1. Introduction

Every aspect of human health and disease bears the imprint of psychosocial processes. Although this is a rather strongly worded statement, it can, I believe, stand up to the hardest scrutiny at both empirical and conceptual levels. The facts leading to this conclusion have been stated by many. Psychosocial factors are related to exposure to harmful agents or environments, to the practice of risk-increasing or decreasing behaviors, to increased susceptibility to particular conditions, progression of disease, help-seeking behavior, access to medical care, adherence to and effect of medical treatments, recovery from serious conditions, and a wide range of other outcomes. Although the quality of the evidence varies
widely, the variety of methodologies and endpoints considered in the literature lends confidence to my first statement.

But, perhaps, this is painting with too broad a brush. Isn't it possible that our emphasis on psychosocial factors merely reflects the "unfinished business" of the biomedical sciences? From this perspective, advances in clinical and laboratory science will account progressively for the leftover variance we now attribute to psychosocial factors. There are a number of important conceptual, methodological, and philosophical issues to be raised in discussion of such a view. For the present, however, I would like to leave those aside and instead concentrate on issues raised by current psychosocial research on chronic illness with particular emphasis on coronary heart disease.

To convince ourselves of the importance of psychosocial factors, we would presumably want to be able to point to a coherent body of literature that underscores the importance of a relatively small number of psychosocial variables. This, however, is no easy task. With the expansion of research interest in psychosocial aspects of health has come an almost geometric increase in the number of factors which are thought to be important. To some extent, the task can be simplified by considering only a portion of the health trajectory.

Consider, for example, health behaviors. A recent attempt (Cummins et al., 1980) to integrate the various factors thought to be related to health behaviors brought together the judgments of nine theorists involved in research on the initiation, maintenance, and practice of these behaviors. Altogether, 109 variables were identified via an examination of the publications of these nine experts. (One blanches at the number which might have been generated by 19 instead of nine experts!) Using a process of multidimensional scaling of similarity judgments made by the nine judges, the authors were able to end up with six domains of variables: accessibility to health care, evaluation of health care, perception of symptoms and threat of disease, social network characteristics, knowledge about disease, and demographic characteristics. In a sense, this represents a kind of consensus conference, and similar efforts could be carried out with respect to etiologic and treatment issues. However, such a process does not clarify the underlying relationships so much as it presents us with the current level of agreement among experts.

When we turn to psychosocial influences of etiologic significance, we are faced with a similarly bewildering task. Again, some limits on the discussion make the task easier. Consider, as we shall for the remainder of this chapter, psychosocial influences on coronary heart disease morbidity and mortality. Jenkins (1971, 1976) reviewed much of the then current literature on "psychologic and social precursors of coronary disease." His 1971 and 1976 reviews covered a total of 250 articles. Were such an attempt to be made in 1983, the number of articles would be in the 500 to 1,000 range! Jenkins identified a number of factors that
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appeared to have some relationship to coronary disease etiology. These ranged from status incongruity to the Type A behavior pattern. To the list of factors considered by Jenkins in those reviews, we would now have to add decision latitude and job demands at work (Karasek et al., 1981, 1982), amount of self-referent speech (Scherwitz et al., 1983), perceived health (Kaplan and Camacho, 1983), hostility (Shekelle et al., 1983; Williams et al., 1980), acculturation (Marmot and Syme, 1976), social network participation (Berkman and Syme, 1979), and perceived control and predictability (Glass, 1977) to name only a few.

Thus we are left with an undifferentiated list of variables, all of which are associated with the development of coronary heart disease. In my view, progress in psychosocial epidemiology will depend to a great extent on our ability to convert long lists of variables to coherent theory or models. Such models of the impact of psychosocial variables on disease incidence, progression, and mortality, if properly constructed, will allow us to see the common themes, the interrelationships between variables, and the direct and indirect pathways of influence, and will provide some focus in our search for pathophysiological mechanisms. Without such unifying attempts, we will be likely to see an increasing accumulation of isolated and disconnected findings—a state of affairs which is not likely to lead to significant advances.

There have been some attempts at integration. Cassel’s (1976) observations in “The Contribution of the Social Environment to Host Resistance,” were one such attempt. He was able to show how findings from a number of studies could be interpreted as demonstrating the joint deleterious effect on health of poor social support and lack of feedback from the environment concerning one’s actions. Syme and his colleagues have similarly made important contributions of this sort. Recently, they (Satariano and Syme, 1981) have argued that a common feature of the Type A behavior pattern, stressful life events such as retirement and bereavement, acculturation, intra- and inter-generational mobility, and other variables of psychosocial importance is social isolation or the disruption of social ties. Similarly, we have recently presented evidence (Kaplan and Camacho, 1983) that perception of one’s level of health may focus the effect of a number of psychosocial variables that have associations with coronary heart disease mortality.

These attempts are important because they can lead to considerable simplification of a large body of evidence. Important as the attempts are, however, they leave many significant questions. For example, we need to understand the linkages between social connections or support and other psychosocial factors that influence health. We need to know if there are interactions of etiologic significance between variables that measure health practices, personality, stress and coping, perceived health, social connections and support, life satisfaction, positive and negative affect, and other variables of psychosocial significance. Such an examination and the resultant simplification would add to our understanding of causal pathways and lead to both intervention methods and further research.
Chapter 14  Kaplan

2. The Human Population Laboratory

We have been examining such questions in our work at the Human Population Laboratory, a unit of the State of California's Department of Health Services. The Human Population Laboratory was established in 1959, its purpose being threefold:

1. to assess the health, including physical, mental, and social dimensions, of persons living in Alameda County, California
2. to ascertain whether particular levels in one dimension of health tend to be associated with comparable levels in other dimensions
3. to determine relationships of various demographic characteristics and ways of living (including personal habits, familial, cultural, economic, and environmental factors) to levels of health.

One of the major efforts of the Human Population Laboratory (HPL) has been a now 18-year-old prospective study of a sample of Alameda County residents (Berkman and Breslow, 1988; Hochstim, 1970). Figure 1 shows the design of this study. It is a three-wave prospective study with measurement of self-reported physical health status, health habits or practices, psychological functioning, and social functioning during each wave. In addition, two mortality clearances have been executed to identify the deaths occurring between the first and second waves (1965-1974) and the second and third waves (1975-1983).

The participants selected in 1965 were chosen on the basis of a three-stage, stratified random sample of household units in Alameda County. This procedure identified approximately 8,800 eligible adults in 4,735 households. Eligibility was defined as non-institutionalized adults, 20 years of age or older (16 if married). Eighty-six percent of the eligible adults returned completed questionnaires, and these 6,928 individuals constitute the cohort that has been followed since 1965. Differences between respondents and non-respondents were examined via an examination of the results of a household enumeration which was done before placing the questionnaire. Non-respondents were generally older, white, male, and single. However, since the non-respondents were such a small portion of the total sample, and the differences were quite small, the respondents were judged to be an adequate representation of the non-institutionalized adults in Alameda County.

This cohort was intensively traced in 1974, vital status was determined for all 1965 respondents who could be found, and an attempt was made to interview all survivors. A computerized death clearance procedure coupled with the tracing efforts identified 717 deaths in the nine-year period. Of the remaining 6,211 respondents from 1965, 98 percent were successfully located in 1974, and completed questionnaires were obtained from 85 percent of those with whom they were placed.

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Through a similar procedure, we have recently ascertained the current vital status of the entire cohort. For the period through 1980, an additional 426 deaths have been discovered, bringing the total deaths for the 15-year period, 1965-1980, to 1,143. We are currently in the process of tracing and reinterviewing a 50 percent random sample of the survivors of the 1974 survey. Our current results indicate a response rate of nearly 90 percent for those with whom questionnaires were placed and approximately 5 percent loss to follow-up.
3. Factors Related to Ischemic Heart Disease Mortality

With this in mind, let me briefly review some findings which have come from the HPL studies of this cohort. I will present data for the 2,352 respondents 50 years or over in 1965, the first nine years of follow-up, and deaths from ischemic heart disease (410-414, International Classification of Diseases, 8th Revision).

*Health Practices.* Previous analyses (Belloc and Breslow, 1972; Belloc, 1973; Breslow and Enstrom, 1980; Wiley and Camacho, 1980; Wingard, Berkman, and Brand, 1982) of this data have shown that not smoking, moderate alcohol consumption, average levels of relative weight for height, moderate leisure-time physical activity, and seven to eight hours of sleep usually are significantly related to both current and future health status. Using univariate and multivariate techniques, it has been shown that people who practiced higher numbers of these health practices had lower levels of both morbidity and mortality than those who did not. Figure 2 demonstrates the strong association between a simple index of the number of health practices engaged in and mortality from ischemic heart disease over the nine-year follow-up period.

*Social Functioning.* Early HPL studies (Berkman, 1971; Renne, 1974) had shown the cross-sectional relationships between social well-being and health. It remained for Berkman and Syme (1979) to demonstrate that an aspect of social functioning—social network participation—was prospectively associated with mortality, even when controls were instituted for health practices, current physical

![Figure 2](image)

Figure 2. Association between Health Practices (0-2 vs. 3-5) and IHD Mortality/100, 1965-1974.
health status, and a variety of other confounders. This finding, which has had a major impact on social epidemiology, has now been replicated in the Tecumseh Study and others (Blazer, 1982; House, Robbins, and Metzner, 1982). Figure 3 shows that this relationship is strong and consistent for ischemic heart disease mortality.

Perceived Health. We have recently shown that an individual’s level of perceived health (i.e., whether he perceives his health as “excellent,” “good,” “fair,” or “poor”) is also associated with future health (Kaplan and Camacho, 1983). Individuals who perceived their health as “poor” in 1965 when compared with those who perceived their health as “excellent” were at approximately twice the risk of death between 1965 and 1974. This is true even when there were controls for 1965 physical health status, health practices, social network participation, depression, life satisfaction, and a number of other potential confounders. Figure 4 shows rates of ischemic heart disease death for different levels of perceived health. This finding has been replicated in other studies (Mossey and Shapiro, 1982; Salonen, personal communication).
Depression. A measure of depression based on the items shown in Table 1 shows a strong gradient of mortality. This measure, derived by Roberts (1981) from the questions available on the HPL questionnaire, has been validated against several other depression scales and gives good reliability and validity. As you can see in Figure 5, those who scored as depressed (one standard deviation or more above the mean) were at significantly increased risk of death from ischemic heart disease over the next nine years.

Life Satisfaction. A measure developed by Berkman (1977) and by Wingard (1980), measuring the extent to which respondents reported being satisfied with various domains of their lives, is also related to risk of death from ischemic heart disease. Individuals who reported low life satisfaction were at increased risk of death from ischemic heart disease (Figure 6).

Helplessness. Table 2 presents the items appearing on another scale. This scale was derived from a factor analysis of psychosocial items on the HPL questionnaire by Berkman (1977) and can be described in a variety of ways. It seems to measure a mixture of helplessness and personal uncertainty. In some ways, it might represent Antonovsky's (1979) concept of coherence. For reasons of simplicity, I'll refer to this as a measure of helplessness. As you can see in Figure 7, it, too, is quite strongly related to mortality from ischemic heart disease.

Socioeconomic Status. Finally, Figure 8 shows that a measure of socioeconomic status based on education (0-8 years versus more) and family income
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Table 1

Items in Depression Index (Roberts, 1981)

- Felt depressed or unhappy
- Appetite poor
- Lonely or remote from others
- Never felt on top of the world
- Too tired to do enjoyable things
- Little enjoyment from leisure time
- Less energy than others
- Never feel pleased about accomplishments
- Felt bored
- Restless
- Felt left out, even in a group
- Never felt excited or interested
- Hard to feel close to others
- Never satisfied with performance
- Cannot relax easily
- Bothered by getting tired in short time
- Felt vaguely uneasy

(inadequate or marginal versus more) is also associated with ischemic heart disease mortality.

4. The "Kitchen Sink" Model

At this point, we are in the customary state of having a list of interesting measures, all of which are associated with ischemic heart disease mortality. Before proceeding further, let me assure you that these associations persist as significant when multivariate analyses are performed with adjustment for age, sex, and 1965 physical health status. The usual strategy at this point is to put all the variables into a single multivariate analysis and examine the independent effect of each variable when there is adjustment for all others. Table 3 presents the results of such an analysis. Again, we are considering only 50+-year-olds and the 217 ischemic heart disease deaths that occurred during the nine-year follow-up. As you can see, health practices, social isolation, perceived health, and helplessness are all significantly associated with ischemic heart disease death. One is tempted to stop here; however, there are a number of conceptual and statistical problems with such an "everything but the kitchen sink" approach. In the real world, variables are
correlated, and this collinearity exacts a substantial cost in multivariate analyses.

Figure 9 shows the extent to which this is true. Practice of a low number of health practices is associated with "poor" perceived health, social isolation, presence of depression and helplessness, low life satisfaction, and low socioeconomic status. Similarly, those who report "poor" perceived health report higher rates of helplessness, depression, social isolation, life satisfaction, low health practices, and low socioeconomic status. Those who are classified as "helpless" report higher rates of low health practices, low life satisfaction, social isolation, "poor" perceived health, low socioeconomic status, and depression. That these variables are correlated should not come as a great surprise; after all, one would expect that various psychosocial measures on the same individual would be correlated, but such patterns of correlation do not signal our defeat. Although they do make the interpretation of cross-sectional data problematic, analyses of prospective data can still be quite valuable.
Figure 6. Association between Life Satisfaction (Low vs. Med./High) and IHD Mortality/100, 1965-1974.

5. Analyses of Direction and Extent of Confounding

When we are examining prospective data, we are able to look in some detail at the nature of the correlations between variables and the extent to which one variable confounds the association between another variable and the outcome of interest. Consider the following. A given risk variable (E) may be related to a disease outcome (D):

a) E → D

However, a confounder variable (C) may be related to both the disease outcome:

b) C → D
and to the risk variable

\[ E \rightarrow C \]

Thus we have the following situation

\[ E \rightarrow D \]

An examination of the extent and symmetry or asymmetry of confounding influences between \( E \) and \( C \) can tell us something about presumed causal relationships. For example, consider first the unadjusted association between \( E \) and \( D \). Now compare this to the association between \( E \) and \( D \) when adjusted for \( C \). At the same time, consider the change in the association between \( C \) and \( D \) when there is adjustment for \( E \). If the adjustment for \( C \) has little or no effect on the association between \( E \) and \( D \), while the adjustment for \( E \) has a great effect on the association between \( C \) and \( D \), then we can postulate the following model:

\[ E \rightarrow D \]

The interpretation of this model is that \( E \) has a direct association with \( D \) while \( C \) has an indirect association, its influence being felt only because of its association with \( E \).

We have used this logic to examine the associations with ischemic heart disease mortality just discussed. To do this, we performed a series of 28 multiple logistic analyses in which each of the seven variables is considered both by itself

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and in conjunction with each of the other six variables. All analyses include simultaneous adjustment for age, sex, and 1965 self-reported physical health status.

Inspection of Table 4 shows that there is considerable variability in the extent to which one variable confounds the association between another variable and mortality. In this table, we show the percentage reduction in approximate relative risk associated with adjustment for another variable. In this case, the baseline value is the relative risk associated with the particular variable of interest when there are controls for age, sex, and physical health status. For example, the approximate relative risk associated with “many” versus “few” health practices decreases 32 percent when there is adjustment for perceived health but only 9.4 percent when there is adjustment for life satisfaction. In some cases, there is considerable asymmetry in these confounding effects. For example, the association between social network functioning and ischemic heart disease mortality is reduced only 4.8 percent when there is adjustment for life satisfaction. On the other hand,

Table 2

Items in Helplessness Index (Berkman, 1977)

I am easily sidetracked from things I start to do.

I have a hard time making up my mind about things I should do.

I keep putting things off, and I don’t get as much done as others do.

Much of the time I’m not sure what I really want.

I have periods of days, weeks, or months when I can’t get going.

I often do things on the spur of the moment without stopping to think.

It seems to me that other people find it easier to decide what is right than I do.
Figure 7. Association between Helplessness and IHD Mortality/100, 1965-1974.

Figure 8. Association between Socioeconomic Status and IHD Mortality/100, 1965-1974.
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Figure 9. Interrelationships between Psychosocial Measures.

The association between life satisfaction and ischemic heart disease mortality is reduced 19.5 percent when there is adjustment for social network functioning. Inspection of these patterns of mutual influence allows us to partition the total association with mortality for a particular variable into components representing a direct association and components representing the influence of other variables—an indirect association.

In the next seven figures, I will demonstrate the results of this type of analysis for each variable we've seen to be related to ischemic heart disease mortality. Let me repeat that the intent is to examine the patterns of confounding between variables and their relationship to ischemic heart disease mortality and, through an examination of the directionality and extent of confounding, to arrive at a model of the way these variables are interrelated in their impact on ischemic heart disease mortality. After we've examined each variable individually, I'll then put all of them together in a way that, I hope, will result in some simplification.
Table 3
Relative Risk of Death from Ischemic Heart Disease, 1965-1974

<table>
<thead>
<tr>
<th>Variable</th>
<th>Relative Risk*</th>
<th>95% C.I.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Health Practices (0 vs. 5)</td>
<td>3.11</td>
<td>(2.68-3.61)</td>
</tr>
<tr>
<td>Social Network Index (Isolated vs. Most Connected)</td>
<td>2.86</td>
<td>(1.65-4.96)</td>
</tr>
<tr>
<td>Perceived Health (Poor vs. Excellent)</td>
<td>1.48</td>
<td>(1.14-1.93)</td>
</tr>
<tr>
<td>Helplessness (Present vs. Absent)</td>
<td>1.53</td>
<td>(1.08-2.18)</td>
</tr>
<tr>
<td>Life Satisfaction (Dissatisfied vs. Else)</td>
<td>1.07</td>
<td>(.64-1.80)</td>
</tr>
<tr>
<td>Depression (Present vs. Absent)</td>
<td>.95</td>
<td>(.67-1.35)</td>
</tr>
<tr>
<td>Socioeconomic Status (Low vs. Else)</td>
<td>1.03</td>
<td>(.83-1.28)</td>
</tr>
</tbody>
</table>

*Adjusted for age, sex, and 1965 physical health status and all variables above.

Table 4
Relative Risk of Ischemic Heart Disease Death with Adjustment for Age, Sex, and 1965 Physical Health Status

<table>
<thead>
<tr>
<th>Variable</th>
<th>RR</th>
<th>%ΔRR adjusted for:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HP</td>
<td>SNI</td>
</tr>
<tr>
<td>Health Practices (HP)</td>
<td>4.24</td>
<td>-19.8</td>
</tr>
<tr>
<td>Social Network Index (SNI)</td>
<td>3.55</td>
<td>-11.8</td>
</tr>
<tr>
<td>Life Satisfaction (LS)</td>
<td>1.49</td>
<td>-7.4</td>
</tr>
<tr>
<td>Depression (DEP)</td>
<td>1.49</td>
<td>-12.1</td>
</tr>
<tr>
<td>Helplessness (HLP)</td>
<td>1.77</td>
<td>-7.9</td>
</tr>
<tr>
<td>Socioeconomic Status (SES)</td>
<td>1.40</td>
<td>-9.3</td>
</tr>
</tbody>
</table>
In these analyses, we have represented a path as direct if it remains significant after adjustment for other variables. Indirect paths are indicated where the adjusted relative risk is reduced by approximately 20 percent or more when there is adjustment for a second variable or where the association of the first variable with the mortality outcome becomes non-significant with adjustment for the second variable. Also, the area of each circle is proportional to the relative risk associated with a particular variable when adjusted for age, sex, and physical health status.

With this in mind, consider first the pathways associated with health practices. Figure 10 shows that the impact of health practices occurs in three ways. One is a direct path not involving any other variables in the analysis. The other two pathways are indirect pathways involving perceived health and social network participation. That is, levels of perceived health and social network participation account for some of the association between the practice of certain discretionary behaviors and ischemic heart disease mortality. Another way of saying this is that people who practice low numbers of health practices are at increased risk, partially because they are also socially isolated and perceive their health as "poor."

Figure 11 shows the results for social network participation. No other variable has a significant effect on the relationship between social network participation and ischemic heart disease mortality. Thus there is only a direct effect.

The situation for perceived health is somewhat more complicated as seen in Figure 12. Here we see that perceived health has a direct pathway and three indirect paths, one through health practices, one through socioeconomic status, and one through helplessness. Thus the increased risk of ischemic heart disease death for people who perceive their health as "poor" rather than "excellent" is due to their level of perceived health as well as the fact that they tend to be lower on the health practices index, belong to lower socioeconomic strata, and feel "helpless." Recall for a moment that health practices also had a path through perceived health. This is the only example we will see of a symmetric confounding relationship between two variables. The fact that it is between perceived health and health practices raises a number of interesting questions which we will discuss later.

Figure 13 shows that the association between socioeconomic status and ischemic heart disease mortality is unaffected by any of the other variables considered, a finding which is in line with other reports (Rose and Marmot, 1981; Salonen, 1982).
A similar finding obtains, as shown in Figure 14, when we consider the helplessness variable. Note that no other variable has a significant effect on the association between helplessness and ischemic heart disease mortality. This is particularly striking when you consider that some of the other variables included in this analysis reflect presence of depression, low life satisfaction, and low health practices.
Different patterns obtain when we consider depression and life satisfaction. In both cases, the association with ischemic heart disease mortality is entirely due to other variables. Thus, in Figure 15, depression exerts its influence indirectly through its association with health practices, perceived health, social networks, socioeconomic status, and helplessness. That is, people who are depressed are at increased risk because they have higher rates of “poor” perceived health, low health practices, social isolation, low socioeconomic status, and helplessness.

Finally, Figure 16 shows the results for life satisfaction. Here again, the associations are all indirect, entirely reflecting the influence of health practices, social networks, perceived health, and helplessness.
Figure 14. Direct Associations between Helplessness and IHD Death.

Figure 15. Indirect Associations between Depression and IHD Death.

Figure 17 shows all of these relationships with ischemic heart disease mortality portrayed simultaneously. Let me first assure you that although this appears to be rather complex, there has been a substantial decrease in complexity from what could have resulted. This model shows five direct pathways with ischemic heart disease mortality, and each of these pathways bears some additional comment.
Socioeconomic Status. Measures of education and income have long been known to be associated with varying rates of coronary heart disease, although the particular directionality of the association is not necessarily consistent over time (Antonovsky, 1968; Morgenstern, 1980). One of the explanations offered to account for this inconsistency is the diffusion of various protective or high risk behaviors to different socioeconomic strata at various points in time (Cassel, Heyden, and Bartel, 1971; Inkeles and Smith, 1974; Morgenstern, 1980). Although a proper analysis of this topic would require a sequential cohort design with information on secular trends in the practice of these high risk behaviors and other variables, our analyses and those of others suggest that such an explanation will not be adequate to account for socioeconomic status-ischemic heart disease mortality associations and changes in these associations over time (Holme et al., 1980; Salonen, 1982). We may need to look at other factors, for example, work related ones. Low socioeconomic status individuals are often exposed to demanding jobs with little control, a situation which Karasek and his colleagues (1981) and others have shown to be associated with increased risk of coronary disease.
Individuals in lower socioeconomic strata also have differential exposure to various harmful substances. A recent publication (Mahaffey et al., 1982) using HANES II data collected by the National Center for Health Statistics demonstrated a substantial relationship between blood lead levels and family income, race, and degree of urbanization. Differential exposure to coronary disease relevant risks associated with different socioeconomic strata should be studied further.

Social Networks. We have demonstrated, as did Berkman and Syme (1979), that in this data set there is a strong relationship between social network participation and mortality. There are several findings here which add to the previous findings. Our results show that this relationship holds beyond the age Berkman and Syme examined (they looked only at individuals aged 30-69; our analyses covered 50-94). Furthermore, the association is strong for mortality from ischemic heart disease. In addition, and more importantly, social network participation is shown to be independent of other risk factors in multivariate analyses and actually accounts for part of the association between health practices,
life satisfaction, and depression and coronary mortality. There are still many unanswered questions about this relationship between social connections and mortality. Our analyses are based on mortality data only and therefore do not allow us to distinguish between factors associated with the development versus progression of coronary heart disease. There is, however, some information that bears upon this issue. Marital status, which is an important component of the measure of social network participation used in these analyses, has been shown in other studies to be related to a wide range of endpoints. Recently, Chandra and colleagues (1982) demonstrated that marital status was related to both hospital case fatality rates and long-term survival in a population of 1,401 myocardial infarction patients, even when there were controls for severity of disease. It seems likely that social network participation is protective both because of internal psychophysiological deregulation present in the hospital environment in those who are isolated, leading to increased hospital case fatality rates and also because of external pathways reflecting the availability of coping resources once released from the hospital, with poorer resources leading to poorer outcome post-discharge.

Some data concerning the role of social connections in the incidence of myocardial infarction are available. An analysis of Finnish hospital discharge records (Koskenuo et al., 1981) demonstrated lower incidence of coronary disease for the married. In preliminary analyses in collaboration with Syme and Salonen of data collection in North Karelia, Finland, on over 10,000 people followed for seven years, we are seeing indications of an association between measures of social connection and the incidence of myocardial infarction. We hope our analyses will clarify some of the issues concerning the role of psychosocial factors in incidence and prevalence of coronary heart disease which have recently been raised (Reed et al., 1983).

Health Practices. The role of health practices continues to be strong in relation to ischemic heart disease mortality. The pathways associated with social network participation and perceived health are particularly interesting. Individuals who practice fewer health practices also tend to be more socially isolated, and this social isolation accounts for some of the association between low health practices and increased risk of death. There is some evidence of a synergistic relationship between the two variables, but further work needs to be done to clarify its nature.

The indirect pathway from health practices through perceived health is an interesting one to speculate about. The bi-directionality of this pathway is particularly interesting. It implies that there is a dynamic relationship between the two variables in their association with heart disease mortality. This may indicate substitutability between the two variables, or it may simply mean that our examination at one point in time of these two variables is masking some temporal process. Perhaps, people who perceive their health as excellent or good over time tend to increase their practice of measures which are protective and decrease their practice of those which are deleterious to their health. Or, perhaps, high levels of
perceived health are a response to increases in health practices. We will be examining these issues in more detail in future analyses of our three-wave data.

Perceived Health. The association between level of perceived health and subsequent mortality is intriguing. This result has now been replicated in several other studies, and I am convinced that it does not simply reflect poor measurement of physical health status. In our analyses, the relationship is strong and consistent across all levels of self-reported physical health status. Analyses by Mossey and Shapiro (1982) of the mortality experience of an over-65-year-old cohort followed for five years arrived at similar conclusions. They were able to control for physical health status using physical exams and medical and hospitalisation utilization data. In analyses of the mortality experience of the North Karelia cohort (Salonen, personal communication), perceived level of health appears to make a strong and independent contribution to ischemic heart disease mortality, even when there are controls for family history, serum cholesterol level, mean arterial pressure, and smoking. Thus the finding seems to be a robust one.

In other unpublished analyses, we have found that many other variables related to coronary heart disease rates are also highly associated with lower levels of perceived health. Upon closer examination, they seem to exert their influence indirectly via perceived health. In this category are measures of job strain, family disruption, negative childhood experiences, and geographical mobility. Perhaps then, perceived health is a variable that focuses the effects of other psychosocial variables over time. We will return to this idea in a moment.

Also of interest are the indirect pathways mediating the impact of perceived health--socioeconomic status and helplessness. It is important to point out that although part of the association between perceived health and ischemic heart disease mortality is an indirect one involving helplessness, when there is control for that measure, the association between perceived health and mortality is strong. Cohort members who perceived their health as "poor" rather than "excellent" are at 1.91 the risk of death in the follow-up period. Similarly, when there is control for socioeconomic status, the increased risk is 1.95. Thus, the association between perceived health and mortality does not appear to be due to factors such as a response set associated with poorer education and income or feelings of helplessness.

Helplessness. Finally, we come to the last variable that has a direct association with ischemic heart disease mortality—that which I've called helplessness. The association between this variable and ischemic heart disease mortality is striking both in its magnitude and in its consistency. Those who are scored as "helpless" have a relative risk of ischemic heart disease death 1.80 that of those who aren't helpless, even when there are controls for age, sex, and physical health status. No other variable is so little influenced by other confounders; the
maximum reduction in relative risk being 7.9 percent with adjustment for social network participation or health practices.

Now, I have to admit that I am not really sure what to call this measure. Berkman (1977) called it "personal uncertainty," but to me there are a variety of other constructs involved. We will have to do some careful work examining this variable in relationship to other psychological measures, but, in the meantime, I think it is important to point out the extent to which it appears to involve other constructs which have figured in the literature. There certainly is the element of helplessness, but there are also strong elements of what Antonovsky (1979) calls coherence and what Kobasa (1982) calls hardiness. In addition, there is a sense of things being out of control and unpredictable (Glass, 1977). We intend to explore the meaning of this variable in much more depth.

Life Satisfaction and Depression. When other controls are instituted, life satisfaction and depression are only indirectly associated with ischemic heart disease mortality. What is particularly interesting is the large number of indirect pathways accounting for the association between these variables and ischemic heart disease mortality. Depression is associated with higher rates via its link with health practices, social network functioning, perceived health, socioeconomic status, and helplessness. Another way of summarizing this is to say that depressed people are at increased risk in a number of ways, and it is these patterns of higher risk levels on other variables which account for their overall increased risk. Of course, we cannot tell from these analyses what the causal relationships are between these variables. For example, does social isolation cause depression, or vice versa, or are they causally unrelated?

Of particular note is the connection with helplessness. This latter variable is one of the pathways by which depression is associated with higher rates of death, but the reverse is not true. That is, depression does not mediate the association between helplessness and ischemic heart disease mortality. This asymmetry lends some confidence to our feelings that we are dealing with two substantively different scales. The helplessness pathway which partially accounts for the effect of depression on ischemic heart disease death is particularly provocative given current thinking about depression (Garber and Seligman, 1980).

Finally, there is life satisfaction. Again, of particular note is that the association between this measure and ischemic heart disease mortality is via indirect pathways involving health practices, perceived health, helplessness, and social network participation. Of note is that social network participation appears to have by far the greatest confounding effect on life satisfaction. It may be that those who are dissatisfied with their functioning in social domains are more isolated, and it is this isolation that primarily increases their risk of death.
6. Discussion and Conclusions

The analyses just presented demonstrate again the significance of psychosocial factors in health. Using data collected on a large community sample which has been followed for nine years, it has been possible to demonstrate that wide range of psychosocial factors is associated with mortality from ischemic heart disease. Furthermore, the analysis of the patterns of confounding between variables has allowed us to gain some insight into the pathways by which such measures are associated with increased mortality risk.

In the introduction to this chapter, we pointed out the need for synthesis in psychosocial epidemiology and proposed an attempt in that direction. It behooves us then to step back and assess to what extent this has been successful. We have shown how it is possible to represent the indirect and direct pathways by which particular measures are associated with risk of ischemic heart disease death, and this has resulted in some simplification; however, the overall picture is still complex. Perhaps, this simply reflects the overall complexity of the subject matter, and we shouldn't expect any great deal of synthesis.

However, certain patterns emerge in Figure 17. The interpretation of some of these is clearer if we return first to Cassel's (1976) seminal paper. He suggested that lack of feedback to the individual concerning consequences of her actions might be associated with deleterious health outcomes. For our purposes, the critical insight has to do not so much with the notion of control over consequences (Antonovsky, 1979; Bandura, 1977; Langer, 1975) as much as it has to do with the notion of feedback. Feedback is, by definition, the flow of information back to the individual from the environment following some action, and it is this element, with its bi-directional and recursive character, which may help us to understand Figure 17. In this view, individuals act both "on" and "in" their environment, and their actions lead to changes in the environment, which affect them as "in" the environment! What is perhaps unique to psychosocial epidemiology is the recognition that these transactional processes with their dense interdependence make the distinction between host and environment problematic.

With this in mind, let us return to Figure 17. Note that health practices and social network participation both involve day-to-day processes in which the individual acts on and gets feedback from the environment, which may or may not lead to changes in the individual and/or environment. In the former case, activities like smoking or jogging lead to the perception of symptoms or bodily states that tell individuals something about their physical condition. In the extreme case, changes in physical activity and the subsequent changes in feeling states have been reported to be one of the ways in which early myocardial infarction patients assess the meaning of anginal pain or "indigestion" (Cowie, 1976; Kaplan, 1981). Similarly, the absence of negative symptoms in smokers and the apparent message that "all is well" may contribute to their difficulty in stopping smoking. Thus discretionary behaviors may be one of the ways in which
individuals get information about their underlying physical state, which is used by them to maintain or modify their actions or environment. In this respect, the bidirectional pathway between perceived health, which we will argue later is a measure of underlying physical health status, and health practices is particularly important.

Social network participation also involves day-to-day activities which provide one with ongoing feedback regarding the structure, meaning, and value of one's social relationships. The data presented here and elsewhere (Berkman and Syme, 1979) as well as that concerning bereavement and other major social losses (Cottingham et al., 1980; Helsing and Szklo, 1981) surely point to the importance of maintenance of these day-to-day social connections. Furthermore, the fact that measures of depression and helplessness do not account at all for the association between social isolation and increased risk suggest that there is something important about the absence of ongoing social feedback which is important far beyond any affective dysfunction. Also of interest is the finding that some of the increased risk associated with a lower health practices score is related to higher rates of social isolation. It may be that those who are unable to use feedback in the social domain to regulate and maintain their social ties are also those who suppress or deny symptoms associated with heavy drinking, smoking, and/or inactivity. Whether this reflects state, trait, or contextual factors such as occupation and stress remains to be seen.

The situation is different when we now turn to helplessness, life satisfaction, and depression. Depression and life satisfaction do seem to carry with them increased risk of ischemic heart disease death but only because of their association with virtually all other measures we have considered. On the other hand, helplessness retains a strong and independent association. It seems reasonable to speculate that whereas health practices and social network participation reflect day-to-day influences, helplessness is more proactively oriented. Individuals who see the world as incoherent and unpredictable and feel helpless and uncertain are likely to appraise new situations as stressful and threatening and also to see themselves as unable to cope. Thus, in this view, the association between this measure and increased risk of ischemic heart disease death may reflect the absence of coping resources with which to meet new environmental or personal demands. The fact that this association is independent of social network participation suggests that it reflects the relative lack of internal resources as opposed to resources associated with other people. Again, it is intriguing to note the association between perceived health and helplessness. Apparently, people who perceive their health as "poor" also feel helpless, and these feelings of helplessness account for some of the increased risk. This finding is consistent with the interpretation of helplessness as an absence of internal coping resources. Individuals who perceive their health as "poor" and who feel helpless are less likely to be able to engage in activities that might improve their health.
Understanding the role of socioeconomic factors in coronary disease continues to be problematic. As pointed out earlier, we agree with other investigators that the increased risk associated with being relatively poor and uneducated is not explainable by any other factors we have measured. Again, some focus on the time dimension may be useful. In our cohort of over-50-year-olds, those who reported being poor and uneducated in 1965 presumably shared certain common features with respect to their past and future. Those who had less than eight years of education had more than 30 years of exposure to the consequences of this, and those who reported "inadequate" or "marginal" income are likely to have also suffered economic privation for some time. Those who reported both, the low socioeconomic strata in our analyses, have thus had considerable exposure to a wide variety of undesirable factors and, what's more, presumably could look forward to similar difficulties in the future. In this view, membership in a low socioeconomic strata exerts a profound influence on past, present, and future. It thus represents a temporal context covering a large part of the individual's history. This type of "contextual" view and analyses of the life experiences, behaviors, and responses following from it will be necessary before we can really understand the role of socioeconomic factors in health. Of interest is that both depression and perceived health were associated with increased risk partially because those who were depressed or perceived their health as "poor" were more highly represented in the low socioeconomic strata.

Finally, we come to perceived health and its pathways. To be sure, judgments of perceived health reflect something about the physical condition of the individual. Individuals who report the presence of chronic conditions or symptoms also report lower levels of perceived health, with the correlation averaging around .40 (Kaplan and Camacho, 1983). However, we have found that measures of negative life events, psychological dysfunction, unhappy childhood experiences, and work stress are also associated with lower levels of perceived health, and it is these lower levels of perceived health which appear to account for the poorer health experience of individuals who report such problems. These findings and others have led us to speculate that levels of perceived health reflect an underlying level of physiologic functioning which is the result of various physical, social, and psychological insults to the individual. In this view, the level of perceived health an individual reports may represent a barometer of host resistance or susceptibility. Recent work in psychoneuroendocrinology (Henry, 1982) and psychoneuroimmunology (Ader, 1981) has pointed to mechanisms which would allow experience at a variety of levels to be focused in such a common way. Our analyses have shown how the association between perceived health and ischemic heart disease mortality includes indirect pathways involving health practices, helplessness, and socioeconomic status. The possibility that an individual's report of perceived health might index a level of physiologic functioning integrating these three levels of functioning seems to us to be quite exciting.
We have tried to show how analyses of the patterns of association between psychosocial measures and ischemic heart disease mortality can help us to understand the pathways by which these factors are associated with increased risk. In the process of these analyses, a number of methodological issues have arisen which bear some comment. Our analyses have been restricted to mortality outcomes, and although there is some evidence that psychosocial factors also exert an influence on the incidence of ischemic heart disease, much more work needs to be done utilizing a variety of health outcomes. It would be valuable to be able to examine in the same data set outcomes reflecting incidence, prevalence, and mortality. Analyses of this sort would go far toward clarifying the biological and social pathways leading to the associations which we have presented.

We also need to explore more fully procedures for examining multiple, correlated measures. In traditional, biomedically oriented, epidemiologic research, we are usually primarily interested in a single or small number of exposure factors, and other measures are considered only insofar as they confound or modify the association between those factors and some health outcome. However, in psychosocial epidemiology, and perhaps in other areas of epidemiology as well, it is becoming clear that there is something about the structure of the relationships between variables which is worthy of interest. We have presented one technique for examining these structural relationships and their association with ischemic heart disease mortality in the present chapter, but much more work needs to be done in this area.

But by far the biggest problem lies within the design of one-wave prospective studies. When we examine a cohort and measure a number of variables at one point in time and then assess the health experience of that cohort at some later point, we have progressed far beyond what would be available in a cross-sectional study. However, we have not gone far enough. Consistently, in the discussion of the patterns of confounding between variables, we have asked questions about the temporal relationships between these variables. For example, do people who practice low numbers of health practices have higher rates of "poor" perceived health because of their low levels of health practices, or vice versa? Do changes in depression or physical health status lead to changes in social network functioning, or vice versa? We could go on and on generating numerous hypotheses about the causal orderings of psychosocial measures and their relationship with health outcomes. Thoits (1982) has pointed to similar problems in the analyses of the "buffering" effect of social support on the consequences of negative life events. What is needed are cohort studies in which the cohort members are assessed at numerous points in time. Such studies would allow us to examine the trajectories of change and influence between psychosocial variables and how these trajectories and patterns of influence are associated with changes in health. Future work at the Human Population Laboratory will utilize our three-wave data covering 18 years in just such a way. We hope our results and others' using this approach will help us to unravel the web of psychosocial causation.
Chapter 14   Kaplan

References


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