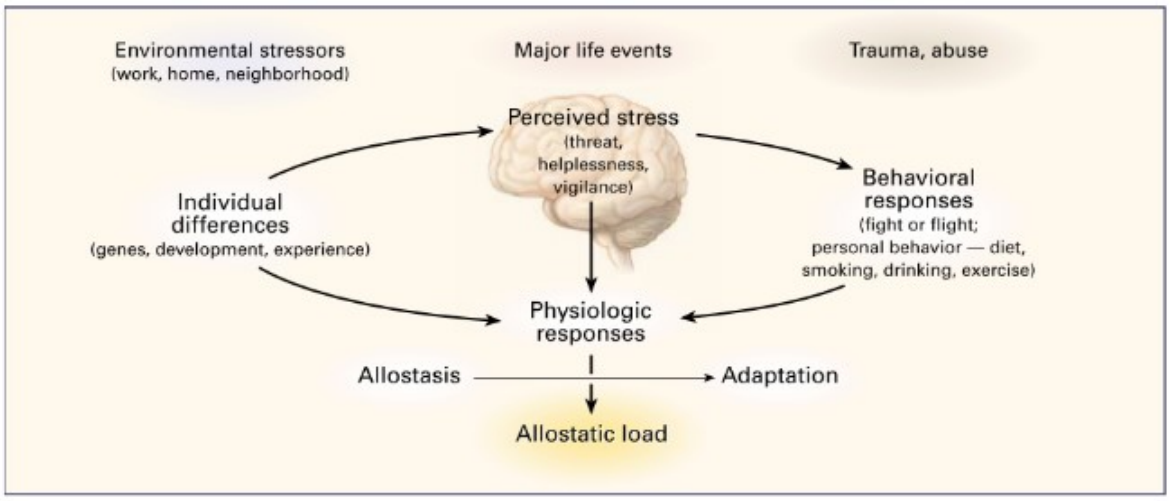

Figure 2: The stress Response and development of allostatic load. How one perceives stress is influenced by individual experiences, genetics, and behavior. The experience of a stressful event initiates physiological and behavioral responses, leading to allostasis and adaption. Over time, the allostatic load can accumulate, and the overexposure to mediators of neural, endocrine, and immune stress can have adverse effects on various organ systems, leading to disease. Illustration adapted from: (41).

Stress and CVDs

There is a large body of evidence linking stress to increased rates of CVDs.

In adults, the most commonly studied stressors are work-related stress and social isolation; a meta-analysis of prospective studies found a 50% (95% CI 1.2–1.9) increased risk for the first-time occurrence of CHD in adults experiencing loneliness and social isolation and a 30% (95% CI 1.2–1.5) increase in people reporting workplace stress (42). During a mean 9.8 year follow-up time, a longitudinal study with 90,164 participants observed a 40% (95% CI 1.1–1.8) risk ratio for incident CHD (defined as first nonfatal MI or coronary death) in people experiencing work-related stress compared with people reporting low work-related stress (43). However, many other stressful life events such as marital problems, care for a sick family member, or the death of a loved one have been linked to an increased risk of CVD as well (44). A Danish cohort study showed that after six years of follow-up, parents who lost a child had a significantly increased risk for MI when compared to parents who did not experience such a traumatic loss. Those parents whose children died unexpectedly had the highest relative risk of MI (45). Many other research findings support the proposed positive correlation between chronic stress and the incidence of CVDs (46,47).

A chronic activation of the body's stress response can lead to the development and progression of CVDs via several pathways. The primary underlying pathology of CVD is atherosclerosis, or the formation of plaque in the artery walls. This plaque can lead to the occlusion of blood vessels, resulting in acute cardiovascular events such as MI. It is known that atherosclerosis is the result of chronic inflammation in the artery walls (48–51). Chronic activation of the HPA axis and the ANS can cause such an inflammatory response in two ways.

First, the noradrenaline released by the activation of the SNS leads to an up-regulation of the transcription of pro-inflammatory cytokines like interleukin-1 (IL-1), tumor necrosis factor α (TNF-α) and interleukin-6 (IL-6). These cytokines in turn promote increases in systemic inflammatory activity (52).

Second, the glucocorticoids released by the activation of the HPA axis–which under normal conditions work as a potent anti-inflammatory substance–can lose their effect through glucocorticoid insensitivity. This insensitivity occurs, when immune

cells, in order to compensate for a chronic secretion by the HPA axis, become less sensitive to glucocorticoids (53).

Furthermore, the activation of the renin-angiotensin-aldosterone system (RAAS) and the secretion of catecholamines (adrenaline and noradrenaline) result in increased blood pressure and heart rate, which further promote endothelial damage and atherosclerosis (Figure 3) (54).

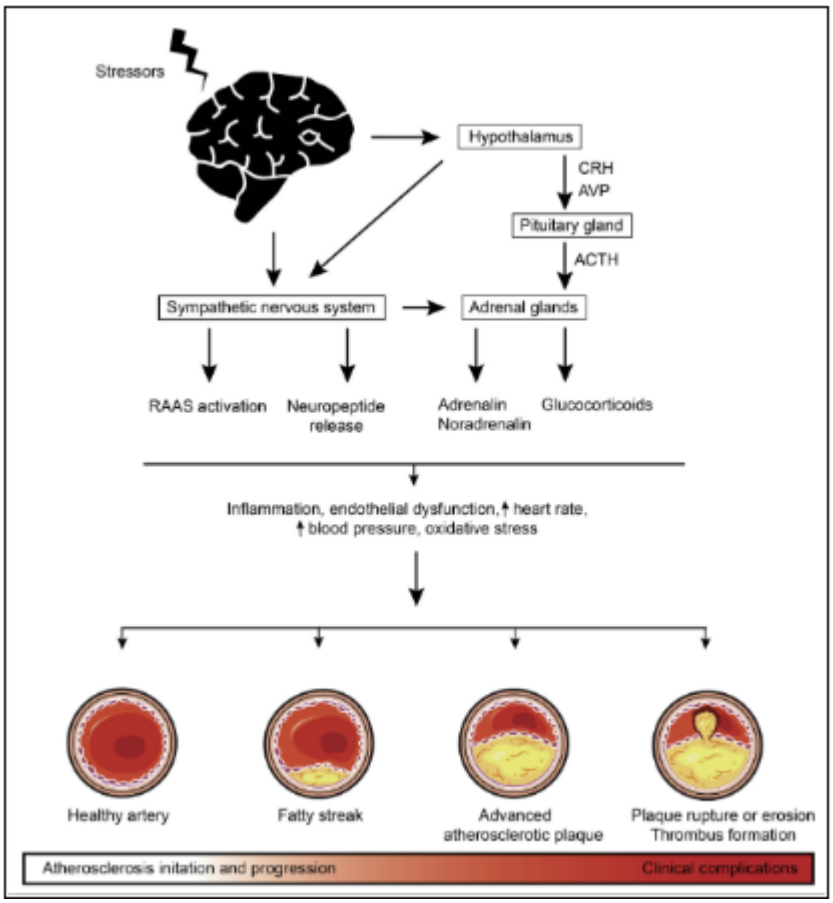

Figure 3: The impact of stress on the development of atherosclerosis. Perceived stress activates the HPA axis (hypothalamus - pituitary gland- adrenal glands) and the sympathetic nervous system, which results in the secretion of catecholamines (adrenaline, noradrenaline) and glucocorticoids. Furthermore, it leads to activation of the renin–angiotensin–aldosterone system (RAAS), while neuropeptides are released from peripheral nerve endings. These reactions lead to an inflammatory response, damage the endothelium (the inner layer of the vessel wall), and cause an increase in heart rate, blood pressure and oxidative stress. All of these factors can in turn induce and/or amplify the development of atherosclerosis, and eventually result in acute cardiovascular events due to plaque rupture or erosion. CRH = corticotrophin releasing hormone, AVP = arginine vasopressin, ACTH = adrenocorticotropic hormone. Illustration adapted from (54).

Stress and Depression

There is robust evidence that the experience of stressful life events increases the risk of depression. The relationship between major life events and depression is particularly well.studied. Such events include stressors like the ending of a close relationship, job loss, a life-threatening medical diagnosis, or a significant financial loss. The common denominator of such events is a substantial disruption to one's life goals, plans, and aspirations (52). A review by Mazure found that approximately 80% of depressive episodes in the general population are preceded by a major life event (55). This association exists even when controlling for genetic and environmental influences. For instance, a study that assessed the occurrence of stressful life events and the onset of depression in monozygotic female twin pairs showed that stressful life events were strongly and significantly associated with subsequent episodes of major depression (56). Stressful events that involve social rejection or interpersonal loss appear to increase the risk of depression particularly strongly (52,57). However, everyday stressors such as job strain (high psychological demands and low decision latitude) also increase the risk of developing depression (58,59). Many other studies support the connection between stress and depression (13,60).

As previously indicated, a frequent or chronic activation of the body's stress system can induce inflammation through up-regulation of pro-inflammatory cytokines and the development of glucocorticoid insensitivity. Many psychobiological models of depression assume that the inflammatory effect of chronic stress can be a trigger for the disease. Smith proposes in his "Macrophage Theory of Depression" that psychological stress, in probable combination with genetic factors, increases cytokine production and leads to depressive symptoms. He assumes that in particular the secretion of IL-1, TNF-α, and interferon-alpha (INF- α) might be the cause of depression (61). In line with this theory are further findings that show that the activity of the vagus nerve–the main contributor to the PNS–is decreased in patients suffering from depression (62). Activity of the vagus nerve has anti-inflammatory properties, and its stimulation via electrical impulses has a positive effect on depressive symptoms. The anti-depressant effect of vagus nerve stimulation (VNS) is explained by an inhibitory influence on the production of pro-

inflammatory cytokines and increases in anti-inflammatory circulating cytokines (63).

Furthermore, Leonard postulated that depression is an inflammatory disease caused by chronic psychological stress (64–66). Since then, many research findings have confirmed inflammation to be a major contributing factor to depression (4). Pro-inflammatory cytokines elicit adaptations in the brain that result in symptoms resembling depression. These behavioral symptoms–often referred to as “sickness behavior”–overlap with those found in major depression and include fatigue, decline in appetite, lack of interest in the physical and social environments, sleeping disturbances, as well as mild cognitive impairment (67–69).

Stress and Anxiety

Although there exists a large research body linking depressive disorders and the body’s stress response, less is known about the connection between anxiety disorders and stress exposure. However, comorbidity between depression and anxiety is highly frequent. Anxiety disorders often precede depression and–as indicated previously–both often occur together with heart diseases (70). The effects of stressful events on the development of anxiety disorders and their prognosis are therefore the subject of current research. In particular, the experience of early life stressors (ELS) seems to be strongly implicated in the alteration of the HPA axis and the manifestation of anxiety disorders (71,72). The term ELS covers a wide range of different traumatic experiences (including parental loss, sexual abuse, or family violence) that occur during childhood and adolescence. Such traumatic experiences may result in a sensitization of the HPA axis and the body’s stress response, which in turn is likely to mediate the increased risk for anxiety disorders in adulthood (73). Individuals who report a history of ELS are at higher risk to develop PTSD in adulthood than those who do not report ELS (74). A review by Carr et al. found that sexual abuse in childhood was correlated with the development of panic disorder and agoraphobia in adulthood (75). It is hypothesized that massive stress during childhood can result in persistent changes to the HPA axis response to stress in adulthood, and that this mechanism can lead to elevated levels of anxiety (71).

Further evidence indicates a correlation between anxiety disorders and inflammatory processes (76). The finding that inflammation is a risk factor for anxiety disorders is especially robust for PTSD. For instance, it has been found that although about 70% of the general US population will experience a traumatic event in their lifetime, only 7.8% will develop PTSD afterward (77). Inflammatory markers such as C-reactive protein (CRP) or cytokines appear to predict an individual's risk of developing PTSD after a traumatic event. A study by Eraly et al. assessed the plasma concentration of CRP as well as symptoms of PTSD in 2,600 Marine and Navy combatants before and after their deployment to a war zone. Results revealed that the baseline CRP had a significant effect on post-deployment PTSD symptom emergence, with higher levels of CRP marking a greater vulnerability to developing these symptoms (78). Supporting these findings, a study examining children immediately after a car accident found that elevated evening free cortisol and morning IL-6 levels within 24 hours after the accident were predictive of PTSD development six months later (79).

The Role of Stress Reduction in Cardiac Rehabilitation

As indicated, heart diseases and psychiatric diseases such as depression and anxiety disorders frequently occur together. There exists a large body of evidence suggesting that experience of severe or chronic stress and the resulting pathophysiological reactions (dysregulation of the HPA axis and ANS and resulting inflammatory processes) might mediate this epidemiological association. Therefore, it is of particular importance to integrate stress reduction measures into the rehabilitation of cardiovascular patients in order to improve the prognosis of both heart disease and mental comorbidities.

Currently, physical exercise, nonspecific psychological interventions, and a Mediterranean diet (MD) are the only secondary prevention strategies for cardiac rehabilitation patients that aim to improve stress management and mental health (80).

A large body of research shows that exercise training in cardiac patients reduces cardiovascular mortality and improves the quality of life (81). Furthermore, evidence

indicates that exercise has anti-depressive, anxiolytic, and stress-reducing effects (82).

One explanation for these beneficial effects is that exercise-based cardiac rehabilitation can reverse chronic dysregulation of the ANS (a component of the body's stress system) by improving sympathovagal balance (83–86).

The MD is one of the best studied diets for cardiovascular health, and an integral component of cardiac rehabilitation programs. The diet is characterized by high consumption of fresh fruit, vegetables, olive oil, nuts, and unprocessed cereals; moderate consumption of sea foods, dairy products, and red wine; and low consumption of red meat (87). Besides the health-promoting effect on the cardiovascular system, there is also strong evidence that the MD has a positive effect on mental health. A randomized controlled trial (RCT) by Parletta et al. found that a three-month intervention of biweekly food hampers and MD cooking workshops with additional fish oil intake, improved mental health and significantly reduced depressive symptoms of the participants when compared to a control group. (88). These results are supported by further studies (89).

The effect of psychological interventions such as stress management and lifestyle change interventions in cardiac rehabilitation is less certain. In a review by Albus et al., the authors summarized the results of 20 RCTs. They compared the effect of exercise-based cardiac rehabilitation with and without additional psychological interventions on depression, anxiety, quality of life, cardiovascular morbidity, cardiovascular mortality, and total mortality in CVD patients. Additional psychological interventions for a lifestyle change or stress management did not have a significant effect on any of the given outcomes (90).

Due to the high therapeutic importance of stress reduction and mental health in heart patients, rehabilitation measures directly aimed at promoting mental health are currently in demand. So called mind-body interventions such as yoga and meditation seem promising for cardiac patients, as they have been shown to reduce symptoms of depression, anxiety and stress in the general population (91).

In the following section, the existing state of research on the effects of yoga and Transcendental Meditation (TM) on psychological parameters will be examined in greater detail.

Yoga, Stress Reduction and Psychological Health

Yoga is an ancient method to promote physical, mental, and emotional health. It combines structured physical postures (asana), breathing techniques (pranayama), and meditation (dhyana) to stimulate a sense of well-being (92,93).

The beneficial effects of regular yoga practice on stress management and psychological health have been well documented. Common gym yoga classes twice a week for 16 weeks significantly reduced stress and improved psychological health in people reporting moderate-to-high levels of stress. When compared to a control group that did not perform any yoga, the yoga practitioners reported less stress, anxiety, depression, insomnia, and distress avoidance behaviors. Even an eight-week yoga intervention had similar significant effects on the experience of stress and subjective health (94). Another study by Rocha et al. found comparable effects on stress, anxiety, and depression when comparing yoga practice to conventional physical exercises in healthy men. They assessed the psychological parameters before and after six months of practice. After the intervention period, the yoga group showed significantly lower scores in depressive symptoms, anxiety, and stress levels when compared to the control group. Importantly, the effects observed in the yoga group do not seem to be solely attributable to the physical practice, as all participants performed regular physical exercise (95). Many other studies confirm the positive effect of yoga on psychological parameters such as depression, anxiety, and the experience of stress (96,97).

The psychological effects of yoga appear to rely on a down-regulation of the body's stress system. Due to the focused attention on the breath and the various movement patterns, yoga exercises seem to cause a shift toward PNS dominance; thereby leading to a down-regulation of the HPA axis and the SNS (92–94).

Furthermore, studies suggest that yoga reverses the negative impact of stress on the immune system. It has been shown that yoga can decrease inflammatory markers like CRP and pro-inflammatory cytokines such as IL-6 in healthy individuals (98), as well as in patients with heart failure (99).

Besides yoga's impact on psychological health, its balancing effects on the HPA axis and the ANS as well as its anti-inflammatory properties make yoga an effective component in the primary and secondary prevention of CVDs (99–101)

TM, Stress Reduction and Psychological Health

Meditation is an umbrella term for a family of emotional and attentional regulatory practices. Meditation practices can be classified into two main styles: focused attention or concentrative meditation, and open monitoring or mindfulness meditation. During focused attention, the practitioner attempts to concentrate on an object (e.g.,, the own breathing sensation), whereas during open monitoring one focuses on the monitoring process itself (e.g., how the attention shifts from one sensation, thought, etc., to another) (102,103). In TM, a mantra–a word without meaning–is silently repeated in order to reach a state of effortless awareness. TM practitioners are advised to meditate for 15 to 20 minutes twice a day. As the TM technique centers on an object–the mantra–it can be broadly included in the focused attention or concentrative meditation category. However, as TM primarily concerns letting go of any effort to maintain the concentration to experience thought- free 'pure consciousness', a third category called automatic self-transcending has been proposed. In contrast to the other two categories, automatic self-transcending is characterized by the absence of focus or effort. The effortless use of a sound without meaning allows the mind to achieve a state of deep relaxation (104).

An important advantage of TM is its standardized format, which is easily accessible even for people without any previous experience with mind-body interventions. Due to its relaxing qualities and straight forward methodology, TM can be seen as a behavioral stress reduction program that incorporates a mind-body approach (105).

TM's beneficial effects on psychological health have been demonstrated in several studies. A RCT by Elder et al. evaluated the effect of TM on perceived stress, depressive symptoms, and burnout in teachers at a therapeutic school. After four months of intervention, the TM group showed significant reductions in each of the three outcome variables when compared to a wait list control group. The observed effect sizes were medium to large, with the largest effect on perceived stress (106). Another RCT that assessed the effects of TM on psychological distress in university students showed comparable results. After three months, the TM group showed significant improvements in total psychological distress, anxiety, depression, anger, and coping ability when compared to the wait list control group (107). Many other studies confirm the beneficial effect of TM on several psychological parameters such

as anxiety (103,108) and perceived stress (105); however, its effect on depressive symptoms is less clear (109).

Furthermore, many studies suggest that TM reduces cardiovascular risk factors like hypertension (110,111) and lowers the risk for mortality, MI, or stroke in CHD patients (110). Therefore, regular TM practice may be clinically useful in the rehabilitation of CVD patients (112).

The underlying mechanisms of the demonstrated impact that TM can have on psychological and cardiovascular parameters still require further investigation. Following the previously described impact of exercise and yoga on the HPA axis and the ANS, some studies found an altered function of the HPA axis within the context of TM. Dougan et al. compared the cortisol awakening response (CAR) of participants who practiced TM for four weeks with a wait list control group. Usually, cortisol levels increase rapidly in the morning upon awakening and decline within an. This physiological response–the CAR–is often atypical in people who suffer from chronic stress and related disorders, such as depression. People with psychological distress most often show higher morning cortisol levels and a higher CAR. During the study, the TM group had significantly lower morning cortisol levels after four weeks and a greater drop in CAR from baseline to week four than the wait list control group (113).

These results suggest that regular TM practice may lead to a down-regulation of the HPA axis and thus could be an effective treatment for chronic stress and its related diseases. However, most existing studies investigating the impact of TM on HPA axis functioning have significant limitations such as unique clinical samples (105) or non-randomized study design (114). To understand the biological mechanisms of concentrative meditation like TM, large RCTs with generalizable samples are needed.

Hypotheses

As indicated, there exists an epidemiological link between cardiovascular and mental illness. The diseases influence each other bidirectionally and worsen the respective prognosis. Chronic or severe stress is a mediator variable for this correlation. Techniques for stress reduction are therefore an integral part of a long-term successful cardiovascular rehabilitation. A reduction of perceived stress and access to effective coping mechanisms improves mental health and thus the prognosis of CVDs. To date, exercise therapy is the only fixed component in cardiovascular rehabilitation programs in Austria that has been proven to contribute to stress reduction and mental health. Further evidence-based measures that specifically contribute to stress reduction and the mental health of CVD patients are in demand. Mind-body interventions such as yoga and TM are among such promising measures. This study aims to contribute to the research into the effectiveness of these therapies in terms of evidence-based medicine (EBM). For this purpose, the psychological effects of a four-week standard rehabilitation were compared to the effects of a four-week standard rehabilitation with additional yoga or TM sessions. Based on the presented state of research, the following hypotheses were derived and examined:

<u>Between-Group Comparison (Standard Therapy vs. TM + Standard Therapy vs. Yoga + Standard Therapy):</u>

1. After four weeks of rehabilitation, the TM and the yoga group show significantly less subjective psychological distress (assessed with the OPD-SQS, ECR-R, BSI-18, ASTS, BDI-II, PSQ-20, PSQI and STAI-6) than the standard therapy group.

2. After four weeks of rehabilitation, the TM and the yoga group show significantly better subjective health (assessed with the SF-36) than the standard therapy group.

3. After four weeks of rehabilitation, the TM and the yoga group show significantly larger improvement in psychological distress (assessed with the OPD-SQS, ECR-R, BSI-18, ASTS, BDI-II, PSQ-20, PSQI and STAI-6) than the standard therapy group.

4. After four weeks of rehabilitation, the TM and the yoga group show significantly larger improvement in subjective health (assessed with the SF-36) than the standard therapy group.

Within-Group Comparison (Baseline vs. After Four Weeks of Rehabilitation)

1. When comparing the baseline test results with the four-week follow-up results, each group will show a significant improvement in subjective psychological distress (assessed with the OPD-SQS, ECR-R, BSI-18, ASTS, BDI-II, PSQ-20, PSQI and STAI-6) and subjective health (assessed with the SF-36).

Material and Methods

Participants

For this study, people who enrolled in a four-week cardiac rehabilitation program at the St. Radegund Rehabilitation Centre were recruited.

The inclusion criteria were as follows:

- Men and women between 40 and 80 years who were admitted to the cardiac rehabilitation center in St. Radegund after MI with ST-elevation (STEMI), after MI without ST-elevation (NSTEMI), after acute coronary syndrome, after coronary artery disease with a percutaneous coronary intervention, or after coronary artery bypass graft.

The exclusion criteria were:

- Patients who must be monitored because of clinical symptoms (subjects with New York Heart Association Functional Classification (NYHA) III, a Mini-Mental score less than 26, or subjects who are not sufficiently mobilized) as well as subjects who regularly perform yoga exercises or any other meditation techniques.

Allocation

After recruitment, participants were randomly assigned to one of three groups.

Control group A received the standard exercise therapy of the rehabilitation center. Intervention group B received TM sessions additional to the standard exercise therapy. Intervention group C received yoga sessions additional to the standard exercise therapy.

Study design

The present study has a 2x3 mixed design with a within-subjects factor *time* and a between-subjects factor *intervention* as shown in Table 1.

		Within-Subjects: Time	
		Baseline	After 4 weeks
Between-Subjects: Intervention	Standard exercise therapy (Group A)		
	Standard exercise therapy + TM (Group B)		
	Standard exercise therapy + Yoga (Group C)		

Table 1: 2x3 mixed design with within-subjects factor time and between-subjects factor intervention. TM = Transcendental meditation.

Study Outcomes

The present data were collected within a pilot study of the Otto Loewi Research Center for Vascular Biology, Immunology and Inflammation. In this study, several surrogate parameters for endothelial function, microbiome and mental well-being as well as EEG data were collected. The present thesis focuses exclusively on the psychometric outcomes of eight standardized self-assessment questionnaires (OPD-SQS, ECR-R, BSI-18, ASTS, BDI-II, PSQ20, PSQI, STAI, and SF-36). For a detailed description of the questionnaires and relevant sub-scores, see the section below.

Material

Operationalized Psychodynamic Diagnostics–Structure Questionnaire Short (OPD-SQS)

The 12-item OPD-Structure Questionnaire was used to assess the structural abilities in personality. With four items per sub-score, the self-survey covers three different dimensions of personality structure: self-perception, relationship model and contact behavior. In terms of content, the self-perception sub-score covers abilities such as self-reflection, emotional differentiation, and emotional tolerance. The relationship model sub-score uses items from the areas of internalization, self-object differentiation, and realistic object perception, and represents relationship experiences combined with corresponding expectations of new relationships. The contact behavior sub-score integrates items from the areas of self-esteem regulation, anticipation, interpersonal contact, and emotional communication. Here, the focus is on interpersonal skills directed at the counterpart combined with aspects of insecurity. Higher scores in the total score are positively correlated with a greater demand for psychological and/or psychiatric therapy (115).

Experiences in Close Relationships–Revised (ECR-R)

The German version of the ECR-R was used to assess adult attachment. This self-reported survey consists of 36 items (Range 1-7), half of which represent attachment anxiety and half of which represent attachment avoidance (116). High scores on the attachment anxiety scale indicate hyper-activating attachment strategies in relevant situations, whereas high scores on the attachment avoidance scale indicate deactivating attachment strategies (117).

Brief Symptom Inventory (BSI-18)

The German version of the BSI-18 was used to assess psychological distress. It is the short form of the Symptom Checklist (SCL-90-R) and a valid self-survey instrument. The BSI-18 consists of 18 items (Range 0-4) and assesses the syndromes of somatization, depression, and anxiety using six items each. Higher scores indicate a higher level of somatization/depression/anxiety in the respective sub-score. The total score is called Global Severity Index (GSI), analogous to the SCL-90-R, and reflects the general psychological strain (118).

Aktuelle Stimmungsskala (ASTS)

The ASTS questionnaire was used to assess the current mood state. It is a shortened version of the “profile of mood state scale” (POMS), and consists of 19 adjectives. Subjects are required to estimate how well an adjective reflects their current feelings on a scale ranging from seven (very strong) to one (not at all). The items are summarized on five different scales. The items can be summed up separately to form five different dimensions: sadness, hopelessness, positive mood, tiredness, and anger. Higher scores in a sub-score indicate higher expression of the respective momentary feeling. Moreover, an overall measure can be formed to describe the current negative mood (119).

Beck’s Depression Inventory-II (BDI-II)

The German version of the BDI-II was used to assess depressive symptoms and their severity. The questionnaire consists of 21 items (Range 0 - 3) about the frequency and/or severity of depressive symptoms, which are aggregated to a total score (Range 0 - 63). A higher score indicates a stronger depressive mood. Various cut-off values are found in the literature as a basis for evaluation. The following values were used for the present study (120,121):

< 14: clinically unremarkable
14-19: mild depressive symptoms
20-28: moderate depressive symptoms
29-63: severe depressive symptoms

Perceived Stress Questionnaire 20 Items Version (PSQ-20)

The PSQ-20 is the validated short version of the Perceived Stress Questionnaire (122) and its German version was used to assess subjectively experienced stress. Its 20 items can be divided into four different sub-scores: worries, tension, joy, and demands. Additionally, all items can be added up to a total score. Higher scores within a sub-score indicate a higher expression of the respective feeling, whereas a higher total score indicates a higher level of perceived stress in general (9).

Pittsburgh Sleep Quality Index (PSQI)

The German version of the PSQI was used to evaluate sleep quality. This is a validated self-report survey consisting of 19 items (Range 0 - 3). These items assess relevant domains of sleep quality over one month, and generate seven different sub-scores: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. In addition to the sub-scores, the sum of scores for the sub-scores yields one global score (Range 0 - 21). Healthy sleepers usually have a total value of no more than five points (124).

6-Item Short Form of the Spielberger State-Trait Anxiety Inventory (STAI-6)

The German version of the STAI-6 was used to assess state anxiety (how anxious one feels at the moment). With six items (Range 1 - 4), a total score can be calculated (Range 20 - 80). Higher scores indicate a higher level of state anxiety (125).

Short Form Health Survey 36 (SF-36)

The German version of the SF-36 was used to evaluate the subjective health of the participants. It is a self-rate questionnaire consisting of 36 items, wherein each item is either a scale itself or represents a part of a scale. The items vary from binary "yes - no" questions to six-level answer scales. The survey covers eight dimensions of subjective health with different item numbers. The dimensions are: physical functioning, bodily pain, role limitations due to physical health problems, role limitations due to personal or emotional problems, general mental health, social functioning, vitality, and general health perceptions. For each sub-score, a score can be calculated (Range 0-100), whereby higher scores define a more favorable health state (126).

Protocol

Upon admission to the rehabilitation clinic, the patients were randomly assigned to one of the three study groups and were required to fill out the questionnaires for the baseline measurement. The control group A received the standard exercise therapy of the rehabilitation center. The intervention group B received TM sessions twice a day in addition to the standard therapy. Intervention group C received yoga sessions twice a day in addition to the standard therapy. After four weeks, when patients were discharged from the rehabilitation clinic, they were given the questionnaires again. For an overview of the study protocol, see Figure 4.

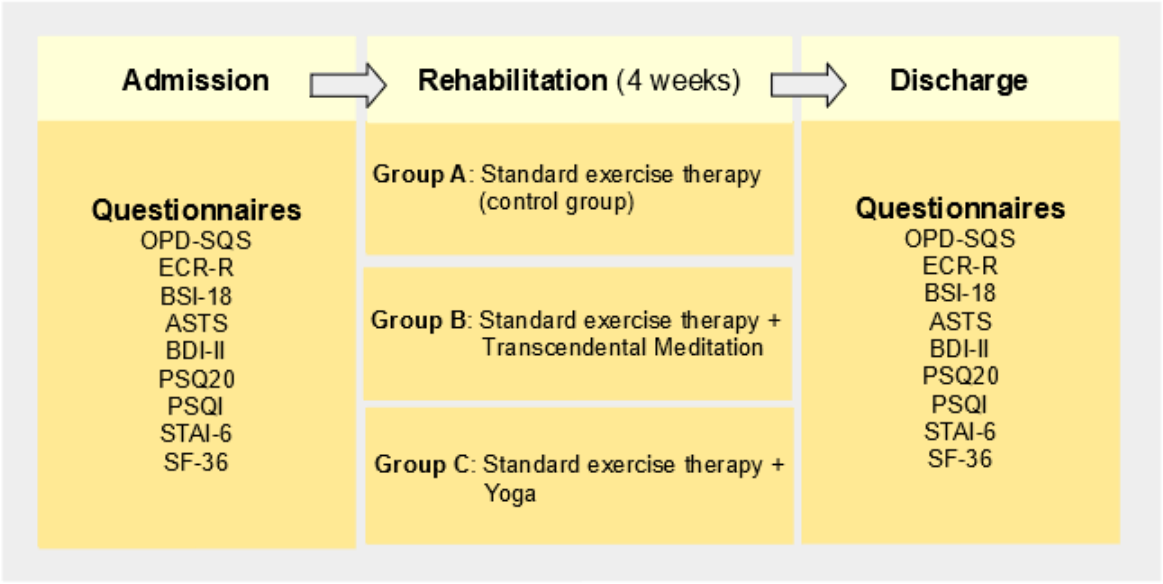

Figure 4: Overview of the study protocol

In addition to the psychometric questionnaires, a number of physiological parameters were collected at both measurement points. To assess vascular functioning, pulse wave velocity, retinal imaging, flow mediated dilatation, and a sit-to-stand test were performed. To detect hormonal changes, scalp hair cortisol was assessed. Furthermore, electroencephalogram (EEG) measurements were taken at baseline and follow-up. These physiological parameters are the subject of further research and will not be discussed in detail below.

Group A: Standard Rehabilitation Exercise Therapy (Control Group)

During the four weeks of cardiac rehabilitation, each patient received an individual therapy plan. On the admission day, all patients had a clinical check-up (general medical history and clinical examination) including electrocardiogram (ECG). According to individual performance, the patients were divided into six preliminary exercise groups. In the first few days, the following examinations were performed: echocardiography, cycle ergometry, and a chest X-ray. Blood was collected and standard laboratory analysis was performed. Afterward, an individual exercise schedule was created for each patient, depending on the performance at the ergometry. Additionally, the patients received physiotherapy sessions (massages, lymphatic drainages, etc.), different educational seminars, psychological assistance, and medical check-ups if necessary (blood analysis, X-rays, ergometry, sonography, 24-hour ECG, and 24-hour blood pressure).

The following sports were available for the patients at the Center for Cardiac Rehabilitation in St. Radegund: bicycle, treadmill, Nordic walking, hiking (in groups), gymnastics, swimming, and strength endurance workouts including weight lifting.

Group B: Standard Rehabilitation Exercise Therapy + TM

At the beginning of the study period, the patients from group B received an introductory lecture about TM, which included general information, the procedure itself, and other techniques of relaxation and personal development.

In this first session, the method was learned by practice, and in the following three meetings additional instructions were given about the correct practice of the TM technique. Each of these four sessions lasted about one to one and a half hours and took place on four consecutive days. In order to correctly learn the method, it was necessary to complete the four meetings in a row.

TM was practiced twice a day for 20 minutes. To guarantee correct performance of TM by the patients, there were two group meetings per week and one individual personal meeting with the TM teacher where questions could be asked.

Group C: Standard Rehabilitation Exercise Therapy + Yoga

Initially, the patients received a 20-minute workshop where they learned how to complete the yoga exercises. After this workshop, yoga sessions were included in the patients' daily timetable. Two yoga sessions were done each day: once in the morning (6:30-6:50) and once in the evening (16:30-16:50); each session lasting for about 20 minutes. During these sessions, the following techniques were used:

- **Asanas** (with a focus on forward bends, inversions, chest openings, neck relaxation and Shavasana)
    - Feet circles
    - Knee to chest pose (Pavanmuktasana)
    - Shoulder circles
    - Neck Release: raise an arm, lower with exhalation and turn the head in the opposite direction
    - Chair Pose (Utkatasana) with hugging
    - Reclining Hand-to-Big-Toe Pose with strap (Supta Padangusthasana)
    - Reclining Abdominal Twist (Jathara Parivartanasana)
    - (Supported) Bridge Pose (Setu Bandhasana)
    - Shavasana: lying on the back with legs spread slightly more than hip-width, arms relaxed to the side, and eyes closed. Usually performed at the end of each yoga session.
- **Body-Scan** (a type of mindful meditation promoting greater awareness of the physical body)
- **Pranayamas:** Alternate Nostril Breathing (alternately inhale with one nostril and exhale with the other while holding the other nostril closed with one finger), Relaxation Breath (breathing through the nose with exhale periods longer than inhale periods), Ujjayi (breathing through the nose with narrowed glottis. The narrowed passage of air creates a "rushing" sound).

Data Analysis

The statistical analysis of the data was carried out using IBM SPSS Statistics, version 26 (127). The relevant scientific publications were researched via the PubMed database and managed using Mendeley (version 1.19.4).

Due to the small sample size of each group (n = 10), the Shapiro-Wilk test was used to test for normal distribution of the dependent variables. If normal distribution was given, single factor analyses of variance (UNIANOVA) were calculated to assess the statistical significance of differences between the three groups (group A vs. group B vs. group C). Three comparisons were made between the groups. First, we compared the average test scores between the groups at baseline (first comparison) and follow-up (second comparison). Furthermore, we calculated the differences between the average scores at baseline and follow-up (follow-up score minus baseline score). Finally, these average differences were compared between the groups (third comparison).

If no normal distribution was given, the non-parametric Kruskal-Wallis test was used to assess the statistical significance of differences between the groups.

To test for statistically significant differences within each group before and after the four-week rehabilitation program, either the t-Test for dependent variables (if normal distribution was given) or the non-parametric Wilcoxon-Test was calculated.

P-values below 0.05 were considered statistically significant.

Results

Descriptive Statistics

A total of 30 patients (7 female) with a mean age of 60 years (*Standard deviation (SD)* = 9.8) participated in this study. After admission, patients were randomly assigned to one of three groups. One person did not take part in the follow-up measurement.

Group A, the control group, received the standard exercise therapy of the rehabilitation center and consisted of 10 patients (1 female) with a mean age of 58.8 years (*SD* = 7.1) and a mean follow-up duration of 26 days (*SD* = 2.7).

Group B received additional TM sessions to the standard exercise therapy and consisted of 10 patients (1 female) with a mean age of 62.1 years (*SD* = 10.4) and a mean follow-up duration of 27 days (*SD* = 0.4). One male participant did not take part in the follow-up measurement.

Group C received additional yoga sessions to the standard exercise therapy and consisted of 10 patients (5 female) with a mean age of 59,2 years (*SD* = 12.1) and a mean follow-up duration of 27 days (*SD* = 3.1).

The descriptive parameters are shown in Table 2.

	n	Gender (female)	Mean Age (years)	Follow-Up Duration (days)
Control Group	10	1 (10%)	58.8	25.78
TM* Group	10	1 (10%)	62.1	26.86
Yoga Group	10	5 (50%)	59.2	27.22
Total	30	7 (23.3 %)	60.03	26.6

*Table 2: Descriptive Parameters. *TM = Transcendental Meditation.*

A Fisher's exact test was calculated to test for differences between the gender distribution among the three groups. No significant group differences regarding the gender distribution could be observed (p = .15).
Furthermore, a single factor analysis of variance showed no significant differences between the groups in terms of average age (F (2, 27) = .32, p = .73, $\eta 2$ = .02), nor did a Kruskal-Wallis test for the follow-up duration (Chi-squares (2) = .85, p = .64).

Operationalized Psychodynamic Diagnostics–Structure Questionnaire Short (OPD-SQS)

The OPD-SQS was used to assess the structural abilities in personality. No significant differences between the three groups in any of the sub-scores or the total score could be observed - neither before nor after the four-week rehabilitation program (see Table 3).

	BASELINE				
	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
Self-perception Mean (SD)	1.8 (2.2)	2.1 (2.5)	2.1 (2.7)	Chi-squares (2) = .07, *p* = .97	
Contact behavior Mean (SD)	4.3 (2.2)	4.2 (1.6)	3.4 (2.5)		F (2,27) = .53, *p* = .60, η2 = .04
Relationship model Mean (SD)	6.2 (2.6)	4.7 (3.2)	5.2 (3.2)		F (2,27) = .76, *p* = .48, η2 = .05
Total score Mean (SD)	12.3 (4.2)	10.6 (7)	11 (6.2)		F (2,27) = .33, *p* = .72, η2 = .02
	AFTER 4 WEEKS				
Self-perception Mean (SD)	1.5 (2.3)	1.1 (2.5)	1.5 (2.8)	Chi-squares (2) = .77, *p* = .70	
Contact behavior Mean (SD)	3.8 (2.4)	2.8 (2.9)	3 (2.3)		F (2,25) = .43, *p* = .66, η2 = .03
Relationship model Mean (SD)	5.1 (3.2)	3.9 (3.8)	4.5 (3.6)		F (2,26) = .28, *p* = .76, η2 = .02
Total score Mean (SD)	10.4 (5.9)	7.3 (8.5)	8.5 (8)	Chi-squares (2) = 1.82, *p* = .41	

*Table 3: Between-group comparisons of the mean OPD-SQS sub-score and total scores. * Kruskal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation.*

Likewise, there were no significant betwee-group differences regarding the point differences between the baseline and follow-up measurement (see Table 4).

	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
Self-perception Mean difference (SD)	- 0.3 (0.8)	- 0.8 (1.5)	- 0.6 (1.2)	Chi-squares (2) = .28, p = .87	
Contact behavior Mean difference (SD)	- 0.5 (2)	- 1.4 (2.8)	- 0.3 (1.4)	Chi-squares (2) = 1.10, p = .58	
Relationship model Mean difference (SD)	- 1.1 (2)	- 0.8 (3.1)	- 0.1 (2.1)	Chi-squares (2) = 1.47, p = .48	
Total score Mean difference (SD)	- 1.9 (3.5)	- 2.8 (6.6)	- 0.8 (3.1)		F (2,24) = .41, p = .67, η2 = .03

*Table 4: Between-group comparisons of the differences in OPD-SQS sub-score and total scores from baseline to four-week follow-up. * Kruskal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation.*

Similar to the between-group results, there were no significant differences before and after the four-week rehabilitation program within each group (see Table 5).

CONTROL GROUP				
	Baseline	After 4 weeks	Wilcoxon Test*	T-test
Self-perception Mean (SD)	1.8 (2.2)	1.5 (2.3)	z = -1.13, *p* = .5	
Contact behavior Mean (SD)	4.3 (2.2)	3.8 (2.4)		t = .81, *p* = .44
Relationship model Mean (SD)	6.2 (2.6)	5.1 (3.2)		t = 1.77, *p* = .11
Total score Mean (SD)	12.3 (4.2)	10.4 (5.9)		t = 1.73, *p* = .12
TM GROUP**				
Self-perception Mean (SD)	2.1 (2.5)	1.1 (2.5)	z = -1.34, *p* = .50	
Contact behavior Mean (SD)	4.2 (1.6)	2.8 (2.9)		t = 1.55, *p* = .16
Relationship model Mean (SD)	4.7 (3.2)	3.9 (3.8)		t = .76, *p* = .47
Total score Mean (SD)	10.6 (7)	7.3 (8.5)	z = -.94, *p* = .42	
YOGA GROUP				
Self-perception Mean (SD)	2.1 (2.7)	1.5 (2.8)	z = -1.51, *p* = .25	
Contact behavior Mean (SD)	3.4 (2.5)	3 (2.3)		t = .71, *p* = .50
Relationship model Mean (SD)	5.2 (3.2)	4.5 (3.6)		t = .15, *p* = .88
Total score Mean (SD)	11 (6.2)	8.5 (8)		t = .75, *p* = .48

*Table 5: Within-group comparisons of the mean OPD-SQS sub-score and total scores. * Wilcoxon tests were calculated for data with no normal distribution. ** TM = Transcendental Meditation.*

Experiences in Close Relationships–Revised (ECR-R)

The ECR-R was used to evaluate adult attachment with its two sub-scores attachment anxiety and attachment avoidance. No significant differences between the three groups in any of the sub-scores could be observed - neither before nor after the 4 weeks rehabilitation program (see Table 6).

	BASELINE				
	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
Attachment anxiety Mean (SD)	11.43 (3.95)	13.13 (4.88)	13.36 (7.2)		F (2,19) = .56, *p* = .58, η2 = .06
Attachment Avoidance Mean (SD)	12.71 (7.37)	14.89 (9.43)	14.22 (7.29)		F (2,20) = .20, *p* = .82, η2 = .02
	AFTER 4 WEEKS				
Attachment anxiety Mean (SD)	10 (4.24)	9.50 (6.68)	10.22 (6.81)	Chi-squares (2) = 1.36, *p* = .52	
Attachment Avoidance Mean (SD)	12.43 (7.12)	14 (10.65)	13.09 (7.63)	Chi-squares (2) = .10, *p* = .95	

Table 6: Between-group comparisons of the mean ECR-R sub-score scores. Kruskal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation.*

Likewise, there were no significant between-group differences regarding the point differences between the baseline and follow-up measurement (see Table 7).

	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
Attachment anxiety Mean difference (SD)	- 0.9 (4.2)	- 2.7 (5.2)	- 5.0 (6.0)		F (2,17) = 1.06, *p* = .37, η2 = .11
Attachment Avoidance Mean difference (SD)	- 0.3 (10.6)	1.1 (8.2)	- 2.0 (3.5)	Chi-squares (2) = .23, *p* = .89	

Table 7: Between-group comparisons of the differences in ECR-R sub-score scores from baseline to four-week follow-up. Krukal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation.*

Similar to the between-group results, there were no significant differences before and after the four-week rehabilitation program within each group (see Table 8).

	CONTROL GROUP			
	Baseline	After 4 weeks	Wilcoxon Test*	T-test
Attachment anxiety Mean (SD)	11.43 (3.95)	10 (4.24)		t = .54, *p* = .61
Attachment Avoidance Mean (SD)	12.71 (7.37)	12.43 (7.12)		t = .08, *p* = .94
	TM GROUP**			
Attachment anxiety Mean (SD)	13.13 (4.88)	9.50 (6.68)	z = -1.16, *p* = .28	
Attachment Avoidance Mean (SD)	14.89 (9.43)	14 (10.65)	z = 0.0, *p* = 1.0	
	YOGA GROUP			
Attachment anxiety Mean (SD)	13.36 (7.2)	10.22 (6.81)	z = -1.83, *p* = .13	
Attachment Avoidance Mean (SD)	14.22 (7.29)	13.09 (7.63)		t = 1.53, *p* = .18

Table 8: Within-group comparisons of the mean ECR-R sub-score scores. Wilcoxon tests were calculated for data with no normal distribution; ** TM = Transcendental Meditation.*

Brief Symptom Inventory (BSI-18)

Due to missing values, no sub-score or total scores could be calculated for the BSI-18. At baseline measurement, only seven out of 30 questionnaires could be scored, and at the second measurement–after four weeks of rehabilitation–none of the tests were filled out sufficiently.

Aktuelle Stimmungsskala (ASTS)

The ASTS questionnaire was used to assess the current mood state on five different sub-scores: sadness, hopelessness, positive mood, tiredness and anger. Higher scores in a sub-score indicate a higher expression of the respective momentary feeling. No significant differences between the three groups in any of the sub-scores could be observed - neither before nor after the four-week rehabilitation program (see Table 9).

	BASELINE				
	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
sadness Mean (SD)	6.80 (5.37)	6.33 (4.56)	6.10 (4.48)	Chi-squares (2) = .14, *p* = .93	
hopelessness Mean (SD)	6.70 (4.79)	5.50 (4.17)	6.10 (4.75)	Chi-squares (2) = .49, *p* = .78	
positive mood Mean (SD)	27.40 (5.62)	29.40 (6.54)	24.60 (8.28)		F (2,27) = 1.22, *p* = .31, η2 = .08
tiredness Mean (SD)	16.10 (6.61)	14.20 (8.63)	12.33 (7.70)		F (2,26) = .57, *p* = .57, η2 = .04
anger Mean (SD)	8.70 (5.79)	5.40 (4.86)	4.80 (2.70)	Chi-squares (2) = 5.45 , *p* = .07	
current negative mood Mean (SD)	50.20 (16.66)	43.11 (14.90)	47.44 (21.66)		F (2,25) = .37, *p* = .69, η2 = .03
	AFTER 4 WEEKS				
sadness Mean (SD)	5.67 (4.87)	4.25 (2.44)	5.00 (3.39)	Chi-squares (2) = .39, *p* = .83	
hopelessness Mean (SD)	5.56 (4.16)	4.00 (1.77)	4.10 (2.60)	Chi-squares (2) = .71, *p* = .70	
positive mood Mean (SD)	30.44 (4.93)	35.25 (4.50)	28.75 (8.16)		F (2,22) = 2.51, *p* = .10, η2 = .19
tiredness Mean (SD)	10.13 (5.94)	8.89 (6.27)	7.40 (4.06)	Chi-squares (2) = 1.71, *p* = .44	
anger Mean (SD)	5.67 (4.98)	4.78 (3.63)	4.30 (2.75)	Chi-squares (2) = 1.06, *p* = .60	
current negative mood Mean (SD)	38.75 (12.09)	26.00 (8.62)	36.25 (16.04)		F (2,20) = 2.05, *p* = .16, η2 = .17

Table 9: Between-group comparisons of the mean ASTS sub-score and current negative mood scores. Kruskal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation.*

Concerning the point differences between the baseline and follow-up measurement, a Kruskal-Wallis test showed a significant difference between the groups for the anger-sub-score. A post-hoc test (Dunn-Bonferroni) was performed and showed that the significant differences exist only between the control and yoga group (z = 2.40, p = .049). Participants who practiced yoga twice a day during a four-week rehabilitation program, had less reduction in feelings of anger after four weeks than participants who only participated in the rehabilitation program.

	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
sadness Mean difference (SD)	- 1.3 (3.5)	- 0.9 (1.3)	- 0.7 (1.5)	Chi-squares (2) = .41, *p* = .82	
hopelessness Mean difference (SD)	- 1.1 (3)	- 0.3 (1.4)	- 2.0 (3.3)	Chi-squares (2) = 1.28, *p* = .53	
positive mood Mean difference (SD)	2.1 (5.3)	5.6 (4.8)	4.4 (6.3)		F (2,22) = .90, *p* = .42, η2 = .08
tiredness Mean difference (SD)	- 7.1 (6.4)	- 4.8 (10.4)	- 5.2 (6.1)	Chi-squares (2) = 1.26, *p* = .53	
anger Mean difference (SD)	- 2.8 (3.2)	- 0.9 (3)	- 0.5 (1.6)	**Chi-squares (2) = 6.28, *p* = .04**	
current negative mood Mean difference (SD)	- 12.8 (15.9)	- 9.2 (13.8)	- 12.0 (13.2)	Chi-squares (2) = .03, *p* = .99	

Table 10: Between-group comparisons of the differences in ASTS sub-score and current negative mood scores from baseline to four-week follow-up. Kruskal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation; Significant results are printed in bold.*

Concerning the within-group comparisons, the control group showed significantly lower scores in tiredness and anger after the four-week rehabilitation program compared to the baseline scores. In the TM group, a significant raise in positive mood could be observed after the four-week program. The yoga group had significantly lower scores in tiredness and current negative mood at the end of the rehabilitation program when compared to the baseline scores (see Table 11).

	CONTROL GROUP			
	Baseline	After 4 weeks	Wilcoxon Test*	T-test
sadness Mean (SD)	6.80 (5.37)	5.67 (4.87)	z = -1.02, *p* = .38	
hopelessness Mean (SD)	6.70 (4.79)	5.56 (4.16)	z = -1,11, *p* = .38	
positive mood Mean (SD)	27.40 (5.62)	30.44 (4.93)		t = -1,21, *p* = .26
tiredness Mean (SD)	16.10 (6.61)	10.13 (5.94)	**z = -2.52, *p* = .01**	
anger	8.70	5.67	**z = -2.38, *p* = .02**	

Mean (SD)	(5.79)	(4.98)		
current negative mood Mean (SD)	50.20 (16.66)	38.75 (12.09)		t = 2.27, *p* = .06
		TM GROUP**		
sadness Mean (SD)	6.33 (4.56)	4.25 (2.44)	z =-1.51, *p* = .25	
hopelessness Mean (SD)	5.50 (4.17)	4.00 (1.77)	z = -.54, *p* = .75	
positive mood Mean (SD)	29.40 (6.54)	35.25 (4.50)		**t = -3.31, *p* = .01**
tiredness Mean (SD)	14.20 (8.63)	8.89 (6.27)	z = -1.35, *p* = .22	
anger Mean (SD)	5.40 (4.86)	4.78 (3.63)	z = -.73, *p* = .63	
current negative mood Mean (SD)	43.11 (14.90)	26.00 (8.62)		t = 1.62, *p* = .17
		YOGA GROUP		
sadness Mean (SD)	6.10 (4.48)	5.00 (3.39)	z = -1.29, *p* = .38	
hopelessness Mean (SD)	6.10 (4.75)	4.10 (2.60)	z = -1.80, *p* = .13	
positive mood Mean (SD)	24.60 (8.28)	28.75 (8.16)		t = -1.60, *p* = .09
tiredness Mean (SD)	12.33 (7.70)	7.40 (4.06)	**z = -2.21, *p* = .03**	
anger Mean (SD)	4.80 (2.70)	4.30 (2.75)	z = -.82, *p* = .75	
current negative mood Mean (SD)	47.44 (21.66)	36.25 (16.04)		**t = 2.57, *p* = .04**

Table 11: Within-group comparisons of the mean ASTS sub-score and current negative mood scores. Wilcoxon tests were calculated for data with no normal distribution; ** TM = Transcendental Meditation: Significant results are printed in bold.*

Beck's Depression Inventory- II (BDI-II)

The BDI-II evaluated depressive symptoms and their severity. A higher score indicates a stronger depressive mood. No significant differences between the three groups in the total scores could be observed - neither before nor after the four-week rehabilitation program (see Table 12).

	BASELINE				
	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
total score Mean (SD)	7.40 (5.62)	8.60 (7.06)	5.00 (4.07)		F (2,25) = .86, *p* = .43, η2 = .07
	AFTER 4 WEEKS				
total score Mean (SD)	3.40 (3.24)	4.25 (5.18)	4.50 (8.17)		F (2,21) = .10, *p* = .91, η2 = .01

Table 12: Between-group comparisons of the mean BDI-II total scores. Kruskal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation.*

Likewise, there were no significant between-group differences regarding the point differences between the baseline and follow-up measurement (see Table 13).

	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
total score Mean difference (SD)	- 4.0 (3.5)	- 5.6 (3.5)	- 3.8 (3.6)		F (2,19) = .59, *p* = .56, η2 = .06

Table 13: Between-group comparisons of the differences in BDI-II total scores from baseline to four-week follow-up. Kruskal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation*

Concerning the within-group comparisons the control group, as well as the meditation group showed a significantly lower total score after the four-week rehabilitation program compared to the baseline score. Total scores in the yoga group did not differ significantly before and after the four weeks of intervention (see Table 14).

	CONTROL GROUP			
	Baseline	After 4 weeks	Wilcoxon Test*	T-test
total score Mean (SD)	7.40 (5.62)	3.40 (3.24)	**z = -2.38, *p* = .02**	
	TM GROUP**			
total score Mean (SD)	8.60 (7.06)	4.25 (5.18)	**z =-2.53, *p* = .01**	

		YOGA GROUP	
total score Mean (SD)	5.00 (4.07)	4.50 (8.17)	z = -1.83 , *p* = .13

Table 14: Within-group comparisons of the mean BDI-II total scores. Wilcoxon tests were calculated for data with no normal distribution; ** TM = Transcendental Meditation; Significant results are printed in bold.*

Perceived Stress Questionnaire 20 Items Version (PSQ20)

The German version of the PSQ20 was used to assess subjectively experienced stress on four different sub-scores (worries, tension, joy, and demands) and a total score. Higher scores in a sub-score indicate a higher expression of the respective feeling, whereas a higher total score indicates a higher level of perceived stress in general. No significant differences between the three groups in the sub-score or total scores could be observed - neither before nor after the four-week rehabilitation program (see Table 15).

			BASELINE		
	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
worries Mean (SD)	28.00 (29.11)	24.00 (25.38)	22.00 (16.94)	Chi-squares (2) = .27, *p* = .88	
tension Mean (SD)	32.00 (20.80)	32.00 (28.60)	34.81 (20.76)	Chi-squares (2) = .51, *p* = .78	
joy Mean (SD)	65.33 (22.18)	73.33 (22.22)	62.00 (22.67)		F (2,27) = .68, *p* = .52, η2 = .05
demands Mean (SD)	34.07 (27.37)	19.26 (12.22)	26.67 (19.52)	Chi-squares (2) = 1.97, *p* = .39	
total score Mean (SD)	30.56 (23.69)	22.59 (17.08)	31.67 (15.58)	Chi-squares (2) = 2.07, *p* = .37	
			AFTER 4 WEEKS		
worries Mean (SD)	21.48 (22.55)	13.33 (12.47)	14.81 (15.91)	Chi-squares (2) = .76 , *p* = .70	
tension Mean (SD)	19.33 (17.05)	14.81 (12.81)	19.26 (20.40)	Chi-squares (2) = .19, *p* = .91	
joy Mean (SD)	75.56 (15.63)	90.37 (14.57)	78.00 (17.79)	Chi-squares (2) = 4.97, *p* = .08	
demands Mean (SD)	22.50 (22.23)	15.56 (15.28)	15.56 (15.63)		F (2,23) = .42, *p* = .66, η2 = .04
total score Mean (SD)	23.33 (20.63)	13.33 (11.21)	17.50 (16.79)		F (2,20) = .71, *p* = .51, η2 = .66

*Table 15: Between-group comparisons of the mean PSQ20 sub-score and total scores. * Kruskal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation.*

Likewise, there were no significant between-group differences regarding the point differences between the baseline and follow-up measurement (see Table 16).

	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
worries Mean difference (SD)	- 8.9 (10.5)	- 7.4 (14.3)	- 7.4 (10.2)	Chi-squares (2) = .57, *p* = .75	
tension Mean difference (SD)	- 12.7 (10.2)	- 16.3 (23.6)	- 15.0 (15.8)		F (2,24) = .11, *p* = .90, η2 = .01
joy Mean difference (SD)	11.9 (15.2)	14.8 (15.9)	16.0 (27.8)		F (2,25) = .10, *p* = .91, η2 = .01
demands Mean difference (SD)	- 10.8 (15.1)	- 0.8 (13.5)	- 10.8 (11.2)		F (2,21) = 1.49, *p* = .25, η2 = .12
total score Mean difference (SD)	- 12.2 (12.1)	- 7.1 (10.5)	- 14.5 (12.5)	Chi-squares (2) = 2.61, *p* = .27	

*Table 16: Between-group comparisons of the difference in PSQ20 sub-score and total scores from baseline to four-week follow-up. * Kruskal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation.*

Concerning the within-group comparisons, the control group showed a significantly lower score in tension and a significantly higher score in joy after the four-week rehabilitation program compared to the baseline score. The meditation group also had significantly higher scores in joy after the four-week program and a significantly lower total score. Participants of the yoga group had significantly lower scores in tension, demands, and in total after the rehabilitation, when compared to their baseline scores (see Table 17).

	CONTROL GROUP			
	Baseline	After 4 weeks	Wilcoxon Test*	T-test
worries Mean (SD)	28.00 (29.11)	21.48 (22.55)	z = -2,05, *p* = .06	
tension Mean (SD)	32.00 (20.80)	19.33 (17.05)	**z = -2,54, *p* = .01**	

joy	65.33	75.56		**t = -2.34, *p* = .047**
Mean (SD)	(22.18)	(15.63)		
demands	34.07	22.50	z = -1.81, *p* = .13	
Mean (SD)	(27.37)	(22.23)		
total score	30.56	23.33	z = -1.18, *p* = .30	
Mean (SD)	(23.69)	(20.63)		
TM GROUP**				
worries	24.00	13.33		t = 1.55, *p* = .16
Mean (SD)	(25.38)	(12.47)		
tension	32.00	14.81	z = -2.0 , *p* = .06	
Mean (SD)	(28.60)	(12.81)		
joy	73.33	90.37	**z = -2.13, *p* = .047**	
Mean (SD)	(22.22)	(14.57)		
demands	19.26	15.56		t = .17, *p* = .87
Mean (SD)	(12.22)	(15.28)		
total score	22.59	13.33	**z = -2.37, *p* = .02**	
Mean (SD)	(17.08)	(11.21)		
YOGA GROUP				
worries	22.00	14.81	z = -1.93, *p* = .09	
Mean (SD)	(16.94)	(15.91)		
tension	34.81	19.26	**z = -2.12, *p* = .047**	
Mean (SD)	(20.76)	(20.40)		
joy	62.00	78.00		t = -1.82, *p* = .10
Mean (SD)	(22.67)	(17.79)		
demands	26.67	15.56		**t = 2.73, *p* = .03**
Mean (SD)	(19.52)	(15.63)		
total score	31.67	17.50		**t = 3.07, *p* = .02**
Mean (SD)	(15.58)	(16.79)		

*Table 17: Within-group comparisons of the mean PSQ20 sub-score and total scores. * Wilcoxon tests were calculated for data with no normal distribution; ** TM = Transcendental Meditation; Significant results are printed in bold*

Pittsburgh Sleep Quality Index (PSQI)

To evaluate sleep quality the PSQI with its seven different sub-scores (subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction) and a total score was used. Healthy sleepers usually have a total value of no more than five points. No significant differences between the three groups in the sub-score or total scores could be observed - neither before nor after the four-week rehabilitation program (see Table 13).

	BASELINE				
	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
subjective sleep quality Mean (SD)	1.00 (0.82)	1.00 (0.47)	1.40 (0.70)	Chi-squares (2) = 2.99, *p* = .22	
sleep latency Mean (SD)	1.90 (1.20)	2.10 (1.20)	2.50 (2.22)	Chi-squares (2) = .17, *p* = .92	
sleep duration Mean (SD)	0.70 (0.95)	0.30 (0.67)	0.60 (0.84)	Chi-squares (2) = 1.61, *p* = .45	
habitual sleep efficiency Mean (SD)	0.44 (0.73)	1.00 (0.82)	1.10 (1.29)	Chi-squares (2) = 2.27, *p* = .34	
sleep disturbances Mean (SD)	1.30 (0.68)	1.33 (0.50)	1.22 (0.44)	Chi-squares (2) = .35, *p* = .87	
use of sleeping medication Mean (SD)	0.00 (0.00)	0.90 (1.45)	0.40 (0.97)	Chi-squares (2) = 3.38, *p* = .18	
daytime dysfunction Mean (SD)	1.11 (0.93)	0.70 (0.68)	0.80 (0.63)	Chi-squares (2) = 1.08, *p* = .59	
total score Mean (SD)	5.75 (2.61)	7.22 (4.27)	7.00 (4.36)	Chi-squares (2) = .40, *p* = .83	
	AFTER 4 WEEKS				
subjective sleep quality Mean (SD)	1.10 (0.57)	0.89 (0.60)	0.60 (0.70)	Chi-squares (2) = 3.29, *p* = .19	
sleep latency Mean (SD)	1.70 (1.42)	1.22 (1.20)	1.20 (1.62)	Chi-squares (2) = 1.07, *p* = .59	
sleep duration Mean (SD)	0.80 (0.63)	0.70 (1.06)	0.20 (0.63)	Chi-squares (2) = 5.20, *p* = .07	
habitual sleep efficiency Mean (SD)	1.10 (1.10)	0.67 (1.00)	0.50 (1.08)	Chi-squares (2) = 2.46, *p* = .30	
sleep disturbances Mean (SD)	1.20 (0.42)	1.22 (0.44)	1.22 (0.44)	Chi-squares (2) = .02, *p* = 1.0	
use of sleeping medication Mean (SD)	0.00 (0.00)	0.33 (1.00)	0.40 (0.97)	Chi-squares (2) = 2.02, *p* = .59	
daytime dysfunction Mean (SD)	0.60 (0.52)	0.11 (0.33)	0.20 (0.42)	Chi-squares (2) = 5.95, *p* = .07	

total score Mean (SD)	6.5 (3.10)	4.89 (2.15)	4.67 (4.61)	Chi-squares (2) = 3.0, *p* = .23	

*Table 18: Between-group comparisons of the mean PSQI sub-score and total scores. * Kruskal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation*

Concerning the point differences between the baseline and follow-up measurement, a Kruskal-Wallis test showed a significant difference between the groups for subjective sleep quality and the PSQI total score. Post-hoc tests (Dunn-Bonferroni) were performed and showed that the significant differences in subjective sleep quality (z = -2.884, p = .01) and the total score (z = -2.937, p = .01) exist only between the control and yoga group. Participants who practiced yoga twice a day during a four-week rehabilitation program had higher improvements in subjective and overall sleep quality than participants who only participated in the rehabilitation program.

	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
subjective sleep quality Mean difference (SD)	0.1 (0.7)	- 0.1 (0.6)	- 0.8 (0.4)	**Chi-squares (2) = 9.33, *p* = .01**	
sleep latency Mean difference (SD)	- 0.2 (1.5)	- 0.7 (1.5)	- 1.3 (1.5)	Chi-squares (2) = 3.04, *p* = .22	
sleep duration Mean difference (SD)	0.1 (1)	0.4 (1.2)	- 0.4 (0.7)	Chi-squares (2) = 3.22, *p* = .20	
habitual sleep efficiency Mean difference (SD)	0.7 (1.1)	- 0.2 (1.2)	- 0.6 (1.1)	Chi-squares (2) = 4.70, *p* = .10	
sleep disturbances Mean difference (SD)	- 0.1 (0.6)	- 0.1 (0.4)	- 0.0 (0.5)	Chi-squares (2) = .29, *p* = .87	
use of sleeping medication Mean difference (SD)	0.0 (0.0)	- 0.3 (1.0)	- 0.0 (0.0)	Chi-squares (2) = 2.22, *p* = .33	
daytime dysfunction Mean difference (SD)	- 0.4 (0.7)	- 0.6 (0.5)	- 0.6 (0.7)	Chi-squares (2) = .52, *p* = .77	

total score Mean difference (SD)	0.8 (2.9)	- 1.8 (4.7)	- 4.0 (2.6)	**Chi-squares (2) = 8.67, *p* = .01**

*Table 19: Between-group comparisons of the differences in PSQI sub-score and total scores from baseline to four-week follow-up. * Kruskal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation; significant results are printed in bold.*

The within-group comparisons showed significant changes from baseline scores to after four weeks only in the yoga group: participants reported a significantly lower score for subjective sleep quality, sleep latency, and total score (see Table 20).

	CONTROL GROUP			
	Baseline	After 4 weeks	Wilcoxon Test*	T-test
subjective sleep quality Mean (SD)	1.00 (0.82)	1.10 (0.57)	z = -.45, *p* = 1.0	
sleep latency Mean (SD)	1.90 (1.20)	1.70 (1.42)		t = .41, *p* = .69
sleep duration Mean (SD)	0.70 (0.95)	0.80 (0.63)	z = -.33, *p* = 1.0	
habitual sleep efficiency Mean (SD)	0.44 (0.73)	1.10 (1.10)	z = -1.66, *p* = .19	
sleep disturbances Mean (SD)	1.30 (0.68)	1.20 (0.42)	z = -.58, *p* = 1.0	
use of sleeping medication Mean (SD)	0.00 (0.00)	0.00 (0.00)	z = 0.0, *p* = 1.0	
daytime dysfunction Mean (SD)	1.11 (0.93)	0.60 (0.52)	z = -1.63, *p* = .25	
total score Mean (SD)	5.75 (2.61)	6.5 (3.10)		t = -.73, *p* = .49
	TM GROUP**			
subjective sleep quality Mean (SD)	1.00 (0.47)	0.89 (0.60)	z = -.58, *p* = 1.0	
sleep latency Mean (SD)	2.10 (1.20)	1.22 (1.20)	z = -1.29, *p* = .38	

sleep duration Mean (SD)	0.30 (0.67)	0.70 (1.06)	z = -1.07, *p* = .50
habitual sleep efficiency Mean (SD)	1.00 (0.82)	0.67 (1.00)	z = -.54, *p* = .78
sleep disturbances Mean (SD)	1.33 (0.50)	1.22 (0.44)	z = -1.0, *p* = 1.0
use of sleeping medication Mean (SD)	0.90 (1.45)	0.33 (1.00)	z = -1.0, *p* = 1.0
daytime dysfunction Mean (SD)	0.70 (0.68)	0.11 (0.33)	z = -2.24, *p* = .06
total score Mean (SD)	7.22 (4.27)	4.89 (2.15)	z = -.85, *p* = .49
			YOGA GROUP
subjective sleep quality Mean (SD)	1.40 (0.70)	0.60 (0.70)	**z = -2.83, *p* = .01**
sleep latency Mean (SD)	2.50 (2.22)	1.20 (1.62)	**z = -2.46, *p* = .02**
sleep duration Mean (SD)	0.60 (0.84)	0.20 (0.63)	z = -1.63, *p* = .25
habitual sleep efficiency Mean (SD)	1.10 (1.29)	0.50 (1.08)	z = -1.60, *p* = .25
sleep disturbances Mean (SD)	1.22 (0.44)	1.22 (0.44)	z = .00, *p* = 1.0
use of sleeping medication Mean (SD)	0.40 (0.97)	0.40 (0.97)	z = 0.0, *p* = 1.0
daytime dysfunction Mean (SD)	0.80 (0.63)	0.20 (0.42)	z = -2.12, *p* = .06
total score Mean (SD)	7.00 (4.36)	4.67 (4.61)	**z = -2.53, *p* = .01**

*Table 20: Within-group comparisons of the mean PSQI sub-score and total scores. * Wilcoxon tests were calculated for data with no normal distribution; ** TM = Transcendental Meditation; Significant results are printed in bold*

6-Item Short Form of the Spielberger State-Trait Anxiety Inventory (STAI-6)

The STAI-6 was used to to assess state anxiety (how anxious one feels at the moment). Higher total scores indicate a higher level of state anxiety. No significant differences between the total scores of three groups could be observed - neither before nor after the four-week rehabilitation program (see Table 21).

	BASELINE				
	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
total score Mean (SD)	36.30 (11.36)	30.33 (11.05)	34.00 (11.63)		F (2,26) = .67, p = .52, η2 = .05
	AFTER 4 WEEKS				
total score Mean (SD)	29.00 (9.82)	25.56 (6.24)	27.41 (6.62)	Chi-squares (2) = .74, p = .70	

*Table 21: Between-group comparisons of the mean STAI-6 total scores. * Kruskal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation*

Likewise, there were no significant between-group differences regarding the point differences between the first and second measurement time (see Table 22).

	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
total score Mean difference (SD)	- 7.0 (11.7)	- 4.1 (6)	- 7.8 (10.7)	Chi-squares (2) = .69, *p* = .71	

*Table 22: Between-group comparisons of the difference in STAI-6 total scores from baseline to four-week follow-up. * Kruskal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation.*

Similar to the between-group results, there were no significant differences before and after the four-week rehabilitation program within each group (see Table 23).

	CONTROL GROUP			
	Baseline	After 4 weeks	Wilcoxon Test*	T-test
total score Mean (SD)	36.30 (11.36)	29.00 (9.82)	z = -1.76, *p* = .10	
	TM GROUP**			
total score Mean (SD)	30.33 (11.05)	25.56 (6.24)	z = -1.83, *p* = .13	
	YOGA GROUP			
total score Mean (SD)	34.00 (11.63)	27.41 (6.62)		t = 2.19, *p* = .06

*Table 23: Within-Group comparisons of the mean STAI-6 total scores. * Wilcoxon tests were calculated for data with no normal distribution; ** TM = Transcendental Meditation.*

Short Form Health Survey 36 (SF-36)

The SF-36 with its eight sub-scores (physical functioning, bodily pain, role limitations due to physical health problems, role limitations due to personal or emotional problems, general mental health, social functioning, vitality, and general health perceptions) was used to evaluate the subjective health of the participants. For each sub-score, higher scores define a more favorable health state. The Kruskal-Wallis test showed a significant difference between the vitality scores of the three groups after the four weeks of rehabilitation. A post-hoc test (Dunn-Bonferroni) was performed and showed that the significant difference exists only between the control and TM group (z = -2.44, p = .04). Participants who practiced TM twice a day during a four-week rehabilitation program, had significantly higher subjective energy levels after four weeks than participants who only participated in the rehabilitation program. No other significant differences between the three groups were observed - neither before nor after the four-week rehabilitation program (see Table 24).

	BASELINE				
	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
physical functioning Mean (SD)	48.33 (23.18)	60.00 (35.36)	57.00 (35.29)		F (2,26) = .33, *p* = .72, η2 = .03
bodily pain Mean (SD)	57.25 (34.81)	72.22 (21.30)	53.75 (25.06)		F (2,26) = 1.16, *p* = .33, η2 = .08

role limitations due to physical health problems Mean (SD)	27.50 (32.17)	41.67 (45.07)	35.00 (45.95)	Chi-squares (2) = .55, *p* = .77	
role limitations due to personal or emotional problems Mean (SD)	74.07 (36.43)	58.33 (46.29)	46.67 (47.66)	Chi-squares (2) = 1.55, *p* = .47	
general mental health Mean (SD)	64.44 (20.34)	76.89 (21.05)	72.80 (15.53)	Chi-squares (2) = 2.47, *p* = .30	
social functioning Mean (SD)	70.00 (24.44)	79.17 (20.73)	78.75 (19.59)	Chi-squares (2) = .95, *p* = .63	
vitality Mean (SD)	47.00 (22.63)	64.44 (20.83)	52.22 (21.52)		F (2,25) = 1.59, *p* = .22, η2 = .11
general health perceptions Mean (SD)	56.50 (19.44)	61.11 (14.95)	60.50 (18.33)		F (2,26) = .19, *p* = .83, η2 = .02
			AFTER 4 WEEKS		
physical functioning Mean (SD)	72.78 (21.95)	80.63 (25.00)	75.50 (25.11)	Chi-squares (2) = 1.05, *p* = .60	
bodily pain Mean (SD)	74.06 (28.91)	85.00 (20.31)	78.13 (25.45)	Chi-squares (2) = .48, *p* = .80	
role limitations due to physical health problems Mean (SD)	56.25 (41.73)	68.75 (45.81)	60.00 (44.41)	Chi-squares (2) = .42, *p* = .82	
role limitations due to personal or emotional problems Mean (SD)	91.67 (23.57)	100.00 (0.00)	81.48 (37.68)	Chi-squares (2) = 2.17, *p* = .43	
general mental health Mean (SD)	84.40 (8.32)	91.11 (9.12)	84.00 (11.47)	Chi-squares (2) = 3.73, *p* = .16	
social functioning Mean (SD)	82.81 (21.06)	92.19 (13.26)	83.33 (24.21)	Chi-squares (2) = .67, *p* = .73	
vitality Mean (SD)	67.00 (9.49)	82.78 (15.23)	70.00 (18.56)	**Chi-squares (2) = 6.39, *p* = .04**	
general health perceptions Mean (SD)	64.50 (14.42)	70.63 (14.99)	66.67 (24.11)		F (2,24) = .25, *p* = .78, η2 = .02

*Table 24: Between-group comparisons of the mean SF-36 sub-score and total scores. * Kruskal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation; Significant results are printed in bold.*

No significant between-group differences regarding the point differences between the baseline and follow-up measurement were found (see Table 25).

	Control	TM**	Yoga	Kruskal-Wallis Test*	UNIANOVA**
physical functioning Mean difference (SD)	19.4 (19.4)	16.9 (26.4)	18.5 (22.7)	Chi-squares (2) = .72, *p* = .70	
bodily pain Mean difference (SD)	12.2 (21.9)	10.9 (15.9)	29.1 (22.1)	Chi-squares (2) = 3.56, *p* = .17	
role limitations due to physical health problems Mean difference (SD)	25.0 (58.2)	21.9 (28.1)	25.0 (31.2)	Chi-squares (2) = .06, *p* = .97	
role limitations due to personal or emotional problems Mean difference (SD)	9.5 (16.3)	38.1 (48.8)	29.6 (53.9)	Chi-squares (2) = .71, *p* = .70	
general mental health Mean difference (SD)	19.1 (19.5)	11.0 (13.5)	11.2 (9.8)		F (2,24) = .89, *p* = .42, η2 = .07
social functioning Mean difference (SD)	14.1 (27.1)	10.9 (27.1)	1.4 (20.2)		F (2,22) = .61, *p* = .55, η2 = .05
vitality Mean difference (SD)	20.0 (21.9)	20.0 (17.1)	16.1 (15.8)		F (2,24) = .13, *p* = .88, η2 = .01
general health perceptions Mean difference (SD)	8.0 (14.2)	4.3 (15.9)	6.7 (12.2)	Chi-squares (2) = .21, *p* = .90	

*Table 25: Between-group comparisons of the difference in SF-36 sub-score and total scores from baseline to four-week follow-up. * Kruskal Wallis tests were calculated for data with no normal distribution; ** UNIANOVA = single factor analysis of variance; TM = Transcendental Meditation.*

The within-group comparisons in the control group showed a significant rise in the mean physical functioning, general mental health, and vitality scores from baseline to after four weeks of rehabilitation. The mean vitality scores also increased

significantly within the meditation and the yoga groups. Additionally, the yoga group showed a significantly higher mean physical functioning score when compared to the baseline measurement (see Table 26).

	CONTROL GROUP			
	Baseline	After 4 weeks	Wilcoxon Test*	T-test
physical functioning Mean (SD)	48.33 (23.18)	72.78 (21.95)		**t = 2.83, *p* = .03**
bodily pain Mean (SD)	57.25 (34.81)	74.06 (28.91)		t = -1.57, *p* = .16
role limitations due to physical health problems Mean (SD)	27.50 (32.17)	56.25 (41.73)	z = -1.22, *p* = .31	
role limitations due to personal or emotional problems Mean (SD)	74.07 (36.43)	91.67 (23.57)	z = -1.41, *p* = .50	
general mental health Mean (SD)	64.44 (20.34)	84.40 (8.32)	**z = -2.20, *p* = .03**	
social functioning Mean (SD)	70.00 (24.44)	82.81 (21.06)	z = -1.48, *p* = .19	
vitality Mean (SD)	47.00 (22.63)	67.00 (9.49)		**t = -2.89, *p* = .02**
general health perceptions Mean (SD)	56.50 (19.44)	64.50 (14.42)		t = -1.78, *p* = .11
	TM GROUP**			
physical functioning Mean (SD)	60.00 (35.36)	80.63 (25.00)	z = -1.98, *p* = .08	
bodily pain Mean (SD)	72.22 (21.30)	85.00 (20.31)	z = -1.84, *p* = .13	
role limitations due to physical health problems Mean (SD)	41.67 (45.07)	68.75 (45.81)	z = -1.84, *p* = .13	
role limitations due to personal or emotional	58.33 (46.29)	100.00 (0.00)	z = -1.63, *p* = .25	

problems Mean (SD)				
general mental health Mean (SD)	76.89 (21.05)	91.11 (9.12)		t = -2.31, *p* = .054
social functioning Mean (SD)	79.17 (20.73)	92.19 (13.26)	z = -1.08, *p* = .38	
vitality Mean (SD)	64.44 (20.83)	82.78 (15.23)	**z = -2.25, *p* = .03**	
general health perceptions Mean (SD)	61.11 (14.95)	70.63 (14.99)		t = -.71, *p* = .50
			YOGA GROUP	
physical functioning Mean (SD)	57.00 (35.29)	75.50 (25.11)		**t = -2.57, *p* = .03**
bodily pain Mean (SD)	53.75 (25.06)	78.13 (25.45)	z = -1.84, *p* = .13	
role limitations due to physical health problems Mean (SD)	35.00 (45.95)	60.00 (44.41)	z = -1.84, *p* = .13	
role limitations due to personal or emotional problems Mean (SD)	46.67 (47.66)	81.48 (37.68)	z = -1.63, *p* = .25	
general mental health Mean (SD)	72.80 (15.53)	84.00 (11.47)	z = -1.90, *p* = .09	
social functioning Mean (SD)	78.75 (19.59)	83.33 (24.21)	z = -1.08, *p* = .38	
vitality Mean (SD)	52.22 (21.52)	70.00 (18.56)		**t = -3.07, *p* = .02**
general health perceptions Mean (SD)	60.50 (18.33)	66.67 (24.11)		t = -1.63, *p* = .14

*Table 26: Within-group comparisons of the mean SF-36 sub-score and total scores. * Wilcoxon tests were calculated for data with no normal distribution; ** TM = Transcendental Meditation; Significant results are printed in bold*

Discussion

The present study investigated the effect of yoga and TM on psychological parameters in cardiac rehabilitation patients. 30 patients suffering from a CVD were randomly assigned to one of three different cardiac rehabilitation programs; participants underwent either four weeks of standard cardiac rehabilitation (control condition), or four weeks of standard rehabilitation with either additional yoga or TM classes. Psychological parameters were assessed via eight standardized questionnaires at baseline and after completion of the four-week program. In the following, the results are summarized and discussed in respect to the hypotheses formulated at the beginning.

Between-Group Comparisons

Effects of TM and Yoga on Psychological Distress

We hypothesized that after four weeks of rehabilitation, the TM and the yoga group would show significantly less subjective psychological distress (assessed with the OPD-SQS, ECR-R, BSI-18, ASTS, BDI-II, PSQ-20, PSQI and STAI-6) than the standard therapy group. However, a between-group analysis indicated no significant difference between TM or yoga and the standard rehabilitation on any respective outcome. Thus, the first hypothesis–that after four weeks the TM and yoga group will show significantly lower scores in tests for psychological distress than the control group–could not be confirmed.

Our hypotheses were based on findings indicating that regular practice of yoga or TM had a positive effect on mental health. For example, a study investigating healthy participants with moderate to high levels of stress showed significant decreases in stress, anxiety, depression, and insomnia after they practiced yoga twice a week for eight weeks (94). In a study conducted by Rocha et al., stress, anxiety, and depression inventories were assessed in healthy men before and after six months of yoga practice. The control group received six months of conventional physical exercise. After the intervention period, the yoga group showed significantly lower scores for psychological parameters when compared to the control group (95). Similar findings could be observed in studies that investigated the effect of TM on mental health. Compared with a wait list control group, participants of a four-month

TM-intervention showed significant reductions in perceived stress, depressive symptoms, and burnout at the end of the intervention period (106). Another RCT assessing psychological distress in college students before and after a three-month TM intervention found that the TM group showed significant improvements in total psychological distress, anxiety, depression, anger, and coping ability, compared to a wait list control (107).

One possible reason why there was no significant difference in psychological distress parameters between the yoga or TM group and the control group after four weeks of intervention could be that all participants were already in generally good mental health at baseline measurement. In their work on the mechanisms of action of mindfulness interventions, Creswell and Lindsay stated that the most pronounced effects of such interventions are observed in participants that report a high stress level. Mindfulness interventions in low-stress participant groups are, by contrast, unlikely to have much impact on health outcomes, according to the authors (128). Given that no group showed clinically remarkable test scores in any of the given distress questionnaires at the beginning of the present study, one might argue that the psychological effect of the interventions was weak because the psychological well-being of the subjects was already quite good. For example, the mean total BDI-II score at baseline was 7.40 in the control group, 8.60 in the TM group, and 5 in the yoga group. These test results are far below the clinically remarkable limit of 14 points (120). In contrast, only one person in the control group and two in the TM group had a BDI-II score of more than 14 at baseline measurement.

Moreover, the existing research on the effects of yoga and TM on mental health is not entirely clear. A randomized study from Javnbakht et al. compared the effect of a two-month yoga intervention (90 minutes of yoga twice weekly) on anxiety and depressive symptoms in women with a wait list control. While the women who participated in yoga classes showed a significant decrease in state and trait anxiety, the depressive symptoms did not differ significantly Between-Groups at post-intervention measurement (96). Likewise, a meta-analysis that evaluated TM's efficacy improving depressive symptoms in adults found no significant difference in depressive symptoms between the TM or control participants (109). Another meta-analysis by Goyal et al. assessed the efficacy of various meditation programs (including TM) in improving stress-related outcomes such as depression, anxiety, or

sleep quality. They included RCTs with clinical populations (depression, anxiety, stress, lower back pain, heart disease, etc.) and an active control. In the 20 RCTs examining comparative effectiveness, there was no evidence that meditation programs were better than any active treatment in improving stress-related health outcomes (129).

In relation to the first hypothesis, our second assumption was that the TM and the yoga group would show significantly larger decreases in psychological distress, than the standard therapy group. We calculated the respective differences between baseline and follow-up measurements (test score after four weeks minus test score at baseline measurement) and compared them between the groups. In accordance with the hypothesis, the subjects of the yoga group showed a significantly larger improvement in subjective and overall sleep quality than the control group. These results are consistent with prior studies indicating a beneficial effect of yoga on sleep quality. A 12-week yoga intervention for older adults showed significant improvement in various sub-scores of the PSQI, including overall sleep quality and subjective sleep quality, when compared with a wait list control group (130). Furthermore, an investigation of subjective sleep quality in long-term yoga practitioners (minimum three years) and subjects without any yoga experience showed that long-term yoga practice is associated with better subjective sleep quality (131). Further studies with similar results confirm the positive correlation between yoga and sleep quality (132,133).

In contradiction to the second hypothesis, the control group showed a stronger decrease in momentary anger than the yoga group. This result may be explained by the fact that of all three groups at baseline measurement, the control group had the highest values for the anger sub-score (mean value = 8.70) while the yoga group had the lowest (mean value = 4.80). Though the group difference at baseline was not significant, it suggests that during the course of the rehabilitation, the control group had more room for improvement than the yoga group.

Effects of TM and Yoga on Subjective Health

In line with our distress-reduction hypotheses, we assumed that after four weeks of rehabilitation, the TM and the yoga group would show significantly better subjective

health (assessed with the SF-36) than the standard therapy group. According to this hypothesis, the TM group showed a significantly larger vitality score (mean value = 82.78) than the control group (mean value = 67) at the follow-up measurement. The beneficial effect of TM on vitality has already been reported in a study including HIV patients. Chhatre et al. found a significant improvement from baseline to follow-up measurement of vitality-scores in HIV patients who participated in a six-month TM program (105). Additionally, the mindfulness-based stress reduction (MBSR) program, an eight-week mindfulness training that includes sitting meditation as well as body scans and gentle yoga, has been shown to be effective in increasing the vitality of breast cancer patients (134). However, the current findings on the impact of TM on vitality are still ambiguous. Other RCTs that compared the effect of TM with a control intervention in different clinical populations could not find a significant difference concerning the participants' improvement in vitality (107,135).

In summary, the majority of parameters on psychological distress and subjective health did not differ significantly between the intervention and control groups – neither before nor after the four weeks of rehabilitation.

One reason why we could not prove a significant effect of yoga and/or TM could be our active control condition. The control group, as well as both intervention groups, received state-of-the-art cardiac rehabilitation, in which each participant was offered an individual training program combined with a health-promoting diet and health education. As already mentioned, regular physical exercise has a positive effect on mental (82) and subjective (81) health. Furthermore, these effects may be caused by physiological mechanisms, similar to those of yoga and meditation; that is improving the sympathovagal balance and reversing chronic dysregulation of the ANS (83–86). In addition, a healthy mixed diet such as the MD can promote not only physical but also mental health (88,89,136). Due to the already positive effect of the standard rehabilitation on the measured outcomes, the additional yoga or meditation sessions probably could not achieve a significant additional therapeutic effect.

The mainly non-significant group comparisons are also in line with prior research on meditation and yoga programs with an active control. A review of RCTs that examined the effectiveness of both yoga and mindfulness-based interventions on stress, depression, and anxiety did find moderate effects of such interventions. However, a subgroup analysis revealed that the effects decreased substantially

when interventions were compared to an active control group as opposed to a wait list control group or no treatment (137). Another systematic review of RCTs by Goyal et al. evaluated study results comparing the effects of various meditation programs (including TM) on mental health parameters and health-related quality of life with an active control group. None of the meditation programs were more effective than a specific active control intervention, such as exercise or progressive muscle relaxation (129).

Within-Group Comparisons

Concerning the comparison of the baseline test results to the four week follow-up within each group, we hypothesized that all groups would show a significant improvement in subjective psychological distress (assessed with the OPD-SQS, ECR-R, BSI-18, ASTS, BDI-II, PSQ-20, PSQI and STAI-6) and subjective health (assessed with the SF-36).

Consistent with this assumption, the within-group analyses demonstrated significant improvements in different sub-scores of the ASTS, the PSQ-20 and the SF-36 in all three groups. Furthermore, the control group as well as the TM group, showed significantly less depressive symptoms (assessed with the BDI-II) after four weeks of rehabilitation than at the beginning of the intervention. Because most of the associated between-group changes were not significant, the current within-group results confirm our assumption that the standard rehabilitation itself had a powerful impact on the participants' psychological and subjective health.

Interestingly, only the yoga group showed a significant improvement in parameters of the PSQI. At follow-up, subjective sleep quality scores, sleep latency, and overall sleep quality were significantly higher than at baseline measurement. These within-group differences are in line with the findings of the between-group comparisons, whereby the yoga group demonstrated a significantly larger improvement in subjective and overall sleep quality than the control group. Therefore, the results suggest that the yoga intervention had a stronger positive influence on the participants' sleep quality than the standard rehabilitation or TM condition. However, within-group analyses should be interpreted cautiously, considering known threats to internal validity (e.g., history or testing effects).

No significant differences were found in any of the groups when comparing the baseline results of the OPD-SQS, ECR-R or STAI-6 to the follow-up scores.

Limitations

There were some limitations to this study. First of all, the duration of the intervention may have been too short to achieve significant effects. Most of the reported studies that could show an impact of yoga or TM on psychological and subjective physical health lasted for at least three months. Therefore, the time period of four weeks might not have been enough to detect substantial changes in emotional regulation and well-being.

Second, this pilot RCT was designed as a feasibility study. Its small group size, with 10 participants in each group, was therefore not adequately powered to detect statistically significant differences.

Another practical problem in implementing the different interventions was that the additional yoga and TM classes could not be integrated into the subjects' digital timetables. Feedback from the investigators suggests that some participants were unsettled by the fact that course times for the yoga and TM sessions were not on their regular schedules. This may have been a stress factor in both intervention groups that was not present in the control group.

Finally, the given test battery with eight questionnaires was possibly too large. Each participant had to answer 162 questions in a row to complete one measurement. This may have led to frustration, reduced focus, and lower compliance while completing the questionnaires. The fact that the BSI-18 was only filled out sufficiently by seven participants at the baseline measurement and not by a single participant at follow-up supports this assumption.

Implications for clinical practice

Cardiovascular and psychiatric diseases often occur together. A common risk factor for this association is chronic stress. Thus, it is of clinical importance to evaluate therapeutic approaches for secondary prevention that have been shown to reduce

perceived stress and related health problems. Yoga and TM are therefore promising interventions.

Despite the limitations, our evidence suggests that yoga and TM can positively affect stress-related health problems like poor sleeping quality or lack of energy. Furthermore, a strength of the current study is its active control group, which should give clinicians greater confidence that the reported benefits are not due to nonspecific effects.

A successful rehabilitation program is not necessarily characterized by quick results, but by lasting changes in the participants' health behavior. During rehabilitation, the density and intensity of health measures are very high; the goal of secondary prevention is achieved when these measures are also implemented in everyday life of the patients after their stay in the rehabilitation clinic. Yoga and meditation are both activities one can easily practice at home without any special equipment and regardless of age or fitness level. Therefore, they might be particularly easy to integrate into everyday life and can serve as an essential addition to secondary prevention of CVDs, both during rehabilitation programs as well as in the long run.

Additionally, the results of this study suggest that yoga was particularly beneficial for sleeping quality while TM was particularly beneficial for increasing vitality. Therefore, future rehabilitation programs could assess the patient's sleep quality and vitality upon admission, and provide targeted yoga or TM interventions based on these results.

Conclusion

The current research showed that additional yoga sessions during a four-week cardiac rehabilitation could enhance participants’ sleeping quality, while TM significantly improved patients’ vitality.

However, the strength of a successful rehabilitation program lies in its ability to lay the foundation for lasting changes in the health behavior of the participants. Longer follow-up intervals are therefore necessary to evaluate whether participants can continue their yoga or TM routine after rehabilitation and how such integration into everyday life affects the prognosis of patients with CVD compared to other stress-reducing interventions of cardiac rehabilitation.

Furthermore, stronger study designs with larger sample sizes are needed to further determine the effects of yoga and TM programs on cardiovascular rehabilitation. It would also be advisable for future studies to collect a more specific set of psychometric data to ensure patient compliance. Psychophysiological parameters of stress such as hair cortisol levels might also be insightful parameters of yoga's and TM's psychological impact with the additional advantage of higher objectivity.

As was the case in this study, further RCTs should compare the yoga and TM interventions with an active control group instead of a wait list control or no treatment.

References

1. Mendis S, Puska P, Norrving B. Global atlas on cardiovascular disease prevention and control. World Heal Organ. 2011;2–14.
2. WHO. The global burden of disease 2004. Updat World Heal Organ. 2004;146.
3. Goldston K, Baillie AJ. Depression and coronary heart disease: A review of the epidemiological evidence, explanatory mechanisms and management approaches. Clin Psychol Rev. 2008;28(2):288–306.
4. Halaris A. Inflammation-Associated Co-morbidity between Depression and Cardiovascular Disease. Curr Top Behav Neurosci. 2016;
5. Colquhoun DM, Bunker SJ, Clarke DM, Glozier N, Hare DL, Hickie IB, et al. Screening, referral and treatment for depression in patients with coronary heart disease: A consensus statement from the national heart foundation of Australia. Med J Aust. 2013;198(9):1–7.
6. Serber ER, Todaro JF, Tilkemeier PL, Niaura R. Prevalence and Characteristics of Multiple Psychiatric Disorders in Cardiac Rehabilitation Patients. J Cardiopulm Rehabil Prev. 2015;29(3):161–70.
7. Hare DL, Toukhsati SR, Johansson P, Jaarsma T. Depression and cardiovascular disease: A clinical review. Eur Heart J. 2014;35(21):1365–72.
8. Ariyo AA, Haan M, Tangen CM, Rutledge JC, Cushman M, Dobs A, et al. Depressive symptoms and risks of coronary heart disease and mortality in elderly Americans. Circulation. 2000;102(15):1773–9.
9. Penninx BWJH, Beekman ATF, Honig A, Deeg DJH, Schoevers RA, van Eijk JTM, et al. Depression and Cardiac Mortality. Arch Gen Psychiatry. 2001;58(3):221.
10. Sundbøll J, Schmidt M, Adelborg K, Pedersen L, Bøtker HE, Videbech P, et al. Impact of pre-admission depression on mortality following myocardial infarction. Br J Psychiatry. 2017;210(5):356–61.
11. Mallik S, Krumholz HM, Zhen QL, Kasl S V., Mattera JA, Roumains SA, et al. Patients with depressive symptoms have lower health status benefits after coronary artery bypass surgery. Circulation. 2005;111(3):271–7.

12. Zhang Y, Chen Y, Ma L. Depression and cardiovascular disease in elderly: Current understanding. J Clin Neurosci [Internet]. 2018;47:1–5. Available from: https://doi.org/10.1016/j.jocn.2017.09.022

13. Joynt KE, Whellan DJ, O'Connor CM. Depression and cardiovascular disease: Mechanisms of interaction. Biol Psychiatry. 2003;54(3):248–61.

14. O'Connor CM, Gurbel PA, Serebruany VL. Depression and ischemic heart disease. Am Heart J. 2000;140(4 SUPPL.):0–6.

15. Cohen BE, Edmondson D, Kronish IM. State of the art review: Depression, stress, anxiety, and cardiovascular disease. Am J Hypertens. 2015;28(11):1295–302.

16. Roest AM, Zuidersma M, De Jonge P. Myocardial infarction and generalised anxiety disorder: 10-Year follow-up. Br J Psychiatry. 2012;200(4):324–9.

17. Todaro JF, Shen BJ, Raffa SD, Tilkemeier PL, Niaura R. Prevalence of anxiety disorders in men and women with established coronary heart disease. J Cardiopulm Rehabil Prev. 2007;27(2):86–91.

18. Celano CM, Daunis DJ, Lokko HN, Campbell KA, Huffman JC. Anxiety Disorders and Cardiovascular Disease. Curr Psychiatry Rep. 2016;18(11).

19. Celano CM, Villegas AC, Albanese AM, Gaggin HK, Huffman JC. Depression and Anxiety in Heart Failure: A Review. Harv Rev Psychiatry. 2018;26(4):175–84.

20. Roest AM, Martens EJ, de Jonge P, Denollet J. Anxiety and Risk of Incident Coronary Heart Disease. A Meta-Analysis. J Am Coll Cardiol. 2010;56(1):38–46.

21. Grace SL, Abbey SE, Irvine J, Shnek ZM, Stewart DE. Prospective examination of anxiety persistence and its relationship to cardiac symptoms and recurrent cardiac events. Psychother Psychosom. 2004;73(6):344–52.

22. Edmondson D, Kronish IM, Shaffer JA, Falzon L, Burg MM. Posttraumatic stress disorder and risk for coronary heart disease: A meta-analytic review. Am Heart J [Internet]. 2013;166(5):806–14. Available from: http://dx.doi.org/10.1016/j.ahj.2013.07.031

23. Martens EJ, De Jonge P, Na B, Cohen BE, Lett H, Whooley MA. Scared to death? Generalized anxiety disorder and cardiovascular events in patients with stable coronary heart disease: The heart and soul study. Arch Gen Psychiatry. 2010;67(7):750–8.

24. Strik JJMH, Denollet J, Lousberg R, Honig A. Comparing Symptoms of Depression and Anxiety as Predictors of Cardiac Events and Increased Health Care Consumption after Myocardial Infarction. J Am Coll Cardiol

[Internet]. 2003;42(10):1801–7. Available from: http://dx.doi.org/10.1016/j.jacc.2003.07.007

25. Tully PJ, Harrison NJ, Cheung P, Cosh S. Anxiety and Cardiovascular Disease Risk: a Review. Curr Cardiol Rep [Internet]. 2016;18(12). Available from: http://dx.doi.org/10.1007/s11886-016-0800-3

26. Edmondson D, von Känel R. Posttraumatic Stress Disorder and Cardiovascular Disease. Lancet Psychiatry. 2017;4(4):320–9.

27. Grippo AJ, Johnson AK. Stress, depression, and cardiovascular dysregulation: A review. Stress. 2010;12(1):1–21.

28. Chauvet-Gelinier JC, Bonin B. Stress, anxiety and depression in heart disease patients: A major challenge for cardiac rehabilitation. Ann Phys Rehabil Med [Internet]. 2017;60(1):6–12. Available from: http://dx.doi.org/10.1016/j.rehab.2016.09.002

29. Selye H. The Stress of Life. McGraw-Hill; 1956.

30. Black PH, Garbutt LD. Stress, inflammation and cardiovascular disease. J Psychosom Res. 2002;52(1):1–23.

31. Chrousos GP. Stress and disorders of the stress system. Nat Rev Endocrinol [Internet]. 2009;5(7):374–81. Available from: http://dx.doi.org/10.1038/nrendo.2009.106

32. Pschyrembel [Internet]. [cited 2020 Aug 29]. Available from: https://www.pschyrembel.de/Stressor/K0LQH/doc/

33. Holmes TH, Rahe RH. The Social Readjusment Rating Scale. J Psychosom Res [Internet]. 1967;11(2):213–8. Available from: https://doi.org/10.1016/0022-3999(67)90010-4

34. Cohen S, Gianaros PJ, Manuck SB. A Stage Model of Stress and Disease. Perspect Psychol Sci. 2016;11(4):456–63.

35. Charmandari E, Tsigos C, Chrousos G. Endocrinology of the stress response. Annu Rev Physiol. 2005;67:259–84.

36. Tsigos C, Chrousos GP. Hypothalamic-pituitary-adrenal axis, neuroendocrine factors and stress. J Psychosom Res. 2002;53(4):865–71.

37. De Kloet ER, Joëls M, Holsboer F. Stress and the brain: From adaptation to disease. Nat Rev Neurosci. 2005;6(6):463–75.

38. Smeets T. Autonomic and hypothalamic-pituitary-adrenal stress resilience: Impact of cardiac vagal tone. Biol Psychol. 2010;84(2):290–5.

39. Antoun M, Edwards KM, Sweeting J, Ding D. The acute physiological stress response to driving: A systematic review. PLoS One. 2017;12(10):1–13.

40. McEwen BS, Stellar E. Stress and the individual. Arch Intern Med. 1993;153:2093–101.

41. McEwen BS. Protective and Damaging Effects of Stress Mediators. N Engl J Med. 1998;338(3):171–9.

42. Steptoe A, Kivimäki M. Stress and cardiovascular disease: An update on current knowledge. Annu Rev Public Health. 2013;34:337–54.

43. Dragano N, Siegrist J, Nyberg ST, Lunau T, Fransson EI, Alfredsson L, et al. Effort-Reward Imbalance at Work and Incident Coronary Heart Disease: A Multicohort Study of 90,164 Individuals. Epidemiology. 2017;28(4):619–26.

44. Kivimäki M, Steptoe A. Effects of stress on the development and progression of cardiovascular disease. Nat Rev Cardiol [Internet]. 2018;15(4):215–29. Available from: http://dx.doi.org/10.1038/nrcardio.2017.189

45. Li J, Hansen D, Mortensen PB, Olsen J. Myocardial infarction in parents who lost a child: A nationwide prospective cohort study in Denmark. Circulation. 2002;106(13):1634–9.

46. Rosengren A, Hawken S, Ôunpuu S, Sliwa PK, Zubaid M, Almahmeed WA, et al. Association of psychosocial risk factors with risk of acute myocardial infarction in 11 119 cases and 13 648 controls from 52 countries (the INTERHEART study): Case-control study. Lancet. 2004;364(9438):953–62.

47. Iso H, Date C, Yamamoto A, Toyoshima H, Tanabe N, Kikuchi S, et al. Perceived mental stress and mortality from cardiovascular disease among Japanese men and women: The Japan Collaborative Cohort Study for Evaluation of Cancer Risk Sponsored by Monbusho (JACC Study). Circulation. 2002;106(10):1229–36.

48. Libby P. History of Discovery : Inflammation in Atherosclerosis. Arter Thromb Vasc Biol. 2012;32(9):2045–51.

49. Yao BC, Meng LB, Hao ML, Zhang YM, Gong T, Guo ZG. Chronic stress: a critical risk factor for atherosclerosis. J Int Med Res. 2019;47(4):1429–40.

50. Liu YZ, Wang YX, Jiang CL. Inflammation: The common pathway of stress-related diseases. Front Hum Neurosci. 2017;11(June):1–11.

51. Glaser R, Kiecolt-Glaser J. Stress-induced immune dysfunction: implications for health. Nat Rev Immunol. 2005;5:243–51.

52. Slavich GM, Irwin MR. From Stress to Inflammation and Major Depressive Disorder: A Social Signal Transduction Theory of Depression. Psychol Bull. 2014;140(3):774–815.

53. Schleimer RP. An overview of glucocorticoid anti-inflammatory actions. Eur J Clin Pharmacol. 1993;45(1 Supplement):3–7.

54. Lagraauw HM, Kuiper J, Bot I. Acute and chronic psychological stress as risk factors for cardiovascular disease: Insights gained from epidemiological, clinical and experimental studies. Brain Behav Immun [Internet]. 2015;50:18–30. Available from: http://dx.doi.org/10.1016/j.bbi.2015.08.007

55. Mazure CM. Life stressors as risk factors in depression. Clin Psychol Sci Pract. 1998;5(3):291–313.

56. Kendler KS, Karkowski LM, Prescott CA. Causal relationship between stressful life events and the onset of major depression. Am J Psychiatry. 1999;156(6):837–41.

57. Slavich GM, O'Donovan A, Epel ES, Kemeny ME. Black Sheep Get the Blues: A Psychobiological Model of Social Rejection and Depression. Neurosci Biobehav Rev. 2010;35(1):39–45.

58. Madsen IEH, Nyberg ST, Magnusson Hanson LL, Ferrie JE, Ahola K, Alfredsson L, et al. Job strain as a risk factor for clinical depression: Systematic review and meta-analysis with additional individual participant data. Psychol Med. 2017;47(8):1342–56.

59. Theorell T, Hammarström A, Aronsson G, Träskman Bendz L, Grape T, Hogstedt C, et al. A systematic review including meta-analysis of work environment and depressive symptoms. BMC Public Health [Internet]. 2015;15(1):1–14. Available from: http://dx.doi.org/10.1186/s12889-015-1954-4

60. Muscatell KA, Slavich GM, Monroe SM, Gotlib IH. Stressful life events, chronic difficulties, and the symptoms of clinical depression. J Nerv Ment Dis. 2009;197(3):154–60.

61. Smith RS. The macrophage theory of depression. Med Hypotheses. 1991;35(4):298–306.

62. Kidwell M, Ellenbroek BA. Heart and soul: Heart rate variability and major depression. Behav Pharmacol. 2018;29(1998):152–64.

63. Breit S, Kupferberg A, Rogler G, Hasler G. Vagus nerve as modulator of the brain-gut axis in psychiatric and inflammatory disorders. Front Psychiatry. 2018;9(MAR).

64. Leonard BE. Inflammation and depression: A causal or coincidental link to the pathophysiology? Acta Neuropsychiatr. 2018;30(1):1–16.

65. Leonard BE. Brain cytokines and the psychopathology ofdepression. Antidepres. Basel: Birkhauser Verlag; 2001. 109–120 p.

66. Connor TJ, Leonard BE. Depression, stress and immunological activation: The role of cytokines in depressive disorders. Life Sci. 1998;62(7):583–606.

67. Miller AH, Maletic V, Raison CL. Inflammation and Its Discontents: The Role of Cytokines in the Pathophysiology of Major Depression. Biol Psychiatry [Internet]. 2009;65(9):732–41. Available from: http://dx.doi.org/10.1016/j.biopsych.2008.11.029

68. Miller G, Blackwell E. Turning up the heat. Inflammation as a Mechanism Linking Chronic Stress, Depression, and Heart Disease. Curr Dir Psychol Sci. 2006;15(6):269–72.

69. Dantzer R, Connor JCO, Freund GG, Johnson RW, Kelley KW. From inflamation to sickness and depression: when the immune system subjugates the brain. Nat Rev Neurosci. 2008;9(1):46–56.

70. Vreeburg SA, Zitman FG, Van Pelt J, Derijk RH, Verhagen JCM, Van Dyck R, et al. Salivary cortisol levels in persons with and without different anxiety disorders. Psychosom Med. 2010;72(4):340–7.

71. Juruena MF, Eror F, Cleare AJ, Young AH. The Role of Early Life Stress in HPA Axis and Anxiety. Vol. 1191, Advances in Experimental Medicine and Biology. 2020. 141–153 p.

72. Tyrka AR, Wier L, Price LH, Ross N, Anderson GM, Wilkinson CW, et al. Childhood Parental Loss and Adult Hypothalamic-Pituitary-Adrenal Function. Biol Psychiatry. 2008;63(12):1147–54.

73. Faravelli C, Lo Sauro C, Lelli L, Pietrini F, Lazzeretti L, Godini L, et al. The Role of Life Events and HPA Axis in Anxiety Disorders: A Review. Curr Pharm Des. 2012;18(35):5663–74.

74. Yehuda R, Flory JD, Pratchett LC, Buxbaum J, Ising M, Holsboer F. Putative biological mechanisms for the association between early life adversity and the subsequent development of PTSD. Psychopharmacology (Berl). 2010;212(3):405–17.

75. Carr CP, Martins CMS, Stingel AM, Lemgruber VB, Juruena MF. The role of early life stress in adult psychiatric disorders: A systematic review according to childhood trauma subtypes. J Nerv Ment Dis. 2013;201(12):1007–20.

76. Michopoulos V, Powers A, Gillespie CF, Ressler KJ, Jovanovic T. Inflammation in Fear-and Anxiety-Based Disorders: PTSD, GAD, and beyond. Neuropsychopharmacology. 2017;42(1):254–70.

77. Keane TM, Marx BP, Sloan DM. Post-traumatic stress disorder: definition, prevalence, and risk factors. Post-Trau. Shiromani PJ, Keane TM, LeDoux JE, editors. New York: Humana Press; 2009.

78. Eraly SA, Nievergelt CM, Maihofer AX, Barkauskas DA, Biswas N, Agorastos A, et al. Assessment of Plasma C-Reactive Protein as a biomarker of PTSD risk. JAMA Psychiatry. 2014;71(4):423–31.

79. Pervanidou P, Kolaitis G, Charitaki S, Margeli A, Ferentinos S, Bakoula C, et al. Elevated morning serum interleukin (IL)-6 or evening salivary cortisol concentrations predict posttraumatic stress disorder in children and adolescents six months after a motor vehicle accident. Psychoneuroendocrinology. 2007;32(8–10):991–9.

80. DGK. S3-Leitlinie zur kardiologischen Rehabilitation im deutschsprachigen Raum Europas LL-KardReha , Anhang (A3): Zusammenfassung der Empfehlungen Inhaltsverzeichnis. 2020;2020:1–56.

81. Anderson L, Oldridge N, Thompson DR, Zwisler AD, Rees K, Martin N, et al. Exercise-Based Cardiac Rehabilitation for Coronary Heart Disease Cochrane Systematic Review and Meta-Analysis. J Am Coll Cardiol. 2016;67(1):1–12.

82. Salmon P. Effects of physical exercise on anxiety, depression, and sensitivity to stress: A unifying theory. Clin Psychol Rev. 2001;21(1):33–61.

83. Besnier F, Labrunée M, Pathak A, Pavy-Le Traon A, Galès C, Sénard JM, et al. Exercise training-induced modification in autonomic nervous system: An update for cardiac patients. Ann Phys Rehabil Med. 2017;60(1):27–35.

84. Mimura J, Yuasa F, Yuyama R, Kawamura A, Iwasaki M, Sugiura T, et al. The Effect of Residential Exercise Training on Baroreflex Control of Heart Rate and Sympathetic Nerve Activity in Patients With Acute Myocardial Infarction. Chest [Internet]. 2005;127(4):1108–15. Available from: http://dx.doi.org/10.1016/S0012-3692(15)34454-8

85. Martinez DG, Nicolau JC, Lage RL, Toschi-Dias E, De Matos LDNJ, Alves MJNN, et al. Effects of long-term exercise training on autonomic control in myocardial infarction patients. Hypertension. 2011;58(6):1049–56.

86. Badrov MB, Wood KN, Lalande S, Sawicki CP, Borrell LJ, Barron CC, et al. Effects of 6 Months of Exercise-Based Cardiac Rehabilitation on Autonomic Function and Neuro-Cardiovascular Stress Reactivity in Coronary Artery Disease Patients. J Am Heart Assoc. 2019;8(17):e012257.

87. Widmer RJ, Flammer AJ, Lerman LO, Lerman A. The Mediterranean diet, its components, and cardiovascular disease. Am J Med [Internet]. 2015;128(3):229–38. Available from: http://dx.doi.org/10.1016/j.amjmed.2014.10.014

88. Parletta N, Zarnowiecki D, Cho J, Wilson A, Bogomolova S, Villani A, et al. A Mediterranean-style dietary intervention supplemented with fish oil improves

diet quality and mental health in people with depression: A randomized controlled trial (HELFIMED). Nutr Neurosci. 2019;22(7):474–87.

89. Antonopoulou M, Mantzorou M, Serdari A, Bonotis K, Vasios G, Pavlidou E, et al. Evaluating Mediterranean diet adherence in university student populations: Does this dietary pattern affect students' academic performance and mental health? Int J Health Plann Manage. 2020;35(1):5–21.

90. Albus C, Herrmann-Lingen C, Jensen K, Hackbusch M, Münch N, Kuncewicz C, et al. Additional effects of psychological interventions on subjective and objective outcomes compared with exercise-based cardiac rehabilitation alone in patients with cardiovascular disease: A systematic review and meta-analysis. Eur J Prev Cardiol. 2019;26(10):1035–49.

91. Nijjar PS, Connett JE, Lindquist R, Brown R, Burt M, Pergolski A, et al. Randomized Trial of Mindfulness-Based Stress Reduction in Cardiac Patients Eligible for Cardiac Rehabilitation. Sci Rep [Internet]. 2019;9(1):1–11. Available from: http://dx.doi.org/10.1038/s41598-019-54932-2

92. Ross A, Thomas S. The health benefits of yoga and exercise: A review of comparison studies. J Altern Complement Med. 2010;16(1):3–12.

93. Guddeti RR, Dang G, Williams MA, Alla VM. Role of Yoga in Cardiac Disease and Rehabilitation. J Cardiopulm Rehabil Prev. 2019;39(3):146–52.

94. Maddux RE, Daukantaité D, Tellhed U. The effects of yoga on stress and psychological health among employees: an 8- and 16-week intervention study. Anxiety, Stress Coping. 2018;31(2):121–34.

95. Rocha KKF, Ribeiro AM, Rocha KCF, Sousa MBC, Albuquerque FS, Ribeiro S, et al. Improvement in physiological and psychological parameters after 6months of yoga practice. Conscious Cogn [Internet]. 2012;21(2):843–50. Available from: http://dx.doi.org/10.1016/j.concog.2012.01.014

96. Javnbakht M, Hejazi Kenari R, Ghasemi M. Effects of yoga on depression and anxiety of women. Complement Ther Clin Pract [Internet]. 2009;15(2):102–4. Available from: http://dx.doi.org/10.1016/j.ctcp.2009.01.003

97. Smith C, Hancock H, Blake-Mortimer J, Eckert K. A randomised comparative trial of yoga and relaxation to reduce stress and anxiety. Complement Ther Med. 2007;15(2):77–83.

98. Kiecolt-Glaser JK, Christian L, Preston H, Houts CR, Malarkey WB, Emery CF, et al. Stress, Inflammation, and Yoga Practice. Psychosom Med. 2010;72(2):1–22.

99. Pullen PR, Nagamia SH, Mehta PK, Thompson WR, Benardot D, Hammoud R, et al. Effects of Yoga on Inflammation and Exercise Capacity in Patients With Chronic Heart Failure. J Card Fail. 2008;14(5):407–13.

100. Christa E, Srivastava P, Chandran DS, Jaryal AK, Yadav RK, Roy A, et al. Effect of Yoga-Based Cardiac Rehabilitation on Heart Rate Variability: Randomized Controlled Trial in Patients Post-MI. Int J Yoga Therap. 2019;29(1):43–50.

101. Hari Krishna B, Pal P, Pal GK, Balachander J, Jayasettiaseelon E, Sreekanth Y, et al. Effect of yoga therapy on heart rate, blood pressure and cardiac autonomic function in heart failure. J Clin Diagnostic Res. 2014;8(1):14–6.

102. Raffone A, Srinivasan N. The exploration of meditation in the neuroscience of attention and consciousness. Cogn Process. 2010;11(1):1–7.

103. Krisanaprakornkit T, Sriraj W, Piyavhatkul N, Laopaiboon M. Meditation therapy for anxiety disorders. Cochrane Database Syst Rev. 2006;(1).

104. Travis F, Shear J. Focused attention, open monitoring and automatic self-transcending: Categories to organize meditations from Vedic, Buddhist and Chinese traditions. Conscious Cogn [Internet]. 2010;19(4):1110–8. Available from: http://dx.doi.org/10.1016/j.concog.2010.01.007

105. Chhatrea S, Metzgera DS, Frankb I, Boyerc J, Thompsonc E, Nidichd S, et al. Effects of behavioral stress reduction Transcendental Meditation intervention in Persons with HIV. AIDS Care. 2013;25(10):1291–7.

106. Elder C, Nidich S, Moriarty F, Nidich R. Effect of transcendental meditation on employee stress, depression, and burnout: a randomized controlled study. Perm J. 2014;18(1):19–23.

107. Nidich SI, Rainforth M V., Haaga DAF, Hagelin J, Salerno JW, Travis F, et al. A Randomized Controlled Trial on Effects of the Transcendental Meditation Program on Blood Pressure, Psychological Distress, and Coping in Young Adults. Am J Hypertens. 2009;22(12):1326–31.

108. Eppley KR, Abrams AI. Differential Effects of Relaxation Techniques on Trait Anxiety: A Meta-Analysis. J Clin Psychol. 1989;45(6):957–74.

109. Gathright EC, Salmoirago-Blotcher E, DeCosta J, Balletto BL, Donahue ML, Feulner MM, et al. The impact of transcendental meditation on depressive symptoms and blood pressure in adults with cardiovascular disease: A systematic review and meta-analysis. Complement Ther Med [Internet]. 2019;46(March):172–9. Available from: https://doi.org/10.1016/j.ctim.2019.08.009

110. Schneider RH, Grim CE, Rainforth M V, Kotchen JM, Alexander CN. Stress Reduction in the Secondary Prevention of Cardiovascular Disease. Circ Cardiovasc Qual Outcomes. 2012;5(6):750–8.

111. Anderson JW, Liu C, Kryscio RJ. Blood pressure response to transcendental meditation: A meta-analysis. Am J Hypertens. 2008;21(3):310–6.

112. Arthur HM, Patterson C, Stone JA. The role of complementary and alternative therapies in cardiac rehabilitation: A systematic evaluation. Eur J Prev Cardiol. 2006;13(1):3–9.

113. Klimes-Dougan B, Chong LS, Samikoglu A, Thai M, Amatya P, Cullen KR, et al. Transcendental meditation and hypothalamic-pituitary-adrenal axis functioning: a pilot, randomized controlled trial with young adults. Stress [Internet]. 2020;23(1):105–15. Available from: http://dx.doi.org/10.1080/10253890.2019.1656714

114. Walton KG, Schneider RH, Nidich S. Review of controlled research on the Transcendental Meditation program and cardiovascular disease: Risk factors, morbidity, mortality. Cardiol Rev. 2004;12(5):262–6.

115. Ehrenthal JC, Dinger U, Schauenburg H, Horsch L, Dahlbender RW, Gierk B. Entwicklung einer Zwölf-Item-Version des OPD-Strukturfragebogens (OPD-SFK). Z Psychosom Med Psychother. 2015;61:262–74.

116. Sibley CG, Liu JH. Short-term temporal stability and factor structure of the revised experiences in close relationships (ECR-R) measure of adult attachment. Pers Individ Dif. 2004;36(4):969–75.

117. Ehrenthal JC, Dinger U, Lamla A, Funken B, Schauenburg H. Evaluation der deutschsprachigen Version des Bindungsfragebogens “Experiences in Close Relationships - Revised” (ECR-RD). Psychother Psychosom Med Psychol. 2008;59:215–23. Available from:

118. Spitzer C, Hammer S, Löwe B, Grabe HJ, Barnow S, Rose M, et al. Die Kurzform des Brief Symptom Inventory (BSI-18): erste Befunde zu den psychometrischen Kennwerten der deutschen Version. Fortschritte der Neurol Psychiatr. 2011;79(9):517–23.

119. Dalbert C. Subjektives Wohlbefinden junger Erwachsener: Theoretische und empirische Analysen der Struktur und Stabilität. Zeitschrift für Differ und Diagnostische Psychol. 1992;13(4):207–20.

120. Hautzinger M, Brähler E, Strauß B. Beck Depressions-Inventar (BDI). Diagnostische Verfahren der Psychother. 2003;1:32–4.

121. Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An Inventory for Measuring Depression. Arch Gen Psychiatry. 1961;4:53–63.

122. Levenstein S, Prantera C, Varvo V, Scribano ML, Berto E, Luzi C, et al. Development of the perceived stress questionnaire: A new tool for psychosomatic research. J Psychosom Res. 1993;37(1):19–32.

123. Fliege H, Rose M, Arck P, Walter OB, Kocalevent RD, Weber C, et al. The Perceived Stress Questionnaire (PSQ) reconsidered: Validation and reference values from different clinical and healthy adult samples. Psychosom Med. 2005;67(1):78–88.

124. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: A New Instrument for Psychiatric Practice and Research. Psychiatry Res. 1988;28:193–213.

125. Marteau TM, Bekker H. The development of a six-item short-form of the state scale of the Spielberger State—Trait Anxiety Inventory (STAI). Br J Clin Psychol. 1992;31(3):301–6.

126. Hays RD, Sherbourne CD, Mazel Rand RM. The Rand 36-Item Health Survey 1.0. Health Econ. 1993;2(3):217–27.

127. IBM SPSS Statistics for Windows. Armonk, NY: IBM Corp.; 2019.

128. Creswell JD, Lindsay EK. How Does Mindfulness Training Affect Health? A Mindfulness Stress Buffering Account. Curr Dir Psychol Sci. 2014;23(6):401–7.

129. Goyal M, Singh S, Sibinga EMS, Gould NF, Rowland-Seymour A, Sharma R, et al. Meditation programs for psychological stress and well-being: A systematic review and meta-analysis. JAMA Intern Med. 2014;174(3):357–68.

130. Halpern J, Cohen M, Kennedy G, Reece J, Cahan C, Baharav A. Yoga for Improving Sleep Quality and Quality of Life for Older Adults. Altern Ther. 2014;20(3):37–46.

131. Vera FM, Manzaneque JM, Maldonado EF, Carranque GA, Rodriguez FM, Blanca MJ, et al. Subjective Sleep Quality and hormonal modulation in long-term yoga practitioners. Biol Psychol. 2009;81(3):164–8.

132. Khalsa SBS. Treatment of chronic insomnia with yoga: A preliminary study with sleep-wake diaries. Appl Psychophysiol Biofeedback. 2004;29(4):269–78.

133. Wang WL, Chen KH, Pan YC, Yang SN, Chan YY. The effect of yoga on sleep quality and insomnia in women with sleep problems: A systematic review and meta-analysis. BMC Psychiatry. 2020;20(1):1–19.

134. Bakhshani NM, Amirani A, Amirifard H, Shahrakipoor M. The Effectiveness of Mindfulness-Based Stress Reduction on Perceived Pain Intensity and

Quality of Life in Patients With Chronic Headache. Glob J Health Sci. 2015;8(4):142–51.

135. Jayadevappa R, Johnson JC, Bloom BS, Nidich S, Desai S, Chhatre S, et al. Effectiveness of Transcendental Meditation on Functional Capacity and Quality of Life of African Americans with Congestive Heart Failure: A Randomized Control Study. Ethn Dis. 2007;17(1):72–7.

136. Uyar GÖ, Coşkun AB, Gökalp G, Köksal E. Association of Mediterranean diet and anthropometric measures with quality of life in coronary artery disease patients. Nutr Hosp. 2019;36(3):674–80.

137. Breedvelt JJF, Amanvermez Y, Harrer M, Karyotaki E, Gilbody S, Bockting CLH, et al. The effects of meditation, yoga, and mindfulness on depression, anxiety, and stress in tertiary education students: A meta-analysis. Front Psychiatry. 2019;10(APR):1–15.