Heart disease is the leading cause of death worldwide (GBD 2015 Mortality and Causes of Death Collaborators, 2016) and is associated with a number of behavioral and psychosocial risk factors. Key among the behavioral risk factors are smoking, lack of exercise, obesity, unhealthy diet, and alcohol consumption. Psychosocial risk factors include depression, anxiety, lack of social support, anger and hostility, stress, and vital exhaustion, which involves excessive fatigue, increased irritability, and feelings of demoralization. A variety of mechanisms relate these risk factors to heart disease, including behavioral mechanisms such as poor health habits and physiological mechanisms such as dysregulation of the sympathetic nervous system, neuroendocrine and hemodynamic responses and inflammation, among others. Behavioral and psychosocial factors are also associated with how people cope with heart disease. One key determinant of survival after a coronary event is getting help in an expeditious fashion. However, many people deny the significance of their symptoms and delay for hours, or even days, before seeking help. After a coronary event cardiac rehabilitation that includes exercise training, nutrition counseling, patient education, risk factor management, and psychosocial support, is an important part of the recovery process. There is also evidence that techniques such as transcendental meditation can speed recovery and improve outcomes.

This chapter addresses the question of behavioral and psychosocial factors in heart disease by first examining different diseases of the heart and then going on to discuss behavioral and psychosocial risk factors for heart disease. We will also be considering the ways in which people cope with heart disease.

Diseases of the Heart

Diseases of the heart fall generally into four categories with each having a distinct set of causes. These four categories are coronary heart disease (CHD), valvular heart disease, cardiomyopathy, and electrical conduction disorders.

Coronary Heart Disease

CHD is the most commonly diagnosed disease of the heart. CHD is a result of the buildup of atherosclerotic plaques on the coronary arteries, which is also known as coronary artery disease (CAD). The buildup of these plaques leads to ischemia or a reduction in blood flow to the heart muscle. When they rupture they can lead to a blockage of the coronary arteries resulting in a myocardial infarction.
Valvular Heart Disease

Valvular heart disease (VHD) refers to any disease process involving the four valves in the heart, the aortic and mitral valves on the left and the pulmonary and tricuspid valves on the right. It is mainly associated with aging (Nkomo et al., 2006) but can also be the result of congenital abnormalities, diseases such as rheumatic heart disease, or physiologic processes during pregnancy (Kovacs et al., 2008). VHD can result in diminished heart functionality, which can vary by the type and severity of the valvular disease.

Cardiomyopathy and Heart Failure

Cardiomyopathy refers to various diseases that affect the heart muscle. In cardiomyopathy the heart muscle becomes enlarged, thick, or rigid and in rare cases may be replaced with scar tissue. As cardiomyopathy worsens the heart is weakened and unable to pump blood efficiently or maintain a proper electrical rhythm (Burke & Tavora, 2011). This can, in turn, lead to heart failure or irregular heartbeats (arrhythmias).

Electrical Conduction Disorders

Disorders of the electrical conduction system come about when the heart beats below 60 beats per minute (bradycardia), beats above 100 beats per minute (tachycardia), or beats in an irregular fashion (Guyton & Hall, 2006). Together these are known as arrhythmias. Many times these irregularities cause no symptoms. Symptoms that may be present are palpitations; feeling a pause between beats; or more seriously the person may experience lightheadedness, passing out, shortness of breath, or chest pain. Most arrhythmias are not serious but in some cases they can lead to stroke, heart failure, or cardiac arrest.

Risk Factors for CHD

Although there is no single cause for heart disease, a variety of risk factors have been identified. These can be divided into non-modifiable factors such as age, sex, and family history of CHD and modifiable factors such as high blood pressure, high blood cholesterol, diabetes and prediabetes, smoking, being overweight or obese, being physically inactive, unhealthy diet, and alcohol intake. This chapter focuses on the modifiable behavioral and psychosocial risk factors for CHD.

Behavioral Risk Factors

Smoking

The association between smoking and heart disease has been observed in a number of studies dating back to the 1970s (Doll & Peto, 1976). Smoking affects CHD by raising triglycerides, lowering “good” cholesterol (HDL), making blood sticky and more likely to clot, which can block blood flow to the heart and brain, damaging cells that line the blood vessels, increasing the buildup of plaque in (MI) or heart attack (Guyton & Hall, 2006). CHD is the leading cause of death worldwide (GBD 2015 Mortality and Causes of Death Collaborators, 2016). Risk factors for CHD include obesity, hypertension, smoking, dyslipidemia and diabetes mellitus with chronic renal disease and chronic inflammatory diseases such as rheumatoid arthritis being increasingly recognized as risk factors (Xu, Stokes, & Meredith, 2016).
Cardiovascular Disease

blood vessels, and causing thickening and narrowing of blood vessels (U.S. Department of Health and Human Services, 2010). Smokers are strongly advised to stop smoking and the evidence shows that quitting smoking significantly reduces CHD risk, bringing it down to levels similar to non-smokers (Cook, Shaper, Pocock, & Kussick, 1986). Secondhand smoke has also been related to CHD, with those breathing secondhand smoke at home or at work showing a 25% to 30% increase in CHD risk (U.S. Department of Health and Human Services, 2014).

Lack of Exercise

Physical activity has been shown to have an important relationship to the prevention of CHD (Varghese et al., 2016). Individuals getting regular exercise show a significant reduction in CHD risk. For example, in the INTERHEART study (Held et al., 2012), it was found that across all global regions, both leisure-time physical activity and mild to moderate (but not heavy) occupational physical activity resulted in lower rates of myocardial infarction (MI). Conversely, ownership of cars and televisions tended to lead to more sedentary behavior, which was associated with an increased risk of MI. In addition to its direct effect on reducing CHD, physical exercise also shows positive effects on other CHD risk factors including blood pressure, cholesterol levels, and cardiorespiratory fitness (Varghese et al., 2016).

Obesity

Obesity has become a world-wide epidemic with an estimated 35% of adults globally being overweight and 10% of men and 14% of women being obese (World Health Organization, 2013). Obesity is often measured by body mass index (BMI) which is measured as weight (kg)/height (m)². Obesity is defined as a BMI of 30 or more with a BMI of 25 being considered overweight. While there is substantial regional variation, it is clear that obesity is a world-wide challenge (World Health Organization, 2013). A recent meta-analysis of 97 cohort studies with a total of more than 1.8 million participants showed that the risk of CHD increased 27% for each 5 kg/m² increment in BMI (Lu et al., 2014).

One mechanism by which obesity is related to CHD concerns the effects of obesity on other CHD risk factors as blood pressure, cholesterol levels, and glucose homeostasis. In addition, obesity is related to inflammation and insulin resistance, which are have been associated with CHD (Abassi, Brown, Lamendola, McLaughlin, & Reaven, 2004; Bastard et al., 2006; Dandona, Aljada, & Bandyopadhyay, 2004). Thus reducing weight can help to decrease the effects of these risk factors as well.

Unhealthy Diet

Diet is another modifiable factor that affects the risk of CHD. First, caloric intake is closely associated with weight gain and the risk of obesity (Warwick & Schiffman, 1992). As such, one can avoid weight gain and obesity by reducing one’s caloric intake. Second, beyond caloric intake, it makes a difference what one eats. In particular, diets high in fat content, especially those high in trans fats, are associated with higher plasma cholesterol and greater CHD (Mozaffarian, Katan, Ascherio, Stampfer, & Willett, 2006). Another dietary component related to CHD is salt intake, which is directly related to blood pressure. It has been estimated that reducing salt intake by 3 g per day would reduce the annual number of new cases of CHD by 10% in the United States (Cappuccio, 2013).

Alcohol Intake

Alcohol has been shown to have a somewhat paradoxical relationship to CHD. Early studies showed that high levels of alcohol consumption were related to increased risk of CHD (Dyer et al., 1981).
However, over time, research has suggested a J-shaped association between alcohol consumption and CHD such that low to moderate alcohol consumption was related to lower rates of CHD than was abstinence (Marmot, 2001; Ronksley, Brien, Turner, Mukamal, & Ghali, 2011). However, evidence from other studies indicates that there may be differences in this relationship between geographical regions. For example, low to moderate alcohol consumption may not be as beneficial for South Asians (Leong et al., 2014).

**Gender Differences in CHD Risk**

There is ample evidence that CHD risk varies by gender. Women tend to develop CHD roughly a decade later than do men (Tan, Gast, & van der Schouw, 2010). Although CHD is often considered to affect males more than females, there are several female-specific risk factors for CHD as well as gender differences in risk factors that are not gender specific.

With respect to female-specific risk factors, one potential risk factor is polycystic ovary syndrome (PCOS). Women with PCOS have a greater tendency to be obese and to have metabolic syndrome, which includes high blood pressure, high blood sugar, and high lipid levels, all risk factors for CHD on their own. CHD risk in women with PCOS is increased by between 26% and 67%, although there is some inconsistency in the results (Tan et al., 2010). Another female-specific risk factor is preeclampsia, which involves the onset of hypertension and proteinuria after the twentieth week of pregnancy. Women experiencing preeclampsia have been found to have twice the risk of later developing CHD compared to those having an uncomplicated pregnancy (Bellamy, Hingorani, & Williams, 2007). Another risk factor unique to women is menopause. Premenopausal women have lower CHD rates than men of the same age. However, after menopause women’s risk of CHD increases by roughly 35% (Tan et al., 2010). One treatment that has been suggested for reducing CHD risk among post-menopausal women is hormone therapy (HT). However, the results of studies of HT on CHD risk have been inconclusive and HT is not currently recommended (Tan et al., 2010). Another potential risk is the use of oral contraceptives (OC). Early studies of high dose contraceptives showed an increase in CHD risk among OC users; later studies, with lower dose OCs, found no increased risk. The current evidence is that use of low dose OC has little effect on CHD risk (Tan et al., 2010).

In addition to these female-specific risk factors there are a number of gender differences in risk factors that affect both men and women. As noted earlier, smoking is a significant risk factor for CHD. Although women tend to smoke less than do men, smoking confers a 60% greater risk of CHD for women as compared to men (Tan et al., 2010). Another key risk factor affecting both men and women is hypertension (high blood pressure). Although hypertension increases CHD risk for both men and women, the increase for older women tends to be greater than that for men of the same age when compared to normotensive individuals (Tan et al., 2010).

Another risk factor that shows stronger effects for women than men is diabetes mellitus. Although men with diabetes show a twofold increase in risk compared to those without diabetes women showed a threefold increase (Huxley, Barzi, & Woodard, 2006). Lipid profiles are also a predictor of CHD for both men and women. However, a meta-analysis of 17 prospective cohort studies found that after adjusting for high density lipoproteins, elevated triglyceride levels were associated with a 37% increased CHD risk for women as compared to a 14% increase in men (Hokanson & Austin, 1996). A fifth risk factor common to men and women is obesity, particularly abdominal fat. Evidence to date indicates that women with central obesity have roughly a 20% greater chance of developing CHD than males with central obesity (Tan et al., 2010). Finally, physical inactivity appears to affect women more than men. Lower fitness levels are related to a higher risk of CHD for women as compared to men (Tan et al., 2010).
Psychosocial Risk Factors

There is growing evidence that CHD risk is significantly influenced by a number of psychosocial factors. These psychosocial factors include depression, anxiety, lack of social support, anger and hostility, the Type D personality, work stress, and vital exhaustion.

Depression

A large number of studies have identified depression as a major risk factor for CHD. Meta-analyses have found that the presence of clinically significant depression can increase the risk of CHD by 30% to 90% among otherwise healthy individuals (Carney & Freedland, 2017). Further, depression significantly increases the risk of adverse coronary events among those with established CHD. The reasons for this relationship are still being established but a number of hypotheses have come to the fore. First, depression is associated with behavioral changes that may increase CHD risk. For example, a number of studies have found that depressed individuals are more likely to be physically inactive than non-depressed individuals which, in turn, is associated with greater risk of CHD (Carney & Freedland, 2017). Individuals who are depressed may be less likely to adhere to medical recommendations such as eating a low-fat diet, getting regular exercise, reducing stress, and taking medications (Dhar, Lambert, & Barton, 2016). Further, people who are depressed have higher rates of smoking (John, Meyer, Rumpf, & Hapke, 2004) and depression is also associated with obesity (Stapelberg, Neumann, Shum, McConnell, & Hamilton-Craig, 2011) and heavier alcohol use (Davidson, 1995), both of which are independent risk factors for CHD.

Depression is also related to biological risk factors. One pathway is through dysregulation of the autonomic nervous system, with depressed individuals showing higher levels of plasma and urinary catecholamines, reduced heart rate variability, and higher resting heart rate compared to individuals who are not depressed (Carney & Freedland, 2017). Beyond this, depression is associated with factors that promote atherosclerosis, such as elevated platelet reactivity (Dhar, Lambert, & Barton, 2016).

It has also been suggested that inflammation may be a common causal pathway that is responsible for both the development of depressive symptoms and CHD (Poole, Dickens, & Steptoe, 2011). Individuals experiencing major depression have higher levels of inflammatory cytokines and C-reactive protein (CRP) than those without depression. This relationship may be bidirectional such that inflammation leads to symptoms of depression while depression tends to be increase inflammation (Carney & Freedland, 2017). These factors likely work together in promoting CHD. In sum, depression is associated with many factors that are themselves related to CHD, but no single factor is invariably associated with poor CHD outcomes (Burg et al., 2013).

Anxiety

Fewer studies have examined the relationship between anxiety and incident CHD, but the results of these studies indicate that anxiety is a significant risk factor. A recent meta-analysis of 20 studies involving 249,846 individuals (Roest, Martens, de Jonge, & Denollet, 2010) found a 26% increase in CHD risk due to anxiety along with a 48% increase in the risk of cardiac death. These effects are comparable effect sizes to those found for depression. These findings are important since anxiety is a common experience. For example, a study of 22 countries found that the prevalence of anxiety varied from 12.0% to 41.8% among men and from 21.5% to 63.7% among women (Kotseva et al., 2009). Particular anxiety syndromes carry different risk for CHD. For example, generalized anxiety disorder (GAD) appears to have a strong association with non-fatal cardiovascular disease whereas panic disorders and phobias are unrelated to CHD (Batelaan, ten Have, van Balkom, Tuithof, & de Graff, 2014).
The mechanisms linking anxiety with CHD are not entirely clear but some plausible links have been suggested (Batelaan et al., 2014). On the one hand, worry such as that found in GAD is associated with muscle tension and disturbed sleep, which may increase CHD risk. Further, anxiety has been associated with several biological processes that can promote CHD including increased cholesterol levels (Papakostas, Ongür, Iosifescu, Mischoulon, & Fava, 2004), hypertension (Player & Peterson, 2011), activation of the immune system (Pitsavos et al., 2006), increased blood coagulability (Geiser et al., 2008), and abnormalities in the autonomic nervous system (Friedman, 2007).

Social Relationships

A large number of studies have documented the relationship between social relationships and CHD, for example, showing that for both men and women having lower social support is associated with an increase in CHD risk. Among the first studies to document this relationship was the Alameda County Study which found that having fewer social ties was associated with death, including CHD deaths, over a nine-year follow-up period (Berkman & Syme, 1979). Later studies with larger samples have replicated these findings with the relative risk being relatively comparable by race and sex.

Some studies use marital status as an indicator of social support. In the National Longitudinal Mortality study, the relative risk ratios for death among individuals 45–64 years of age who were divorced or separated were higher compared with currently married individuals (and were relatively comparable by race and sex). The relative risk ratios were smaller for those over 65 but remained statistically significant. Another large scale study examined mortality patterns in the Netherlands from 1950 through 1970 and found a significant increase in both all-cause mortality as well as CHD mortality for both men and women who were unmarried as compared with their married counterparts (von Poppel & Young, 2001).

A number of studies have found a relationship between social isolation and adverse events and death in cardiac patients. A review of studies of morbidity and mortality following a heart attack found relative risk ratios of 2.0 to 3.0 between individuals who were socially isolated or lacked a social support network and those who were more socially integrated (Mookadam & Arthur, 2004). A recent meta-analysis of 148 studies relating social relationships to mortality found that individuals with stronger social relationships showed a 50% increase in survival in the face of life-threatening conditions (Holt-Lunstad, Smith, & Layton, 2010).

Two classes of mechanisms have been identified to explain this relationship. Analyses of the Third National Health and Nutrition Examination Study found that individuals with greater social support were more likely to be tested for hypertension and cholesterol, to eat recommended levels of fruits and vegetables, and to be physically active, and were less likely to be smokers than those with low levels of social support (Ford, Ahluwalia, & Galuska, 2000). A meta-analysis of 122 studies relating social support to medical regimen adherence (DiMatteo, 2004) found that several dimensions of social support, including practical support, emotional support, marital status, family cohesion and family conflict were strongly related to medicine adherence, with adherence being 1.74 times higher in patients from cohesive families and 1.53 times lower in patients from families in conflict.

A second possible pathway is through physiological mechanisms. In particular, low social support has been associated with alterations in cardiovascular, neuroendocrine, and immune functioning (Uchino, 2006). Many studies have indicated that social support is associated with lower cardiovascular reactivity to stress and also with lower blood pressure during everyday life (Uchino, 2006). Much of this can be attributed to the stress reducing properties of social support. Social support is also associated with lower overall cortisol levels (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003) as well as lower plasma and urinary catecholamine levels and higher levels of oxytocin (Grewen, Girdler, Amico, & Light, 2005), and better immune functioning (Uchino, 2006).
Anger and Hostility

There is now substantial research showing a significant relationship between anger and hostility and the occurrence of CHD. Although the hypothesis goes back to at least the 19th century (Smith, Glazer, Ruiz, & Gallo, 2004), empirical work began in earnest after identification of the key components of the Type A behavioral pattern (Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985). The Type A behavior pattern consists of several components, including time pressure, hard-driving behavior, competitiveness, and free-floating anger and hostility. More recent research has focused on anger and cynical hostility as the “active ingredients” for CHD. For example, one early study followed 255 physicians over a period of 25 years and found a nearly fivefold difference in CHD incidence between those high and low in hostility as measured by the Cook and Medley Hostility Scale (Barefoot, Dalstrom, & Williams, 1983). Since then numerous other studies have obtained findings consistent with a significantly increased rate of CHD among those high in anger and hostility (Smith et al., 2004).

A number of hypotheses have been put forward to explain the relationship between anger/hostility and CHD. Of these the most comprehensive is the transactional model put forward by Smith and his colleagues (2004). The transactional model argues that the effects of anger and hostility on CHD can be understood as a transaction between the person and the environment. Individuals high in anger and hostility tend to have lower social support and higher interpersonal conflict than individuals low in anger and hostility. Further, they tend to create situations of higher conflict and show stronger cardiovascular responses to stressful situations, which puts them at greater risk for CHD (Suarez & Williams, 1989).

In addition, there is evidence that individuals high in anger and hostility tend to engage in less healthy behaviors than do those lower in anger and hostility. For example, Miller, Karkides, and Ray (1995) found hostility to be associated with heavy drinking and current cigarette smoking among Mexican Americans. Siegler et al. (2003) found that hostility was associated with avoidance of exercise and a high-fat diet. Further, there is growing evidence that individuals higher in anger and hostility may be at higher CHD risk due to their genetic make-up. Research with genes related to serotonergic function have found these genetic variants to be associated with hostility as well as cardiovascular reactivity, blood pressure, and coronary events (Boyle et al., 2015; Brummett et al., 2013).

Stress

The role of stress in CHD has been the subject of discussion for many years. However, it has only been in the last few decades that scientific evidence has convincingly linked both acute and chronic stress levels to CHD (Dimsdale, 2008). For example, the daily number of deaths attributed to CHD increased dramatically on the day of the Northridge, California, earthquake of 1994 as compared with the same date in previous years (Leor, Poole, & Kloner, 1996). Likewise, studies of chronic stressors have shown a relationship with incident CHD. For example, the INTERHEART study found that individuals reporting chronic stress at home or at work had more than 2.1 times the risk of developing an MI compared to those without such stressors (Rosengren et al., 2004). Work stress has also been pinpointed as a particularly relevant stressor with individuals in jobs having high job strain but low decision latitude being particularly likely to develop CHD (Karasek, 1979).

The mechanisms connecting stress with CHD are still under investigation. However, it is clear that stress can lead to changes in health-related behaviors such as smoking (Byrne & Mazanov, 2016) and excessive alcohol intake (Sillaber & Henniger, 2004), which put one at higher risk of CHD. Further, the neuroendocrine and hemodynamic responses to stress also put strain on the heart. The classic physiological response to stress is characterized by the release of catecholamines and corticosteroids, increases in heart rate, activation of the hypothalamic-pituitary-adrenal axis and increases in
cardiac output and blood pressure as well as platelet aggregation and coronary artery vasoconstriction (Moksnes & Espnes, 2016). Such changes also predispose a person to cardiovascular events by inducing endothelial dysfunction, increasing atherosclerosis, promoting plaque rupture, and by setting off lethal arrhythmias (Krantz, Whittaker, & Sheps, 2012).

Vital Exhaustion

Vital exhaustion refers to excessive fatigue, increased irritability, and feelings of demoralization (van Diest & Appels, 1991). Although these symptoms seem to have much in common with depression there is evidence that they are actually two distinct constructs. In particular, individuals experiencing vital exhaustion generally don’t exhibit the sad mood that is characteristic of depression (van Diest & Appels, 1991). Studies of the relationship of vital exhaustion to CHD have found that initially healthy individuals exhibiting vital exhaustion are 50% more likely to develop CHD than are individuals without this trait. Further, individuals with CHD who exhibit vital exhaustion are twice as likely to have recurrent cardiac events (Frestad & Prescott, 2017). One factor that seems to be related to vital exhaustion is social class, with low education and income strongly associated with vital exhaustion (Skodova et al., 2008).

The research evidence linking vital exhaustion to CHD has been inconsistent. On the one hand, vital exhaustion may influence health behaviors that affect influence CHD. For example, Igna, Julkunen, and Vanhanen (2011) found a relationship between vital exhaustion, unhealthy behaviors such as inactivity, smoking, alcohol use, and poor diet, as well as levels of triglycerides, which are an independent risk factor for CHD. A path model suggested that the relationship between vital exhaustion and triglycerides was mediated by the unhealthy behaviors. On the other hand, Koertge, Ahnve, Schenck-Gustafsson, Orth-Gomer, and Wamala (2003) found no relationship between vital exhaustion and lifestyle variables.

Another possible mechanism linking vital exhaustion to CHD is dysregulation of the hypothalamus-pituitary-adrenal (HPA) axis due to prolonged stress. However, here again the evidence is contradictory (Frestad & Prescott, 2017). Finally, it is possible that vital exhaustion is associated with inflammatory processes, which are in turn related to CHD (Janszky, Lekander, Blom, Georgiades, & Ahnve, 2005).

Coping With Cardiovascular Disease

We now turn to the question of how people deal with CHD, looking first at the problem of denial and delay in seeking treatment and then addressing ways in which patients cope with heart disease and make a good recovery.

Cardiac Denial and Delay in Treatment

It is a well established fact that the sooner help is received for cardiac symptoms, the better the person’s chances of survival. It is desirable to get treatment within one to two hours of the onset of symptoms (Moser, Dracup, & Wu, 2012). However, delay time is often two to six hours (Moser et al., 2006) and there are a large number of people who delay for days. The evidence indicates that people are most likely to delay if they are older, female, have less education, are minorities, and have low socioeconomic status (Moser et al., 2012). Further, they are more likely to delay when they have a history of angina (chest pain), and have risk factors such as hypertension or diabetes, or when symptom onset is at home and they consult with a physician or family member. One of the things that happens is that people with cardiac symptoms tend to wait for those symptoms to go away, have concern about troubling others, and fear the consequences of seeking help. When people attribute
symptoms to cardiac sources they are likely to delay less and when they engage in self-treatment they tend to delay more (Moser et al., 2012).

Another issue related to delay in seeking treatment is the problem of denial. It is not unusual for people experiencing symptoms of a heart attack to deny the significance of their symptoms (Moser et al., 2012). However, it is unclear the extent to which denial results in delay in seeking treatment. While some studies have shown a relationship between denial and delay in seeking treatment, others have not. For example, Perkins–Porras, Whitehead, Strike, and Steptoe (2008) found that denial was related to longer delays. However, Carney, Fitsimons, and Dempster (2002) found no relationship between denial and delay. As such it is an open question as to whether denial leads to longer delays in seeking treatment.

**Cardiac Rehabilitation**

An important part of recovery from CHD involves changes in lifestyle and behavior. Cardiac rehabilitation (CR) typically consists of a number of components including structured exercise training, nutrition counseling, patient education, risk factor management, and psychosocial support, the aim of which is to produce significant changes in lifestyle and behavior. A review of CR practices worldwide found that most CR programs lasted for a median of 20 sessions and were generally delivered by physicians, nurses, and physiotherapists (Pesah, Supervia, Turk-Adawi, & Grace, 2017).

CR programs have been shown to be both clinically effective and cost effective in improving outcomes for CHD patients. For example, a clinical trial of a community-based CR program in the Netherlands showed significant improvement in risk factors such as lack of exercise, smoking and being overweight as compared with usual care (Minneboo et al., 2017). Another comprehensive lifestyle program was effective in reducing coronary events in heart patients over a four-year period when compared to individuals receiving usual care (Vizza, 2012). Further, CR has been shown to be cost effective when compared to usual treatment as well as an exercise program alone (Dang, Ji, Jhamnani, & Wang, 2017).

**Exercise**

Just as a sedentary lifestyle is a risk factor for CHD so, too, it is important for cardiac patients to engage in levels of exercise that will reduce cardiovascular risk. The importance of this is illustrated by a world-wide study involving more than 15,000 adults that examined the relationship between self-reported physical activity and both cardiovascular and all-cause mortality (Steward et al., 2017). In this study, doubling exercise volume was associated with a reduction of 17% in cardiac mortality. Current guidelines from the European Society of Cardiology recommend moderate-to-vigorous intensity aerobic exercise at least three days per week for 30 minutes per session (Montalescot et al., 2013). While these guidelines are for the minimum amount of recommended physical activity, it is clear that, except for extreme levels of physical activity, more activity is better. Although improvements in cardiopulmonary fitness are the most likely reason for these results there is also evidence that exercise helps fight symptoms of depression, which are often a problem for heart patients (Stein, 2012).

**Stress Management**

Stress is a psychosocial risk factor for incident CHD and also appears to play a role in recurrent coronary events (Dimsdale, 2008). As such, stress management training can play an important role in helping individuals cope with CHD even though it is not currently a routine component in CR. One study of bypass graft patients in Singapore found significant reductions in depression, trait
anger, and cardiovascular reactivity to stress three months after a workshop intervention that taught patients how to manage stress and build stronger relationships with cardiovascular reactivity reduced 60% (Bishop et al., 2005). The importance of this for clinical outcomes is shown in a recent study by Blumenthal and his colleagues (2016). CHD patients were randomly assigned to either a standard CR program or a CR program plus stress management and followed for up to five years. Patients receiving the additional stress management component showed significantly fewer adverse coronary events compared with those in the standard CR program and those not receiving any CR. This has led to the recommendation that stress management be incorporated as a routine component in CR programs (Lim, 2016).

Treatment for Depression

As noted earlier, depression is a significant risk factor for the development of CHD. Evidence also shows that depression among CHD patients is a significant predictor of recurrent coronary events, with depression conferring a two to three times risk for mortality or non-fatal coronary events (Lett et al., 2004). Despite such data there is a paucity of evidence that treating CHD patients for depression leads to better outcomes. Studies of treatment of CHD patients for depression have generally not reduced the number of recurrent coronary events or reduced mortality, even when the intervention leads to a reduction in depression (Burg & Czajkowski, 2012; Lett et al., 2004).

The question arises as to why this is the case. One possibility is that the timing of the intervention needs to be fine tuned to address patients’ needs. For example, in the ENRICHD study (Burg & Czajkowski, 2012), treatment for depression was initiated shortly after their MI and those treated did not differ from the control group with respect to recurrent coronary events. In retrospect, this may not have been the optimal time to assess and intervene, as there was a good chance of detecting an adjustment disorder or transient depressed mood rather than a true underlying depression. Also immediately after an MI, patients may be less inclined to want to deal with the issues related to depression. This suggests that assessment and intervention at a later time might provide more accurate diagnosis of depression and that treatment would be more effective. It also suggests that it may be better to treat depression with medication or exercise rather than psychotherapy (Lett et al., 2004).

Transcendental Meditation

Meditation has been practiced for centuries for its reputed psychological and health benefits. Recent evidence suggests that Transcendental Meditation is an effective way for CHD patients to reduce their risk of recurrent coronary events (Orme-Johnson, Barnes, & Schneider, 2012). Studies have shown that TM is associated with reduced sympathetic nervous system activation, reduced cortisol secretion, reduced blood pressure, reduced cardiovascular responses to stress and favorable changes in metabolic syndrome, trait anxiety, depression, alcohol consumption, and tobacco use (Orme-Johnson et al., 2012). Most importantly, TM has been found to reduce myocardial ischemia and carotid atherosclerosis and to reduce cardiovascular clinical events and mortality (Orme-Johnson et al., 2012; Schneider et al., 2012). This strongly suggests the inclusion of TM in standard CR regimens.

Conclusion

The evidence is clear that CHD is as much a behavioral and psychosocial condition as it is a medical one. There are multiple behavioral and psychosocial risk factors for CHD that health psychologists can intervene to change. On the behavioral side, smoking, lack of exercise, obesity, an unhealthy diet, and excessive alcohol intake are associated with higher levels of CHD. Looking at psychosocial factors, we find that depression, anxiety, lack of social support, anger and hostility, stress and vital...
exhaustion are all significantly related to the occurrence of CHD. Such data suggest possible ways of reducing the risk of CHD and suggest interventions for stopping smoking, getting more exercise, reducing obesity, changing diet and reducing alcohol intake. They also suggest that addressing problems of depression, anxiety, lack of social support, anger and hostility, stress, and vital exhaustion should be effective in reducing CHD risk.

Moving beyond the risk factors for incident CHD it is also important to consider the factors involved with coping with a CHD diagnosis. One major problem in reducing CHD mortality is to reduce the amount of delay in seeking help for CHD symptoms. It is clear that getting rapid treatment is essential to reducing CHD mortality, but also that many people delay for hours and even days before seeking help. Once diagnosed with CHD it is important for patients to engage in cardiac rehabilitation, increase their exercise, and get treated for depression. It is also helpful to engage in transcendental meditation. All of these can help to reduce the likelihood of recurrently coronary events and extend the person’s lifespan.

References


