PSYCHOLOGIC AND SOCIAL ASPECTS OF CARDIOVASCULAR DISEASE

Kathleen B. King, R.N., Ph.D.
University of Rochester

ABSTRACT

In summarizing the evidence, it becomes apparent that several psychologic and social variables are related to coronary heart disease (CHD). Coronary prone behavior pattern, in particular the hostility component, appears to be related to the development and perhaps expression of CHD, whereas it is not reliably related to outcomes after CHD is manifest. Depression clearly has been shown to be related to outcomes after CHD has declared itself. Lack of social ties appears to be related to mortality, whereas emotional social support has been shown to be related to recovery from coronary events. It also seems apparent that there are subsets of vulnerable individuals who might be best served by targeted interventions. Interventions are proposed as suggested by the prevailing evidence.

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INTRODUCTION

Research examining the connection between psychologic and social factors and coronary heart disease (CHD) has steadily grown over the past two decades. The majority of the research done to date has been descriptive and can be categorized into one of two types of samples studied. One group of studies focuses on relationships between psychologic and social factors and the development and/or initial manifestation of CHD. These studies generally use population-based samples without regard to CHD disease status. Central to this line of inquiry is the identification of risk factors that may be amenable to primary prevention efforts. A second group of studies address psychologic and social factors in relation to outcomes after CHD has become manifest, usually expressed as a myocardial infarction (MI) or coronary artery bypass grafting (CABG) surgery. These studies are limited to samples of individuals known to have CHD. Here, the identification of factors that may be amenable to secondary preventive efforts is a key objective.

In addition to the descriptive work, a small number of experimental studies have tested interventions designed to improve outcomes after a coronary event. The bases for these experiments can be found in earlier empirical work, but also are derived from clinical observation.

Using these different approaches, a few content areas related to psychologic and social factors and CHD have received significant attention. Psychologic factors include Type A behavior pattern and depression. Social factors include social support and social isolation. While other factors have received some attention in the literature, the above few were chosen for review because sufficient data exist to draw some preliminary conclusions. Thus, this article presents a summary of the evidence within each of these content areas, with attention to how relationships may be dependent on the samples studied, as well as the methodologic approach used. Where possible, existing reviews of the literature are used and updated with studies that have appeared more recently.

TYPE A BEHAVIOR PATTERN AND CHD

Descriptive Studies

Friedman and Rosenman first described Type A behavior pattern in the 1960s, which was based on their clinical observations of men who suffered MI. Since then, this concept has been the focus of numerous reports aimed at explicating the relationship between Type A behavior pattern and CHD. Studies using all three of the methodologies outlined above can be found. The majority of studies have used cross-sectional designs, thereby limiting interpretation of the direction of relationships. However, a number of prospective studies have been done in which it is possible to estimate how well Type A behavior predicts outcomes. Population-based studies have examined the capacity of Type A behavior to predict the onset of CHD. As well, studies limited to individuals with known CHD have examined the influence of Type A behavior on illness outcomes. Several reviews of this literature were published in the late 1980s, and a consensus regarding Type A behavior began to emerge (1–3). One particularly elegant and often-cited meta-analysis was published by Matthews (1). Only prospective studies were included and studies were weighted for sample size (4–24). Studies were separated into population-based samples versus samples of individuals with known CHD. Separate analyses were conducted for types of measurement used to classify Type A behavior [i.e. the Structured Interview (Type A-SI) and the Jenkins Activity Scale (Type A-JAS)]. In addition, studies that measured hostility (via the Cook–Medley Scale of the Minnesota Multiphasic Personality Inventory), depression, and anxiety were included. No significant relationship was detected between Type A behavior and CHD when all studies were analyzed together. However, several significant relationships were found when the subset analyses were done. The Type A-SI measure predicted CHD, whereas the Type A-JAS measure did not predict CHD. Combined, both measures of Type A behavior predicted CHD incidence in population-based studies, but were not related to mortality or recurrent events in studies limited to subjects with known CHD. Of particular interest, hostility measured by either the Type A-SI or the Cook-Medley Scale was a reliable predictor of CHD incidence in the population-based studies. Matthews suggested that the Type A-SI may have predicted CHD incidence because it includes measurement of hostility, whereas the Type A-JAS does not.

In another review, Williams (2) suggested that global measures of Type A behavior pattern are not the best measure of coronary prone behavior. He proposed that the "hostility complex," consisting of cynicism, angry feelings, and aggressive responding to provocation, may be the underlying factor that

Reprint Address: K. B. King, R.N., Ph.D., Associate Professor of Nursing and Psychology, University of Rochester, School of Nursing, 601 Elmwood Avenue, Box SON, Rochester, NY 14642.

predicts CHD. He also summarized evidence that suggests the relationship between the hostility complex and CHD is stronger in younger adult men compared to older men.

Since these papers were published, additional original research reports have examined the relationship between Type A behavior pattern and CHD. Friedman and Booth-Kewley (25) compared 50 post-MI men with 50 healthy men using a cross-sectional design. Type A-SI discriminated between men with and without CHD, whereas Type A-JAS did not. However, hostility was not related to CHD. Barefoot and colleagues (26) conducted a cross-sectional study of 50 men who were asymptomatic but at high risk and underwent coronary angiography. Half of these men were diagnosed with CHD. Neither Type A-SI nor hostility discriminated between those with and without CHD. However, in a subset of non-smokers, those categorized as Type A were more likely to have CHD. Brackett and Powell (27) followed 862 patients who had survived an MI for an average of 4.5 years. Type A-SI was associated with sudden death, but not with non-sudden death or recurrence of MI. Comparing sudden to non-sudden death, Type A-SI was the most significant factor. Barefoot and colleagues (28) interviewed 1,201 men and 266 women with documented CHD and followed them for six years. Neither Type A-SI, Type A-JAS, nor hostility were associated with cardiac mortality or non-fatal events. All of these studies controlled for at least one known physical indicator of disease severity, such as left ventricular function.

Two of these studies offer some evidence that Type A-SI, although not hostility, is related to CHD; although neither study is a prospective or a population-based study (25,26). The other two studies add evidence that Type A does not reliably predict increased cardiac mortality or morbidity in those with known CHD, although one study did predict sudden death (27,28). These studies add confidence to the conclusions drawn from the earlier reviews. It seems increasingly clear that Type A behavior, as measured by Type A-SI and likely reflecting the hostility component, is a significant independent predictor of the incidence of CHD. It is also evident that Type A behavior, no matter how it is measured, is not a reliable predictor of morbidity or mortality in those who already have developed CHD.

While not enough time has elapsed to predict the development of CHD from measures of Type A behavior pattern in children, some work has been done to examine the relationship between Type A behavior pattern and coronary risk factors. Several studies have found no difference between children classified as Type A or Type B in terms of resting blood pressure (29–31). Kennard and colleagues (32) found that children (aged 6–16 years) classified as Type A actually had lower systolic and diastolic blood pressure than children classified as Type B. Most recently, Lee and colleagues (33) assessed Type A behavior and blood pressure in school-aged children and followed them through high school. Type A behavior pattern was not associated with elevated blood pressure at the time of the original measurement (school-age) nor did it predict increases in blood pressure over a ten-year period. In fact, both males and females classified as Type B had higher diastolic blood pressure than those classified as Type A.

Hayman and colleagues (29) found no differences between Type A and Type B school-aged children on several indices of obesity. In addition, Type A behavior, measured using the Matthews Youth Test for Health (MYTH), was associated with lower total and LDL-cholesterol and apolipoprotein-B levels. The impatience–aggression component of the MYTH also was related to lower serum lipid levels. Similarly, Kennard's group (32) found that children classified as Type A had lower total serum and LDL-cholesterol levels. Of particular interest, they found that Type A children tended toward better exercise endurance on the treadmill and had significantly higher social competence skills and less behavioral disturbance than Type B children.

Consistent with the studies of children and younger adolescents, hostility (Cook–Medley Hostility Scale) was not found to be related to serum cholesterol levels or physical fitness in older adolescents and young adults (18–30 years) in the 1985–1986 cross-sectional sample of the Coronary Artery Risk Development in Young Adults (CARDIA) Study (34). However, hostility was strongly related to smoking. Longitudinal data from the CARDIA Study demonstrated that persons with higher hostility scores were more likely 21 to 23 years later to be current smokers and have higher serum lipid levels (35).

The above studies are quite consistent in demonstrating that Type A behavior pattern is not related to a higher physiologic risk factor profile in children and adolescents. In fact, it appears that some components of the Type A behavior pattern may be beneficial to children. On the other hand, hostility has been shown to be related to a major behavioral risk factor, smoking, and to higher lipid levels in young adults.

Several explanations have been offered for why Type A behavior/hostility is related to incidence of CHD but far less consistently to outcomes in persons with known CHD. It may be that there is less variance in Type A behavior pattern in persons with known CHD, thus making it unreliable as a predictor in these studies. Another explanation is that the hostility component of Type A influences initial events, but not later events (1). Even if this is so, it is not clear whether the influence is on the development of atherosclerosis over time or on the precipitation of an acute event such as MI. Interestingly, two of the current studies offer data that suggest opposing mechanisms. Based on the finding that Type A-SI discriminated between asymptomatic men with and without CHD, Barefoot and colleagues (26) proposed that hostility may be linked to the pathogenesis of CHD rather than to factors that produce symptoms or trigger coronary events. On the other hand, because Type A-SI was related to sudden death after MI but not non-sudden death or recurrence of MI, the Brackett and Powell (27) data suggest that Type A behavior may influence the onset of an acute event more so than the process of atherosclerosis. Although somewhat different in terms of the time frame, both explanations fall within the psychophysiologic model for linking this behavior pattern to CHD. This model hypothesizes that Type A, in particular hostility, heightens cardiovascular and neuroendocrine reactivity [see Smith (36) for review]. To date, most of the research in this area is laboratory-based. Little has been done longitudinally or in patients with established CHD. Obviously, there is a need for continued exploration of the possible mechanisms by which Type A influences CHD incidence.

Perhaps more intriguing is that a few studies have demonstrated associations between Type A behavior and better outcomes after MI, as well as between Type B behavior and poorer outcomes. Data from the Western Collaborative Group Study showed that among 231 post-MI patients, those categorized as Type B had higher mortality than Type As (19). In another study of 1,201 men and 266 women for a subset of patients with the highest severity of disease scores after MI, those categorized as Type A (Type A-SI) also had better survival (28). Most recently, Ahern and colleagues (37) reported that Type B behavior was associated with higher mortality and cardiac arrest in a sample 265 post-MI patients followed for one year. They used the Bortner Type A–B Scale

which measures time pressure and hard driving components of behavior (but not hostility) and is controlled for standard physical variables. Ahern's group speculates that those individuals who withdraw from activities may be at highest risk for mortality or cardiac arrest, not those who are ambitious and engaged in life. Given these findings, it would be reasonable to consider Barefoot and colleagues' suggestion that teaching post-MI patients to decrease Type A behavior may not necessarily be in their best interest (28).

Intervention Studies

Several experiments have been conducted to test the efficacy of altering Type A behavior. All of these studies were conducted in samples of individuals with known CHD and predate the recent consensus that Type A behavior is not a reliable predictor of outcomes after MI. In reviewing 10 studies, Nunes and colleagues identified 8 different types of interventions, with 17 distinct treatment groups using a variety of combinations of 8 types of intervention strategies (38–48). Despite the variation in interventions, meta-analysis showed that Type A behavior could be significantly decreased. The studies were analyzed to ascertain which treatments were most effective in reducing Type A behavior. Treatments were successful to the extent that they included an educational message, a coping method (relaxation or cognitive), and a behavioral intervention (imagery or behavioral modification), thus suggesting that a comprehensive approach is most effective. Lastly, the authors examined the effect of treatment on morbidity and mortality. Interventions were effective in reducing mortality measured three years later but not one year later, although this finding was based on only two studies. Since this meta-analysis was reported, Friedman and colleagues (49) published an extension of their work in which they compared post-MI patients randomly assigned to either cardiac counseling plus training to alter Type A behavior or cardiac counseling alone. They also used a non-random comparison group. The short-term results of this study were included in the above meta-analysis. Additional findings showed that those receiving counseling plus Type A training had fewer cardiac recurrences over the 4.5 years of follow-up than either the counseling only or the non-random comparison group. While no differences in cardiac deaths were apparent for the first year post-MI, those in the Type A training group suffered significantly fewer deaths between 1 and 4.5 years after MI.

It is difficult to judge how to interpret or apply these results in light of the more recent data demonstrating the unreliability of the relationship between Type A behavior and outcomes in persons with known CHD. Part of the challenge lies in the fact that all the interventions used to date are multifaceted, including cognitive and environmental restructuring, behavioral components, and education aimed at reducing Type A behavior. In addition, these interventions are usually incorporated into programs that address other aspects of risk reduction for CHD. Thus, it is hard to know what component(s) of the interventions are responsible for the effect and how best to structure data-based interventions in the future.

Conclusions

It seems clear that Type A behavior holds some relationship to the incidence of CHD. Over the last few years the terminology has begun to shift from "Type A behavior pattern" to "coronary prone behavior pattern," acknowledging that only part(s) of the original concept, hostility being the leading contributor, may be detrimental to one's health. There is also evidence to suggest that Type A behavior can be altered, however the data addressing the extent to which changing behavior affects disease course and outcome are equivocal. What is not altogether clear are the mechanisms that explain the demonstrated relationships. The most widely proposed hypothesis is the psychophysiologic model. Other mechanisms have been proposed, including the psychosocial vulnerability model, the transactional model, and the health behavior model (36). The psychophysiologic model suggests that hostility heightens cardiovascular and neuroendocrine activity. Heightened reactivity may take its toll in the long run by initiating and fueling the atherosclerotic process and/or in the short run by triggering symptoms or acute events. The psychosocial model postulates that certain types of psychosocial variables such as low social support or high interpersonal conflict are related to hostility and form a profile that may make one vulnerable to disease. The transactional model integrates the psychophysiologic and psychosocial models and postulates that people high in hostility not only respond to the environment with heightened physiologic reactivity, but create more frequent and provocative environments by their own thoughts and actions. Lastly, the premise of the health behavior model is that hostile people have poorer health habits, putting them at greater risk for disease. The data demonstrating that hostility in young adults was found to be related to smoking behavior supports this model. While no studies have directly tested these models, they provide the frameworks necessary to examine mechanisms in future research. Given the number of possible explanations, they also suggest that the mechanisms are probably complex and multidimensional and that a search for simple, direct relationships may fall short of the mark.

DEPRESSION AND CHD

The link between depression and CHD has received increasing attention in the past decade. Most of the research in this area has focused on the relationship between depression and recovery from coronary events, and all of it has been descriptive. However, there is a small amount of evidence that links depression to the incidence of CHD. In one population-based study, Anda and colleagues (50) demonstrated that people with depressed affect were at increased risk of both fatal and non-fatal ischemic heart disease, even after adjusting for standard risk factors and demographics.

In an extensive review addressing depression and MI, Fielding (51) showed that a significant number of patients suffer depression following their MI. The prevalence of post-MI depression varies widely across studies (from 16% to 64%) depending on the measurement approach used, as well as whether major and/or minor depression were included within the depressed category. In general, lower prevalence of depression is found when strict diagnostic criteria are used (52). Nonetheless, post-MI and post-CABG depression are significant clinical problems for a number of people.

It is well recognized that depressive disorders negatively affect quality-of-life and can pose a major obstacle in returning to a fully functional life-style after a cardiac event (51). Since Fielding's review, other evidence substantiates this notion. For example, depression has been shown to be related to impairment in activities of daily life shortly after MI, as well as to impaired social functioning in the year after MI (53).

In addition to the toll that depression takes on the patient and the family's quality-of-life, it has been demonstrated that depression increases the risk for mortality after MI. In a subset of 335

patients partaking in the Cardiac Arrhythmia Pilot Study (CAPS), Ahern and colleagues (37) found that depression was an independent predictor of death or cardiac arrest. In a sample of 153 men and 49 women post-MI, depression was also found to be an independent predictor of mortality six months post-MI, controlling for baseline clinical variables (54). Of particular interest, depressed patients did not differ from non-depressed patients on severity of cardiac disease, although previous MI and left ventricular function also predicted mortality. These same patients were followed through 18 months post-MI (55). In-hospital depression continued to predict death up to 18 months after MI, although the largest effect was between MI and the 6-month follow-up.

A question often posed is whether depression experienced by patients predates their CHD event versus whether it is a reaction to either MI or surgery and the ensuing hospitalization (51). Schleifer and colleagues (56) interviewed 283 men and women within two weeks after MI. Although only 8% of subjects reported a prior psychiatric history, the prevalence of post-MI depression was high (45%). Prior history was not associated with post-MI depression. In contrast, in a sample of 129 men and women, Forrester and colleagues (52) found 19% of patients to be depressed after MI, and post-MI depression was related to a personal history of mood disorder, although not to a personal history of any psychiatric disorder. Lesperance and colleagues (55) found 15.8% of patients to be depressed one week after MI. Patients with a pre-MI history of depression were more likely to be depressed in-hospital and were more likely to become depressed after discharge from the hospital. This is the only study that examined CHD outcomes related to prior and recurrent depression. Although pre-MI depression was not related to post-MI mortality, those with recurrent depression at the time of their MI (versus those with first-time depression) were at significantly increased risk for mortality 18 months after their MI (40% versus 10%). Those with recurrent depression also had higher depression scores than those experiencing first time depression. It is notable that the two studies using the strictest criteria for diagnosis of depression found the lowest prevalence and demonstrated a positive association between prior history of depression and post-MI depression. It has been proposed that patients with major depression have a genetic predisposition that puts them at higher risk for recurrence depression and poorer prognosis after MI (57).

In summary, there is substantial evidence that depression is a problem for a subset of people after a cardiac event and that post-MI depression is related to mortality. The data suggest that use of a reliable measure of clinical depression can enhance the identification of those who may be at most risk for poor outcomes. Although more evidence is necessary before firm conclusions can be drawn, the data also suggest that patients experiencing recurrent depression in-hospital should receive priority attention.

SOCIAL SUPPORT/SOCIAL ISOLATION

Common typologies that are referred to under the general heading of social support have been extensively reviewed and critiqued elsewhere (58,59) and only will be briefly summarized here. Support from the social environment can be measured in terms of either structure or function. Structural support usually refers to the existence of and/or interconnections between social ties. Examples of structural type variables include marital status, living arrangements, and number of social contacts, and are often indexed together to represent social integration. Functional support usually refers to whether the social ties provide specific functions. Examples of functional type variables include emotional support, tangible aid, feeling of belonging, and informational support.

Descriptive Studies

Despite the variety of ways in which social support has been conceptualized and measured, consistencies in empirical findings have emerged. In a recent review, Berkman (60) showed that social isolation and lack of social ties consistently carries a higher risk of mortality from all causes in population-based studies. Using a population-based sample and focusing only on prediction of CHD morbidity and mortality, Orth-Gomer and colleagues (61) have shown that social support, measured as social integration, predicted the incidence of CHD in a sample of 736 men free of heart disease at study intake. Subjects were followed for six years and social support remained a significant predictor controlling for other risk factors. In another prospective, population-based sample of 2,603 adults, Vogt and colleagues (62) examined the incidence of and mortality from ischemic heart disease as predicted by social networks in terms of network scope (i.e. the number of domains in which people had social relationships), network size, and frequency of contacts. Only scope of networks predicted five-year mortality. None of the social support measures predicted incidence of cardiac disease. Thus, the evidence to date indicates that social ties are related to mortality, but whether they are related to incidence of CHD is less clear. Given the relatively few studies examining the relationship between social ties and incidence of CHD, it is probably premature to draw any conclusions.

As is true with coronary prone behavior and hostility, it is not clear what mechanisms underlie the relationships that have been demonstrated, nor how time plays into the picture. For example, how does lack of social ties influence mortality if not through the development of disease? On the other hand, could social ties influence the development of health behaviors and risk factors, rather than directly influencing the incidence of clinical manifestation of disease (60)? In addition, studies are needed to ascertain whether lack of social ties can be altered, and if they can, whether altering them will improve health and health outcomes. Crucial to the development of interventions is an understanding of these mechanisms.

Data also are accumulating that suggest a relationship between lack of social ties and/or social isolation with mortality and morbidity after a CHD event. Berkman (60) reviewed five post-MI studies published through 1992 and found that lack of social support predicted mortality in each study (63–67). In one study, lack of emotional support was related to increased risk of mortality for both men and women after MI, although the relationship was stronger for men (63). One study also demonstrated a relationship between living alone and non-fatal recurrent cardiac events (64). All five studies controlled other medical variables traditionally shown to predict outcomes after MI. Since this review was published, one study has confirmed earlier findings. A prospective study of 232 people undergoing cardiac surgery found that lack of participation in social groups predicted mortality within six months of surgery, controlling for prior surgery, age, and impaired activities of daily living, although no relationship was found between mortality and marital status or having no confidant (68). However, in the CAPS study, the measure of social support was not related to cardiac arrest or death (37).

All of the above studies use mortality or cardiac morbidity as outcomes. Studies using other outcomes, such as functional and emotional outcomes after cardiac events, have demonstrated that social support is related to recovery. In a study of 155 men and

women undergoing CABG and 103 of their spouses, naturally occurring support which enhanced self-esteem was related to both emotional and functional outcomes in patients and spouses up to one year after surgery (69). Others also have shown social support to be related to outcomes after CABG, measuring support as a unidimensional construct (70–72), as well as when using a multidimensional measurement strategy (73).

Intervention Studies

In a series of papers, Frasure-Smith and colleagues (74–76) reported the results of a randomized experiment including 453 men post-MI. The intervention was structured to monitor stress levels and intervene when they were assessed to be high during the year after MI. Subjects in the intervention group had lower stress scores and fewer deaths occurred at the one-year follow-up. No differences between the groups were detected for the rate of hospital readmissions or length of admission, although the experimental group had more health care visits during the year after MI (74). Following these subjects longitudinally, the mortality rate was not different between the groups between one and five years after MI (75). Experimental group subjects did have fewer recurrent MIs from two to five years after the index MI, but again the groups did not differ in rate of hospital readmissions. In a secondary analysis of these data, the sample was divided by level of stress (76). This approach indicated that the intervention had its effect for both risk of cardiac death and recurrence of MI in the high-stress group, but not in the low-stress group. Because the intervention was atheoretical by design, Frasure-Smith and colleagues suggest that the type of intervention may not matter as much as the fact that subjects receive regular, concerned, and supportive attention. This is not helpful in understanding what mechanisms may be at play; however, results of the last analysis suggest that patients with high levels of stress benefit most from the intervention. As such, they should receive priority for attention-oriented interventions.

Conclusions

The question arises as to how to use the social support data to enhance health care and outcomes. The evidence bears out that social support, even considering the wide variety of ways it has been measured, is predictive of cardiac and all-cause mortality. Although less plentiful, the evidence also is consistent in demonstrating a relationship between social support and other types of recovery outcomes after cardiac events. What is notable is that, by far, the majority of studies measure social support as it naturally exists in each person's environment. A recent review of social support and cancer may be helpful in putting this fact in perspective (77). This review demonstrated that while correlational studies suggest a strong relationship between naturally-occurring emotional support and psychological outcomes, studies of peer discussion group interventions aimed at providing emotional support outside the context of the patient's usual support system are not as convincing. On the other hand, educational interventions aimed at providing information did positively affect outcomes, although not as strongly as might be expected given the findings from the descriptive, correlational studies. Provider-based educational interventions may be more effective because they meet the needs of a greater proportion of patients, whereas emotional support interventions are disproportionally needed by those lacking this type of support in existing relationships.

The question for the health care provider remains—where are social support interventions best aimed? Helgeson and Cohen (77) suggest that interventions focused on altering or enhancing existing emotional supports, when they are found to be inadequate, may prove most beneficial rather than trying to create new social relationships that are provider-based. When this is not possible to accomplish, which is inevitable in some cases, provider-based support may be central to enhancing outcomes.

Fortunately, questions regarding how best to treat depression and social isolation in people with CHD is being addressed in a study funded by the National Institutes of Health (78). This multicenter, randomized clinical trial, titled "Enhanced Recovery in Coronary Heart Disease Patients (ENRICHD)," began recruiting subjects in the fall of 1996. Targeted to post-MI patients, the study will test whether the risk of cardiac death and recurrent MI can be reduced by ameliorating depression and/or low social support. The experimental intervention is designed to begin individual cognitive–behavioral therapy within days of the index MI. Individual therapy will be tailored to each subject's unique needs and problems, and length of treatment will vary depending on patient progress. Persons with major depression will be evaluated for concurrent pharmacotherapy. Subjects will progress to group therapy, intended to provide a socially supportive environment in which subjects can practice skills acquired during individual therapy. Subjects in both the experimental and control arms will receive health education about CHD risk factor modification. Findings from the trial will enhance the ability to specify psychologic and social interventions and continue to improve patient outcomes.

FUTURE OPPORTUNITIES

While some consistent relationships have emerged, there are a few problems in this body of literature. The way in which psychologic and social variables are measured is indeed variable. As an example, in the five post-MI studies reviewed by Berkman, five different strategies were used to measure social support (60). Conceptualizations ranged from measuring social networks (quantity of support) to social interaction (quality of support). This same problem exists in the coronary prone behavior literature, as well as in studies of depression and CHD. Differences in methodology and measurement strategy can be seen as a limitation. On the other hand, confidence in study results is enhanced given that they are fairly consistent despite these variations. Regardless, use of valid and reliable measures will help in future research.

Much of the research is limited to middle-aged, Caucasian men. This is especially true of studies examining Type A behavior pattern, hostility, and depression (79), although studies examining the effects of social support in women are also limited. Increasingly, women are included in studies, but often the effects of gender are not examined. A far larger problem is the inclusion of subjects other than Caucasians. Much work needs to be done to understand how psychologic and social variables influence CHD in diverse populations.

Lastly, many studies focus on only a few psychologic or social factors that have the potential to affect CHD. Despite this, there is a growing consensus that the influence of psychologic and social factors on both the development of and recovery from CHD is multifactorial and that the literature would be enhanced by a multivariate approach. In addition, there is a great need to further the understanding of mechanisms involved in the relationship between psychologic and social variables and CHD. Undoubtedly, this understanding will be enhanced by theory-driven research.

SUMMARY

It is apparent that several psychologic and social variables are related to CHD. Coronary prone behavior pattern, in particular the hostility component, appears to be more closely related to the development and perhaps expression of CHD, whereas depression has been shown to be related to outcomes after CHD has declared itself. Lack of social ties appears to be related to mortality, whereas emotional social support has been shown to be related to recovery from coronary events. It also seems apparent that there are subsets of vulnerable individuals who might be best served by targeted interventions. For example, those with recurrent depression after a coronary event warrant special attention. How best to structure those interventions is the next piece of the puzzle.

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