Advances in the Conceptualization of the Stress Process
Advances in the Conceptualization of the Stress Process:
Essays in Honor of Leonard I. Pearlin

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Springer
Preface

In 1981, Leonard Pearlin and his colleagues published an article that would rad-
ically shift the sociological study of mental health from an emphasis on psychiatric
disorder to a focus on social structure and its consequences for stress and psycho-
logical distress. Pearlin et al. (1981) proposed a deceptively simple conceptual
model that has now influenced sociological inquiry for almost three decades. With
his characteristic penchant for reconsidering and elaborating his own ideas, Pearlin
has revisited the stress process model periodically over the years (Pearlin 1989,
1999; Pearlin et al. 2005; Pearlin and Skaff 1996). One of the consequences of this
continued theoretical elaboration of the stress process has been the development of
a sociological model of stress that embraces the complexity of social life. Another
consequence is that the stress process has continued to stimulate a host of empirical
investigations in the sociology of mental health. Indeed, it is no exaggeration to
suggest that the stress process paradigm has been primarily responsible for the
growth and sustenance of sociological research on stress and mental health.

Pearlin et al. (1981) described the core elements of the stress process in a brief
paragraph:

The process of social stress can be seen as combining three major conceptual domains: the
sources of stress, the mediators of stress, and the manifestations of stress. Each of these
extended domains subsumes a variety of subparts that have been intensively studied in
recent years. Thus, in the search for sources of stress, considerable interest has been
directed to life events and to chronic life strains, especially the former; in work concerned
with conditions capable of mediating the impact of stressful circumstances, coping and
social supports have had a rather dramatic rise to prominence; and as for stress and its
symptomatic manifestations, the expanding volume of research ranges from the microbi-
ological substrates of stress to its overt emotional and behavioral expressions (p. 337).

With these three sentences, a paradigm was launched – one that has emerged as
the dominant perspective in the sociology of stress and mental health.
This initial specification of the stress process is now widely known. Pearlin and
his colleagues described how stressful life events and more chronic life strains
diminish individuals’ self concepts and their sense of mastery. They also argued
that two types of psychosocial resources, social support and coping, play important
roles in protecting individuals from the consequences of their stressful experiences.
Pearlin et al. (1981) made the important assertion that “there are several junctures at which the mediators can conceivably intervene: prior to an event, between an event and the life strains that it stimulates, between the strain and the diminishment of the self-concept, or prior to the stress outcome” (p. 341). Thus, at this early stage in the development of the stress process paradigm, the complexity of a seemingly simple model was apparent. Although some of the constructs and dynamics of the stress process had been introduced earlier by Pearlin (Pearlin 1980, 1983; Pearlin and Lieberman 1979; Pearlin and Schooler 1978), the synthesis of these ideas into a model and the presentation of an empirical test of that formulation clearly catapulted the paradigm into the forefront of thinking and research in medical sociology and the sociology of mental health.

In a subsequent article, Pearlin (1989) more explicitly discussed the central importance of the social context in which the stress process operates. In so doing, he highlighted the distinctive sociological perspective that the stress process brings to the study of stress and its manifestations. He also elaborated on the interplay among stressful life events and chronic strains and continued to explore the locations in the stress process where mediators could be expected to exert their influence. In this paper, Pearlin clearly establishes the sociological character of the stress process.

Pearlin’s (1999) contribution to the Handbook of the Sociology of Mental Health (Aneshensel and Phelan 1999) provides a comprehensive reflection on the stress process paradigm approximately two decades after its creation. In this chapter, Pearlin identifies three key assumptions that underlie the model. First, the stress process is dynamic in nature: changes in one set of factors produce changes in others. Second, Pearlin argued persuasively that social stress is by no means unusual or abnormal; indeed, it is typical of ordinary life. Stress arises out of commonly-held social roles of everyday life and in typical social contexts. Third, the origins of stress are in the social world. This directs the sociological study of stress to a greater emphasis on social context than on history or biology.

Pearlin then systematically reviews the major components of the stress process. He reiterates the importance of social and economic statuses as crucial structures that influence human experience. He draws attention to the importance of the neighborhood context as a kind of crucible in which life experiences occur. He further elaborates the domain of stressors by noting that other dimensions of stress require consideration within the paradigm and he articulates the concept of stress proliferation (having earlier provided an empirical demonstration of this process in Pearlin et al. 1997). In this chapter, he also clarifies the conceptual distinction between resources as mediators and resources as moderators of the stress–distress relationship. He concludes with a succinct justification of the advantages of examining psychological distress as the primary outcome in stress process research.

These three major statements in 1981, 1989, and 1999, together with Pearlin’s program of empirical research, provided sociologists with a well-articulated model that was soon applied to a variety of issues. His emphasis on the social context in which the stress process unfolds became one of the dominant perspectives for understanding the social patterning of mental health and illness. His careful consideration of the many sources of stressors in people’s lives and the variations in the
availability of mediating and moderating resources provided sociologists with a rich source of ideas for empirical investigation. Leonard Pearlin’s work has been particularly noteworthy in the ways that it has fostered innovation in the study of social roles, especially those related to the family and work. His ideas have also stimulated studies of the social structural determinants of psychosocial resources such as social support and mastery.

Remarkably, this was only the beginning. In a seminal paper, Pearlin et al. (1997) demonstrated how the stress process could be applied to the study of caregiving. In subsequent studies of people giving care to persons with HIV/AIDS (Pearlin et al. 1997; Turner et al. 1998) and caregivers to persons with Alzheimer’s disease or other dementias (Aneshensel et al. 1993, 1995; Pearlin 1992; Skaff and Pearlin 1992; Skaff et al. 1992), the utility of the stress process for understanding the stress of caregiving was documented empirically. This work not only introduced the stress process paradigm to social scientists interested in caregiving and family dynamics, but it also brought the paradigm to the attention of researchers in the health sciences and other disciplines concerned with family-based care. In short order, research based on the stress process paradigm increased exponentially.

The influence of this paradigm spread further as Leonard Pearlin began to explore the ways in which the stress process might be aligned with ideas from the life course perspective. Pearlin and Skaff (1996) suggested a number of ways in which principles central to the life course perspective could be integrated with key elements of the stress process to examine how individuals’ exposure to stressors. They suggested that as people move through the life course, individuals’ lives are restructured. As their statuses and roles change, so too do the stressors they encounter and the mediating resources to which they have access.

These ideas have been elaborated; Pearlin et al. (2005) specify elements of the stress process that may affect stress and health across the life course. These include the effects of economic strains and discriminatory experiences, stress proliferation, and the intersection of status attainment and stress exposure. This synthesis of the stress process with the life course has been stimulating to research in the sociology of mental health. Most recently, Turner and Schieman (2008) have assembled a wide-ranging set of papers that explore the interface of the stress process with the life course.

It is no exaggeration to assert that this vast body of research on stress and mental health is due in large part to the imagination of Leonard Pearlin. The richness of his theoretical ideas and his apparent comfort with investigating the complexities of social life have called a generation of sociological researchers to action. The work continues and a second generation has emerged to carry on this research. And there is little doubt that the generative nature of Len’s responses to the work of others has facilitated the continuing significance of the stress process.

In honor of Leonard Pearlin’s significant contributions to sociological theory and research, we invited some of his colleagues, collaborators, students, and friends to contribute essays that attest to Len’s influence on their work. We also encouraged these researchers to tell us what their future lines of inquiry might be and how Leonard Pearlin’s ideas have shaped these new directions.
Initially, the authors came together in Boston in August 2008, for a day of celebration with Len. The day began with a breakfast hosted by Jean Shin, Director of the American Sociological Association (ASA) Minority Affairs Program. Len Pearlin has been a long-time supporter of the Minority Fellowship Program. The breakfast provided new MFP Fellows with the opportunity to meet a number of sociologists with research interests in stress and health.

Sally Hillsman, Executive Officer of ASA presented Len with a plaque that acknowledged his contributions to the MFP program. She also noted that Len Pearlin has been a member of ASA for 58 years. We then presented our papers, shared memories with Len, and conclude with a celebratory dinner. We have included a picture of the entire group. The essays that appear in this book are all dedicated to Len Pearlin, colleague, mentor and friend.

We wish to acknowledge the American Sociological Association for providing meeting space for the one-day event and Jean Shin for hosting the MFP breakfast. We also wish to thank Teresa Krauss and Katie Chabalko at Springer for their support of this project. Special thanks to Kathleen Lynch for her assistance in the final editing process.

Finally, we wish to acknowledge the efforts of our colleagues in contributing to this book. Their cooperation has been stellar. Over many years, this group of stress researchers has met regularly at the American Sociological Association Annual Meetings where the Section on the Sociology of Mental Health has become a vibrant forum for the exchange of ideas. We will contribute our share of royalties from the sale of this book to the Section in recognition of its continued support of sociological research.

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References

Front Row (left to right): Melissa Milkie, Alex Bierman, Leonard Pearlin, Scott Schieman, Heather Turner
Second Row (left to right): Carol Aneshensel, Peggy Thoits, Leslie Caplan, Elizabeth Menaghan, Jay Turner, Joseph Mullan
Third Row (left to right): Elena Fazio, William Avison, Carmi Schooler, K. A. S. Wickrama, Blair Wheaton, Marilyn Skaff
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Part I

Conceptual and Methodological Developments
Introduction

Evidence revealing racial and socioeconomic disparities in health has long been available and continues to accumulate. Among those that are now well documented are Black-white inequities in overall health, all-cause mortality and life expectancy, low birth weight, infant mortality, reproductive health, hypertension and heart disease, as well as various psychiatric and substance use problems. Similar disparities are found across socioeconomic status (SES). Although race and SES are associated, prior research has documented substantial health disparities across SES within race and across race within SES (Geronimus et al. 1996; Williams 1999). This paper argues that progress in understanding the origins of such consequential health disparities can be materially enhanced by adopting the theoretical guidance embodied in the work of Leonard I. Pearlin. It is hypothesized that health disparities arise to a substantial degree from differences in lifetime exposure to social stress. For more than a quarter century, Pearlin’s stress process model has represented the dominant perspective of researchers attempting to identify potentially modifiable social contingencies in mental health. The high degree of the success of the model in accounting for variations in depressive symptoms and psychological distress suggests its potential power for advancing our understanding of racial and SES health disparities. These disparities have a massive impact in terms of unequal suffering and dramatic social and economic costs. It is thus no surprise that substantial research has accumulated aimed at identifying the origins of such disparities. It is clear that racial and SES differences in the availability, use, and effectiveness of medical care (e.g. Escarce et al. 1993; Ferguson et al. 1997; Fincher et al. 2004; Johnson et al. 1993; Klabunde et al. 1998; Peterson et al. 1997), and in the level of trust in health care institutions and physicians, are implicated (Doescher et al. 2000; Kao et al. 1998a, b; Saha et al. 2003; Thom and Campbell 1997), as are differences in a variety of health...
behaviors (Fraser et al. 1997; Healthy People 1990; McGinnis and Foege 1993). However, it is also clear that adjustments for these collective differences leave the majority of racial and SES health disparities unexplained (e.g. Lynch et al. 1996; Marmot et al. 1997; Lantz et al. 1998; Lantz et al. 2001). Available evidence points to the conclusion that potentially modifiable social factors play a fundamental role in racial and SES health disparities – a role that includes but goes substantially beyond their significance for such well established risk factors as poor nutrition, smoking, sedentary lifestyle, and obesity. However, no consensus has yet emerged about the identity or nature of these social factors or how they might be effectively addressed. It will be argued that this state of affairs arises from several significant deficiencies that have characterized most prior studies, including the failure within studies of physical health and general health outcomes to take advantage of the conceptual insights of Leonard I. Pearlin.

This paper proposes a strategy for more adequately evaluating the social origins of racial and SES health disparities by more fully addressing the stress hypothesis through utilization of an elaborated version of Pearlin’s stress process model. Multiple strands of evidence have been accumulating in support of the stress hypothesis (e.g. Adler et al. 1993; Lantz et al. 1998; Wilkinson 1996) and it appears to have emerged as a leading contender for the mechanism by which minority status and low social status are translated into relatively poor health (Dowd and Goldman 2006).

Despite the availability of a substantial array of evidence confirming the health significance of social stress, it is contended that the explanatory significance of stress with respect to health disparities has never been effectively tested for several reasons. This includes, most importantly, the crucial fact that the problem of misclassification in the disordered versus the well distinction has not been effectively addressed, and that differences in exposure to stressors have most often not been adequately estimated (Turner and Avison 2003; Turner et al. 1995). It will be suggested that dealing with these and other impediments to progress in the context of the stress process model has the potential to yield a significant forward leap toward identifying potentially modifiable factors associated with increased or decreased health risk within and also across race and socioeconomic status (SES). The specific model to be proposed, which is an elaboration of Pearlin’s model, is presented as Fig. 1.1. It will be argued that this model may substantially overcome the misclassification and stress measurement problems and that there is compelling evidence for most of the linkages shown.

**Background**

**The Problem of Misclassification**

As the scientific foundation of public health efforts, the goal of epidemiologic research has been to identify factors implicated in the causation of the particular disorder under investigation. However, both racial and SES health disparities
involve a substantial array of often overlapping physical and emotional disorders and problems. An assumption underlying much of what is argued here is that the multiplicity or generality of these disparities suggests that the major contributing factors (both risk and protective) may also be quite general in nature. Based on a review of extant animal and human studies, Cassel argued more than thirty years ago that the social environment acts to raise or lower susceptibility to all forms of distress and disorder in general and that the nature of the particular disorders that occur is determined on other grounds (Cassel 1974, 1976). Evidence accumulated over the intervening years provides strong support for Cassel’s claim. Guided by this premise, what is required is that research goes beyond conventional practice. The tremendous public health contributions of investigations based on the standard social etiology model notwithstanding, its utility for identifying fundamental determinants of wide ranging racial and SES health disparities may be limited. This is so because risk and protective factors are typically identified through contrasting the social experiences, socioenvironmental contexts, personal attributes, and to a limited extent, the genetic make up of those with and without the disorder under investigation. Those not qualifying for the clinically defined target disorder, including those for whom the disorder has not quite reached a detectable stage are implicitly, and frequently erroneously, classified as “well.” The crucial point is that the most important factors contributing to health disparities may not be linked to a specific disorder, or set of related disorders, to the exclusion of others. Following Aneshensel (Aneshensel 2005; Aneshensel et al. 1991), it is argued that the misclassification of individuals with unmeasured or undetected forms of distress or illness as non-disordered is likely to have obscured or yielded underestimates of the significance of causally relevant social, contextual, and dispositional factors. Research that avoids such misclassification may well provide a significant forward leap in our understanding of the factors, other than inequities in health services and differing health behaviors, that underlie racial and SES health disparities. The strategy proposed
seeks to solve the misclassification problem through combining consideration of certain biomarkers, to estimate current physical health status, with measures of both psychiatric and substance disorders and problems.

Because most disorders that are significantly implicated in health disparities have insidious rather than abrupt onsets, a related problem is one of establishing the time of onset. This is a crucial difficulty because in non-experimental community-based research the pursuit of causal inferences requires the establishment of the temporal antecedence of the risk/protective factors being evaluated (Kenny 1979). What is required, therefore, is a prospective design – one in which analytic outcomes include first onsets of physical, psychiatric, or substance disorders, both individually and collectively considered, and continuous measures that allow reliable assessment of changes in health status over time. As a large body of research utilizing the stress process model has demonstrated, this can be achieved within the mental health and substance abuse domains by assessing the lifetime and recent occurrence of DSM IV psychiatric and substance disorders, employing multidimensional measures of psychological symptomatology, and evaluating quantity/frequency of substance use, along with the counts of problems associated with such use.

With respect to physical health outcomes, establishing temporal order and assessing changes in health status over time have been highly problematic, especially within large-scale community studies and where interest goes beyond one or more particular disorders. Although studies that have focused on self appraisals of health status have yielded interesting findings, they have not been revealing of factors that may account for racial and SES health disparities. It is suggested that it may now be possible to overcome the daunting measurement problem that has long impeded our capacity for causal interpretation with respect to general physical health status. Based on the concept of allostatics (Sterling and Eyer 1988), McEwen and colleagues (McEwen 1998; McEwen and Stellar 1993; McEwen and Seeman 1999; Seeman et al. 1997) formulated the concept of allostatic load, referring to “the cumulative wear and tear on the body’s systems owing to repeated adaptation to stressors” (Geronimus et al. 2006). Allostatic load is thus thought to provide a meaningful description of the long-term biological consequences of chronic stress (McEwen and Seeman 1999; Seeman et al. 1997, 2004). The individual’s response to stress exposure results in dysregulation that is reflected by a change in the set-point of physiological markers (Dowd and Goldman 2006). When such changes endure over time the consequence is health deterioration. Allostatic load has been shown to be associated with increased mortality (Karlamanga et al. 2006; Seeman et al. 2004), lower SES, and the occurrence of depressive disorder (McEwen 2003). This and other evidence led to the “weathering” hypothesis initially proposed by Geronimus (1992) to account for the observation of earlier health deterioration among African Americans. “The stress inherent in living in a race-conscious society that stigmatizes and disadvantages Blacks may cause disproportionate physiological deterioration, such that a Black individual may show the morbidity and mortality typical of a white individual who is significantly older” (Geronimus et al. 2006, p. 826). Two categories of biomarkers are used to derive estimates of allostatic load – primary mediators involving substances released by the body in response to
stress, including norepinephrine, epinephrine, cortisol, and dehydroepiandrosterone sulfate (DHEA-S) and a secondary set of mediators that are generated from the effects of the primary mediators (e.g. elevated systolic and diastolic blood pressure, cholesterol levels, glycated hemoglobin levels, and waist to hip ratio) (Seeman et al. 1997). These categories of markers are labeled as mediators because they are the paths or physiological mechanisms by which adverse social experiences are translated into risk for mortality and for wide ranging forms of clinically detectable disease. However, because allostatic load may be taken to constitute a useful summary measure of “weathering,” it may represent a meaningful physical health outcome measure. Geronimus et al. (2006) have presented a clear rationale for such a perspective. They note, “An allostatic load algorithm is conceptually suited for the study of weathering. Because the stress response disrupts regulation of various systems throughout the body – for example, the cardiovascular, metabolic, and immune systems – the concept of weathering encompasses multiple systems and includes impacts on them that might not yet register clinically. Similarly, allostatic load is measured across physiological systems and includes sub-clinical indicators of the body’s response to stress – responses that increase the risk of morbidity” (Geronimus et al. 2006, p. 826).

As “weathering” refers basically to premature aging it may also be captured by measures of cell aging. As Aviv (2006) has noted, mean leukocyte telomere length, an index of cell aging, may be an indicator of biological age. As such it yields information beyond chronological age about risk for developing diseases of aging – diseases that reduce life span such as coronary heart disease, and hypertension (Benetos et al. 2001, 2004; Samani et al. 2001). Stimulated by the demonstrated linkage between chronic stress and poor health, Epel and colleagues (Epel et al. 2004) addressed the question of whether stress accelerates aging at the cellular level. Noting recent research that has pointed to the crucial roles of telomeres (DNA-protein complexes that cap chromosomal ends and that shorten with each replication and with age in all replicating somatic cells that have been examined) (Frenck et al. 1998) and telomerase (a cellular enzyme with direct telomere-protective functions), they tested the hypothesis that stress impacts health by modulating the rate of cellular aging. Assessing cell aging in terms of telomere length and the level of telomerase, Epel et al. (2004, p. 17312) found women with the highest level of perceived stress to have “telomeres shorter on average by the equivalent of at least one decade of additional aging compared to low stress women” (see also Mays et al. 2007; Seeman 2008). In a subsequent study, Epel and colleagues (Epel et al. 2006) found low telomerase activity, occasioned at least in part by chronic stress exposure, to be associated with major risk factors for cardiovascular disease and proposed that low leukocyte telomerase constituted an early marker for CVD risk and perhaps for shortened telomeres. Thus, current physical health status can be estimated by telomere length and the level of leukocyte telomerase as well as by allostatic load. Because these biomarkers can be taken to represent current health status and can be treated as continuous variables, they allow analyses in which the temporal order of variables can be established with confidence and provide means for measuring changes in health status over time. A strategy of considering both
Biomarkers would also allow evaluation of the concordance between cell aging and allostatic load and the assessment of their relative predictive efficacy and that of the components that comprise these two approaches for estimating “weathering.”

If it is accepted that these measures represent meaningful estimates of current physical health status, two scientifically crucial advances might be achieved – resolution of the misclassification problem and effective evaluation of the utility of the stress process model for advancing understanding of the origins of racial and SES health disparities. Misclassification can be avoided by evaluating the predictors, cross-sectionally and over time, of the presence and/or severity of problematic status on one or more of the three health dimensions – physical health, mental health, and substance use disorders and problems. This strategy would yield the unique opportunity to distinguish those who have some form of a significant health problem from those who do not. Such a multidimensional measurement strategy would also allow assessment of the possibility of cultural and sociodemographic variation in the propensity to express the consequences of stress exposure in physical, emotional, or behavioral ways. An ability to test this possibility may advance our understanding of the well-established but anomalous finding that, despite strong evidence predicting elevated mental health risk among African Americans, lower rather than higher rates of psychiatric and substance disorders are observed (e.g. Kessler et al. 1994; Turner and Gil 2002).

A multidimensional measurement strategy such as that described would also allow a unique consideration of patterns of comorbidity and concordance across alternative indices of health, and an examination of the risk significance of the prior occurrence of physical, psychiatric, and substance disorders for current general health status. It is argued that this measurement approach is likely to complement traditional disease-specific approaches and that it promises an advance in understanding potentially modifiable factors of relevance across a range of health problems that underlie racial and SES health disparities.

**Improved Estimation of Stress Exposure**

Available evidence leaves little doubt that exposure to social stress increases risk for poor health, regardless of the dimension of health under consideration. However, despite the reliability with which the stress–health linkage has been observed, both available evidence and medical predilection have led to a widespread assumption that the magnitude of the contribution of exposure differences toward explaining observed variations in health risk ranges from trivial to modest (Rabkin and Struening 1976; Turner et al. 1995).

Although measures of recent life events have long been criticized for ignoring other forms of social stress, among other shortcomings (e.g. Raphael et al. 1991; Sandler and Guenther 1985; Moos and Swindle 1990), it is clear that such measures remain dominant today in terms of use, and that most of what is known about the health significance of stress exposure, is based on the checklist measures of recent events
(Turner and Wheaton 1995). However, recent research has clearly demonstrated that checklist scores yield substantially biased estimates of total stress exposure across race/ethnicity, gender, and SES, at least among the young. Specifically, limiting stress measurement to a checklist of recent events has been shown to significantly overestimate total stress exposure among women relative to men, and systematically underestimated such exposure among African Americans relative to whites, and among persons of lower SES relative to their more advantaged counterparts (Turner and Avison 2003). In contrast to recent events, which suggest that women experience significantly higher levels of stress than men, estimated total stress reveals men to have significantly higher exposure. Total stress, assessed in terms of recent events, chronic stressors, discrimination stress, and the lifetime occurrence of major and potentially traumatic events, estimated the elevation in stress exposure among African Americans relative to whites to be 2.6 times greater than that estimated by scores on recent life events alone. Importantly, the substantial contribution of differential stress exposure toward explaining race differences in distress was observed even when discrimination stress was excluded from the analysis (Taylor and Turner 2002). The corresponding comparison of those in the upper and lower SES categories indicated that the total stress score estimated an elevation in exposure in the lower SES category that is three hundred percent higher than estimated on the basis of recent events alone (Turner and Avison 2003).

There seems a good basis for contending that the failure of prior research to take account of a range of social stressors has significantly biased estimates of status differences in exposure and resulted in the systematic underestimation of the contributions of stress exposure to the occurrence of health problems and racial and SES disparities in health. As already noted above, it is contended that the stress hypothesis has never been effectively tested primarily because of the misclassification problem and because of our failure to effectively estimate differences in stress exposure. As has elsewhere been argued, the relative absence of research that has gone beyond recent life events or a known-groups strategy for assessing differences in stress leaves open the question of the relative contributions to health disparities of variation in exposure to stress and differences in vulnerability to stress. This is so because unmeasured differences in stress exposure across race or SES will masquerade within research findings as differences in adaptational ability (Turner et al. 1995). Accordingly, poor measurement of exposure differences tends to lead toward conclusions that locate the source of health disparities largely within the skins of the victims.

In prior work, attempts have been made to improve on this circumstance by going beyond recent events in estimating level of stress exposure (Taylor and Turner 2002; Turner and Avison 2003; Turner and Lloyd 1999; Turner and Wheaton 1995; Turner et al. 1995) by adding measures of chronic stress, of lifetime exposure to major and potentially traumatic events, and of discrimination stress. It is suggested that effective evaluation of the contribution of differences in stress exposure to racial health disparities may also require consideration of additional forms or types of stress exposure such as colorism and hyper vigilance associated with uncertainty about covert discrimination.
The Promise of the Stress Process Model

As noted above, Fig. 1.1 presents an elaboration of the stress process model suggested by Pearlin. It reflects a health outcome measurement strategy which, as argued above, may effectively address the misclassification problem, as well as the multidimensional assessment of stress exposure that may minimize underestimation and biased estimate of stress effects.

Stress Exposure

Hundreds of investigations have reported relationships between exposure to social stress, primarily estimated by checklists of recent life events, and both mental and physical health status (Dohrenwend and Dohrenwend 1974; Jemmott and Locke 1984; Jenkins 1976). With respect to mental health, high levels of stress exposure have consistently been found to predict higher level of psychological distress (Avison et al. 2007; McLean and Link 1994; Thoits 1983; Turner and Wheaton 1995) and to account for a substantial portion of observed variation in psychological distress across SES and race (e.g. Turner and Avison 2003; Turner and Lloyd 1999). Moreover, cumulative adversity assessed by a lifetime of exposure to major and potentially traumatic events has been shown to substantially increase risk for the subsequent onset of psychiatric disorder, drug dependence, and alcohol dependence (Lloyd and Turner 2008; Turner and Lloyd 2003, 2004).

With respect to physical health disparities, evidence supporting the stress hypothesis is also widespread. There is now an extensive body of research, employing both human and non-human animal models, that addresses specific forms of disease or disorder. These studies reveal clear linkages between exposure to social stress and the onset and persistence of numerous chronic health problems including cardiovascular disease (Jenkins 1978; Kaplan et al. 1982; Kaplan et al. 1983; Nerem et al. 1980; Rozanski et al. 1999; Vitaliano et al. 2002), multiple sclerosis (Grant et al. 1989; Stip and Truelle 1994; Warren et al. 1982), diabetes mellitus (Hagglof et al. 1991; Leaverton et al. 1980; Mooy et al. 2000; Themlund et al. 1995), high blood pressure (Karlsen and Nazroo 2002; Krieger and Sidney 1996), fibromyalgia (Kivimaki et al. 2004), rheumatoid arthritis and osteoarthritis (Rogers et al. 1980; Zautra et al. 1994), Graves’ thyroid disease (Harris et al. 1992; Kung 1995; Sonino et al. 1993; Winsa et al. 1991), and respiratory illness (Cohen et al. 1998; Cohen et al. 2002; Karlsen and Nazroo 2002).

Thus, three persistently observed associations converge in support of the plausibility of the stress hypothesis, (1) the clear disparities in health across race and SES; (2) the compelling evidence, partially reviewed above, suggesting a potentially causal linkage between social stress and varying aspects of health, and (3) the strong evidence that exposure to substantially elevated levels of social stress is characteristic among African Americans (Turner and Avison 2003) and persons of
lower socioeconomic position (Kessler and Cleary 1980; Seeman and Crimmins 2001; Turner and Lloyd 1999; Turner et al. 1995). Indeed, as reviewed above, considerable evidence has accumulated over the past two decades indicating that the task of persistently coping with eventful and chronic stressors can profoundly affect one’s health (e.g. James 1994; James et al. 1992).

As Pearlin (1989) long ago argued, it is increasingly clear that stress exposure arises out of the context of people’s lives and thus that it is differentially distributed across contexts defined by social status, including race and SES (Turner and Avison 2003; Turner et al. 1995). Because stress exposure is generated or conditioned by social factors, the possibility of interventions aimed at reducing such exposure should, in our view, command substantially more attention in research than they have so far received. Supportive of this contention are findings that an important portion of the protective significance of family structure and of cultural factors in relation to depression and substance use problems is explained by the differences in stress exposure (Barrett and Turner 2005, 2006; Turner et al. 2006). Development of effective interventions, however, requires an understanding of the relative significance of different forms and sources of social stress, and for whom various forms are more and less important. A core objective of future research should be to identify the forms or aspects of stress exposure that most contribute to premature aging and thus to racial and SES health disparities. In this regard, it is important to note that resolution of the health outcome misclassification problem and more adequate estimation of the level of stress exposure are of crucial significance for effectively evaluating the significance of social stress for racial and SES health disparities.

**Mediating/Moderating Influences**

Regardless of whether variations in stress exposure can be fully and reliably measured, both evidence and everyday experience make clear that we would still observe cases where individuals are relatively unaffected in the face of substantial stress exposure and cases of adverse behavioral, emotional, and/or physical health outcomes where the magnitude of exposure appears minimal. Clearly, individuals differ importantly in their experience of, and how effectively they deal with, given environmental occurrences and circumstances. As Pearlin et al. (1981) long ago noted, this fact has pointed toward hypotheses that various factors may moderate or mediate the connection between social stress and health related outcomes.

_**Social Support.**_ A huge literature is now available attesting to the direct and stress moderating significance of social support in relation to physical and mental health (e.g., Cohen and Wills 1985; Kessler et al. 1985; Turner 1983; Turner and Marino 1994; Turner and Turner 1999; Uchino et al. 1996; Vaux 1988; Veiel and Baumann 1992). Indeed, on the basis of a careful review of prospective mortality studies that included consideration of various alternative hypotheses, House et al. (1988, p. 544) have concluded that “social relationships have a predictive, arguably causal, association with health in their own right.”
There is also specific and consistent evidence that lack of social support is a risk factor for coronary heart disease (CHD) onset and prognosis (Bunker et al. 2003), and is associated with reduced immunological function (Uchino et al. 1996; Cohen et al. 1997). In addition, findings have been reported suggesting that social support demonstrates a main effect with respect to blood pressure (Strogatz et al. 1997) and also buffers the impact of high stress on systolic blood pressure (Karlin et al. 2003; Berkman et al. 1993). These findings are consistent with the argument of Rowe and Kahn (1987) proffered more than two decades ago that lack of social support may be associated with greater biological aging (or “weathering” in Geronimus’ terms), and hence with increased susceptibility to the diseases of aging. Finally, social support, primarily in the form of supportive or positive family relations, has been shown by a number of investigators to be of significance for substance abuse and other problem behaviors (e.g. Jessor et al. 1995; Resnick et al. 1997; Wills et al. 1997).

This mass of evidence documenting the health significance of social support notwithstanding, it is now clear that not all relationships, even those that are very close, are uniformly positive (Rook 2003) and that negative aspects of relationships may be more consequential than positive aspects, at least with respect to mental health outcomes (Finch et al. 1999; Rook 1984; Newsom et al. 2005). Accordingly, researchers should routinely assess both positive and negative aspects of primary relationships.

Self-esteem and Mastery (Personal Control). Primary, among other variables that have shown either direct or moderating/mediating power with respect to mental health and substance use problems in a broad range of populations, are those of mastery (Pearlin and Schooler 1978; Pearlin et al. 1981; Gecas 1989; Turner and Roszell 1994) and self-esteem (Kaplan 1975, 1980; Rosenberg et al. 1989; Turner and Roszell 1994). With respect to physical health, a variety of studies have found mastery to be a strong predictor of general physical health status (Caputo 2003; Forbes 2001; Pudrovska et al. 2005). In addition, a 35-year longitudinal study found mastery to be inversely related to blood pressure and to be a significant predictor of cardiovascular well-being (Russek et al. 1990). There is also research demonstrating a small but consistent relationship between self-esteem and physical health (Antonucci and Jackson 1983; Gidron et al. 2006; Krol et al. 1994).

Additional personal resources/attributes that may directly influence physical and mental health or condition the effects of social stress have received somewhat less attention. These include optimism, mattering, emotional reliance, and “John Henryism.”

Optimism. Based on both animal and human research, it has been suggested that optimism is associated with immune function, risk for cancer, and longevity (Seligman 1990). Other research employing largely prospective designs has confirmed a linkage between optimism and both physical and mental health (Scheier and Carver 1992), and evidence for its significance for the course of symptoms and disorder (Fournier et al. 2002; Scheier and Carver 1985; Scheier et al. 1989; Segerstrom 2007). There are grounds for hypothesizing that optimism constitutes an effective moderator of the health impact of adverse experiences and circumstances.

Mattering. Rosenberg and McCullough (1981)conceptualized mattering as a primary motivator of the self-concept rooted in beliefs that (1) others are dependent upon us;
we are the object of others’ attention; (3) we are important to others; and (4) that others see our lives as an extension of their own. The perception of mattering, “simply put, is an existential belief in our own relevance to others” (Lewis and Taylor 2009, p. 275). This perception shares conceptual linkages with other aspects of the self such as self-esteem and mastery, which have been studied in far greater detail. Self-esteem and mastery may be viewed as important, if not necessary, requisites for establishing the satisfying and mutually-supportive relationships that foster the perception of mattering. Mattering has been found, however, to be empirically distinct from self-esteem and mastery (Elliott et al. 2004; Marcus 1991; Rosenberg and McCullough 1981; Taylor and Turner 2001), supporting Rosenberg and McCullough’s (1981) hypothesis that, “To feel that we matter to others is conceptually distinct from feeling that they think well of us” (p. 168). In addition, the perspective that mattering, like other dimensions of the self, is an important dimension of psychological well-being is supported by research demonstrating that perceptions of mattering are negatively associated with psychological distress or depressive symptoms (Pearlin and LeBlanc 2006; Rosenberg and McCullough 1981; Schieman and Taylor 2001; Taylor and Turner 2001; Turner et al. 2004). There appears to be little or no extant research assessing the significance of mattering for physical health status.

**Emotional Reliance.** This term represents the principal dimension of Hirschfeld and colleagues’ (Hirschfeld et al. 1977) construct of “interpersonal dependency.” The central hypothesis associated with the construct is that individuals who rely almost exclusively on the approval and attention of others for their sense of personal worth are more vulnerable. They found such reliance to be predictive of depression. Subsequent research has reported that emotional reliance increases risk for poor health and substance problems as well as depression (Bornstein 1992; Hirschfeld et al. 1983; Turner and Turner 1999). Although little specific evidence is available, it has been argued elsewhere that the effects of social stress may be importantly amplified by the level of emotional reliance (Turner et al. 2004). I am not aware of any studies that have examined race differences in emotional reliance or in the health significance of such reliance. However, some evidence suggests that higher levels of SES are associated with lower levels of emotional reliance. Given the linkage between race and SES, emotional reliance is also likely to be unequally distributed across race, raising a question of whether emotional reliance differentially influences risk for adverse health outcomes.

**John Henryism.** John Henryism, referring to “a strong behavioral predisposition to cope in an active, effortful manner with the psychosocial stressors of everyday life” (James et al. 1992, p. 59), appears to be implicated in racial differences in blood pressure (James 1994; James et al. 1983; James et al. 1984; James and Thomas 2000). Among African Americans scoring high in John Henryism, lower levels of SES have been found to be associated with increased risk for hypertension. This synergism between SES and this behavioral predisposition suggests that the significance of social stress for health, variously and collectively defined, may be elevated in the presence of high levels of John Henryism. Adoption of the relatively comprehensive assessment of variations in stress exposure specified in the Fig. 1.1 model would allow estimation of the extent to which this personal predisposition
or attribute amplifies the stress-health linkage and an examination of the social, contextual, and familial circumstances under which such amplification is minimized and maximized.

The preceding review of the components of the model specified in Fig. 1.1 represents an elaboration of Pearlin’s work in just three respects. First, it extends the explanatory application of the model beyond emotional and behavioral problems, where most work has focused, to include physical health status, thereby allowing at least some progress in resolving the misclassification problem. Second, it expands efforts to more adequately estimate variations in stress exposure. Finally, it expands somewhat on the range of personal resources considered. While these elaborations may well advance our capacity to uncover the origins of racial and SES health disparities, the model and the assumptions that underlie it remain those set forth by Leonard I. Pearlin. Principal among these assumptions, which have informed more than a generation of mental health researchers, are that stress is a process involving substantially more than the number and severity of stressors and that both stress exposure and the model factors hypothesized to mediate or moderate the health consequences of social stress arise out of the conditions of life to which the individual has been and is being exposed. As suggested above, the case that the model presented offers real promise of advances in the service of reducing health disparities owes a great deal indeed to the theoretical and empirical contributions of Leonard Pearlin.

Concluding Comment

It is a significant social advance for the National Institutes of Health to highlight race and SES disparities in health as a problem of monumental significance that both deserves and requires the highest priority among both service providers and health researchers. Quite aside from, and independent of, the goal of honoring the work and achievements of Leonard I. Pearlin, the objective of this paper has been to demonstrate the immense promise of the model he contributed for advancing the contribution of sociology toward understanding the origins of such disparities. As documented above, there are considerable grounds for contending that the principal conceptual path contributed by Pearlin, that has guided the work of many researchers across nearly three decades, promises significant future contributions. There can be no greater legacy than work of enduring utility in the effort to reduce health related misery and its unequal distribution across race/ethnicity and socioeconomic status. Thank you Len.

References