Annu. Rev. Psychol. 1997. 48:411–47

HEALTH PSYCHOLOGY: What is an Unhealthy Environment and How Does It Get Under the Skin?

Shelley E. Taylor and Rena L. Repetti

Department of Psychology, 1283 Franz Hall, University of California, Los Angeles, Los Angeles, California 90095-1563; e-mail: taylor@psych.sscnet.ucla.edu

Teresa Seeman

Andrus Gerontology Center, University of Southern California, Los Angeles, California 90089-0191

KEY WORDS: family, social support, SES, community, work

ABSTRACT

This review explores the role of environments in creating chronic and acute health disorders. A general framework for studying the nesting of social environments and the multiple pathways by which environmental factors may adversely affect health is offered. Treating socioeconomic status (SES) and race as contextual factors, we examine characteristics of the environments of community, work, family, and peer interaction for predictors of positive and adverse health outcomes across the lifespan. We consider chronic stress/allostatic load, mental distress, coping skills and resources, and health habits and behaviors as classes of mechanisms that address how unhealthy environments get "under the skin," to create health disorders. Across multiple environments, unhealthy environments are those that threaten safety, that undermine the creation of social ties, and that are conflictual, abusive, or violent. A healthy environment, in contrast, provides safety, opportunities for social integration, and the ability to predict and/or control aspects of that environment.

0084-6570/97/0201-0411$08.00

CONTENTS

HEALTH PSYCHOLOGY: WHAT IS AN UNHEALTHY ENVIRONMENT AND HOW DOES IT GET UNDER THE SKIN?

The role of the environment in health and illness has been known since the time of Hippocrates. With the discovery that infectious agents produce disease, physicians and public health researchers directed their attention to the environmental conditions that give rise to these agents and permit them to breed. Following breakthroughs in water treatment, sewage control, food storage, and waste disposal, the incidence of many infectious diseases declined substantially, soon to be replaced by the slower-developing chronic illnesses of heart disease, cancer, and diabetes, among others. These diseases have come to be known as diseases of lifestyle, because behavioral risk factors are clearly involved in their etiology and progression. An unintended consequence of the

focus on lifestyle has been to divert attention away from the role of the environment in producing disease in favor of an emphasis on behavior. As health psychologists have increasingly identified what risk factors people incur and how they incur them, the focus of health prevention has moved from environmental interventions to individual behavior. Some scientists have argued that this focus has led to a culture of blame, whereby individuals are held responsible for good health and blamed or discredited for their illnesses (Becker 1993).

The role of the environment in producing chronic as well as acute disease merits renewed attention in the context of the current concerns addressed by health psychology. Specifically, as research has identified the individual difference predictors of chronic illness, including health behaviors, use of health services, social factors such as social support, and psychological factors such as hostility and depression, it has become clear that these predictors are nested within geographic, developmental, occupational, and social environments. In this review, we focus attention on these environments and the ways environmental characteristics may influence health and also influence individual characteristics or behaviors that pose risks for health.

In so doing, our analysis gives primary status neither to environmental characteristics nor to their concomitant effects on individuals. Not all individuals in the same environment are affected by that environment in the same way, nor will all individuals in a given environment sustain health risks. Thus, we explicitly reject the notion that the health effects of environments can be reduced to or explained by individual-level factors. Rather, we maintain that individual characteristics are nested within social environments (see Figure 1), with each level of analysis revealing information about the causes of health and illness that consideration of one level alone cannot provide.

As we note below, social class and race provide a context for understanding the effects of environment. These characteristics are well-established predictors of all-cause mortality and a variety of specific diseases, and they are also reliably associated with individual differences in exposure to stress, the practice of health behaviors, coping strategies, and other factors of interest to psychologists (Adler & Matthews 1994, Williams & Collins 1995). With SES and race as background, we then examine community, family, work, and peer groups as specific environments that have a contributing role in health and illness (see Figure 1). Within each environment, we ask the question, “How do the health-relevant characteristics of this environment get under the skin?” For example, some children live in conflict-ridden families and others do not, and the health risks sustained differ between the two groups. We ask how that environmental characteristic may translate into risks that have the identified health consequences. In so doing, we consider several general pathways in

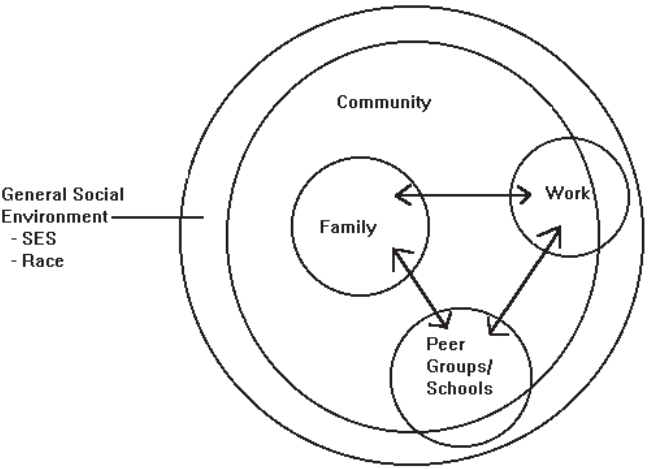

Figure 1 Diagrammatic representation of social environments that have health-relevant implications. This representation assumes that the proximal environments of family, work, and peer groups are nested or partially nested within neighborhood and fully nested within a larger social environment in which such factors as socioeconomic status (SES) and race have health implications.

each of the environments. These general pathways, as well as more complex combinations of them, are illustrated in Figure 2.

Environments exert direct effects on health (Figure 2, Segment a) that may be largely unmediated or unmoderated by psychological and social processes, except insofar as they lay the initial groundwork for their occurrence. For example, the poor and African-Americans are disproportionately likely to contract certain kinds of cancers because of differential exposure to toxins at work or in their neighborhoods. At present, research has not progressed to the point of identifying whether there are social or psychological factors that contribute to these adverse effects.

Because this is a psychological review, the routes on which we focus most of our attention are psychosocial pathways for the development of health risks. A first route whereby environments may get under the skin is by differentially exposing people to chronic stress (Segments fk). That chronic stress may have a cumulative effect on the body was first observed by Hans Selye (1956) in his articulation of the General Adaptation Syndrome. Selye maintained that individuals respond to stressful events with nonspecific reactions that, over time, produce wear and tear on the system. Repeated cycling through the three-phase syndrome of alarm, resistance, and exhaustion, Selye argued, leads to cumula-

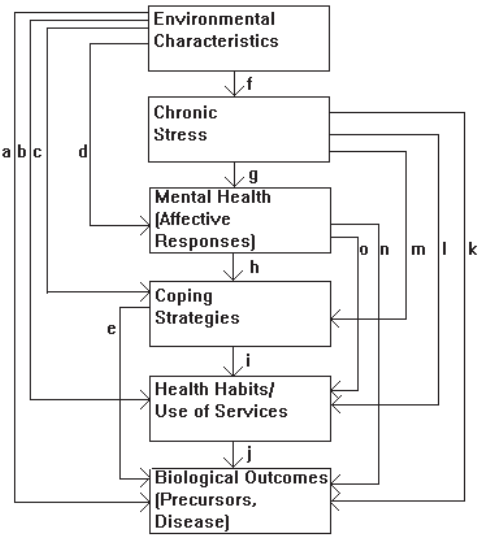

Figure 2 A range of models by which environmental characteristics may come to exert adverse biological outcomes on individuals. Segments b–o represent partial pathways that may concatenate to form more complex pathways.

tive damage to the organism. Building on these ideas, McEwen & Stellar (1993) proposed that, beginning early in life, there are cascading relationships between environmental factors and genetic predispositions that lead to large individual differences in susceptibility to stress and, in some cases, to disease. Physiological systems within the body fluctuate to meet demands from external forces, a state termed allostasis. Over time, the allostatic load builds up, which is defined as the physiological costs of chronic exposure to fluctuating or heightened neural or neuroendocrine responses that result from repeated or chronic environmental challenges that an individual reacts to as stressful. For example, the hypothesized links between recurrent changes in autonomic reactivity produced by stress and the subsequent development of chronic hypertension (Anderson et al 1991) may be thought of as an allostatic load model. The allostatic load formulation, then, explicitly argues that stress produces cumulative identifiable damage that results in increased pathology (Seeman et al 1996).

A second route by which environments may have adverse health effects is via an impact on mental health or mental distress (Melamed 1995) (Segments dn). Negative emotions, such as depression, anxiety, and hostility, appear to

play a significant role in health risks, including all-cause mortality (Martin et al 1995) and especially coronary heart disease risk (Booth-Kewley & Friedman 1987). These relations are apparently not due to behavior changes associated with affective disorders, such as increased smoking or alcohol consumption. Major depression, depressive symptoms, history of depression, and anxiety have all been related to the likelihood of cardiac events (Frasure-Smith et al 1995), and depression is a risk factor for mortality following myocardial infarction, independent of cardiac disease severity (Frasure-Smith et al 1995). State depression and clinical depression have both been related to sustained suppressed immunity (Herbert & Cohen 1993). Anger appears to be significant in the development of coronary artery disease and hypertension, at least among some individuals (Frasure-Smith et al 1995). These health effects of negative emotions may result from the activation of both the sympathetic-adrenal-medullary (SAM) system and the hypothalamic-pituitary-adrenocortical (HPA) axis. The former is manifested in increased blood pressure, heart rate, circulating levels of epinephrine and norepinephrine, and constriction of peripheral blood vessels. SAM activation is believed to contribute to the development of coronary artery disease (Manuck et al 1995), essential hypertension (Krantz & Manuck 1984), and susceptibility to infectious disease (Cohen & Herbert 1996). The activation of HPA leads to high circulating levels of corticotrophin-releasing hormone, adrenocorticotropic hormone, and cortisol. HPA activation has been linked to atherosclerosis (Troxler et al 1977) and chronic inflammatory responses, as are found in rheumatoid arthritis and reactivity of the airways in people with asthma (McNeil 1987). Although biologically based predispositions appear to play some role in the relation of affective diseases to physical state changes, environmental factors are also reliably related to sustained depression, anxiety, and anger. As such, mental health/distress constitutes a second important route by which environments may get under the skin.

Coping strategies constitute a third explanatory route that may clarify how unhealthy environments adversely affect health (Segments ce). Individuals who find constructive ways of coping with stress, such as taking direct action or finding meaning in their experience, may be better able to withstand the potential adverse effects of stressful circumstances. In addition, there may be stressor-specific coping styles, such as expressing hostility or suppressing anger, that have health implications both generally and for specific disorders, such as cardiovascular disease and hypertension. In addition, environments influence the development of coping strategies, especially those involved in managing conflict and stress, and the ability to develop social ties.

Health habits are heavily implicated in the development of illness, especially chronic illness, as numerous reviews have documented (e.g. Adler &

Matthews 1994) (Segments bj). They include smoking, alcohol and drug abuse, diet, exercise, the use of preventive and secondary health services, and adherence to treatment recommendations. Environments constitute the contexts in which health habits are learned, encouraged, and practiced. The family is an important context for the acquisition of health habits, and it lays the groundwork for a broad array of healthy and unhealthy behaviors (Taylor 1995). The peer group, first in adolescence and then in adulthood, is an important context within which many health-compromising behaviors are acquired and enacted, including smoking, alcohol, and drug abuse.

These, then, are the routes we consider in each of the environments analyzed. These routes are not independent or even discrete pathways by which unhealthy environments affect health; all represent routes in potential causal chains involving two or more of these processes. Thus, for example, a chronically stressful family environment may prevent the development of effective coping strategies (Segments fm), compromise the learning of health habits (Segments fl), and produce chronic anxiety and/or depression (Segments fg), all of which feed into enhanced health risks (Segments e, j, and n). In most areas, research has not progressed to the point where these complex pathways can be identified or tested in the context of particular health problems or disorders. Thus, Figure 2 is offered largely as a representation of potential routes, rather than established routes, by which pernicious environments exact adverse health effects.

SOCIOECONOMIC STATUS AND RACE

We begin our consideration of the question "What is an unhealthy environment?" by examining SES and race differences in health. Because SES and race heavily determine the more proximal environments in which people live, such as neighborhood and work, they provide important contexts for understanding the features of these more proximal environments that may compromise health (Williams & Collins 1995).

SES is traditionally measured by education, income, and occupation. Using these criteria, an extensive, highly consistent literature documents the negative health outcomes that result as one moves lower on the SES gradient (for reviews, see Adler et al 1993, Williams & Collins 1995). Analyses of potential measurement artifacts or natural or social selection suggest that these inequalities are real and affected little by selection factors (see Macintyre 1997). SES is related to higher prevalence and incidence of most chronic and infectious disorders and to higher rates of nearly all major causes of morbidity and mortality across populations and across time (Adler et al 1993, Macintyre 1997, Williams & Collins 1995). Moreover, SES differentials in morbidity and

mortality appear to be widening rather than narrowing (e.g. Marmot 1994, Wagener & Schatzkin 1994).

The association of SES with morbidity and mortality is, for the most part, linear, with increasingly better health outcomes as one ascends the SES continuum. As such, the relation of SES to health and mortality involves more than the obvious role of inadequate financial resources or poor and dangerous living conditions associated with poverty. While SES differentials may be found among children (Durkin et al 1994) and among the elderly (e.g. Seeman et al 1994), the largest social inequalities in health and mortality are seen most frequently for those aged 40 to 65.

Substantial race differences also exist in health. On virtually every major index of health status, African-Americans look worse than whites, and these differences in health occur across the lifespan (Williams & Collins 1995). With the exception of race-specific disorders such as sickle-cell anemia, these black-white differences are apparently not due primarily to genetic or biological factors that differentiate blacks from whites (for discussion, see Anderson et al 1991, Williams & Collins 1995). Socioeconomic differences account substantially for these health status differences, inasmuch as approximately one third of the African-American population lives in poverty, compared with 11% of the white population. Nonetheless, poverty does not fully account for black-white differences in health (e.g. Rushing et al 1992). Within every level of SES, African-Americans typically have worse health than whites (Williams & Collins 1995).

Although SES and race are not themselves environments, they provide an important and often overlooked context for understanding the more immediate environments within which people live, namely communities, work, family, and social life. As such, they provide the environmental backdrop against which more specific environmental encounters occur.

COMMUNITY

Certain characteristics of the communities in which people live have been shown to have adverse effects on health. Many of these arise because of the social class and racial composition of those communities. Consequently, they act as the proximal manifestation of these characteristics. For example, low-SES neighborhoods have higher rates of cancer, hypertension, heart disease, and upper-respiratory disorders, including asthma, bronchitis, and emphysema (Adler et al 1993). Other health-enhancing or health-compromising characteristics of environment are less dependent upon community social class or racial composition.

Chronic Stress

The degree of chronic stress experienced by individuals is heavily influenced by the characteristics of their communities. Residents of middle- and upper-income communities typically have access to high-quality housing, an abundance of shops, banks, health-care services and transportation. In poor neighborhoods, these resources are less likely to be available (Troutt 1993). Consequently, the lower one is on the SES continuum, the greater the amount of hassle and time needed to address basic tasks of living. Further contributing to the chronic stress of lower SES communities are characteristics such as police-documented higher crime rates (Macintyre et al 1993), greater perceived threat of crime and more local problems (Sooman & Macintyre 1995), more refused services (e.g. taxi, credit, ambulance; Sooman & Macintyre 1995), and poorer transportation and recreational facilities (Macintyre et al 1993). Lower SES neighborhoods also have been linked to greater exposure to physical hazards such as air and water pollutants, hazardous wastes, pesticides, and industrial chemicals (Calnan & Johnson 1985) and to greater crowding and exposure to noise (Evans 1997).

Lack of available housing typically leads to overcrowding, both in the form of high density within the neighborhood and crowding within the home (defined as 1.5 persons or more per room). High density is associated with higher all-cause mortality (Levy & Herzog 1974) and with higher rates of mortality due to cancer and stroke, but not heart disease (Levy & Herzog 1978). High-density living is also associated with death due to homicide, but negatively associated with death due to suicide, which appears to be tied to isolation and loss of family ties (Levy & Herzog 1978). A potential mediating pathway for these effects is suggested by the fact that high-density living is associated with both higher reports of chronic stress and with biochemical indices of stress, such as urinary excretion of norepinephrine and epinephrine (Fleming et al 1987). Crowding in the home has been related to increased likelihood of infections and to higher death rates from heart disease, to respiratory disorders, and to all-cause mortality (Levy & Herzog 1978). In addition to its association with crowding, substandard housing may affect health in other ways. Older buildings with poorly lit hallways and debris pose risks to safety and to health by attracting rodents and insects that may spread infection, for example.

Community studies of the effects of noise on physical health have identified few clear-cut relations to disease morbidity in the adult population. Acute noise produces short-lived elevations in cardiovascular and neuroendocrine functioning, but many adults appear to habituate to chronic noise. However, some studies suggest a relation of chronic noise to the development of hypertension, links that necessitate further investigation (Evans 1997). Recent evidence suggesting a relation of noise exposure to abnormal fetal development

also merits continued consideration (Evans 1997). As noted below, noise may adversely affect health habits as well.

High rates of crime and delinquency are associated with a variety of adverse health characteristics, including a high rate of infant mortality, low birth rate, tuberculosis, and child abuse (Sampson 1992). Women living in high-violence neighborhoods are significantly more likely to experience pregnancy complications than women living in neighborhoods with little violence (Zapata et al 1992).

Because a number of the adverse characteristics of neighborhoods are intercorrelated, investigators have attempted to operationalize the concept of high-stress neighborhood. Harburg et al (1973) defined areas characterized by low socioeconomic status, high population density, high geographic mobility, high rates of marital breakup, and high crime as high stress; low-stress areas had more favorable ratings on all these variables. Harburg found higher rates of hypertension in the high-stress than low-stress locales, especially among dark-skinned black men. Others (e.g. Troutt 1993) have argued that in such neighborhoods there are low levels of economic opportunity, poor marriage pools, and poor transportation, which erodes the ability of residents, especially single mothers, to seek employment in the neighborhood, to marry, or to move to a better neighborhood. As noted below, such characteristics may have adverse effects on parenting, which affects the health of children.

Mental Health/Distress

Little research has addressed the impact of community characteristics on mental health, and still less has tied such effects to physical health outcomes. To date, the only community characteristic to receive systematic study is exposure to violence, especially for children. Osofsky (1995) argued that exposure to chronic community violence directly and adversely affects children's mental health. Children's reports of psychological distress are significantly related to their reports of witnessing acts of violence (Richters & Martinez 1993). Children who live in violent neighborhoods show signs of posttraumatic stress disorder, including disrupted patterns of eating and sleeping, difficulties in controlling attention and relating to others, anxiety responses and fear, and reexperiences of the violent episodes they have witnessed (for a review, see Osofsky 1995). Among adults, sleep disturbances, nightmares, and manifestations of anxiety are common (Pynoos 1993). Exposure to violence may also adversely affect the mental health of children and adults because of the need to deal with losses and to cope with grieving for family members, neighbors, and friends who have been killed. Such grieving may compromise immune functioning (Kiecolt-Glaser et al 1994).

Coping Skills and Resources

Community characteristics may affect coping skills and resources in several ways that have health implications. Repeated arousal of intense negative emotions due to exposure to chronic violence, for example, may lead to difficulty in the effective regulation of emotions among children (Osofsky 1993). The adverse effects of chronic exposure to violence may be partially offset if an individual has at least one supportive person in the neighborhood, a protective place to go, or certain personal resources such as an adaptable temperament or a high level of intelligence (Osofsky 1993). Low-SES neighborhoods may also compromise the development of coping skills relating to the recruitment and effective use of social contacts. Parents who are concerned about exposing their children to drugs and crime may keep their children inside and otherwise restrict social behavior. Thus, the ability to develop or utilize a supportive network may be somewhat compromised (Sampson 1992). Whether these disruptions in the formation of social ties have negative effects on health in childhood or cumulative effects on adult health is unknown.

The inherently social nature of communities affects coping and health at both the community level and the individual level. In particular, characteristics of communities influence the degree to which social capital may be created (Coleman 1990). Social capital exists when the relations among people make possible individual or joint achievements that otherwise would not be possible (Coleman 1990). Social capital involves parents and children in friendship networks and community organizations, both formal and informal, which are characterized by a set of obligations, expectations, and social ties that connect the adults, and that help to bring about the control and supervision of children. Through such a process, it is argued, information is exchanged, norms are established, and informal systems of social control are laid down. As such, a child is raised in a neighborhood environment with norms and sanctions that are not or cannot be brought about by one adult in isolation. The creation of social capital is virtually impossible to develop in unstable communities, and mere density of a community is insufficient to establish it. Social capital, then, constitutes a community-level coping variable.

"Social impoverishment," the absence of social capital, has been tied to several health-related outcomes. Such indicators predict high rates of child abuse (Garbarino & Sherman 1980), which is important as a cause of morbidity and mortality and also as a predictor of violence in adulthood (Widom 1989). Indicators of social capital are inversely related to levels of adolescent aggression and delinquency which, in turn, are predictors of homicide and suicide. Low levels of social organization are associated with high levels of adult crime as well. Social disorganization also predicts whether young girls will become pregnant (Osofsky 1990); whether they will be supported by a

network in limiting high-risk maternal behavior such as smoking, drinking, and drug abuse (Wallace 1990); and whether they will abuse their children after birth (Sampson 1992). Overall, the more formal and informal ties exist in a community, the more dense and multiplex the networks and the greater the constraints on deviant, often health-compromising behavior, both for adolescents and adults (Sampson 1992).

On the individual level, the lack of social capital may erode the quality of social support available to an individual. Rapid mobility in and out of an area reduces the marriage pool initially, and the likelihood of remarriage following divorce. Fear of crime fosters a distrust of others that can contribute to social isolation (Krause 1992). Social isolation has, in turn, been related to an array of adverse health outcomes (House et al 1988a), and it is known to compromise immunologic functioning (Kiecolt-Glaser et al 1994). In contrast, social opportunities provided via information networks, intergenerational networks, churches, and other community organizations may foster the creation of individual social ties that have a health-protective effect (Sampson 1992).

Health Habits and Behaviors

Community characteristics influence the degree to which certain health habits may be practiced. Because poorer neighborhoods have fewer facilities and resources, the adoption of public health recommendations such as obtaining a healthy diet and obtaining regular exercise sometimes cannot be met. High-stress neighborhoods, characterized by high density, high crime, and high mobility, may also lead to the development of health-compromising behaviors that act as efforts to cope with stress, such as smoking, alcohol consumption, and drug abuse. Noise appears to increase rates of smoking and the use of some drugs (Evans 1997).

Health habits are also affected by the social capital generated within communities. For example, pregnant women, especially young, single pregnant women, show higher levels of prenatal care in neighborhoods with strong social networks, perhaps because these networks put pressure on them to avoid compromising the health of their fetus and also provide them with more information about what constitutes effective prenatal care (Sampson 1992). Other health habits may be similarly affected.

The availability of health care varies substantially by neighborhood. Consistently poor health services are found in low-income, minority, and transient areas (Macintyre et al 1993, Williams 1990, Wyke et al 1992). Especially for children, the combination of rapid population loss coupled with inadequate health services is devastating. Communities experiencing poverty, overcrowding, and rapid population change show rises in infant mortality and low birth weight (Wallace & Wallace 1990). In contrast, the information networks that

develop in stable communities transmit knowledge of and linkages to clinic services (Sampson 1992), including prenatal services, child health services, and general child care.

Conclusion

Documenting the effects of community characteristics on health is difficult. Studies that control for demographic characteristics, such as SES and race, on the one hand, and more proximal family characteristics, such as family income, on the other hand, may statistically underestimate the contribution of community to health by virtue of drawing off variance into these more distal and proximal predictors (G Duncan, J Connell & P Klebanov, unpublished observations). Moreover, estimating the independent contribution of highly correlated community characteristics, such as population density, population mobility, and community SES level creates problems of data analysis and interpretation. In addition, community characteristics do not have uniformly positive or negative effects that consistently translate into health outcomes. For example, the presence of middle-SES neighbors appears to have a protective effect in reducing aggression and delinquency among low-SES youngsters, especially black males from low-income, single-parent homes, but it may simultaneously adversely affect the ability to develop social relationships with their peers (Kupersmidt et al 1995).

Despite the difficulties with drawing inferences from community studies, the evidence suggests several community characteristics that have adverse effects on health, including crowding, air pollution, exposure to violence, and the absence of social networks and social ties. In addition, the protective effects of informal and formal social ties and networks appear to be robust. Exactly how community and neighborhood characteristics affect health is still largely unknown (Jencks & Mayer 1990); the evidence is strongest for chronic stress, the erosion of social ties, and development of poor health behaviors, although the links to physiology have yet to be established. The role of mental distress and coping skills other than those relating to social capital are less well studied.

THE FAMILY SOCIAL ENVIRONMENT

The family environment clearly influences children's health. The link can be direct, as when a parent's behavior exposes a fetus to drugs in the womb (Neuspiel et al 1989). We focus on indirect links and highlight four characteristics of the family social environment that appear to influence child and adolescent health: (*a*) the quality of parenting, especially emotional aspects of the parent-child relationship; (*b*) the family's social climate, especially the

amount of conflict and violence in the home; (*c*) the parents' mental health and other behavioral characteristics of the parents; and (*d*) variables associated with social-economic conditions of the household, such as whether the child lives with one or both parents and the educational level of parents.

Chronic Stress

Characteristics of a family environment that appear to be associated with health problems in children include a lack of warmth and emotional support from parents and a high level of conflict and violence. A cold and unresponsive parenting style has been associated with retarded infant growth and increased rates of illness in childhood (Bradley 1993, Gottman & Katz 1989). A stressful family environment can even influence prenatal development (Collins et al 1993). Quarreling and fighting at home have been linked to psychosomatic symptoms such as headaches and stomachaches in adolescents (Mechanic & Hansell 1989). When family dysfunction and conflict escalate to the point of abuse, the direct and indirect effects on child health can be lethal. Children with histories of physical abuse and neglect have an elevated mortality risk for all causes of death, including homicide, transportation injury, other unintentional injury, and disease (Sorenson & Peterson 1994). A history of sexual abuse can have health consequences years later, leading to increased rates of psychological distress, headache, asthma, diabetes, arthritis, HIV infection, gynecological problems, and various somatic symptoms (Braaten 1996). Some of the effects of sexual abuse on health may be mediated by poor health habits. Women with a history of childhood sexual abuse are more likely to smoke, abuse drugs, and engage in risky sexual behavior, and they are less likely to use medical care (Springs & Friedrich 1992). Other indirect effects of abuse may be mediated by conditions in the family. For example, the instability and social isolation that is often found in abusive families appear to mediate the effects of maltreatment on children's poor academic performance (Eckenrode et al 1995) and may influence health as well (House et al 1988b).

Repeated interference with homeostatic processes may be one physiologic pathway through which a chronically stressful family environment causes health problems in children. Experimental manipulations resembling two of the family characteristics considered, namely the presence of anger and conflict and the absence of emotional warmth and responsiveness, can disrupt patterns of cardiovascular and neuroendocrine regulation in children. For example, preschoolers respond to videotapes of angry adult interactions with increases in heart rate and blood pressure (El-Sheikh et al 1989), and separations from mothers lead to increased heart rates as well as elevations in norepinephrine and cortisol, particularly among socially shy children (Kagan et al 1987, 1988). However, a secure infant-mother attachment predicts lower

stress response (as indexed by salivary cortisol) to new or strange situations (Hertsgaard et al 1995). Cortisol responses to separation are also attenuated in the presence of a responsive caregiver (Gunnar et al 1992). A buildup in allostatic load experienced by a child who is responding physiologically to repeated social challenges at home may help to explain the poor development and high rates of illness observed in children from stressful family environments.

Mental Health/Distress

Depression is clearly associated with deleterious health outcomes among children and adolescents, such as increased acute illness and physical symptoms and unhealthy behaviors such as smoking and substance abuse (Gore et al 1992, Lewinsohn et al 1994). Household economic conditions influence both the risk of childhood depression and its link to health. Risk factors for childhood depression include living in a single parent household, parental unemployment, and parents' poor educational background (Gore et al 1992, Kaslow et al 1994, Lewinsohn et al 1994). Depression is more strongly linked to poor health among adolescents with a lower standard of living (Gore et al 1992).

More depressed children are also found among families whose members provide one another with little or no support, families that do not experience a sense of cohesiveness, and families characterized by high levels conflict, especially marital conflict (Kaslow et al 1994, Lewinsohn et al 1994). Characteristics of the parent-child relationship that are associated with depression include low levels of behavioral and emotional involvement, high levels of conflict and hostility, and a parenting style that is more autocratic, dominant, and controlling (Kaslow et al 1994, Lewinsohn et al 1994). Children of depressed mothers appear to be at increased risk for both depression and suicidal behavior, as well as a variety of other psychiatric diagnoses (Kaslow et al 1994). The behavior of depressed parents may contribute to this risk. Most studies find depressed mothers to be less responsive, more critical, negative, and irritable, and more controlling and intrusive with their children (Downey & Coyne 1990, Nolen-Hoeksema et al 1995).

Abuse within the family is also associated with depression and suicide (Malinosky-Rummell & Hansen 1993). Evidence suggests that the connection between a childhood history of family violence and recurrent depressions in adulthood is mediated by chronic interpersonal problems in the adult's life (Kessler & Magee 1994).

In summary, the links between family and parenting characteristics and depression in children and adolescents are clear. There is also evidence for negative short-term health effects in the form of increased rates of acute illness and suicide. Chronic or intermittent depression in childhood and adolescence

may be associated with patterns of coping and/or physiologic responses to stress that contribute to long-term adverse health outcomes as well.

Coping Skills and Resources

Davies & Cummings (1994) suggested that emotionally secure children are better able to regulate their emotions in the face of stress and therefore cope more effectively with daily problems. According to their model, emotional security is threatened by destructive forms of family conflict, such as conflicts between parents that involve physical aggression, and by parent-child relationships marked by instability and a lack of parental warmth and responsiveness. Family social environments with features such as these, including abuse, discord, and parental psychopathology, are associated with maladaptive coping in children, in particular difficulty with anger regulation (Crittenden 1992, Cummings & El-Sheikh 1991, Zahn-Waxler et al 1984). Because anger and its regulation have been tied to the development of heart disease and hypertension, there may be health risks associated with growing up in homes that have these characteristics.

Family experiences also influence how children learn to negotiate interpersonal situations involving frustration and anger. Violent boys are more likely to live in mother-headed households, or to have fathers who use spanking for discipline and rarely express affection for their sons (Sheline et al 1994). Prospective research indicates that the development of hostile attitudes and behaviors in male adolescents is associated with family interactions that are nonsupportive and have a negative affective tone (Matthews et al 1996). Parent-child conflict and ineffective parenting practices foster problems in social information processing and social skill deficits in children that, in turn, lead to poor coping in school, in particular during interactions with peers (Patterson et al 1989). Children from families in which there is greater organization and consistency in the home use fewer aggressive coping strategies in response to everyday stress (Hardy et al 1993).

Dysfunctional coping strategies in children and adolescents may persist into adulthood. Thus, maladaptive coping styles that are first acquired in response to a stressful family environment in childhood may be associated with greater autonomic reactivity and poorer health outcomes throughout the lifespan. For example, hostility measured in adolescence is linked to coronary risk factors (such as high lipid ratios, larger body mass, smoking, and caffeine consumption) assessed more than 20 years later (Siegler et al 1992).

Health Habits and Behaviors

The abuse of substances, such as alcohol, cigarettes, and illicit drugs, and risky sexual behavior are two health-threatening classes of behavior that are usually

first observed during adolescence. In addition to their direct effects on health, these behaviors are linked with patterns of sleep, diet, and physical activity that indicate an unhealthy adolescent lifestyle (Donovan et al 1991).

The use of drugs by family members, both parents and siblings, is a reliable risk factor for adolescent drug usage (Denton & Kampfe 1994). Although genetics may play a role in cigarette smoking (Rowe & Linver 1995), the family's influence on adolescents' use of other drugs may be mediated by social environment factors. In addition to the imitation of behaviors observed at home, teens whose parents abuse substances appear to be more vulnerable to stress (Barrera et al 1995) and to acquire attitudes and coping styles that lead to increased affiliation with substance-using peers (Wills et al 1994). The experience of maltreatment in the home is also a risk factor for adolescent drug use (Malinosky-Rummell & Hansen 1993). In contrast, supportive and cohesive families help protect adolescents with problem-drinking fathers (Farrell et al 1995).

Drug-abusing teens are more likely to live in single-parent homes and homes from which they feel alienated (Denton & Kampfe 1994). A lack of support and sense of rejection and detachment from parents has been associated with adolescent substance use (Barrera et al 1993, Turner et al 1993). Deficits in parental support partially mediate the association between low parental education and increased substance use among teens (Wills et al 1995b). Prospective longitudinal data suggest that fathers' difficulty controlling anger predicts sons' future alcohol and drug usage (D'Angelo et al 1995). Maternal behavior rated as cold, unresponsive, and underprotective when children were five years old has been associated with frequent drug usage during adolescence (Shedler & Block 1990). Findings like these do not rule out the possibility that the same stable personality traits in a child that undermine the development of supportive family relationships may also increase the child's propensity to abuse substances (Wills et al 1995a).

There are conflicting findings regarding the role that parental control, supervision, and monitoring play in adolescent drug use. On the one hand, studies suggest that the homes of some abusing adolescents are overly controlling and that the children experience a lack of autonomy (Denton & Kampfe 1994, Webb et al 1991). On the other hand, longitudinal evidence relates abuse of drugs and smoking by teens to homes with less consistent enforcement of rules, less parental monitoring of the child's behavior, fewer parental demands of the child, and less imposition of structure and organization (Biglan et al 1995, Stice & Barrera 1995). In addition, some parents respond to their teen's use of drugs with fewer attempts at control and less support, which may signal the parents' acquiescence or sense of helplessness (Stice & Barrera 1995).

Many of the characteristics of families that are linked to substance abuse are also linked to adolescent sexual behavior. Teens growing up in single-parent households and those with histories of physical or sexual abuse show an increased probability of engaging in risky sexual behavior (Cunningham et al 1994, Jemmott & Jemmott 1992). Teens engage in more frequent sexual activity and more risky sexual behaviors when there is less parental monitoring and more permissiveness at home (Jemmott & Jemmott 1992, Metzler et al 1994). However, there is also evidence of increased sexual activity among adolescents whose parents overprotect them and fail to help them to learn to function independently (Turner et al 1993).

Conclusion

Research findings consistently point to three characteristics of family environments that can undermine the health of children and adolescents: (*a*) a social climate that is conflictual and angry or, worse, one that is violent and abusive; (*b*) relationships, particularly parent-child relationships, that are unresponsive and lacking in cohesiveness, warmth, and emotional support; and (*c*) parenting that is either overly controlling and dominating on the one hand or uninvolved with little monitoring of the child and little imposition of rules and structure on the other hand. These dimensions of a family environment are stressful for children, and they are associated with depression, maladaptive ways of coping with negative affect, and health-threatening behaviors in adolescence. The family characteristics identified as contributors to poor health outcomes in childhood are often embedded within households characterized by economic strain and few resources. Evidence suggests that parenting behavior may mediate some of the effects of economic strain (Huston et al 1994); however, economic strain may mediate the effects of other family characteristics, such as the number of parents in the home (Gore et al 1992).

THE PEER SOCIAL ENVIRONMENT

Chronic Stress and Mental Health/Distress

Characteristics similar to the factors identified in stressful communities and families predict chronic stress in the peer and school environments: less adult attention and stability at school, and more conflict with and less acceptance by peers. For example, children who show greater cardiovascular reactivity are more prone to develop respiratory illnesses when they are enrolled in preschools that are unstable and unable to provide individual adult attention (e.g. those with high teacher-child ratios, high staff turnover, more part-time teachers, etc) (Boyce et al 1995). As highly reactive children in less stable and less attentive preschools repeatedly respond to social stressors in the environment, they may build up high allostatic load, which may result in more illnesses.

Adolescents spontaneously mention interpersonal stressors involving peers—such as conflicts with friends, feeling lonely or left out of peer groups, and boyfriend/girlfriend problems—as among the most common and most distressing problems that they face in daily life (Repetti et al 1996). Being neglected or rejected by peers has been associated in longitudinal studies with both increased aggression and depression (Kupersmidt & Patterson 1991). The complex interactions between the family and peer social environments are illustrated by research indicating that poor parenting practices often result in child behavior problems that can, in turn, lead to peer rejection (Patterson et al 1989).

Coping Skills and Resources

As youngsters move from childhood into their teenage years they spend less time with their families and (especially for girls) more time with peers (Larson & Richards 1991). Not surprisingly, then, adolescents often turn to their friends for support, especially when there is turmoil at home (Aseltine et al 1994). The presence of supportive peer relationships is usually associated with better mental health among children and adolescents, although that association partly reflects the impact of psychological functioning on the development of supportive friendships (Hirsch & DuBois 1992). The peer group may facilitate children's coping by enhancing self-esteem, perceptions of control, and the perceived security of social relations (Sandler et al 1989b). Empirical evidence for the stress-buffering role of peer social support has been mixed. Some studies suggest that support from friends can provide protection from the negative impact of stress on children's psychological adjustment (Dubow & Tisak 1989), but others do not find evidence of a moderating role of peer support (Cumsille & Epstein 1994). The effectiveness of peer support is likely to vary as a function of the stressor with which the adolescent is coping (Gore & Aseltine 1995).

Health Habits and Behaviors

Adolescents' beliefs about the prevalence and acceptance of alcohol and drug use by peers are risk factors for alcohol and drug use, with peer influence increasing as children age (Bailey & Hubbard 1990, Donaldson 1995). In addition to passive social pressures exerted by the perceived behavior and attitudes of peers, social contact with certain individuals may increase the availability of these substances (Dolcini & Adler 1994). For example, residence in a fraternity or sorority and the adoption of a party-centered lifestyle are reliable predictors of binge drinking among college students (Wechsler et al 1995).

Peer social influence affects other behaviors that pose significant threats to adolescent health. For example, high school students are more likely to engage

in risky sexual behavior if their friends are sexually active or if they believe that a majority of their peers have had intercourse (Walter et al 1992). Consistent with data on other risky behaviors, one of the best-known predictors of adolescent smoking is association with peers who smoke (Biglan et al 1995). The appeal of smoking may derive, in part, from its function as a social signal suggesting adult role status and independence from parents (Rowe & Linver 1995).

The influence exerted by peers may be at least partially shaped by the family social environment. On the one hand, children are more likely to affiliate with substance-abusing peers if their parents abuse substances or inadequately supervise their activities (Biglan et al 1995, Wills et al 1994). On the other hand, the effect of peer drug use is much weaker when parenting is authoritative (i.e. parents are involved, make demands, and supervise while demonstrating acceptance and warmth) (Mounts & Steinberg 1995).

Conclusion

The social environments provided at school and by peers affect child and adolescent health at each point in Figure 2. There is particularly strong evidence for the stressfulness of conflicts and rejection by peers and for the social influence of peers over several categories of health-threatening behavior. An important finding is that the effects of the peer social environment must be understood within the context of the family environment.

ADULT SOCIAL ENVIRONMENT

A broad range of social ties has been examined concerning adult health. These ties include not only immediate family and other close relatives and friends but also ties to larger formal and informal groups. An extensive literature documents the range of negative health outcomes that accrue to those whose social environment is either structurally impoverished (i.e. characterized by fewer social ties) or functionally impoverished (i.e. characterized by a dearth of socially supportive interactions with others) (Berkman 1995, Broadhead et al 1983).

Although social ties have generally been seen as serving a health-promoting role, the social environment can also be a source of increased and potentially chronic stress, which contributes to increased rather than decreased health risks; that is, social relationships are characterized by costs as well as benefits. Such costs can take the form of requests/demands for assistance, criticism, or other forms of interpersonal conflict (Averill 1982). When such characteristics predominate in the social environment, they can result in increased risks for

poor mental and physical health (for discussion and review, see Burg & Seeman 1994, Seeman et al 1996).

Chronic Stress

The health-promoting effects of the social environment are generally hypothesized to result from the stress-reducing effects of social integration within a nurturant, supportive milieu (Cohen 1992). Evidence linking social environment characteristics to physiologic stress responses supports this view, pointing to the potential importance of the social environment in the accumulation of allostatic load.

Although being married has been generally associated with better immune responses as measured by various parameters of immune reaction (Kiecolt-Glaser et al 1994), spousal interaction characterized by greater hostility and conflict has been associated with greater cardiovascular and neuroendocrine reactivity and lower immune function (Kiecolt-Glaser et al 1994). The stress-inducing combination of emotional and physical demands of caring for a sick spouse has also been related to lowered immune function (e.g. among those with a spouse with cancer or those who are caring for a spouse with Alzheimer's disease) as well as elevated lipids and increased endocrine reactivity (for a review, see Kiecolt-Glaser et al 1994). Ambulatory monitoring of blood pressure at work and at home indicates that men show a reduction in blood pressure in the home environment whereas women do not (Unden et al 1991), which may be due to the greater demands of home and child care frequently assumed by women. Beyond the marital relationship, individuals reporting lower levels of support from close friends and family also exhibit higher heart rate and systolic blood pressure (Dressler et al 1986, Unden et al 1991), higher serum cholesterol and lower immune function (Thomas et al 1985), and higher levels of neuroendocrine activity (Seeman et al 1994).

Experimental studies also demonstrate the physiologic impacts of social interactions. Interpersonal challenge or hostility have been shown to elicit increased neuroendocrine and cardiovascular activity (Brown & Smith 1992, Krantz et al 1986). The presence of a friend or supportive confederate, however, generally attenuates cardiovascular reactivity in subjects confronted with challenging laboratory tasks (for a review, see Seeman & McEwen 1996). By contrast, the presence of a stranger observing the testing session produces increased reactivity (Snydersmith & Cacioppo 1992) as does the presence of others (friends or strangers) who disagree with the subject about some aspect of the session (Back & Bogdonoff 1964, Gerin et al 1992). Reactivity appears to be reduced by the presence of even one other person who agrees with the subject (Back & Bogdonoff 1964, Gerin et al 1992).

Mental Health/Distress

The positive and negative effects of the social environment on mental health have been extensively documented (for a review, see George 1989). Greater social integration, particularly as reflected in the presence of primary ties with spouse, children, and other kinds of supportive significant others are associated with lower risk of depression (George 1989), while marital disruption—either through bereavement, marriage dissolution, or the cognitive impairment of one's spouse—is associated with increased risks for psychological distress (Aseltine & Kessler 1993, Bloom et al 1978, Moritz et al 1989). The quality of existing relationships, however, appears equally important in predicting mental health outcomes. Relationships that are characterized by criticism, unwelcome advice or conflict, or demands for caregiving have been associated with increased psychological distress (Kessler et al 1985). Depression and/or negative affect in significant others is also associated with increased depression and psychological distress (Joiner 1994). Even in the absence of overtly negative social interactions, failure of the family or friends to provide anticipated or expected support can result in increased psychological distress (Brown & Harris 1989).

Women may be especially vulnerable to the psychological consequences of nonsupportive interactions with family or friends. Although men and women both report more supportive than negative social interactions, women report relatively more negative interactions with nondiscretionary family or kin ties (Lefler et al 1986, Schuster et al 1990) and appear to be more emotionally distressed by such negative interactions (Lefler et al 1986, Wethington et al 1987). Men benefit more consistently from greater social integration (House et al 1988a).

Coping Skills and Resources

Throughout life, one's family and friends serve as the social context within which events are appraised and coping strategies are evaluated and initiated (Cohen 1992). Family and/or friends can affect coping efforts through provision of actual instrumental and/or informational support and can serve as sources of emotional support (Cohen 1992). Such social resources may be particularly important in coping with lower-SES environmental demands: The relationship between lower occupational status and greater psychological vulnerability to life events disappears for lower-status women reporting high social support (Turner & Noh 1983).

The influence of the family or friends on coping, however, may not always be health promoting. The assistance provided by family and friends in coping with illness and/or disability can result in increased dependency and disability (for reviews, see Seeman et al 1996, Thompson & Sobolew-Shubin 1993) or

poorer disease control. Diabetic men with larger support networks exhibit poorer control of their diabetes (e.g. significant increases in glycosylated hemoglobin, cholesterol and triglycerides, glucose and weight; Kaplan & Hartwell 1987) as do teenagers reporting greater satisfaction with their social-support systems (Kaplan et al 1985), possibly because members of the support network undermine diabetes-related diet behaviors. Studies also document limits to the effectiveness of social support in promoting better coping. In the face of severe stressors such as life-threatening disease, even the presence of close and apparently supportive relationships is not always associated with reductions in psychological distress or greater physical recovery (Bolger et al 1997, Coyne & Fiske 1992).

Health Habits and Behaviors

The social environment also serves as a source of learning and reinforcement for attitudes and behaviors that affect health. People who are more socially integrated exhibit greater preventive health behavior, including less smoking and drinking (Broman 1992) and more cancer screening (for a review, see Berkman 1995) and more successful risk reduction efforts such as reducing dietary fat, exercising, and stopping smoking (Bovbjerg et al 1995, Sallis et al 1989). Supportive family environments have also been related to better adherence in hemodialysis treatment (Christensen et al 1992). However, the social environments provided by family and friends also carry the potential for encouraging more detrimental health behaviors such as problem drinking (Seeman et al 1988), less successful efforts to quit smoking (Cohen 1992), and as indicated above, poorer control of diabetes. Family environments can also present more direct health threats in the form, for example, of second-hand smoke exposure from living with a smoker (Sandler et al 1989a) and physical abuse (Mercy & Saltzman 1989).

Evidence linking social environment characteristics to health-care utilization is neither extensive nor consistent. Consultations with family and friends can result in increased or decreased utilization, depending on the degree to which their attitudes and behaviors favor such utilization as well as their ability to provide assistance to facilitate utilization (Geertsen et al 1975, Penning 1995, Sampson 1992).

Conclusion

The characteristics of the social environment that relate most strongly to adult health outcomes—lack of social integration and poor quality of social relationships—also exhibit links with postulated pathways for these health effects. Specifically, social relationships characterized by conflict and hostility are associated not only with increased mental distress but also with increased

physiologic arousal and lowered immune function, profiles of physiologic activity with known links to disease pathology (McEwen & Stellar 1993). In contrast with these health-damaging effects of impoverished social environments, environments characterized by supportive relationships appear to serve a stress-reducing, health-promoting function, enhancing psychological functioning and reducing physiologic arousal. In addition, the social environment can impact directly and importantly on health behaviors and health-care utilization.

WORK

The work environment represents an important life arena for adults that contributes to life satisfaction. Controlling for SES and health, those who work report a higher quality of life than those who do not (Ruchlin & Morris 1991). Moreover, full-time employment predicts slower declines in perceived health and in physical functioning for both men and women (Ross & Mirowsky 1995). Nonetheless, adverse characteristics of the work environment have long been suspected to contribute to ill health. Work stressors are among the most common and upsetting stressors that people report, and because the majority of adults work full-time, they may be exposed to the health-compromising effects of these conditions over the long term.

The work environment may directly affect illness precursors or illness, including injuries, cancers, and respiratory and cardiovascular disease, by exposing workers to physical, chemical, and biological hazards (House & Smith 1985). Because this voluminous literature has not yet implicated psychosocial mechanisms in disease pathology, apart from the fact that such adverse exposure is strongly linked to SES and race, we do not review it here.

Chronic Stress

Chronic stress is the mechanism most commonly offered to explain the adverse effects of the work environment on health. Work overload is one of the chief factors studied. Workers who feel required to work too long and too hard at too many tasks report more stress (e.g. Caplan & Jones 1975), practice poorer health habits (Sorensen et al 1985), and report more health complaints than do workers not suffering from overload (Repetti 1993). Work overload appears to trigger neuroendocrine and cardiovascular reactions that, over time, can increase the likelihood of cardiovascular disease. Working 40 or more hours a week is a risk factor for producing babies of low birth weight among employed women (Peoples-Sheps et al 1991). Work pressure also predicts ill health. Men reporting high job pressure or demands sought medical attention more and showed more documented signs of pathology; a follow-up investigation revealed these men were more likely to die in the following decade

(House et al 1986). Consistently, however, research demonstrates a stronger relationship between reported work overload and physical health complaints than between number of hours worked and poor health (e.g. Herzog et al 1991), which raises the possibility that psychological distress or negative affectivity is implicated in this relation.

Role conflict and role ambiguity have also been tied to illness precursors and illness (Caplan & Jones 1975). Role conflict occurs when an individual receives conflicting information about work tasks or standards. Chronically high blood pressure and elevated pulse have been tied to role conflict and role ambiguity (French & Caplan 1973).

Responsibility for others may contribute to ill health. For example, a study comparing illness rates of air-traffic controllers and second-class airmen found that hypertension was four times more common and diabetes and peptic ulcers twice as common among the air-traffic controllers than among the airmen, who did not have responsibility for the fates of others. Moreover, all three diseases were diagnosed at a younger age among the air-traffic controllers. Hypertension and ulcers were more common among controllers at busier airports (Cobb 1976).

The perception that one's career or job development has been inadequate may also contribute to ill health. People who feel they have been promoted too quickly or too slowly, who feel insecure about their jobs, and who feel that their ambitions are thwarted are more likely to report stress, to seek help for psychological distress, and to show higher rates of illness, especially cardiovascular disease (Catalano et al 1986).

Research has especially focused on the effects of a high-strain work environment defined as one with a high level of demands and a low level of decision latitude (Karasek & Theorell 1990). Considerable research has supported the hypothesized relation of high work strain to poor health (e.g. Landsbergis et al 1992). Work strain is significantly negatively associated with health-related quality of life, including physical functioning, role functioning related to physical health, vitality, social functioning, and mental health (Lerner et al 1994). Job strain has been linked to higher fibrinogen levels among working middle-aged women (Davis et al 1995) and to low birth weight (Woo 1994). Frankenhaeuser (1991) found that the catecholamine/cortisol balance is different in high- versus low-control situations, with cortisol levels lower in high-control situations. Not all studies find adverse effects of high levels of job demands and strain, however (Albright et al 1992), and studies relating job demands and decision latitude to coronary heart disease risk factors (cholesterol, smoking, and systolic and diastolic blood pressure) are inconsistent (Alterman et al 1994, Pieper et al 1989). Nonetheless, research suggests that the risk that role overload will lead to heart disease may be

reduced when people are given a high degree of control in the work environment (Karasek & Theorell 1990).

Job uncertainty and unemployment have been associated with a variety of adverse mental and physical health outcomes, including depression, physical symptoms, physical illness (Hamilton et al 1990), alcohol abuse (Catalano et al 1993), and a heightened mortality rate (Sorlie & Rogot 1990). The negative effects of unemployment appear to be generated partly by the financial strain produced by unemployment and by the fact that unemployment creates vulnerability to other life events. Being unstably employed is also related to poor health (Pavalko et al 1993, Rushing et al 1992).

Research has focused increasingly not only on work stress but on its interactive effects with the stress induced by other roles, especially into the family. Much of this work has focused on married women with young children who are employed and who are chiefly responsible for household and child-care tasks. Overall, combining marital, parental, and occupational roles does not appear to significantly affect mortality (Kotler & Wingard 1989). However, attempting to juggle heavy responsibilities at both work and home reduces the enjoyment of all tasks (Williams et al 1991). On the other hand, employment can also be beneficial for women's well-being (Repetti et al 1989). Whether the effects of multiple roles are positive or negative appears to depend heavily on resources available. Having control and flexibility over one's work, having a good income, and having someone to help with the housework or child care all reduce the likelihood that combining multiple roles will produce psychological costs (Lennon & Rosenfield 1992, Rosenfield 1992). Although less research has been conducted on men, what evidence there is suggests that for men, multiple roles are protective (Adelmann 1994).

Mental Health/Psychological Distress

Mental health or psychological distress has typically not been examined as a pathway to poor health in the work environment but has been treated as an outcome in its own right. This is because psychological distress often translates into outcomes of importance to employers, specifically job dissatisfaction, absenteeism, disability claims, and high rates of turnover. Overall research suggests that the factors that compromise physical health also compromise mental health (for a review, see Taylor 1995). Whether anxiety and depression generated by job stress constitute a route by which the chronic stress of the work environment translates into poor health outcomes is unknown.

Coping Skills and Resources

The role of coping strategies in moderating the relation between chronic work stress and adverse health outcomes is an understudied area. This may be true

partly because objective work demands are often so powerful that individual coping strategies have little room in which to operate. In addition, understanding how people cope with job stress may necessitate the examination of coping strategies specific to a particular job (Dewe & Guest 1990). Nonetheless, active coping strategies have been associated with more effective coping in a variety of situations (for a review, see Haidt & Rodin 1995), and the Karasek model of work strain is consistent with such an argument in positing that the degree to which individuals have decision latitude reduces work strain. Nonetheless, under conditions of high psychological demands, decision latitude may have negative effects.

Social support has been extensively studied in the work environment, and its effects are generally beneficial. Social support can have both direct (e.g. Ganster et al 1986, Loscocco & Spitze 1990) and moderating effects (e.g. Repetti et al 1989) on reported stress and health problems in the workplace. Those who report being unable to develop satisfying relationships report more negative affect at work (Buunk et al 1993) and poorer physical and mental health (Repetti 1993). Poor social relations at work have been tied directly to heightened catecholamine levels (Cooper & Marshall 1976), to risk for coronary heart disease (Repetti 1993), and to heightened fibrinogen levels in working women (Davis et al 1995). A study of government workers found that high workload was related to high blood pressure, but this relationship was attenuated among employees who had supportive relationships with their supervisors (House 1981). Adverse effects of unemployment also appear to be moderated by the seeking of social support (Turner et al 1991). Social support may be especially important for buffering work stress for minority group members (Gutierres et al 1996).

Health Habits and Behaviors

As noted, health habits are adversely affected by job stress and as such, may also play a moderating role in the relation between work stress and adverse health outcomes. People who feel they have more control over work are less likely to engage in risky health behaviors (Wickrama et al 1995). Substance abuse in the work environment has been extensively studied. Muntaner et al (1995) found that after adjusting for SES, alcoholism, and certain work conditions, drug abuse was higher in individuals with high-strain jobs. Job strain may not lead people to start smoking, but it does appear to lead to heavier smoking (Green & Johnson 1990). High job dissatisfaction is associated with heavy drinking, and negative consequences of drinking are associated with the job characteristics of low autonomy, little use of capacities, and lack of participation in decision making (Greenberg & Grunberg 1995). However, some research suggests that such abuse may be more related to general feelings of

powerlessness, alienation, and lack of commitment than to specific job characteristics (Seeman et al 1988). Problem drinking in response to job characteristics may also depend on the degree to which alcohol consumption is perceived to be a useful means of coping (Greenberg & Grunberg 1995). Most studies relating job characteristics to health habits are cross-sectional, so it is difficult to determine whether job strain and health habits may be due to some third factor, such as emotional distress.

Conclusion

Research links work stress to a broad array of acute diseases, and evidence that it contributes to chronic disease such as cardiovascular disease, is mounting (Repetti 1993). The main psychosocial pathway from the work environment to poor health that has been studied involves chronic stress. Other potential routes, such as mental health or emotional distress, coping strategies, and health habits, have been studied not as pathways but as outcomes in their own right. The research on occupational stress could clearly profit from a consideration of more complex and multiple causal routes in relating chronic stress to adverse health outcomes.

CONCLUSIONS

Individual experiences and behaviors predictive of health outcomes are nested within geographic, social, developmental, and economic environments. The initial context for this observation is the recognition that social class and race are related to all-cause mortality and morbidity associated with a wide range of disorders. Those in the lower ends of the social class distribution and African-Americans disproportionately live in high-stress communities, occupy jobs characterized by high demands and low control, and live in family and social environments where they are disproportionately exposed to violence, conflict, and abuse. The type of community in which one lives and the demands of the work environment feed into the family environment and the kinds of social ties external to the family that may be developed. Thus, social environments influence health in a complex and interactive fashion.

The health effects of individual characteristics such as hostility, or the health effects of a family environment characteristic such as a high level of conflict, must be understood within the larger environments in which these behaviors are learned and expressed. At the very least, such an analysis should alert the psychologist to the potential risks of “psychologizing” or “biologizing” health-predictive variables without considering the contexts in which they occur. For example, recent empirical studies identifying a genetic component to the perception of social support (Kendler et al 1991) and suggesting that social support is partly a function of individual dispositions in personality

(Cohen et al 1986) must be balanced by the recognition that environments play an important role in fostering or undermining the ability to create social ties. Psychosocial predictors of health outcomes do not occur and should not be studied in an economic, racial, developmental, and social vacuum. With respect to interventions, the implications of such a viewpoint are complex. On the one hand, considering multiple levels of analysis simultaneously suggests multiple intervention points ranging from the individual through the family to the community. On the other hand, any intervention focus must acknowledge the interrelatedness of these environments and the fact that change induced at one level may have modest long-term effects, if corresponding changes do not occur at other levels.

Substantial evidence relates characteristics of environments to health-relevant outcomes, including all-cause mortality and a wide range of chronic diseases. Manifold pathways, reviewed above, have been implicated, including the exposure of individuals to chronic stress and the increased allostatic load that may result, the creation of chronic or intermittent emotional distress, the development or use of ineffective coping strategies and the inability to form and make use of social ties, and the acquisition and practice of health habits that are dependent upon environmental factors that limit personal and social resources. Empirically, the links from environmental characteristics to chronic stress to identifiable biological endpoints have most commonly been made. Pathways involving the other routes are less well studied, and more complex concatenations of these pathways, as suggested by Figure 2, have barely been studied at all. Each of the environments studied provides suggestive evidence about likely pathways that may guide research in the future.

At the outset, we asked "What is an unhealthy environment and how does it get under the skin?" Answering such a question definitively is currently beyond the scope of any review, because much of the research one would like to see has not yet been conducted. Nonetheless, despite the gaps in the literature, the beginning of an answer is emerging. Consistently across the environments examined—community, family, work, and peers—those that threaten personal safety; that limit the ability to develop social ties; or that are characterized by conflictual, violent, or abusive interpersonal relationships are related to a broad array of adverse health outcomes. These effects appear to occur across the lifespan, beginning prenatally and carrying through into old age. Likewise, a picture of the healthy environment is coming into view. Environments are healthy to the degree that they provide safety and opportunities for social integration. In addition, the ability to experience a sense of personal control may be important, within certain parameters.

People have evolved as social animals and as such appear to be sensitively "tuned in" to others in the social world. Therefore, it should not be surprising

that the social environment has such potential to affect physiology, both positively and negatively. Evidence continues to accumulate that throughout the lifespan, the structure and quality of social interactions have profound effects on psychological, behavioral, and physiologic functioning, and ultimately on our health and well-being.

ACKNOWLEDGMENTS

Funds for this review were provided in part by the MacArthur Foundation's SES Planning Initiative on Health. Preparation of this manuscript was also supported by MH 42152 from the National Institute of Mental Health to the first author and by FIRST Award R29-48593 from the National Institute of Mental Health to the second author. The third author was supported, in part, by a grant from the MacArthur Research Network on Successful Aging and by NIA-SERCA grant AG-00586. The authors gratefully acknowledge the helpful comments of Sheldon Cohen on a prior draft.

Literature Cited

Adelmann PK. 1994. Multiple roles and psychological well-being in a national sample of older adults. *J. Gerontol.: Soc. Sci.* 49: S277–85

Adler NE, Boyce WT, Chesney MA, Folkman S, Syme SL. 1993. Socioeconomic inequalities in health: no easy solution. *J. Am. Med. Assoc.* 269:3140–45

Adler NE, Matthews KA. 1994. Health and psychology: Why do some people get sick and some stay well? *Annu. Rev. Psychol.* 45:229–59

Albright CL, Winkleby MA, Ragland DR, Fisher J, Syme SL. 1992. Job strain and prevalence of hypertension in a biracial population of urban bus drivers. *Am. J. Public Health* 82:984–89

Alterman T, Shekelle RB, Vernon SW, Burau KD. 1994. Decision latitude, psychologic demand, job strain, and coronary heart disease in the Western Electric study. *Am. J. Epidemiol.* 139:620–27

Anderson NB, McNeilly M, Myers H. 1991. Autonomic reactivity and hypertension in blacks: a review and proposed model. *Ethn. Dis.* 1:163–70

Aseltine RG, Kessler RC. 1993. Marital disruption and depression in a community sample. *J. Health Soc. Behav.* 34:237–51

Aseltine RH Jr, Gore S, Colten ME. 1994. Depression and the social developmental context of adolescence. *J. Pers. Soc. Psychol.* 67:252–63

Averill JR. 1982. *Anger and Agression: An Essay on Emotion.* New York: Springer-Verlag

Back KW, Bogdonoff MD. 1964. Plasma lipid responses to leadership, conformity, and deviation. In *Psychobiological Approaches to Social Behavior*, ed. HP Leiderman, D Shapiro, pp. 24–42. Stanford, CA: Stanford Univ. Press

Bailey SL, Hubbard RL. 1990. Developmental variation in the context of marijuana initiation among adolescents. *J. Health Soc. Behav.* 31:58–70

Barrera M, Chassin L, Rogosch F. 1993. Effects of social support and conflict on adolescent children of alcoholic and nonalcoholic fathers. *J. Pers. Soc. Psychol.* 64: 602–12

Barrera M, Li SA, Chassin L. 1995. Effects of parental alcoholism and life stress on Hispanic and non-Hispanic Caucasian adolescents: a prospective study. *Am. J. Commmunity Psychol.* 23:479–507

Becker MH. 1993. A medical sociologist looks at health promotion. *J. Health Soc. Behav.* 34:1–6

Berkman LF. 1995. The role of social relations in health promotion. *Psychosom. Med.* 57: 245–54

Biglan A, Duncan TE, Ary DV, Smolkowski K. 1995. Peer and parental influences on adolescent tobacco use. *J. Behav. Med.* 18:315

Bloom BL, Asher SJ, White SW. 1978. Marital disruption as a stressor: a review and analysis. *Psychol. Bull.* 85:867–94

Bolger N, Foster M, Vinokur AD, Ng R. 1997. Close relationships and adjustment to a life crisis: the case of breast cancer. *J. Pers. Soc. Psychol.* In press

Booth-Kewley S, Friedman HS. 1987. Psychological predictors of heart disease: a quantitative review. *Psychol. Bull.* 101:343–62

Bovbjerg VE, McCann BS, Brief DJ, Follette WC, Retzlaff BM, et al. 1995. Spouse support and long-term adherence to lipid-lowering diets. *Am. J. Epidemiol.* 141:451–60

Boyce WT, Chesney M, Alkon A, Tschann JM, Adams S, et al. 1995. Psychobiologic reactivity to stress and childhood respiratory illness: results of two prospective studies. *Psychosom. Med.* 57:411–22

Braaten LS. 1996. Sexual dysfunction in sexually traumatized women. *Health Psychol.* 18:6

Bradley RH. 1993. Children's home environments, health, behavior, and intervention efforts: a review using the home inventory as a marker measure. *Genet. Soc. Gen. Psychol. Monogr.* 119:437–90

Broadhead EW, Kaplan BH, James SA, Wagner EH, Schoenbach VJ, et al. 1983. The epidemiologic evidence for a relationship between social support and health. *Am. J. Epidemiol.* 117:521–37

Broman C. 1992. Social relationships and health-related behavior. *J. Behav. Med.* 16: 335–50

Brown GW, Harris TO, eds. 1989. *Life Events and Illness.* New York: Guilford

Brown PC, Smith TW. 1992. Social influence, marriage, and the heart: cardiovascular consequences of interpersonal control in husbands and wives. *Health Psychol.* 11: 88–96

Burg MM, Seeman TE. 1994. Families and Health: the negative side of social ties. *Ann. Behav. Med.* 16:109–15

Buunk BP, Doosje BJ, Jans LGJM, Hopstaken LEM. 1993. Perceived reciprocity, social support, and stress at work: the role of exchange and communal orientation. *J. Pers. Soc. Psychol.* 65:801–11

Calnan M, Johnson B. 1985. Health, health risks, and inequalities: an exploratory study of women's perceptions. *Sociol. Health Ill.* 7:55–75

Caplan RD, Jones KW. 1975. Effects of work load, role ambiguity, and Type A personality on anxiety, depression, and heart rate. *J. Appl. Psychol.* 60:713–19

Catalano R, Dooley D, Wilson G, Hough R. 1993. Job loss and alcohol abuse: a test using data from the epidemiological catchment area. *J. Health Soc. Behav.* 34: 215–25

Catalano RA, Rook K, Dooley D. 1986. Labor markets and help-seeking: a test of the employment security hypothesis. *J. Health Soc. Behav.* 27:277–87

Christensen AJ, Smith TW, Turner CW, Holman JM Jr, Gregory MC, Rich MA. 1992. Family support, physical impairment, and adherence in hemodialysis: an investigation of main and buffering effects. *J. Behav. Med.* 15:313–25

Cobb S. 1976. Social support as a moderator of life stress. *Psychosom. Med.* 38:300–14

Cohen S. 1992. Stress, social support, and disorder. In *The Meaning and Measurement of Social Support,* ed. HOF Veiel, U Baumann, pp. 109–24. New York: Hemisphere

Cohen S, Herbert TB. 1996. Health psychology: psychological factors and physical disease from the perspective of human psychoneuroimmunology. *Annu. Rev. Psychol.* 47:113–42

Cohen S, Sherrod DR, Clark MS. 1986. Social skills and the stress-protective role of social support. *J. Pers. Soc. Psychol.* 50:963–73

Coleman JS. 1990. *Foundations of Social Theory.* Cambridge, MA: Harvard Univ. Press

Collins NL, Dunkel-Schetter C, Lobel M, Scrimshaw SCM. 1993. Social support in pregnancy: psychosocial correlates of birth outcomes and postpartum depression. *J. Pers. Soc. Psychol.* 65:1243–58

Cooper CJ, Marshall J. 1976. Occupational sources of stress: a review of the literature relating to coronary heart disease and mental ill health. *J. Occup. Psychol.* 49:11–28

Coyne JC, Fiske V. 1992. Couples coping with chronic and catastrophic illness. In *Family Health Psychology,* ed. TJ Akamatsu, SC Crowther, SE Hobfoll, MAP Stevens, pp. 129–49. Washington, DC: Hemisphere

Crittenden PM. 1992. Children's strategies for coping with adverse home environments: an interpretation using attachment theory. *Child Abuse Negl.* 16:329–43

Cummings EM, El-Sheikh M. 1991. Children's coping with angry environments: a process-oriented approach. In *Life-Span Developmental Psychology: Perspectives on Stress and Coping,* ed. EM Cummings, AL Greene, KH Karraker, pp. 131–50. Hillsdale, NJ: Erlbaum

Cumsille PE, Epstein N. 1994. Family cohesion, family adaptability, social support, and adolescent depressive symptoms in outpatient clinic families. *J. Fam. Psychol.* 8:202–14

Cunningham RM, Stiffman AR, Dore P, Earls F. 1994. The association of physical and sexual abuse with HIV risk behaviors in adolescence and young adulthood: implications for public health. *Child Abuse Negl.* 18:233–45

D'Angelo LL, Weinberger DA, Feldman SS. 1995. Like father, like son? Predicting male adolescents' adjustment from parents' distress and self-restraint. *Dev. Psychol.* 31:883–96

Davies PT, Cummings EM. 1994. Marital conflict and child adjustment: an emotional security hypothesis. *Psychol. Bull.* 116: 387–411

Davis MC, Matthews KA, Meilahn EN, Kiss JE. 1995. Are job characteristics related to fibrinogen levels in middle-aged women? *Health Psychol.* 14:310–18

Denton RE, Kampfe CM. 1994. The relationship between family variables and adolescent substance abuse: a literature review. *Adolescence* 29:475–95

Dewe PJ, Guest DE. 1990. Methods of coping with stress at work: a conceptual analysis and empirical study of measurement issues. *J. Organ. Behav.* 11:135–50

Dolcini MM, Adler NE. 1994. Perceived competencies, peer group affiliation, and risk behavior among early adolescents. *Health Psychol.* 13:496–506

Donaldson SI. 1995. Peer influence on adolescent drug use: a perspective from the trenches of experimental evaluation research. *Am. Psychol.* 50:801–2

Donovan JE, Jessor R, Costa FM. 1991. Adolescent health behavior and conventionality-unconventionality: an extension of problem-behavior theory. *Health Psychol.* 10:52–61

Downey G, Coyne JC. 1990. Children of depressed parents: an integrative review. *Psychol. Bull.* 108:50–76

Dressler WW, Dos Santos EJ, Viteri FE. 1986. Blood pressure, ethnicity, and psychosocial resources. *Psychosom. Med.* 48:509–19

Dubow EF, Tisak J. 1989. The relation between stressful life events and adjustment in elementary school children: the role of social support and social problem-solving skills. *Child Dev.* 60:1412–23

Durkin MS, Davidson LL, Kuhn L, O'Connor P, Barlow B. 1994. Low-income neighborhoods and the risk of severe pediatric injury: a small-area analysis in northern Manhattan. *Am. J. Public Health* 84: 587–92

Eckenrode J, Rowe E, Laird M, Brathwaite J. 1995. Mobility as a mediator of the effects of child maltreatment of academic performance. *Child Dev.* 66:1130–42

El-Sheikh M, Cummings EM, Goetsch V. 1989. Coping with adults' angry behavior: behavioral, physiological, and self-report responding in preschoolers. *Dev. Psychol.* 25:490–98

Evans GW. 1997. Environmental stress and health. In *Handbook of Health Psychology*, ed. A Baum, T Revenson, JE Singer. Hillsdale, NJ: Erlbaum

Farrell MP, Barnes GM, Banerjee S. 1995. Family cohesion as a buffer against the effects of problem-drinking fathers on psychological distress, deviant behavior, and heavy drinking in adolescents. *J. Health Soc. Behav.* 36:377–85

Fleming I, Baum A, Davidson LM, Rectanus E, McArdle S. 1987. Chronic stress as a factor in physiologic reactivity to challenge. *Health Psychol.* 6:221–37

Frankenhaeuser M. 1991. The psychophysiology of workload, stress, and health: comparison between the sexes. *Ann. Behav. Med.* 13:197–204

Frasure-Smith N, Lesperance F, Talajic M. 1995. The impact of negative emotions on prognosis following myocardial infarction: is it more than depression? *Health Psychol.* 14:388–98

French JRP Jr, Caplan RD. 1973. Organizational stress and the individual strain. In *The Failure of Success*, ed. AJ Marrow. New York: AMACON

Ganster DC, Fusilier MR, Mayes BT. 1986. Role of social support in the experience of stress at work. *J. Appl. Psychol.* 71:102–10

Garbarino J, Sherman D. 1980. High-risk neighborhoods and high-risk families: the human ecology of child maltreatment. *Child Dev.* 51:188–98

Geertsen R, Klauber MR, Rindflesh M, Kane R, Gray R. 1975. A re-examination of Suchman's view on social factors in health care utilization. *J. Health Soc. Behav.* 16: 226–37

George LK. 1989. Stress, social support, and depression over the life-course. In *Aging, Stress, Social Support, and Health*, ed. K Markides, C Cooper, pp. 241–67. London: Wiley

Gerin W, Pieper C, Levy R, Pickering TG. 1992. Social support in social interaction: a moderator of cardiovascular reactivity. *Psychosom. Med.* 54:324–36

Gore S, Aseltine RH Jr. 1995. Protective processes in adolescence: matching stressors with social resources. *Am. J. Community Psychol.* 23:301–27

Gore S, Aseltine RH Jr, Colten ME. 1992. Social structure, life stress and depressive symptoms in a high school-aged population. *J. Health Soc. Behav.* 33:97–113

Gottman JM, Katz LF. 1989. Effects of marital discord on young children's peer interaction and health. *Dev. Psychol.* 25:373–81

Green KL, Johnson JV. 1990. The effects of psychosocial work organization on patterns of cigarette smoking among male chemical plant employees. *Am. J. Public Health* 80: 1368–71

Greenberg ES, Grunberg L. 1995. Work alienation and problem alcohol behavior. *J. Health Soc. Behav.* 36:83–102

Gunnar MR, Larson MC, Hertsgaard L, Harris ML, Brodersen L. 1992. The stressfulness of separation among nine-month-old infants: effects of social context variables and infant temperament. *Child Dev.* 63: 290–303

Gutierres SE, Saenz DS, Green BL. 1996. Job stress and health outcomes among Anglo and Hispanic employees: a test of the person-environment fit model. In *Stress in the 90's*, ed. G Keita, S Sauter. Washington, DC: Am. Psychol. Assoc. In press

Haidt J, Rodin J. 1995. *Control and Efficacy: An Integrative Review*. Rep. John D and Catherine T MacArthur Found. Prog. Ment. Health Hum. Dev. Univ. Michigan, Ann Arbor

Hamilton VL, Broman CL, Hoffman WS, Renner DS. 1990. Hard times and vulnerable people: initial effects of plant closing on autoworkers' mental health. *J. Health Soc. Behav.* 31:123–40

Harburg E, Erfurt JC, Havenstein LS, Chape S, Schull WJ, Schork MA. 1993. Socio-ecological stress, suppressed hostility, skin color, and black-white male blood pressure: Detroit. *Psychosom. Med.* 35:176–96

Hardy DF, Power TG, Jaedicke S. 1973. Examining the relation of parenting to children's coping with everyday stress. *Child Dev.* 64:1829–41

Herbert TB, Cohen S. 1993. Depression and immunity: a meta-analytic review. *Psychol. Bull.* 113:472–86

Hertsgaard L, Gunnar M, Erickson FM, Nachmias M. 1995. Adrenocortical responses to the strange situation in infants with disorganized/disoriented attachment relationships. *Child Dev.* 66:1100–6

Herzog AR, House JS, Morgan JN. 1991. Relation of work and retirement to health and well-being in older age. *Psychol. Aging* 6: 202–11

Hirsch BJ, DuBois DL. 1992. The relation of peer social support and psychological symptomatology during the transition to junior high school: a two-year longitudinal analysis. *Am. J. Community Psychol.* 20:333–47

House JS. 1981. *Work Stress and Social Support*. Reading, MA: Addison-Wesley

House JS, Landis KR, Umberson D. 1988a. Social relationships and health. *Science* 241:540–45

House JS, Smith DA. 1985. Evaluating the health effects of demanding work on and off the job. In *Assessing Physical Fitness and Physical Activity in Population-Base Surveys*, ed. TF Drury, pp. 481–508. Hyattsville, MD: Natl. Cent. Health Stat.

House JS, Strecher V, Meltzner HL, Robbins CA. 1986. Occupational stress and health among men and women in the Tecumseh Community health study. *J. Health Soc. Behav.* 27:62–77

House JS, Umberson D, Landis KR. 1988b. Structures and processes of social support. *Annu. Rev. Sociol* 14:293–318

Huston AC, McLoyd VC, Coll CG. 1994. Children and poverty: issues in contemporary research. *Child Dev.* 65:275–82

Jemmott LS, Jemmott JB III. 1992. Family structure, parental strictness, and sexual behavior among inner-city black male adolescents. *J. Adolesc. Res* 7:192–207

Jencks C, Mayer S. 1990. The social consequences of growing up in a poor neighborhood. In *Inner-City Poverty in the United States*, ed. L Lynn, M McGeary, pp. 111–86. Washington, DC: Natl. Acad.

Joiner TE Jr. 1994. Contagious depression: existence, specificity to depressed symptoms, and the role of reassurance seeking. *J. Pers. Soc. Psychol.* 67:287–96

Kagan J, Reznick JS, Snidman N. 1987. The physiology and psychology of behavioral inhibition in young children. *Child Dev.* 58: 1359–473

Kagan J, Reznick JS, Snidman N. 1988. Biological bases of childhood shyness. *Science* 240:167–71

Kaplan M, Chadwick MW, Schimmel LE. 1985. Social learning intervention to promote metabolic control in Type I diabetes mellitus: pilot experiment results. *Diabetes Care* 8:152–55

Kaplan RM, Hartwell SL. 1987. Differential effects of social support and social network on physiological and social outcomes in men and women with Type II diabetes mellitus. *Health Psychol.* 6:387–98

Karasek R, Theorell T. 1990. *Healthy Work: Stress, Productivity, and the Reconstruction of Working Life*. New York: Basic Books

Kaslow NJ, Deering CG, Racusin GR. 1994. Depressed children and their families. *Clin. Psychol. Rev.* 14:39–59

Kendler KS, Kessler RC, Heath AC, Neale MC, Eaves LJ. 1991. Coping: a genetic epidemiological investigation. *Psychol. Med.* 21:337–46

Kessler RC, MacLeod JD, Wethington E. 1985. The costs of caring: a perspective on the relationship between sex and psychological distress. In *Social Support: Theory, Research and Applications*, ed. IG Sarason, BR Sarason, pp. 491–506. Dordrecht: Martinus Nijhoff

Kessler RC, Magee WJ. 1994. Childhood family violence and adult recurrent depression. *J. Health Soc. Behav.* 35:13–27

Kiecolt-Glaser JK, Malarkey WB, Cacioppo JT, Glaser R. 1994. Stressful personal rela-

tionships: immune and endocrine function. In *Handbook of Human Stress and Immunity*, ed. R Glaser, J Kiecolt-Glaser, pp. 321–39. San Diego: Academic

Kotler P, Wingard DL. 1989. The effect of occupational, marital, and parental roles on mortality: the Alameda County study. *Am. J. Public Health* 79:607–12

Krantz DS, Manuck SB. 1984. Acute psychophysiologic reactivity and risk of cardiovascular disease: a review and methodologic critique. *Psychol. Bull.* 96:435–64

Krantz DS, Manuck SB, Wing RR. 1986. Psychological stressors and task variables on elicitors of reactivity. In *Handbook of Stress, Reactivity, and Cardiovascular Disease*, ed. KA Matthews, SM Weiss, T Detre, TM Dembroski, B Falkner, et al, pp. 85–107. New York: Wiley

Krause N. 1992. Stress and isolation form close ties in later life. *J. Gerontol.* 46: S183–94

Kupersmidt JB, Griesler PC, DeRosier ME, Patterson CJ, Davis PW. 1995. Childhood aggression and peer relations in the context of family and neighborhood factors. *Child Dev.* 66:360–75

Kupersmidt JB, Patterson CJ. 1991. Childhood peer rejection, aggression, withdrawal, and perceived competence as predictors of self-reported behavior problems in preadolescence. *J. Abnorm. Child Psychol.* 19: 427–49

Landsbergis PA, Schnall PL, Deitz D, Friedman R, Pickering T. 1992. The patterning of psychological attributes and distress by "job strain" and social support in a sample of working men. *J. Behav. Med.* 15: 379–414

Larson R, Richards MH. 1991. Daily companionship in late childhood and early adolescence: changing developmental contexts. *Child Dev.* 62:284–300

Lefler A, Krannich RS, Gillespie DL. 1986. Contact, support and friction: three faces of networks in community life. *Sociol. Perspect.* 29:337–55

Lennon MC, Rosenfeld S. 1992. Women and mental health: the interaction of job and family conditions. *J. Health Soc. Behav.* 33:316–27

Lerner DJ, Levine S, Malspeis S, D'Agostino RB. 1994. Job strain and health-related quality of life in a national sample. *Am. J. Public Health* 84:1580–85

Levy L, Herzog A. 1974. Effects of population density and crowding on health and social adaptation in The Netherlands. *J. Health Soc. Behav.* 15:228–40

Levy L, Herzog A. 1978. Effects of crowding on health and social adaptation in the city of Chicago. *Hum. Ecol.* 3:327–54

Lewinsohn PM, Roberts RE, Seeley JR, Rohde P, Gotlib IH, Hops H. 1994. Adolescent psychopathy. II. Psychosocial risk factors for depression. *J. Abnorm. Psychol.* 103: 302–15

Loscocco KA, Spitze G. 1990. Working conditions, social support, and the well-being of female and male factory workers. *J. Health Soc. Behav.* 31:313–27

Macintyre S. 1997. The black report and beyond: What are the issues? *Soc. Sci. Med.* In press

Macintyre S, Maciver S, Solomon A. 1993. Area, class and health: Should we be focusing on places or people? *Int. Soc. Policy* 22:213–34

Malinosky-Rummell R, Hansen DJ. 1993. Long-term consequences of childhood physical abuse. *Psychol. Bull.* 114:68–79

Manuck SB, Marsland AL, Kaplan JR, Williams JK. 1995. The pathogenicity of behavior and its neuroendocrine mediation: an example from coronary artery disease. *Psychosom. Med.* 57:275–83

Marmot MG. 1994. Social differentials in health within and between populations. *Health Wealth: J. Am. Acad. Arts Sci.* 123: 197–216

Martin LR, Friedman HS, Tucker JS, Schwartz JE, Criqui MH, et al. 1995. An archival prospective study of mental health and longevity. *Health Psychol.* 14:381–87

Matthews KA, Woodall KL, Jacob T, Kenyon K. 1996. Negative family environment as a predictor of boys' future status on measures of hostile attitudes, interview behavior, and anger expression. *Health Psychol.* 15:30–37

McEwen BS, Stellar E. 1993. Stress and the individual: mechanisms leading to disease. *Arch. Intern. Med.* 153:2093–101

McNeil GN. 1987. Depression. In *Handbook of Psychiatric Differential Diagnosis*, ed. SM Soreff, GN McNeil, pp. 57–126. Littleton, MA: PSG

Mechanic D, Hansell S. 1989. Divorce, family conflict, and adolescents' well-being. *J. Health Soc. Behav.* 30:105–16

Melamed BG, ed. 1995. Special section: the interface of mental and physical health. *Health Psychol.* 14:371–426

Mercy JA, Saltzman LE. 1989. Fatal violence among spouses in the United States 1976-85. *Am. J. Public Health* 79:595–99

Metzler CW, Noell J, Biglan A, Ary D, Smolkowski K. 1994. The social context for risky sexual behavior among adolescents. *J. Behav. Med.* 17:419–38

Moritz DJ, Kasl SV, Berkman LF. 1989. The health impact of living with a cognitively impaired elderly spouse: depressive symptoms and social functioning. *J. Gerontol: Soc. Sci.* 44:S17–27

Mounts NS, Steinberg L. 1995. An ecological

analysis of peer influence on adolescent grade point average and drug use. *Dev. Psychol.* 31:915–22
Muntaner C, Anthony JC, Crum RM, Eaton WW. 1995. Psychosocial dimensions of work and the risk of drug dependence among adults. *Am. J. Epidemiol.* 142:183–90
Neuspiel DR, Rush D, Butler NR, Golding J, Buur PE, Kurzon M. 1989. Parental smoking and post-infancy wheezing in children: a prospective cohort study. *Am. J. Public Health* 79:168–71
Nolen-Hoeksema S, Wolfson A, Mumme D, Guskin K. 1995. Helplessness in children of depressed and nondepressed mothers. *Dev. Psychol.* 31:377–87
Osofsky JD. 1990. *Gender issues in the development of deviant behavior: the case for teenage pregnancy*. Presented at the Prog. Hum. Dev. Crim. Behav., Radcliffe Coll.
Osofsky JD. 1993. Applied psychoanalysis: how research with infants and adolescents at high psychosocial risk informs psychoanalysis. *J. Am. Acad. Psychoanal.* 41: 193–207
Osofsky JD. 1995. The effects of exposure to violence on young children. *Am. Psychol.* 50:782–88
Patterson GR, DeBaryshe BD, Ramsey E. 1989. A developmental perspective on antisocial behavior. *Am. Psychol.* 44:329–35
Pavalko EK, Elder GH Jr, Clipp EC. 1993. Worklives and longevity: insights from a life course perspective. *J. Health Soc. Behav.* 34:363–80
Penning MJ. 1995. Health, social support, and the utilization of health services among older adults. *J. Gerontol.* 50B:S330–39
Peoples-Sheps MD, Siegel E, Suchindran CM, Origasa H, Ware A, Barakat A. 1991. Characteristics of maternal employment during pregnancy: effects on low birthweight. *Am. J. Public Health* 81:1007–12
Pieper C, LaCroix AZ, Karasek RA. 1989. The relation of psychosocial dimensions of work with coronary heart disease risk factors: a meta-analysis of five United States data bases. *Am. J. Epidemiol.* 129:483–94
Pynoos RS. 1993. Traumatic stress and developmental psychopathology in children and adolescents. In *American Psychiatric Press Review of Psychiatry*, ed. JM Oldham, MB Riba, A Tasman, Vol. 12. Washington, DC: Am. Psychiatric Assoc.
Repetti RL. 1993. The effects of workload and the social environment at work on health. In *Handbook of Stress*, ed. L Goldberger, S Bresnitz, pp. 368–85. New York: Free Press
Repetti RL, Matthews KA, Waldrun I. 1989. Employment and women's health. *Am. Psychol.* 44:1394–401
Repetti RL, McGrath EP, Ishikawa SS. 1996. Daily stress and coping in childhood and adolescence. In *Handbook of Pediatric and Adolescent Health Psychology*, ed. AJ Goreczny, M Hersen. Allyn & Bacon. In press
Richters JE, Martinez P. 1993. The NIMH community violence project: Vol. 1: children as victims of and witnessses to violence. *Psychiatry* 56:7–21
Rosenfield S. 1992. The costs of sharing: wives' employment and husbands' mental health. *J. Health Soc. Behav.* 33:213–25
Ross CE, Mirowsky J. 1995. Does employment affect health? *J. Health Soc. Behav.* 36:230–43
Rowe DC, Linver MR. 1995. Smoking and addictive behaviors: epidemiological, individual, and family factors. In *Behavior Genetic Approaches in Behavioral Medicine*, ed. JR Turner, LR Cardon, JK Hewitt, pp. 67–84. New York: Plenum
Rushing B, Ritter C, Burton RPD. 1992. Race differences in the effects of multiple roles on health: longitudinal evidence from a national sample of older men. *J. Health Soc. Behav.* 33:126–39
Ruchlin HS, Morris JN. 1991. Impact of work on the quality of life in community-residing young elderly. *Am. J. Public Health* 81: 498–500
Sallis JF, Hovell MF, Hofstetter CR, Faucher P, Elder JP, et al. 1989. A multivariate study of determinants of vigorous exercise in a community sample. *Prev. Med.* 18:20–34
Sampson RJ. 1992. Family management and child development: insights from social disorganization theory. In *Facts, Frameworks, and Forecasts: Advances in Criminological Theory*, ed. J McCord, 3:63–93. New Brunswick, NJ: Transaction
Sandler DP, Helsing KJ, Comstock GW, Shore DL. 1989a. Factors associated with past household exposure to tobacco smoke. *Am. J. Epidemiol.* 129:380–87
Sandler IN, Miller P, Short J, Wolchik SA. 1989b. Social support as a protective factor for children in stress. In *Children's Social Networks and Social Support*, ed. D Belle, pp. 277–304. New York: Wiley
Schuster TL, Kessler RC, Aseltine RH Jr. 1990. Supportive interactions, negative interactions, and depressed mood. *Am. J. Community Psychol.* 18:423–38
Seeman M, Seeman AZ, Budros A. 1988. Powerlessness, work, and community: a longitudinal study of alienation and alcohol use. *J. Health Soc. Behav.* 29:185–98
Seeman TE, Berkman LF, Blazer D, Rowe J. 1994. Social ties and support and neuroendocrine function: MacArthur Studies of Succesful Aging. *Ann. Behav. Med.* 16:95–106

Seeman TE, Bruce ML, McAvay G. 1996. Social network characteristics and onset of ADL disability. *J. Gerontol.: Soc. Sci.* In press

Seeman TE, McEwen BS. 1996. Social environment characteristics and neuroendocrine function: the impact of social ties and support on neuroendocrine regulation. *Psychosom. Med.* In press

Selye H. 1956. *The Stress of Life.* New York: McGraw-Hill

Shedler J, Block J. 1990. Adolescent drug use and psychological health: a longitudinal inquiry. *Am. Psychol.* 45:612–30

Sheline JL, Skipper BJ, Broadhead WE. 1994. Risk factors for violent behavior in elementary school boys: Have you hugged your child today? *Am. J. Public Health* 84: 661–63

Siegler IC, Peterson BL, Barefoot JC, Williams RB. 1992. Hostility during late adolescence predicts coronary risk factors at mid-life. *Am. J. Epidemiol.* 136:146–54

Snydersmith MA, Cacioppo JT. 1992. Parsing complex social factors to determine component effects. I. Autonomic activity and reactivity as a function of human association. *J. Soc. Clin. Psychol.* 11:263–78

Sooman A, Macintyre S. 1995. Health and perceptions of the local environment in socially contrasting neighborhoods in Glasgow. *J. Health Place.* 1:15–26

Sorensen G, Pirie P, Folsom A, Luepker R, Jacobs D, Gillum R. 1985. Sex differences in the relationship between work and health: the Minnesota heart survey. *J. Health Soc. Behav.* 26:379–94

Sorenson SB, Peterson JG. 1994. Traumatic child death and documented maltreatment history, Los Angeles. *Am. J. Public Health* 84:623–27

Sorlie PD, Rogot E. 1990. Mortality by employment status in the national longitudinal mortality study. *Am. J. Epidemiol.* 132: 983–92

Springs FE, Friedrich WN. 1992. Health risk behaviors and medical sequelae of childhood sexual abuse. *Mayo Clin.* 67:527– 32

Stice E, Barrera M Jr. 1995. A longitudinal examination of the reciprocal relations between perceived parenting and adolescents' substance use and externalizing behaviors. *Dev. Psychol.* 31:322–34

Taylor SE. 1995. *Health Psychology.* New York: McGraw-Hill. 3rd ed.

Thomas PD, Goodwin JM, Goodwin JS. 1985. Effect of social support on stress-related changes in cholesterol level, uric acid level, and immune function in an elderly sample. *Am. J. Psychiatry* 142:735–37

Thompson SC, Sobolew-Shubin A. 1993. Perceptions of overprotection in ill adults. *J. Appl. Soc. Psychol.* 23:85–97

Troutt DD. 1993. *The Thin Red Line: How the Poor Still Pay More.* San Francisco: Consum. Union US West Coast Reg. Off.

Troxler RG, Sprague EA, Albanese RA, Fuchs R, Thompson AJ. 1977. The association of elevated plasma cortisol and early atherosclerosis as demonstrated by coronary angiography. *Atheroscleroses* 26:151–62

Turner JB, Kessler RC, House JS. 1991. Factors facilitating adjustment to unemployment: implications for intervention. *Am. J. Community Psychol.* 19:521–42

Turner RA, Irwin CE Jr, Tschann JM, Millstein SG. 1993. Autonomy, relatedness, and the initiation of health risk behaviors in early adolescence. *Health Psychol.* 12: 200–8

Turner RJ, Noh S. 1983. Class and psychological vulnerability among women: the significance of social support and personal control. *J. Health Soc. Behav.* 24:2–15

Unden AL, Orth-Gomer K, Elofsson S. 1991. Cardiovascular effects of social support in the work place: twenty-four-hour ECG monitoring of men and women. *Psychosom. Med.* 53:50–60

Wagener DK, Schatzkin A. 1994. Temporal trends in the socioeconomic gradient for breast cancer mortality among US women. *Am. J. Public Health* 84:1003–6

Wallace R. 1990. Urban desertification, public health and public order: "planned shrinkage," violent death, substance abuse and AIDS in the Bronx. *Soc. Sci. Med.* 31:801–13

Wallace R, Wallace D. 1990. Origins of public health collapse in New York City: the dynamics of planned shrinkage, contagious urban decay and social disintegration. *Bull. NY Acad. Med.* 66:391–434

Walter HJ, Vaughan RD, Gladis MM, Ragin DF, Kasen S, Cohall ST. 1992. Factors associated with AIDS risk behaviors among high school students in an AIDS epicenter. *Am. J. Public Health* 82:528–32

Webb JA, Baer PE, McLaughlin RJ, McKelvey RS, Caid CD. 1991. Risk factors and their relation to initiation of alcohol use among early adolescents. *J. Am. Acad. Child Adolesc. Psychiatry* 30: 563–68

Wechsler H, Dowdall GW, Davenport A, Castillo S. 1995. Correlates of college student binge drinking. *Am. J. Public Health* 85:921–26

Wethington E, McLeod JD, Kessler RC. 1987. The importance of life events in explaining sex differences in psychological distress. In *Gender and Stress*, ed. RC Barnett, LB Biener, GK Baruch, pp. 144–56. New York: Basic Books

Wickrama K, Conger RD, Lorenz FO. 1995.

Work, marriage, lifestyle, and changes in men's physical health. *J. Behav. Med.* 18: 97–112
Widom C. 1989. The cycle of violence. *Science* 244:160–66
Williams DR. 1990. Socioeconomic differentials in health: a review and redirection. *Soc. Psychol. Q.* 53:81–99
Williams DR, Collins C. 1995. US socioeconomic and racial differences in health: patterns and explanations. *Annu. Rev. Sociol.* 21:349–86
Williams KJ, Suls J, Alliger GM, Learner SM, Wan CK. 1991. Multiple role juggling and daily mood states in working mothers: an experience sampling study. *J. Appl. Psychol.* 76:664–74
Wills TA, DuHamel K, Vaccaro D. 1995a. Activity and mood temperament as predictors of adolescent substance use: test of a self-regulation mediational model. *J. Pers. Soc. Psychol.* 68:901–16
Wills TA, McNamara G, Vaccaro D. 1995b. Parental education related to adolescent stress-coping and substance use: development of a mediational model. *Health Psychol.* 14:464–78
Wills TA, Schreibman D, Benson G, Vaccaro D. 1994. Impact of parental substance use on adolescents: a test of a mediation model. *J. Pediatr. Psychol.* 19:537–56
Woo G. 1994. *Strain in daily activities during pregnancy: associations of physical exertion, psychological demand, and personal control with birth outcomes.* PhD thesis. Univ. Calif., Los Angeles
Wyke S, Campbell G, MacIver S. 1992. Comparison of the provision of, and patient satisfaction with, primary care services in a relatively affluent and a relatively deprived area of Glasgow. *Br. J. Gen. Pract.* 42: 271–75
Zahn-Waxler C, Cummings EM, McKnew DH, Radke-Yarrow M. 1984. Altruism, aggression, and social interactions in young children with a manic-depressive parent. *Child Dev.* 55:112–22
Zapata BC, Rebolledo C, Atalah E, Newman B, King M-C. 1992. The influence of social and political violence on the risk of pregnancy complications. *Am. J. Public Health* 82:685–90