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Racial Differences in Preterm Delivery

Developing a New Research Paradigm

Edited by
Diane Rowley
Heather Tosteson

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Foreword
Racial Differences in Preterm Delivery

Sherman A. James, PhD

This supplement to the journal features eight articles discussing various aspects of the problem of black/white differences in preterm delivery. Although each article makes a unique and important contribution, as a group, they are unified by one singularly important theme: the paradigms that currently guide research on black/white differences in preterm delivery have failed to illuminate the most important causes for the persistent black excess prevalence observed for this disorder. Furthermore, the authors argue, in the absence of a more intellectually penetrating paradigm—grounded in historical analysis as well as in the contemporary life experiences of black women—black infants will likely continue to die at twice the rate of white infants. To the authors, this is unacceptable, especially for a society that professes to value all human life equally. Recognizing the need to go beyond critique, however, these eight articles specify a creative and challenging research agenda, one that holds unusual promise for changing the way researchers think about the underlying causes of preterm delivery in black women.

In addition to their common emphasis on the need for a new research paradigm, two other themes which effectively provide specificity to the first theme are apparent. These are (1) the likely role of the *combined* effects of race, sex, and class oppression on the health of African-American women and (2) the need for researchers to establish genuine partnerships with black communities, to reduce the current high level of mutual distrust and to facilitate the grounding of research questions in the everyday life experiences of community members. With an emphasis on these three mutually reinforcing themes, I offer brief comments on several of the salient points raised in these articles.

The articles by Rowley et al., Wise, and Krieger et al. speak most directly to the underresearched *joint* influence of race, sex, and social class status on the health of African-American women. Rowley et al., for example, point out that the paradigm based on poverty—which sought to explain the greater incidence of poor birth outcomes among many black mothers in terms of their social class disadvantages—advanced, to a degree, our understanding; however, this particular paradigm breaks down in the face of the elevated risks also observed for college-educated black women.[1] Since genetic factors linked to "race" are unlikely to fully explain these results, Rowley et al. describe a conceptual model that emphasizes the unique, simultaneous exposure of black women (including those of middle-class status) to both racism and sexism as the most parsimonious potential explanation of these findings. Discovering exactly how this process works—that is, how these unique social stressors affect the biological functioning of black women during pregnancy—is the main objective of the interdisciplinary program of research described in the concluding section of the authors' article.

Krieger et al. provide a masterful analysis of the major contours of race, gender, and class oppression in advanced industrial societies like the United States. The length of the article is more than justified by the monumental intellectual effort any group of scholars must undertake in order to provide an in-depth critique of the existing research paradigm. The latter, the authors argue, not only fails to engage the concept of social class in an intellectually rigorous manner but completely ignores the corrosive effects of racism and sexism on health. Moreover, in a major departure from most reviews of this type, Krieger et al. provide clear theory-based definitions of both "racism" and "sexism" and then proceed to describe how each has interacted, historically, with social class oppression to create and maintain the current "structure of domination" that characterizes our own society and those of other advanced capitalist countries. The persistently poorer health of working-class people, as well as the manner in which race and gender exacerbate this social inequality in health, can only be understood, they maintain, if the social, economic, and political factors that reinforce the current structure of domination are illuminated through rigorous, sustained scholarly research. Pragmatically, they acknowledge that any meaningful reduction in health inequalities will not only require new theoretical (and empirical) insights into the problem but committed public health action in the political arena as well.

One final comment on the corrosive effects of sexism on the

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health of black women and black communities is warranted. As the crisis in black America deepens, prominent African-American scholars[2,3] are speaking out with increasing directness about how sexism on the part of black men—their misguided complicity with white men to preserve male power and privilege—not only undermined the Freedom Movement of the 1960s and 1970s but, to this day, continues to add to the psychosocial stress with which black women must contend daily. Krieger et al. grasped this point with unusual clarity, and the paradigm shift which they call for exposes the health-eroding effects of all forms of oppression that affect black women, whether the source of that oppression comes from within or from outside black communities.

If, as Krieger et al. argue, "race" and "gender" are social constructions whose meanings are subject to manipulation in order to preserve nonegalitarian social structures, Wise invites readers to consider an identical proposition for "science." The latter, he argues, is also a social construction, with judgments about what is "good" versus "bad" science, "truth" versus "falsehood" being largely a matter of what leading researchers in a given discipline can agree upon at any one time. As a deeply human activity, therefore, "science" is not invariably linked to egalitarian or progressive social movements. Naive claims about adherence to scientific objectivity aside, "science" is frequently an unwitting partner of other ideological forces that serve to reinforce hierarchical worldviews and strengthen nonegalitarian social arrangements.

Several of the articles (Hargraves and Thomas, Hatch et al., Dressler, Gamble, and Krieger et al.) agree on the above point, but Wise argues that the deeply "social" nature of science deserves our serious attention because the antagonisms that periodically emerge, pitting advocates of "social" etiologies of disease against those who insist upon the preeminence of "biological" explanations of disease, are frequently rooted in subterranean conflicts over the relative power and prestige of various academic disciplines. This point is well worth remembering. According to Wise, most of these "either/or" perspectives on health (including infant health) are really "false" antagonisms—health is indivisibly biological *and* social.

Finally, Wise reminds us that only a relatively small percentage of adverse birth outcomes in African-American women can be attributed to ill-advised behaviors by the mother: teenage pregnancy, inadequate use of prenatal care, use of harmful substances during pregnancy, etc. Rather, the problem of preterm births among the minority of all black women who give birth is a "mainstream" affair. That is, black women who give birth to preterm babies engage in a wide variety of "appropriate" maternal behaviors and yet do not carry their babies to term. For Wise, the key explanatory factors must reside in common (i.e., "mainstream") exposures, and this, of course, is precisely the point that Rowley et al. and Krieger et al. make.

The third and final theme concerns the potential benefits of establishing genuine research partnerships with black communities when conducting health-oriented research in these communities. This theme is implicit in the discussions by Rowley et al. and McLean et al. in their discussions of factors to be considered when developing research instruments for use with black or other nonwhite, largely non-middle-class populations. Krieger et al. also touch upon this theme in their discussion of the need to recognize and appreciate "agency" within working-class communities of color. Indeed, the very concept of "partnership" presupposes a mutual acknowledgment of "agency" by parties to the agreement, with each partner recognizing both the desire and the capability of the other to act with enlightened self-interest.

The most extensive and most explicit discussions of what the concept of "research partnership" entails are provided by Hatch et al. and Dressler. According to these authors, the potential benefits are clear; they include, among other things, a significantly increased likelihood that research questions will be grounded in the actual, rather than the presumed, life experiences of the study participants. Second, "disempowering" stereotypes of working-class individuals and people of color are much less likely to receive reinforcement in published articles when the research was based on the concept of a genuine egalitarian partnership. As noted in several of the articles (Rowley et al., Hatch et al., Dressler, McLean et al., and Krieger et al.), here "ethnographic" (or other qualitative) methods of research can play an important role in opening up communication and building trust between researchers and study communities. The opportunity for study participants to tell their own stories, in their own words, gives them a "voice," indeed "empowers" them, potentially strengthening their investment in the information the study is designed to produce and the social utility of that information.

Obviously, the problems that must be confronted when researchers attempt to establish partnerships with communities are as real as the above benefits. Both Hatch et al. and Dressler, for example, warn that a struggle for control of the research agenda could occur. Resolution of this crisis will take time, patience, and a predisposition on the part of researchers to engage in "straight talk" about who will benefit from the research and when these benefits are likely to occur. It is clearly important, then, for researchers to decide beforehand if they are willing to invest, up front, the time and energy required if the benefits of a genuine partnership are to be reaped later on.

While these and other potential problems discussed by Hatch et al. and Dressler are very real, the urgent collective call for a paradigm shift expressed by authors of the articles in this issue of the journal leaves little room for continued adherence to traditional, "hierarchical" models of research, models wherein principal investigators and their staffs have little or no meaningful exchanges with communities whose health they wish to protect and improve. By raising a series of challenging questions and by sharing their insights on how some of these questions can be approached, these authors have made a distinctive contribution to our ability to "see" the problem of preterm delivery among black women in a different light. We await, with interest, empirical findings to be generated by the new research paradigm they seek to foster.

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Preterm Delivery Among African-American Women: A Research Strategy

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Preterm delivery (pregnancy < 37 weeks gestation) is a major contributor to infant mortality and morbidity. In 1988, 10.2% of all liveborns of known gestational age were delivered preterm.[1] Over 18,000 fetal deaths at 20 to 36 weeks gestation were reported in 1988.[2] Although fewer than 1% of all singleton infants born in 1980 to U.S. residents were very low birthweight (VLBW) (< 1500 g), a marker for preterm births, that 1% accounted for 40% of all infant deaths that year.[3]

Black infants are disproportionately represented in preterm delivery and VLBW. In 1988, 18% of black live births and 8.5% of white livebirths were preterm.[1] Thirty-one percent of all preterm fetal deliveries occurred to black women.[2] In recent years, preterm delivery has been increasing among African-American women but staying stable among white women. The black-white gap in infant mortality could be reduced 25% if deaths among infants weighing 750 g or less were prevented and 62% if deaths among infants weighing less than 1500 g could be prevented.[4,5]

Although applying existing knowledge and interventions can help reduce the black-white infant mortality gap, particularly in the postneonatal period, the gap will not substantially lessen until the rate of preterm delivery among black women declines.[6] Explaining the disparity in black and white preterm delivery requires a new paradigm that takes into account social and biologic factors associated with preterm birth.

The Current Paradigm: A War on Poverty

In concluding a review of the progress our nation has made over this century in reducing racial and ethnic differentials in infant mortality, Kessel et al. wrote: "The Children's Bureau's 75-year-old inquiry on infant mortality demonstrated the coincidence of higher infant mortality with low earnings, poor housing, the employment of the mother outside the home, and large families. . . . For all of our progress, some things just don't change."[7] This paradigm of poverty has defined the public health agenda for maternal and child health in this century. The major constituency for any public health problem is the vulnerable group—in this case, the disadvantaged and the poor, because infants born into poor families have higher rates of mortality and low birthweight.[8–10]

Public health action is now directed by this poverty-driven paradigm.[10,11] But is the explanation valid? First, statistical confirmation of its premises is called for. For example, we can provide statistics to demonstrate that social class differentials exist in specific risk factors for infant mortality, such as nutritional deficits, poor access to preventive health care, and higher risks for infectious disease.[11–15] Second, the poverty paradigm requires us to demonstrate that ameliorative programs do work. Thus, we have shown that immunizations prevent infectious disease morbidity and mortality, that safe home environments reduce the risk of injuries, and that use of seat belts saves lives.[16–18] Third, we need to show that ameliorative programs are not universally available to the poor, not a difficult task. Many poor and near-poor families lack access to primary health care services for pregnant women and infants.[19] The obvious conclusion is that public health programs must be made universally available to poor people to counteract their increased risks for poor pregnancy outcome.

However, we must admit that even our known interventions have limitations. For example, smoking cessation interventions need to be available to poor people, especially since more poor people than rich people in this country smoke cigarettes.[20–22] Yet our smoking cessation intervention programs have helped only a minority of smokers kick the habit.[22,23] Improving interventions is one public health goal that remains current within this paradigm.[12,24]

The paradigm of poverty as the cause of differential health outcomes begins to break down when it is applied to all health problems of the black community, particularly those related to pregnancy outcome.[25,26] Public health professionals have tended to equate being black in the United States with being poor and,

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by analogy, have blamed poverty for the excess infant mortality among black babies. This explanation works best for excess postneonatal (day 28 to the first birthday) mortality, especially among babies born weighing 2500 g or more.[26–28] Although prolonging the life of very ill newborns in neonatal intensive care units has tended to postpone deaths of those infants until the postneonatal period,[29] postneonatal mortality rates still help us assess the quality of health care in prevention of infectious diseases, injuries, and other causes of infant death.[30]

To try to control for the contributing effect of poverty on the gap in black infant mortality, Schoendorf et al. examined infant mortality among singleton infants whose parents were college-educated.[26] The preventable infant mortality rate, created by calculating the postneonatal mortality rate of infants weighing 2500 g or more at birth and excluding those who died of birth defects, was equal for black and white infants. Furthermore, the two groups had similar infant mortality rates for many causes, including sudden infant death syndrome (SIDS), injuries, and respiratory diseases. Thus, the poverty hypothesis makes sense only when it is applied to mortality due to causes unrelated to low birthweight. Programs such as Healthy Start that provide expanded services, garner community support, and attempt to improve home environments should reduce the disparity in death rates from SIDS, infections, and injuries.

The Schoendorf study suggests that poverty influences postneonatal mortality among normal birthweight black infants. However, among infants born to this highly educated group of parents, those born to black college graduates had an 80% higher risk of dying during their first year of life than babies born to white college graduates. This excess was related to a higher rate of low birthweight; most notably, the VLBW rate was three times higher in the African-American college-educated population.[26]

Although the authors' assumptions behind stratifying by education to adjust for socioeconomic status (SES) can be questioned (the two groups may have different cultural, social, and economic constructs of SES),[31] the high rate of VLBW among all black babies regardless of maternal education suggests that SES is not a marker for the racial disparity in preterm delivery. And so the poverty hypothesis does not explain much of the excess black infant mortality that is due to preterm delivery. If being poor does not explain the excess risk of preterm delivery among black women, what is the explanation?

An Approach to Research on Preterm Delivery Among African-American Women

In the previous discussion, "black" and "white" are terms used by convention to discuss racial designations scientific research related to the disparity in infant mortality, preterm delivery, and low birthweight. The inconsistency in acting upon concepts of race (biological) and ethnicity (cultural) in scientific research causes confusion, especially when the meaning of race is not clearly stated and social, cultural, and environmental differences are discussed in research results.[32,33] Race is routinely used in scientific research even though it is difficult to define and is a poor maker for genetic variation.[34,35]

From this point forward, we use the term "African-American" to emphasize ethnicity. Our use of ethnicity is based on self-definition and group identification defined from within and describes socioeconomic, cultural, and environmental characteristics.[36] Although the use of the term "white" appears inconsistent with this position, we were unable to come up with a satisfactory ethnic alternative for "white."

As noted by Cooper, "racial differences reflect different social environments, not different genes."[35] We therefore attribute any disparity in the rate of preterm delivery between African-Americans and whites to biologic mechanisms other than genetic factors associated with being African-American. We believe that at this stage, studies of ethnic differences between African-American women and other groups will not be informative, since our understanding of the constellation of factors influencing preterm delivery in the African-American community is limited. Furthermore, because the majority of African-American women do not experience a preterm delivery but live within the same culture and environment as those who do, they constitute the appropriate comparison group for studying etiologic risk factors for preterm delivery.

We view the high rate of preterm delivery as a sociobiological problem. Different social environments can account for different health states. As John Cassel noted, aspects of the social environment "are capable of producing profound effects on host susceptibility to . . . disease."[37] This generalized susceptibility encompasses many diseases and is caused by the interaction of a disease agent and the host's susceptibility to disease. In Cassel's view, host susceptibility is profoundly affected by both the social and physical environments. For example, pregnancy outcome can be affected by the quality of care a woman receives for a pregnancy-threatening condition like pregnancy-induced hypertension or an increased risk of exposure to lead. Cassel also postulated that psychosocial processes act at the immunoneuroendocrine level either to enhance or diminish susceptibility to disease. Thus, protective factors in the context of a woman's psychological and social environment could strengthen her resistance to a pregnancy-threatening condition, whereas psychosocial insults could reduce her resistance to such a condition.

A large body of literature suggests a link between maternal psychosocial stress and pregnancy outcome.[38–42] This complex relationship probably functions directly by altering physiologic responses and indirectly by promoting high-risk behaviors such as smoking.[24,43] One measure of psychosocial stress during pregnancy is lack of social support, which has been fairly consistently associated with adverse pregnancy outcome, including preterm delivery.[44,45] Yet intervention trials to reduce psychosocial stress through providing social supports and medical attention have found little evidence of increased birthweight or reduced rates of preterm delivery. As Oakley indicates, studies need to provide careful documentation and definition of the meaning of social support, explore the differential effect of social support on different subgroups of women, and determine whether some forms of social support may be detrimental to some women.[45]

Psychosocial stress may also result from negative social interactions, such as racism.[46] We agree with Dressler's hypothesis that "social and psychological factors . . . are related to health outcome, independently from behavioral factors that mechanically increase individual exposure to physical or chemical insults."[47]

One important clue to our lack of understanding about how psychosocial stressors and mediating factors affect preterm delivery may lie in the observations of Mervin Susser, who has written that in epidemiologic studies, the "variables analysed are multiple, but they are often divorced both from biological

substrate and from societal context."[48] Susser calls for epidemiologists to "fill the gaps . . . between the disease manifestation . . . and social behaviour, political structure and economic forces." He points out that the "penetration of these many strata . . . enhances the prospects of control and prevention."

Recent work by Polednak supports the importance of political structure and economic forces on the biological substrate.[49] He found that the gap between African-American and white infant mortality is narrower in metropolitan areas that are more residentially integrated, independent of the economic level of the community.

Such ecological analysis suggests important areas for further research into the interrelationships of institutional and interpersonal experiences of racism and pregnancy outcomes. Health may be influenced by the extent to which individuals experience or perceive personal and institutional racism and use different adaptive responses.[50] Racial inequality may function both as a psychosocial stressor that directly alters physiologic response and as a structural factor that limits access to quality health care.[51]

Clearly, the agent/host/environment relationship is not being elucidated in our current understanding of the causes of preterm delivery. Physical stressors—nutritional deficits, environmental toxins, inadequate housing, occupation, and physical activity—may also influence the risk of preterm delivery and interact with psychosocial stressors.[50,52–56]

Very little information has been published on immunoneuroendocrine responses to the social environment. However, stress does alter neuroendocrine function and may affect the immune system.[57–60] Stress may trigger a number of physiologic responses according to its duration and timing, genetic variability, the individual's coping style, social support, personal control, and the nature of the stressful stimuli.[61,62]

Only a few studies of stress and pregnancy have included physiologic markers.[63,64] These studies suggest that biologic markers that quantitate physiologic responses to the social environment are available and deserve further exploration.

Defining A Conceptual Model

Our approach requires identification of the pathways that lead to a preterm delivery. Although VLBW has been used as a marker for preterm delivery, VLBW infants do not represent the end product of a common final pathway.[65] Future research should be restricted to infants and fetuses delivered before 37 weeks gestation.

In fact, the etiology of preterm delivery is poorly understood. A preterm delivery may result from idiopathic preterm labor, preterm premature rupture of the membranes or other medically indicated early cause for delivery.[66,67] These pathways may have both different and common risk factors and underlying pathologies. For example, infections such as chorioamnionitis may result in either preterm premature rupture of membranes or idiopathic preterm labor.[68–72] It is important to estimate the risk of preterm delivery among African-American women for any of these pathways. The contextual framework for our analysis of why African-American women are at increased risk of preterm delivery builds on a model proposed by Sherman James.[73]

We view a person in the context of a social environment in which social behavior and cultural, historical, political, and economic forces influence health and disease. Gender, race, and social class thus affect a woman's health, and each is associated with pregnancy outcome. She is exposed to a myriad of stressors within her environment. These stressors must be measured in the context of her environment and understood through the filter of her race, gender, and cultural, social, and political condition. She responds to the stressors in her environment, either consciously or subconsciously, as she seeks protection through support systems and attempts to reduce stress through exercise or other healthful mechanisms or through smoking, drinking, or other unhealthful mechanisms. Other responses are physiologic—the "flight or fight" response at the neuroendocrine level.

All of these responses must be viewed within the context of the woman's environment: how the insult was delivered, how the woman perceived it, and what protection she had at the time of the insult. Each of the stress management techniques she uses can affect her ability to carry her pregnancy to term. We need to understand the social forces that elicit the stress, the adaptive mechanisms employed to manage the stress, and the underlying physiologic responses that increase (or reduce) the risk for preterm delivery.

We propose that future research focus on the environmental exposures and the social context that create a higher risk of preterm delivery for African-American women. This evolving model requires an interdisciplinary approach that expands the contribution of the social sciences to the biophysical model.[74] This approach emphasizes the need (1) to describe more fully the sociocultural, psychological, and behavioral influences on maternal health during pregnancy; (2) to improve the definition and measurement of the psychosocial constructs and the physical and environmental stressors that may be associated with pregnancy outcomes in African-American women; (3) to identify in each of the component pathways physiologic markers associated with a preterm delivery; and (4) to identify statistical approaches that permit estimates of the contribution of both individual behaviors and social forces to the risk of preterm delivery.

Epidemiologic variables, such as age, sex, or occupation, have specific social, cultural, and historical contexts that are important for understanding disease processes in a community.[48,75] The social phenomena that create stressors and the cultural responses that are adaptive mechanisms for handling environmental threats must be described.[76] Limitations in the description of SES and in the definition and measurement of psychosocial constructs, along with the lack of information on how social effects of being African-American in the United States may lead to adverse health, indicate a need for qualitative studies to identify the constellation of risk factors and forces that may influence preterm delivery. Qualitative studies do more than inform the development of better variables for quantitative studies.[77,78] They contribute uniquely to development of appropriate intervention strategies.

Earlier studies of stress and adverse pregnancy outcomes explored a series of constructs such as stressful life events, locus of control, personality traits, ways of coping, and social support networks.[38–42] We need a clearer picture of the effects of chronic stress, acute stress, and chronic role strain. Major sources of stress among African-American women (racial and sexual discrimination) and protective responses may not be captured by previously used scales. Furthermore, although previous study suggests that physical activity, workload, environmental

toxins, and psychosocial factors contribute to poor pregnancy outcome,[52–56] we need studies that explore the interactive effects of all these factors. Future studies also need to explore whether the timing of stress affects pregnancy outcome, since stress during certain intervals of the gestational period may influence the risk of preterm delivery.

If, in accordance with Cassel, we postulate that environmental stressors are not disease-specific but may alter immunoneuroendocrine functions, we must identify potential physiologic markers that are associated with stress. These markers may indicate that some women are at higher risk for clinical/subclinical infections, autoimmune reactions, or neuroendocrine reactions that contribute to a higher risk of preterm birth. Physiologic markers may be also pathway-specific. For example, preliminary evidence suggests that elevated plasma corticotropin-releasing hormone is associated with idiopathic preterm labor but not with preterm labor associated with infection.[79]

The research approach we envision calls for simultaneously determining the contribution of social, environmental, and cultural forces, of psychosocial constructs, and of behavioral and physiologic responses to the risk of preterm delivery; thus, commonly used mathematical models that produce statistical associations between variables obtained through traditional quantitative epidemiologic research are of limited value in describing the potential causal factors. These models estimate the contribution of individual risks to the probability of disease but fail to estimate the degree to which social forces increase or decrease the risk of disease. Given that the model we are evolving describes a complex network of factors whose interplay may vary with contextual exposures, we need more appropriate statistical models that can incorporate individual and contextual contributions to the risk of disease.

CONCLUSIONS

Higher rates of preterm delivery account for much of the disparity in infant mortality between African-American infants and white infants. Although all infant survival has improved since the 1970s as a result of improvements in medical technology that keep small babies alive,[80] The twofold excess risk of African-American infant mortality has not declined. The medical model successfully provides therapeutic strategies for improving birthweight-specific survival but not strategies to reduce the incidence of the preterm delivery. The search for etiologic risk factors for preterm delivery requires a conceptual model that includes the contribution of the social and physical environment to disease susceptibility and resistance.

The prevailing public health model that explains the racial disparity in infant mortality suggests that racial disparities in health outcomes are due either to differences in SES or individual behavior. The public health model may support a modest reduction in the black/white disparity in infant outcomes,[6] but it does not capture the contribution of social origins to the disparity. We are left with a limited foundation for developing prevention strategies.

We at the Centers for Disease Control and Prevention have attempted to set the scene for developing a new conceptual model that expands the medical and public health models to include the role of contextual, social, and physical environments in defining the disparity in infant mortality. The evolving conceptual model requires input from professionals across a diverse set of disciplines, including public policy, sociology, psychology, anthropology, physiology, epidemiology, biostatistics, health education, history, community organization, and the clinical sciences. The collaboration also includes the women we want to study.

In 1991 and 1992, we asked people from these disciplines to contribute to discussions of our evolving conceptual model. Some were asked to develop background articles that will guide the conduct of our research. These articles review methodologic issues in epidemiologic research on black women from the contextual framework of racism, sexism, and social class;[81] explore health policy strategies related to reducing infant mortality;[82,83] discuss bioethical issues in conducting research with black women;[84] review the measurement of psychosocial risks during pregnancy;[85] and promote collaboration between the community of black women, public health researchers, and policy makers.[86,87] The articles provide the theory for new research that will contribute to prevention strategies to reduce preterm delivery.

Applying this new research approach is a large and daunting task. To narrow the frame of reference to permit contextual study, research may need to focus on one or two well-defined communities of African-American women. Once we have a better understanding of these communities, we hope to apply our knowledge to all African-American women. Undoubtedly that application will require modification, within the context of environmental insults. With this newly gained knowledge, we can develop more effective approaches to the prevention of excess preterm delivery.

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Confronting Racial Disparities in Infant Mortality: Reconciling Science and Politics

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Somewhat paradoxically, it is its reverence for science that makes a society so vulnerable to science's misuse. The more prominent science becomes in shaping societal perceptions and institutions, the greater the potential that the lexicon of science will be extended far beyond its empirical purview and be used to frame virtually all matters of public debate. Of special concern is the extension of science into social relations, and in turn, public policy. For here, appeals to scientific principles have almost uniformly been used to recast inherently social phenomena as stirrings of some biologically determined "natural order."[1,2] The more central science becomes as an idiom of societal discourse, the more likely it will be invoked to consecrate hierarchical world views or justify policy determinations. The history of science is replete with reminders that, in the exercise of power, science can be transformed from social tool to social weapon, from a collective instrument for societal advancement to a technical guarantor of the status quo[3] (see also Gamble's cogent argument in this supplement[4]).

To understand the meaning of infant mortality is to confront this issue and this history. Far more than any other health statistic, the infant mortality rate derives its meaning and utility from a complex discourse between the technical and political domains. Pathophysiologic mechanisms leading to infant death continue to receive intensive scientific exploration. In recent years, these efforts have given rise to some of modern medicine's most technically intensive clinical environments.

The "political" meaning of infant mortality is derived from two related attributes. First, infant mortality implies tragedy. Few things in life are as tragic as the death of an infant. The accompanying sense of loss transcends any particular worldview or political persuasion. Regardless of who or what is held responsible for an infant's death, the fact that a newborn died is generally seen as a "shame." In this sense, the infant mortality rate represents not just a simple health statistic but the quantification of a population's collective tragedy.

However, in addition to its essential tragedy, the infant mortality rate has been traditionally viewed as an inherent reflection of social conditions and societal equity, speaking to issues of social well-being and distributive justice. This second component extends the social meaning of the infant mortality rate from merely the tragic, from being a "shame," to an intensely "political" form of social judgment, to being "shameful."

This dual nature—as tragedy and social mirror—gives the infant mortality rate a powerful public presence and explains why the infant mortality rate is as much at home on the front page of the local newspaper as it is in the most esoteric of medical journals. The duality also sets into motion a powerful, though often subtle, dynamic that can create enormous tension in the manner that infant mortality, and therefore disparities in infant mortality, is understood and ultimately addressed.

Even though these tensions can often seem abstract, their impact is not. Infant mortality has always been an extremely accessible health statistic, implying that problems related to its analysis and interpretation are likely to be played out in a variety of practical ways in communities across the country. This discussion is a response to these practical considerations. I attempt to identify those tensions in science and politics that have increasingly hindered the constructive translation of our empirical understanding of infant mortality into effective public policy and public health practice. I do not provide exhaustive review but rather a strategic critique of the growing mythology and antagonisms that currently undermine a coherent public understanding of racially disparate infant mortality rates and, therefore, the collective commitment needed to mount an effective response.

Historical Roots of Disciplinary Antagonism

Although the struggle to find meaning in infant death has likely existed since women and men began to value the promise of birth, biblical references provide some early insight into its social meaning and, through infanticide, its relation to the exercise of power. Pharaoh commanded that "Every son that is

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born ye shall cast into the river" (Exodus 1:22), while in the New Testament, Herod's order to slay the young children of Bethlehem in a vain attempt to kill "the babe" destined to be "King of the Jews" (Matthew 2:16) is commemorated as the Feast of the Holy Innocents.

However, the modern emergence of infant death as a focus of technical and social concern is largely rooted in the social reform movements of the early and mid-nineteenth century. Relying heavily on a newly established vital registration system and a growing capability to generate population-based statistics, Victorian reformers generated a number of public reports documenting alarming elevations in the mortality rates of poor populations in Europe's burgeoning industrial cities.[5–7] Culminating in the classic *Report on the Sanitary Condition of the Labouring Population of Great Britain* in 1842, these technical reports altered the empirical foundation of the political debate regarding the urban poor.[8] Not only did they tie the miserable sanitary conditions to health but also, through their tables and charts, they documented unequivocally that poverty meant more than hardship: it also meant death.

The emergence of infant mortality as an indicator of special importance emanated from an interesting marriage of technical and political concerns. Technically, the alarmingly high rates of mortality among the young, by themselves, accounted for the majority of excess deaths in poor populations. In addition, the causes of these deaths were more directly tied to sanitary conditions of the home and neighborhood.[8–10] Politically, the universal innocence and frailty of young children imbued measures of young child mortality with an emotional content of great benefit in advocating for social reform. Here, technical insights were coupled with political utility.[11] Together, they gave the infant mortality rate a special character in public discourse, combining in one simple statistic the legitimacy of empirical science and the urgency of a profound human tragedy.

The growing importance of the infant mortality rate within the reform movement, however, only served to highlight more prominently the inherent tensions over the rate's interpretation. One important group of sanitary reformers, which included Edwin Chadwick and Florence Nightingale, pointed less to social conditions than to the direct environmental contamination related to crowding, unclean water, and decaying refuse and sewage in the streets as the principal cause of urban morbidity and mortality. Consequently, the need for sanitary reform could be advocated with little regard for the political or social claims of the affected inhabitants.[12] Not surprisingly, the remedies that emerged from this position were primarily technical, with a strong emphasis on public engineering rather than political reform. Indeed, the investigations of Chadwick and his colleagues tended to elevate hygiene above privation and sanitation above injustice and, though serving the cause of reform, constrained its purview to the effects of unsanitary local conditions, not the economic forces that created them.

Although this direct focus on reducing illness and mortality lay at the heart of the sanitary reforms of the mid-nineteenth century, an important dissident group used the growing body of mortality data as evidence of inequitable economic relations and social injustice. With special attention to the young child mortality rate, Friedrich Engels and others interpreted differential mortality as an empirical reflection of societal relations and urged reformers to look beyond technical or programmatic interventions to more basic changes in the underlying fabric of industrial Europe.[13]

These differences in the interpretation of infant mortality data only intensified during the early part of the twentieth century as the issue increasingly became the province of public institutions and the competing claims of emerging professions (see the article by Hargraves and Thomas[14] in this supplement for an important and eloquent historical perspective on this issue). In the United States, the Children's Bureau chose infant mortality as the subject of its first major study. Significantly, this effort expressly linked analysis to action, recommending a variety of technical programs yet still linking infant mortality to unacceptable levels of social deprivation. However, by the early 1930s, the inclusive vision and administrative power of the Children's Bureau had largely been replaced by the mainstream medical community and its tight focus on technical solutions.[15] The dominance of the medical community in defining responses to infant mortality only increased with the phenomenal growth of the Medicaid program in the late 1960s and 1970s and an unprecedented expansion in medical knowledge and clinical capacity.

However, at the very same time the medical community was gaining increased control overt technical approaches to infant mortality, the political struggles of the 1960s initiated a demand that infant mortality data be used more explicitly within the political arena to strengthen calls for social reform. This political focus was particularly evident in efforts to establish community health center systems and other community development programs under the Office for Economic Opportunity. Expanded political utility reinforced the infant mortality rate's public presence and helped generate a new surge in empirical research focused on the social causation of disparate infant mortality.

Addressing Critical Analytic Antagonisms

The problem has been, however, that this recent, and rather remarkable, development of the technical and political perspectives of infant mortality has largely occurred in parallel, not convergent, lines, with little substantive interaction. Indeed, as these two domains grew in strength, so too did their disciplinary isolation. Both their perspectives and constituencies diverged and provided a conceptual and programmatic dissonance that today, more than ever, functionally undermines the struggle for shared understanding and collective action.

Chaotic epidemiology and the marginalization of infant mortality causation. Increasingly, health policy has looked to epidemiology for empirical justification. Epidemiology, on the other hand, has looked to health policy for social relevance. This relationship has enhanced the direct political impact of epidemiology and has provided health researchers with unprecedented access to the public arena. Not surprisingly, the more politically charged a health indicator is, the more multifactorial its determinants will appear because indicators prominent in the public debate of social and health policy are more likely to attract empirical attention. Indicators of intense political utility, such as infant mortality, tend to become "empirically sticky" because of public attention bestowed on any issue or variable so associated. This phenomenon has helped fuel a remarkable proliferation of studies reporting associations between infant mortality and a vast array of singular variables or risks. Stan-

dard discussion of infant mortality generates long lists of "risk factors" with little regard for their respective prevalences or how these risks interact to actually determine infant mortality patterns in the real world. Rather, the focus on singular risk associations has meant that the higher a factor's associated relative risk (RR), the more dramatic the factor's entry into the public debate.

But a focus on the highest RRs tends to distract policy's empirical gaze toward the extremes of risk determination. For example, teenage pregnancy, complete lack of prenatal care, and maternal drug use have all gained widespread acceptance as principal determinants of social disparities in infant mortality rates in the United States. The reality, however, is that, although these issues are associated with high RRs for infant mortality, they make relatively small contributions to disparate rates in most areas of the country, because the risk for infant death associated with teen pregnancy, for example, tends to be inversely related to its prevalence in the community.[16,17] In communities with relatively high rates of teen pregnancy, the risk for infant death becomes substantial only in the very youngest group of women, generally younger than 17 years of age, who are not likely to contribute the majority of births in that community. In most urban areas of the United States, the elimination of the risk associated with teenage pregnancy would reduce social and racial disparities in infant mortality by less than 10%.[17] The impact of no prenatal care would be similar.[18] Although much remains to be learned regarding the impact of maternal drug use in pregnancy, its role as an independent determinant of extreme prematurity and neonatal death remains largely unimpressive.[19,20] The scale of disparities in infant mortality today is similar to that prior to the crack cocaine epidemic. Clearly, teenage pregnancy, lack of prenatal care, and maternal drug use represent major problems and require expanded services. However, they are not major contributors to disparities in infant mortality. Rather, the bulk of disparate infant mortality occurs in the mainstream of women who are not teenagers, who receive some prenatal care, and who do not use illicit drugs.

The uncritical interpretation of singular risk associations has led to a kind of tyranny of the "*P*" value, by which statistically significant risk associations push the public debate to the margins and frame the public understanding of infant mortality as the product of deviant maternal behavior. Not only has this "marginalization" played into destructive stereotypes of maternal responsibility for infant death,[21] but it has also helped generate in many communities a host of specialized programs designed to provide services to a relatively small group of "high-risk" women. Although these programs are greatly needed, they distract attention and resources from the basic infrastructure of comprehensive health care and social service provision in these same communities. It was no coincidence that the original funding scheme for the federal infant mortality reduction effort, the Healthy Start Initiative, was supposed to have derived from migrant and community health center funding. In communities across the country, programs designed to address the extremes of risk have been implemented at the very same time that funding for local neighborhood health centers and general primary care programs deteriorated.

Social versus medical causation. Perhaps the most consistent and debilitating expression of disciplinary antagonism in addressing disparities in infant mortality is the pervasive tension between social explanations and medical explanations for observed differences in outcome, often termed a choice between "social models" and "medical models" of infant mortality causation. Social models stress the power of social variables to determine infant survival and the importance of structural change in overcoming disparate outcomes. Medical models stress pathways of frank pathophysiology and their potential interruption through clinical interventions. In this framework, social factors, such as poverty, are cast as inherently alternative explanations to clinical factors, such as chorioamnionitis or respiratory distress syndrome.

This conceptual tension between social and medical explanations has found diverse expression in empirical research, clinical care, and the development of public policy and has led to a disturbing isolation between the social and biologic sciences. A vast sociologic, anthropologic, and demographic literature exists on the causes of infant mortality. However, it is rarely tapped in the exploration of clinical pathways to adverse birth outcomes. Similarly, the social sciences continue to make little use of the clinical literature in refining the search for relevant social and behavioral influences. Efforts to reduce infant mortality have also been plagued by deep divisions between those who advocate social strategies, such as community development, and those who propose expanded clinical services, such as enhanced prenatal monitoring for high-risk women. Too often, those who elevate the role of social determinants indict clinical technologies as failed strategies. But devaluing clinical intervention diverts attention from the essential goal that it be provided equitably to all those in need. Belittling the role of clinical care tends to unburden policy of the requirement to provide equitable access to such care. The antagonism between social and medical explanations, therefore, involves more than conceptual concerns. It enhances disciplinary provincialism and reinforces tensions in the development of ameliorative policy.

Preventive versus therapeutic strategies. Antagonisms between social and medical explanations for infant mortality have also caused tensions between preventive and therapeutic interventions. Although preventive strategies are undoubtedly more desirable, the public discussion of infant mortality has been increasingly characterized by a false perception that therapeutic strategies have not improved infant mortality.[11] This tension is expressed somewhat more subtly as an underlying impression that poor children somehow benefit less from therapeutic, and particularly "high technology," interventions than do their wealthier counterparts. There can be little question that preventive efforts have been seriously ignored and must be broadly enhanced. However, it is also important to recognize that therapeutic strategies, particularly neonatal intensive care services, have contributed greatly to improving survival of both poor and wealthy infants alike.[22,23] The false notion that therapeutic efforts have been ineffective is particularly troubling in light of profound social inequities in the provision of greatly expanding intensive care capabilities in many developing countries and the unraveling of regionalized neonatal intensive care systems in some areas of the United States based on the patient's ability to pay. This "social deregionalization" of neonatal intensive care deserves focused attention since between 60% and 80% of the total decline in neonatal mortality in the

United States over the past two decades has been ascribed to the impact of improved management of the high-risk delivery and intensive care for ill newborns.[23,24] Even small social disparities in access to high technology care could have an important impact on differential infant outcome.

The divide between the clinical and public health communities. A schism between clinical and public health communities has resulted from the tensions between social and medical explanations of disparate infant outcome. Public health efforts to address infant health have generally involved financial incentives to enhance the health care use and a variety of community-based initiatives, including outreach, home visitation, and social service programs.[25] However, in many settings, these efforts have been developed and implemented without links to clinical services or facilities. Indeed, clinical providers may not even know that many of their patients may be enrolled in such programs. Clearly, the potential for the fragmentation of services is significant. However, the schism may also create problems in service planning, since enhanced outreach efforts may only aggravate already strained clinical facilities.[26] Moreover, this lack of clinical and public health collaboration represents a lost opportunity for a joint, expanded commitment to improving infant health.

Birthweight, Biology, and the Interpretation of Racial Disparities in Infant Mortality

The incoherent mix of the technical and political aspects of infant mortality has proven most destructive in the search for the causes of racial disparities in infant mortality. This result is not surprising, given the troubled history of scientific approaches to race[3,27] and the contentiousness of the ongoing struggle to confront racism. Indeed, in the United States, social disparities in infant mortality have largely been defined by the issue of race, since it continues to be associated with profound differences in wealth and social status. In addition, vital statistics data in the United States contain information on race but little on social status. Race, therefore, has been used as a proxy variable for analyses addressing social differences in birth and mortality.[28,29] In 1989, the infant mortality rate for blacks in the United States was 18.6 deaths per 1,000 live births, whereas the rate for whites was 8.1, yielding a risk ratio of 2.3.[17] In general, the trend over the past three decades has been for the absolute difference between black and white rates to fall while the relative disparity has remained largely intact.

Traditionally, the racial disparity in infant mortality has been considered a direct reflection of disparate social conditions. However, the numerous empirical studies concerned with racial disparities in infant morality have increasingly been interpreted as questioning this social explanation. Although the elements of these arguments are manifold and at times quite subtle, there are several issues that seem central to the debate and are discussed below.

Assessing the social content of race. Using multivariate techniques, a number of studies have attempted to assess the influence of race on infant outcome while controlling for social variables such as income or education.[22,30–33] Almost uniformly, these studies have reported a persistent elevation in unsuccessful birth outcomes among African-Americans even when selected social and economic variables are taken into account. Similarly, the relatively low rates of low birthweight and infant mortality among Mexican-American populations, despite significant social deprivation,[34] have also been interpreted as suggesting nonsocial causes for the elevated African-American rates.

In a sense, despite the cautions of their authors, these studies are being interpreted as statistically stripping race of its social content, leaving behind a residual biologic component. Technically, the uniformly crude social variables used in these analyses are not likely to have captured fully the social meaning of race in American society. Most commonly, maternal level of education, health insurance coverage, and areal estimates of family income have been employed as the principal indicators of social status. Yet health researchers have not adequately sought out the insights of the social sciences in analyzing racial influences and have not fully recognized the nuanced social character of race so well documented in the sociologic, psychological, and anthropologic literature.[35,36] (See the definitive critical review by Krieger et al.[36] in this supplement.) Even with similar family incomes, African-American families possess far fewer assets, are less likely to own their homes, and receive fewer intensive-care services when hospitalized than their white counterparts. College-educated African-Americans have significantly lower incomes than whites with the same level of education.[37] Indeed the persistence of racial disparities in health outcomes, despite the simultaneous analysis of social variables, is more the rule than the exception and has characterized patterns of mortality as a result of pedestrian injury and homicide, for example.[17] The empirical experience from a variety of disciplines suggests that the social character of race is exceedingly complex and that persistent racial effects should be interpreted cautiously.

The prominence of birthweight. Approximately two thirds of the difference between black and white infant mortality rates in the United States is currently due to differences in neonatal mortality. Slightly more than two thirds of this neonatal differential is, in turn, accounted for by the elevated birth rate of black neonates born with birthweights of $< 1{,}500$ grams.[17] This strong association between very low birthweight (VLBW) and disparate infant mortality has led to a strong analytic interest in factors that influence birthweight.

The central reason to measure birthweight is because it remains a powerful predictor of infant morbidity and mortality. However, as the public appreciation of birthweight's role has grown, some forget that its importance rests solely on its relationship with outcome. Too often, birthweight is considered an outcome variable per se, with little regard for the inherent elasticity between birthweight and morbidity and mortality.[33] When this occurs, a 50-gram birthweight difference may be considered "significant" on the basis of statistical tests regardless of whether it has any actual impact on outcome. This tendency to establish birthweight as an outcome variable is more than a technical concern. Rather, it has helped confuse the etiology and possible resolution of disparate infant mortality rates in the United States.

It is important to remember that birthweight is generally employed as a proxy measure of gestational age, since gestational age is often difficult to measure accurately on a population basis. However, birthweight also can reflect intrauterine growth effects independent of the length of gestation. "Small for gestational age" or "intrauterine growth retardation" both describe diminished fetal growth for a designated gestational age. The complex relationship between birthweight, gestational age, and mortality must be addressed carefully, particularly

when proxy uses are intended.[38–40] Nevertheless, in terms of mortality, and particularly neonatal mortality, birthweight effects relate to extreme prematurity and are generally captured within the VLBW category (< 1,500 grams).

More worrisome, however, is the failure to distinguish between factors that affect mean birthweight and those that affect the probability of delivery at birthweights incurring a high risk of morbidity or mortality (e.g., < 1,500 grams). To begin, birthweight differences near the mean may reveal effects that are statistically significant but wholly irrelevant to functional outcomes. For example, a 50-gram difference in mean birthweight may reflect a discernible influence of a studied factor, but whether a population's mean birthweight is 3,225 grams or 3,275 grams would not likely make much practical difference. The attraction of using mean birthweight or broad categories (e.g., "low birthweight," defined as < 2,500 grams, or "prematurity," defined as < 37 weeks gestation) is to enhance the numbers of such infants in any studied population. But the majority of infants so categorized are located near the upper boundaries of these categories, precisely the group least likely to experience morbidity or mortality.

Further, no evidence suggests that the actual pathophysiologic mechanisms affecting birthweights near the mean bear any resemblance to mechanisms resulting in the delivery of infants at extremely short gestations. This distinction is often overlooked in interpreting findings that relate to overall birthweight distributions or mean birthweight. Not only has this problem generated considerable disorder in identifying the major causes of disparate infant mortality rates in the United States but it has also contributed to the confusion surrounding possible biologically determined causes of disparate infant mortality, including inherited influences.

In this context, great concern has arisen about the suggested use of race-specific birthweight standards in comparing the birthweight distributions of blacks and whites.[41–43] These suggestions have been based on the observation that currently examined populations of blacks and whites have different mean birthweights and that blacks generally have higher survival rates at low birthweights and lower survival at normal or high birthweights when compared with their white counterparts. Race-specific standards would assess any group of births in relation not to some general standard, such as 2,500 grams, but rather to their position along the birthweight distribution of their racial group. Because African-American populations have been observed under current conditions to have lower mean birthweights than white populations, the adjustment process would have the effect of using different low birthweight standards for African-Americans and whites, thereby reducing the disparity in low birthweight rates.

The notion that race-specific standards should be used and that they would, through technical manipulation, erase a significant portion of the racial disparity in low birthweight rates has alarmed many, particularly those impressed by the political importance of disparate low birthweight rates. Although any proposal to use race-specific standards for any health outcome warrants intense scrutiny, the suggestion regarding birthweight does not, in fact, threaten the scope or ultimate meaning of racial disparities in neonatal and infant mortality. Rather, this controversy has been fueled by a mistranslation of empirical findings and a continued fragmentation into technical or political interpretations of infant mortality.

Perhaps the most common expression of this fragmented approach has been the erroneous perception in the policy arena that race-specific birthweight standards are tantamount to race-specific mortality standards. This stems from the tendency to view birthweight as a concrete outcome and assumes incorrectly that the reduction in low birthweight rates through race-specific adjustment implies an associated reduction in the disparity in mortality. In reality, any such adjustment in birthweight distribution implies a concomitant adjustment according to birthweight's predictive relationship to mortality. In essence, by reducing the importance of general birthweight differences, race-specific birthweight standards necessarily increase the contribution of differences in survival within defined birthweight categories (i.e., birthweight-specific mortality). Although this causative tilt from birthweight distribution to birthweight-specific mortality raises some important questions, it is crucial to recognize that no adjustment in birthweight can change the magnitude of the overall difference in infant mortality.

Another important consideration in assessing the practical impact of race-specific birthweight standards is its relevance to the relatively small group of extremely premature infants who account for the majority of neonatal deaths in the United States. This concern relates directly to the distinction between factors that shape mean birthweights and those that determine rates of highly pathologic deliveries. In general, VLBW infants have been excluded from the primary birthweight distributions, which are dominated by the 98% of births not falling into the VLBW group. It is useful to remember that the mean birthweight in Japan is similar to that for African-Americans in the United States and yet Japan has the lowest infant mortality rate in the world.[44] One must question, therefore, the practical significance of birthweight adjustments determined by, and most applicable to, birthweight categories that contribute relatively little to disparate mortality.

Practically speaking, then, race-specific birthweight standards may be useful in selected analytic settings and should be viewed as a technical innovation worthy of consideration and empirical evaluation. However, on technical grounds alone, they cannot alter either the scale of the social character of excess infant mortality and therefore present little real substrate for controversy in policy. Ironically, it is through their misinterpretation that race-specific birthweight standards pose a meaningful threat. The concerns generated by such standards have only deepened the tensions between social and biologic perspectives and exacerbated the distrust held by those working from the political perspective for technical explanations of disparate infant death.

Intergenerational effects. Although not specifically concerned with the issue of race or social status, another important epidemiologic issue regarding biologically determined explanations of disparate infant mortality has been the suggestion that intergenerational mechanisms may help shape current disparities in birthweight and, therefore, infant mortality. In general, these studies have revealed differences in mean birthweight or a higher risk of delivering a low birthweight infant if the mother herself had been born at low birthweight.[45,46] Although these findings are intriguing, they can be easily misinterpreted as suggesting that current disparities in birth outcomes are somehow biologically fixed on the basis of prior birth experiences. Significantly, these studies have generally suggested intergenerational effects for mean birthweight and low birthweight rates. How-

ever, there has been little evidence that such effects occur in determining VLBW or extreme prematurity, the primary contributors to neonatal and infant death. Rather, the findings relate almost exclusively to birthweight and gestational age categories that have little impact on disparities in morbidity or mortality. Moreover, despite creative attempts to account for intergenerational social effects, these studies' efforts to control for these important, though often subtle, influences remain incomplete.

The veil of heterogeneity. Social disparities in infant mortality have also been framed as unavoidable because of the complex heterogeneity of American society. Comparisons of international infant mortality rates have often been used to highlight the disturbing freefall of the U.S. relative position over the past several decades. The rates of Japan, Sweden, Norway, and some 20 other countries have fallen below that of the United States. Some respond that such comparisons are inappropriate because these populations, unlike that of the United States, are "homogeneous." This response implies that the provision of services in the United States is somehow so complicated because of social, cultural, and geographic heterogeneity that efforts to address infant health are doomed to only mediocre success. Although the provision of services in the United States may indeed involve greater administrative complexity, such qualifications have rarely proven acceptable in comparing other social indicators related to children, including growth, athletic achievement, or measures of educational performance. The acceptance of disparate infant mortality rates as the inevitable product of heterogeneity, though seemingly logical, is actually more closely related to ideology and politics. There is little evidence for the contention that administrative complexity is less likely to affect scholastic achievement than to affect our ability to prevent unnecessary child death.

Reconciling Social and Medical Models of Infant Mortality Causation

In a setting of profound poverty, the intention of clinical intervention is not to alleviate poverty but reduce its power to alter health outcomes; thus, clinical interventions attack on the tragedy of infant mortality will be successful only when *social* influences are no longer expressed in differential outcomes. The ultimate contribution of the clinical world to the reduction of racial disparities in infant mortality is thereby defined as racial equity in infant survival, regardless of the scale of persistent racial disparities in social status. The result would be the elimination of the reflective component, the mechanism by which the infant mortality rate articulates social differentiation. This dynamic relationship between medical and social determinants of infant mortality underscores the essential need for analytic approaches that can integrate both.

A suggested analytic model. If epidemiology is concerned with truth, policy is concerned with truth that is relevant. This duality implies that a certain structure, or discipline, is required to ensure that technical insights will serve directly the needs of policy. In addressing disparities in infant mortality, therefore, it is useful to create such an analytic structure, particularly in light of the difficult interaction between the political and technical perspectives regarding infant mortality's import. Many analytic approaches are possible and many have been proposed. However, for the purposes of understanding disparities in infant mortality, certain analytic capabilities are particularly useful.

First, an analytic approach should distinguish between the causes of infant mortality and the causes of disparities in infant mortality. Second, an analytic approach should link the epidemiology of disparate mortality and arenas of intervention. Third, a policy-based analytic approach must embrace both social and biologic influences but not segregate them artificially into distinct analytic boxes. Fourth, it must guide deliberations at a variety of levels; it must serve the needs of national policy as well as those of local action. Last, it must be accessible across disciplines. This requirement, perhaps the most difficult, implies a certain simplicity and eclecticism, characteristics that can often disconcert those more comfortable with the complexities and conformities typical of disciplinary specialization.

As I have described in a previous publication,[47] at a basic level, disparities in infant mortality can be created by two general mechanisms: (1) the elevation of risk in a population and (2) the reduction of access to interventions capable of modulating the impact of elevated risk on outcome. These two mechanisms, or "dual currencies" of disparity creation, can in turn, affect both preventive and therapeutic interventions.[47] Using this approach, an analytic framework for examining disparities in neonatal mortality, the primary determinant of infant mortality, can be constructed that responds to the special requirements of policy and community-based action. Such an analytic framework is presented graphically in Figure 1 and described in detail below.

As noted earlier, neonatal mortality, defined as deaths of liveborn infants < 28 days of age, depends heavily on birthweight distribution, particularly VLBW (< 1500 grams). This association with birthweight allows the neonatal mortality rate and, significantly, disparities in the neonatal mortality rate to be stratified into two components: birthweight distribution and birthweight-specific mortality. This is a traditional stratification technique and has served many analytic purposes well.

To meet the needs of policy, however, these two components must be related to intervention. Empirically, birthweight-specific mortality has been linked with perinatal interventions, particularly neonatal intensive care and the obstetrical care of high-risk deliveries. This relationship is depicted along the upper arm of the model depicted in Figure 1. Birthweight distribution, virtually by definition, is closely tied to prenatal interventions, including preconceptual care, nutrition supplementation, and prenatal health care. This link appears as the lower arm of the model. In this manner, the relationship between prenatal interventions and birthweight distribution represents preventive mechanisms, whereas the relationship between perinatal interventions and birthweight-specific mortality primarily represents therapeutic response.

Both the preventive and therapeutic relationships are sensitive to the two fundamental mechanisms of social influence. Differences in maternal/fetal risk in populations can take the form of differential prevalences of such medical conditions as hypertension, diabetes, or asthma, of demographic factors such as young maternal age, or of maternal behaviors such as smoking or heavy alcohol use. These maternal/fetal risk factors are profoundly shaped by social conditions and can influence both the preventive and therapeutic relationships, although in this case, the effect on the preventive arm, expressed as birthweight distributive differences, is far more significant.

Reduced access represents differences in factors that determine the use of relevant interventions. Along the preventive

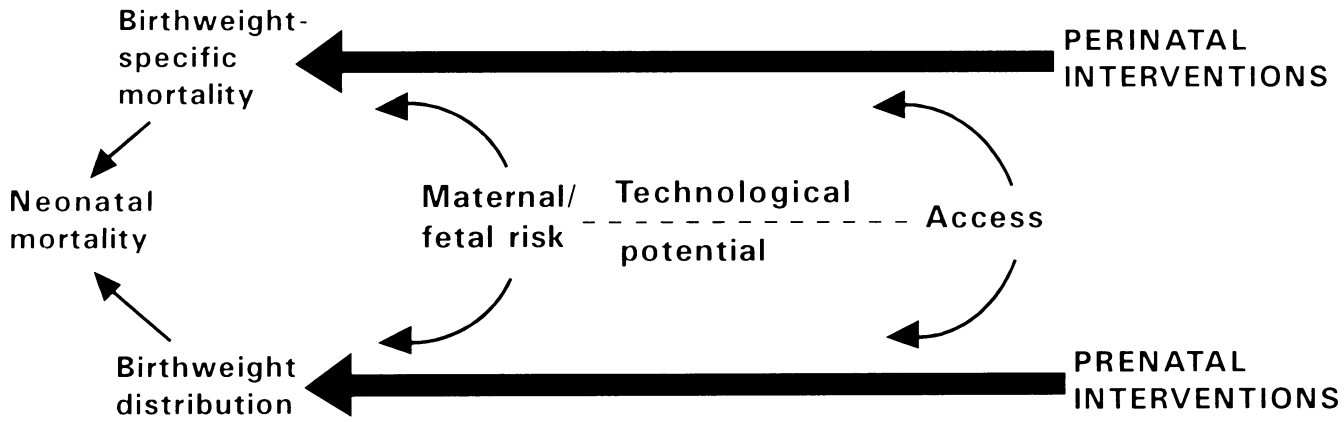

Figure 1. Technological and social interactions affecting neonatal mortality. This figure has been published previously in a slightly modified form (Wise PH. Poverty, technology and recent trends in the United States infant mortality rate. Paediatric and Perinatal Epidem 1990;4:390–401).

arm, reduced access to prenatal interventions has been clearly recognized as being shaped by social considerations. Indeed, the lack of progress in improving the percentage of women who receive early prenatal care in the United States has attracted considerable recent attention and has generated numerous pleas for expanded access to such services. Along the upper arm, access to perinatal interventions, largely the domain of regionalized high-risk obstetrical systems and neonatal intensive care, has not received sufficient attention as an area where social forces could be of great potential consequence. As mentioned earlier, strong financial pressures have created incentives for hospitals in some areas of the country to reduce the availability of intensive-care services to high-risk women and newborns who lack adequate health insurance coverage. Beyond the fundamental injustice of such policies, it is critical to recognize that even small differences in access to perinatal interventions through this "social deregionalization" of perinatal services based on the ability to pay could, therefore, produce dramatic disparities in neonatal mortality.

Of crucial importance, and what ultimately determines the relative power of risk and access effects to shape outcome, is the efficacy, or technologic potential, of the intervention in question. Interventions wholly without efficacy are not likely to create disparities in outcome even if differences in access exist. When the technologic potential of an intervention is high, differences in access to the intervention will dominate disparities in outcome. When the technologic potential of an intervention is relatively low, differences in maternal/fetal risk will dominate disparities in outcome. In this manner, differences in the capacity to alter the linkage of risk to outcome will ultimately determine the relative importance of risk or access effects.

The dynamics of this model operate in both local and national settings. By applying the model to local patterns of disparate neonatal mortality, the relative importance of perinatal or prenatal influences can be gauged and the role of differential risk status and access to highly efficacious interventions assessed. In addition, the model is helpful in developing evaluative strategies needed to assess local interventions or programs by isolating changes in risk and access over time. In the face of growing risk, even a successful access enhancement program could be associated with a rise in infant death. Indeed, since the technologic potential of any intervention is rarely complete, increasing risk can almost always overwhelm enhanced access.

The model also helps frame the dynamics of national policy as it illustrates the organic interaction of advocacy and efficacy. Advocates' calls for expanded access to services obviously accept the efficacy of those services. A lack of consensus about the efficacy of the services in question generally reduces the strength of arguments for expanded access. For example, over the past few years, the greatest threat to advocacy pleas to expand access to prenatal care has been not cost but questions regarding the actual efficacy of prenatal care to improve birth outcome. This, in turn, shifts the public discussion away from access issues, along the model's central axis, toward differential risk status, usually framed in terms of adverse maternal behaviors, such as illicit drug use, smoking, and illegitimacy. It is not surprising, therefore, that within the public arena, the efficacy of prenatal care is both defended and assaulted vociferously with little regard for the growing body of evaluative and analytic evidence that suggests a far more complex picture.

The model also emphasizes how growing technical efficacy can either reduce or exacerbate extant disparities in outcome because the greater the technical capacity to respond to risk, the greater the burden on the system to provide it equitably. This point holds important lessons for the research community and policymakers considering directions for health care reform, for clinical innovation demands an increasingly responsive system of equitable provision. Regardless of their preventive or therapeutic character, new interventions—particularly those with great efficacy—can widen disparities in outcome if differentials in access to these new interventions are allowed to persist. The introduction of surfactant replacement therapy, for example, has been associated with a profoundly enhanced efficacy to improve survival among newborns suffering from respiratory distress syndrome. While this therapy would be expected to reduce overall neonatal mortality rates, its ultimate effect on social and racial disparities in neonatal mortality remains unclear. Similarly, the introduction of new immunizations could, in the face of worsening inequities in the provision of primary care services, actually enhance current disparities in morbidity and mortality from the illnesses targeted by these new, highly effective interventions.

Reconciling biologic and social etiologies. The model makes no distinction between biologic and social influences on outcome, an important fact since inherently biologic etiologies can be altered profoundly by social influences. Similarly, social etiologies ultimately have biologic expression and therefore provide opportunities for clinical intervention.

However, because of the long and troubled history of biologic explanations of racial and social differences in health outcomes, some people—those who believe survival differences directly reflect profound social inequity—distrust biologic insights into the causes of racial disparities in infant mortality. However, these biologic arguments must be understood and integrated into a comprehensive view of disparate infant mortality because biologic explanations will inevitably be invoked often and broadly whether empirical evidence supports them or not.

The primary tension emanates from the fundamental confusion of biologic influence and biologic determination. Although there is clear consensus that biologic processes are involved in infant death, there is considerable resistance, rightly so, to determinative explanations implying that the scale of disparity is somehow biologically fixed. At times, these biologic explanations are interpreted as genetic, or inherited, factors, a transition often associated with issues of race.

Despite speculation, little current evidence supports the contention that racial disparities in infant mortality are based on genetic, or otherwise biologically determined, pathways. Moreover, one recent study revealed that elevated African-American VLBW rates were due not to a singular condition but rather to an across-the-board elevation in virtually all conditions directly associated with premature birth.[48] This suggests that if some inherited predisposition does exist, it is only partial or heavily interactive such that it is ultimately expressed as multiple clinical conditions. The primary sources for determinative speculation are derived from reports that have been interpreted far more expansively than their methods allow or their authors intended. Nevertheless, the notion that racial disparities in infant mortality are generated by immutable biologic forces continues to reverberate in scientific and political circles. The profound tragedy here is that this specious debate has continued to distract policymakers and freeze local agencies in their efforts to address disparities in infant survival.

Sweeping biologic explanations are dangerous because they are commonly viewed as inherently exculpatory, relieving policy of the responsibility for disparate infant survival. Such speculation based on current evidence can, if not framed constructively, prove irresponsible, and, inadvertently at times, support ideological positions dedicated to public inaction and the status quo. However, current biologic insights, rather than providing new justification for the status quo, actually strengthen an activist agenda by helping to generate new opportunities for potential intervention.

Indeed, the interactive nature of social and biologic influences, itself, undermines deterministic views of racial disparities. As the presented model shows, the distinctions between social and biologic etiologies are less important to policy formation than the capacity to alter outcome. For example, phenylketonuria (PKU) is a potentially lethal disorder of complete genetic origin. However, because an effective intervention exists (dietary alterations), any observed disparities in PKU mortality are likely social in origin. Here, a completely genetic condition is characterized by completely social disparities in outcome. Similarly, possible intergenerational effects, if relevant, will likely be amenable to clinical intervention. Biologic processes do not undermine social etiologies; they merely modulate mechanisms of influence.

Biologic determination is less about biology than it is about limits. Confronting such perspectives therefore, should not rely on an impulsive refutation of all biologic influence, but rather on the informed unmasking of ideological predilections masquerading as fundamental scientific insights. Without such a purposeful approach, the divisions between the social and medical perspectives will continue to deepen, and the potential for a shared agenda will be undermined. In such a fragmented setting, appeals to biologic determinism will gain credibility in policy, ultimately transforming the public perception of disparities in infant mortality from the inherently unjust to the merely inevitable.

Elevating Women's Health

Another major problem is the striking disinterest of the mainstream infant mortality research and policy communities in the health of women when they are not pregnant. In many ways, this lack of commitment stems from a central tenet of child advocacy that elevates the claims of the child above those of the parent.[49] Policy-based and programmatic approaches to infant mortality reduction have thus been confined primarily to the prenatal period.

With a tight focus on the prenatal period, the health of reproductive-age women has been addressed only to the extent that it is likely to affect that of newborns. The numerous programs designed to improve birth outcomes have invariably expressly focused on infant well-being, even though they all operate by providing services to women. In research as well, a tight focus on the direct intrauterine effects has resulted in a paucity of information about the impact of conditions both before conception and after delivery. Even the idea of preconceptual care refers to the infant.

We must recognize that, in some large measure, problems with infant ill health are a legacy of women's ill health generally. Cross-disciplinary investigations that can examine the interactions between the general health of women and childbearing are needed urgently. Measures that can adequately assess the interaction of risk, utilization, and chronic illness across women's reproductive experience are required. Issues of reproductive health, including contraception, sexually transmitted diseases, and abortion services, should no longer be dramatically separated from the mainstream of infant mortality investigation and programs. The adverse effects of unhealthy behaviors—such as smoking, heavy alcohol ingestion, and illicit drug use—on birth outcome should be more closely linked to why these behaviors were initiated and their direct effects on the health of the woman.

Expanded focus on women's health would also likely enhance the effectiveness of current prenatal care initiatives. The heterogeneous causation of VLBW suggests that singular therapeutic interventions are not likely to address this problem. Indeed, according to one study, almost three quarters of the racial disparity in VLBW deliveries was associated with conditions that generally preclude continued pregnancy once they emerge. More successful approaches are likely to lie in comprehensive strategies, particularly preventive efforts that begin long before pregnancy occurs.

We should recognize the growing proportion of all neonatal deaths caused by extreme prematurity and VLBW.[50] In many

communities, indeed in some states, half of the total racial disparity in neonatal mortality occurs in newborns with birthweights < 600 grams and gestational ages < 26 weeks. In most large-scale community-based prenatal interventions, by the time a woman recognizes she is pregnant, makes a prenatal care appointment, is seen and screened, and seeks a referral, a home visit or other similar intervention will on average occur close to 22–24 weeks of gestation. This places an enormous burden on prenatal strategies to achieve efficacy in a shrinking time frame. Clearly, with the epidemiology of disparate neonatal mortality shifting to lower and lower birthweight groups, an expanded commitment to women's health will only continue to gain importance. Moreover, even in terms of prenatal care, the best guarantee for the early initiation of such care is for the pregnant woman to have had a strong relationship with a high-quality health care system long before conception occurs.

In essence, an expanded commitment to women's health would help transform prenatal care from the first component of *child* health care to merely one component, albeit an important one, of *women's* health care across a lifetime. Such an effort would require both significant collaborations across disciplines and a greater willingness of those concerned with infant outcome to confront the central determinants of women's health, including gender equity and universal access to contraception, abortion, and all forms of preventive and therapeutic health care, regardless of pregnancy status.

Summary

The public debate surrounding disparities in infant mortality has resulted from a profound failure to seek a common wisdom. Because of its essential social roots, infant mortality will always remain the province of fundamental ideological and political conflict. However, without a more integrated analytic approach, progress in reducing disparate infant mortality will remain limited by internecine struggles for disciplinary purview and false claims of societal relevance. For in the end, the struggle to address disparate infant mortality will be advanced best by integrated technical and political strategies[51,52] that recognize that the pursuits of efficacy and justice are inextricably linked.

I developed this manuscript to accompany the article by Martha C. Hargraves in this supplement.

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Infant Mortality: Its History and Social Construction

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For 130 years, America has recognized infant mortality as a problem. However, methods of defining and studying the problem have changed significantly, including changes in who is studied within the population, what is studied (low birthweight or preterm birth), and how the problem is researched, whether demographically or medically. This article presents the natural history of infant mortality as a social problem as it was defined through the activities of three interest groups in the late nineteenth and early twentieth centuries. It describes the individuals involved—the winners and losers—and the process of turning claims into public policy, as well as the reasons behind the outcomes.

Progressivism. The first interest group influential in defining infant mortality as a social problem was the educated, middle-class American women involved in the Progressive movement in England and the United States. These women were the first to recognize infant mortality as a social problem. Needing ways to use their knowledge and energy, they became involved in the settlement house movement, which created homes for needy immigrant men, women, and children; developed links between the middle and working classes; and unified members of both these classes to fight for better conditions. The results included legislation to provide public health care and the development of government agencies, which regulated working conditions and created a healthier environment for infants and children.

Public health and medical professionals. Public health and medical professionals joined to yield a circumscribed view that continues to define the perspective of medical disciplines today. This group responded to the health care needs of infants and children by creating the field of pediatrics and by beginning to practice preventive medicine.

Black women. Finally, black women organized in response to racism, directed in particular at African Americans and expressed through segregation, an exclusionary device for ignoring groups or screening out causes and remedies inconsistent with the values and prejudices of the prevailing social order. Black women worked to overcome discrimination and increase awareness of poor conditions for African Americans, as well as further the public health agenda and educate and care for black populations.

The following pages describe how these three influential segments of the American population defined, measured, and responded to a condition that troubles us still: infant mortality.

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Progressivism

The English model. English sanitary reform significantly affected Americans. The social, political, and economic developments that generated reform in England were similar to those in the United States. The two countries shared a common liberal political tradition, and both had welfare systems originating in the Elizabethan Poor Law Statutes. Two schools propagated the rationale for these reform movements: (1) the social conservatives and romantic moralists, who interpreted the need for reform as a consequence of "moral" degeneration inspired by the "civilized" temptations of the city; and (2) the anti-industrialists and economic radicals, who blamed emerging industrial capitalism for dehumanizing workers, thereby creating poverty, ill health, and other social miseries.

Sanitarian ideas developed as a compromise between these two interpretations. While accepting that social progress could not proceed without moral progress, sanitarians reversed the moralist equation and constructed a rationale for government intervention. Conceding that education and moral influence were necessary if the new urban underclass was to escape its social miseries, sanitarians argued that both were nonetheless useless while the conditions in which that class lived were rife with physically degenerative influences.[1] They interpreted poverty and ill health as related to environmental and hygienic factors rather than moral or economic ones.

England's influence and America's response. The British conducted an extensive study of mortality rates among their population. The study's results, combined with statistics published by the General Register Office, fueled Americans' concern about their own urban health. Thus, in 1849, the American

Medical Association (AMA) issued a report showing that mortality rates in the nation's 10 largest cities were considerably higher than those elsewhere in the country.[2] Unfortunately, data collection methods were insufficient; mortality included all deaths reported in cities, with little comparable data available for rural areas. Maternal mortality was higher than expected, and the term *infant* often included children 0–5 years of age.

The American discourse differed from its English counterpart in several respects, however. For example, while Britons involved in the discourse on public health wielded considerable political influence and promptly institutionalized their reform ideas within centralized government agencies, American sanitarians were largely outside the political framework. Not until the 1860s did states and municipalities begin to form permanent boards of health, thereby giving American sanitary reform an institutional base. Moreover, American concern created little response until well into the twentieth century, and even that response was fractured by the lack of national coordination, since public health reform was primarily a state and municipal matter. In contrast, England's first National Board of Health, then the Privy Council, and finally the Local Government Board provided unity, direction, and impetus to sanitary reform and the development of public health programs at the local level.

America's discourse was also less comprehensive than its English counterpart. Almost entirely absent was the radical economic interpretation prominent in England. Not until after the Civil War did American sanitarians begin to consider seriously English theories that the poor could not rectify the conditions that influenced disease attending urban poverty and that a disproportionate amount of the excessive morbidity and mortality occurred among infants and young children.

Between the end of the depression of the 1890s and the conclusion of World War I, the Progressive Era occurred. Progressivism was not a single movement, but rather an endless variety of movements—the women's rights movement, the single-tax movement, the labor movement, the social justice movement, the antimonopoly movement, the Women's Christian Temperance movement, the Social Gospel movement, and the conservation movement, among others.

Moralism and idealism were mixed with realism, pragmatism, and "efficiency." Individualism jostled with nationalism; ideas of justice, with racism and nativism. Most importantly, citizens' groups—angry at poor public facilities, tax favoritism, and corruption—waged local campaigns against business privileges and excesses that laid the basis for reform in the next decade.[3] With this new era came the principle that America's children are her most valuable resource, an idea developed primarily by a group of middle-class white women.

Women and reform. Historically, maternal health and infant mortality were placed in the feminine domain in the late nineteenth century because of America's Victorian cultural norms, which restricted women to the work of "childbearing, charitable activities through volunteerism, and nursing the wounds sustained by both individual men and communities in battles for political and economic advantage."[4] Women were to be pious, pure, and submissive. If men tended to corruption while women tended to moral perfection, however, some women reasoned that the Republic needed them for other duties, as well.[4]

This rationalization provided women with the justification to escape domesticity and submissiveness and break into the public sphere. Because women were prohibited from voting and participating in political parties and business enterprises, they operated essentially from all-female volunteer groups dedicated to safeguarding the community from degradation. Organizations sent women as missionaries to the American West and China. Women taught elementary education, petitioned for temperance laws, attempted to save other women from prostitution, condemned slavery, published religious tracts, and founded religious groups. In their public lives, most of them avoided male-operated organizations to resist male domination.[5]

After the Civil War, middle-class women built large voluntary organizations and gained admission to higher education. They justified the need for education as necessary for improving their capability as wives and mothers. This claim provided an impetus to open such women's colleges as Mills College in 1852, Vassar College in 1865, Wellesley and Smith in 1875. During the 1880s, Harvard opened the Women's Annex, Columbia University opened Barnard, and Brown opened Pembroke. Concurrently, many American colleges and universities became coeducational, partly in response to women's demands for higher learning but mostly to create stiff competition for students. In 1870, one third of American colleges and universities admitted both women and men; by 1890, two thirds did. Before the turn of the century, women accounted for 36% of America's undergraduates and 13% of its graduate students.[6,7]

These institutions created environments where middle-class women could learn, be nurtured, challenge the typical circumstances of women, and create friendships. They emphasized a sense of duty that included humility, relationships, care, and service.[4] Upon completion of their education, however, most women were shocked at the lack of opportunity for professional careers. They either taught or returned to the family's requirements for social visitation and volunteer work.

Jane Addams and Ellen Gates Starr, who met at Rockford Female Seminary, were both disappointed in the thwarting of their personal medical ambitions. In response, they opened America's first officially recognized settlement, Hull House, in 1889 in Chicago. Others followed.

Settlements satisfied two needs of women who resided in the various houses established in Chicago, Boston, and New York. First, they allowed the young educated women who operated them to escape from limiting societal roles. Second, they provided a connection between middle-class white women and working-class immigrant women. Through gifts and fellowships sponsored by wealthy women patrons, Hull House created a new dimension for poor and working-class immigrant women and children as well as a new sphere of professionalization for educated women.

Lillian Wald's founding of the settlement for visiting nurses in New York City brought with it a specific focus on the needs of poor immigrants. Stunned by the poverty in the immigrant neighborhood, Wald quit medical school and, with another nurse, began a practice to provide nursing skills for patients who could not pay. Overwhelmed by the need, the women recruited more nurses, and, by 1900, 15 nurses and several residents staffed the Nurses' Settlement and made the Henry Street Settlement famous.[8] Wald was later credited with creating the first independent public health nursing service, establishing the independence of nurses from doctors, and defining nursing as a dignified profession for American women.[9]

By 1891, Florence Kelly, as a divorcee and mother of two

children, joined Hull House as a resident. She differed from other residents because, as the daughter of a congressman, she had the necessary knowledge to "play" the political games and acquire a place on the policymakers' agenda on behalf of women who worked in the state's sweatshop system. Hull House provided Kelly with the resources she needed to define a new position for herself and other women within American society. In 1892, the Illinois Bureau of Labor Statistics hired her to study the sweatshops in Chicago's garment industry, and, at the same time, the Federal Commissioner of Labor recruited her to survey Chicago's slums for a larger investigation of workers' living conditions.[10] Kelly, her fellow residents, and members of the Illinois Woman's Alliance (an organization for women's unions and voluntary societies) worked to persuade the state legislature to regulate the sweatshop industries[11] by publicizing their findings and lobbying at the state capital. In 1893, a law was passed prohibiting child labor, limiting women's work hours, and regulating conditions at the sweatshops.[10]

Historians have concluded that Florence Kelly, more than any other resident, transformed Hull House from a philanthropic organization into an engine of social reform, and her approach to social problems became the model for the settlement's reformers. Kelly taught them to investigate specific problems, arm themselves with facts, publicize the issues, offer solutions supported by public opinion, lobby the government for programs to enact the solutions, and then demand their own appointments to administer the new program. This was a route both to social reform and professional success for women.[12,13]

The Children's Bureau. The idea of an agency to oversee the needs of mothers and children became the primary focus of Kelly and Lillian Wald. They mobilized the network of women who aided them in other causes, including Jane Addams, and collected support from individuals working with Charities, the National Conference of Charities and Corrections, the General Federation of Women's Clubs, the Mothers' Congress, and the National Child Labor Committee. From these efforts came the Children's Bureau in 1912, undergirded by Kelly's claim that "[t]he noblest duty of the Republic is . . . so cherishing all its children that they . . . may become self-governing citizens. . . . The care and nurture of childhood is thus a vital concern of the nation."[13] The bureau was established in the Department of Commerce and Labor, with Julia Lathrop as director, to "investigate and report upon all matters pertaining to the welfare of children and child life among all classes of our people."[14] Among the issues suggested for study were "infant mortality, the birth rate, orphanages, juvenile courts, desertion, dangerous occupations, accidents, and diseases of children in the several States and Territories."[14] Thus, this act legitimized infant mortality as a social problem.

The bureau's work primarily targeted the immigrant population of the cities and middle-class women who did not work outside the home. Its programs included infant mortality studies beginning in 1916 among immigrants, the development of educational material on the care and feeding of infants, and the establishment of a birth registration program that launched a national campaign to register all births. The work of the Children's Bureau encompassed home visitation, education, and the training of professional women who worked with the bureau.

Some women welcomed the intervention of child-welfare workers, while others resisted and resented the intrusions. Resistance was located particularly in communities of African-American, Native American, and foreign-born women. A study of the female dominion between 1917 and 1924 shows not only the persistent interrelationship between reform and professional opportunities for women but also the racial and class identities that divided American women and empowered some at the expense of others.[4] Nonetheless, the Children's Bureau developed the support and power to develop and lobby to pass the first federal legislation to provide for the health care of women and infants, the Sheppard-Towner Maternity and Infancy Act in 1921, the first federal welfare program to precede the New Deal.

Professionalization/Medicalization

Women of the Progressive Era defined the problem of infant mortality and conceptualized it as essentially an urban problem, and, in particular, a poor immigrant problem, most visible in the Northeast and near western cities. How the problem was addressed was determined mainly by reform women, who "professionalized" the movement for social reform as a means to upward mobility. In doing so, however, they ignored the needs expressed by other subgroups and much of rural America to play a part in the definition of this problem. Their professionalization process was enacted through the mission of the Children's Bureau. This mission established the foundation for standards of practice that determined the quality of professional care to the nation's children. Additionally, it provided the women a base of power and scope of operation never attained before. They adopted a strategy of organized advocacy that Conrad and Schneider have referred to as "interest politics," an activity that encompasses "the promotion, directly or indirectly, of definitions of deviance that specifically support and buttress certain class or status interests."[15] White women in the reform movement defined deviance, or need, as contrasting with what they posited as "correct," normal, and appropriate for children's optimal development. However, the living conditions and sanitary requirements reform women prescribed as necessary for child welfare were often unattainable for poor women. For example, they defined as requirements for child health certain costly "baby-saving devices" out of reach for almost all of the women who needed them most. Consequently, many American women did not fit the mold the bureau developed and hence were labeled as deviant in the process.

Through the acquisition of scientific expertise in defining the problems facing mothers and their infants and of skills to manage the data that defined the magnitude of those problems, these white women developed universal standards of care in feeding, bathing, homemaking, and hygiene that systematized child care, home management, play, and care of orphans and delinquent and abandoned children. With these accomplishments, reform women successfully persuaded the public of the magnitude of the troubling condition and won its place on the public policy agenda. Moreover, they created, and hence captured control of, the labor market for child-welfare practitioners. Larson defines this accomplishment as professionalization, "the process by which producers of special services sought to constitute and control the market for their expertise."[15] Having established expertise to combat the problem, standards to reduce the problem, and data to measure its existence and change, these women succeeded in establishing welfare reform as a science and concomitantly a profession over which they claimed control.

In contrast, public health practitioners and the medical profession explained the existence of the problem of infant mortality by blaming the environment—impure air, contaminated food, waste, and a lack of parental nurturing on the part of the poor. The idea that some universal law demanded a certain number of infant deaths and the imprecision in data collection regarding infant mortality played a role in the lack of public health response. Further, the distinctive health needs of women and children were not acknowledged. In 1873, William Clendenin told the American Public Health Association that "there are not general causes existing and affecting persons of one age, that do not exist at every other period of life."[16] Clearly, Clendenin provided an excuse for continuing to neglect the needs of women and of their infants.

Thus, American public health reformers entered the last quarter of the nineteenth century increasingly convinced that infant mortality was a pressing urban health problem, yet uncertain whether sanitary engineering and regulation or parental education would cause more results. Throughout the 1870s, they complained that taxpayers, landlords, and corrupt municipal officials blocked every effort they made to lower the infant death rate through improving the housing conditions of the poor. Furthermore, they lamented what they believed to be the poor's persistent apathy about improving their own condition or observing even the most basic rules of hygiene and sanitation. The American public health reformers wondered how the problem could be reduced without fundamentally reordering urban social and political configurations and effecting massive behavioral change.

As the last quarter of the nineteenth century began, American public health officials regarded the levels of infant mortality prevailing in their cities with concern heightened by a sense of impotency. Having refined the definition to include only those infants younger than one year and having made that age reduction a measure of their sanitary program's effectiveness, they found the still high infant mortality rate frustrating. As a consequence, they hesitantly began to move toward treating infant mortality as a health problem that demanded special, separate measures.[17]

The construction of a new medical specialization. While women in the settlement house movement were developing and marketing their unique professional skills to care for the needs of women and infants, pediatrics had generated enough professional interest and won enough scientific respectability for the AMA to create a special section; thus, in 1887, 43 of the most prominent American experts on infantile and childhood diseases formed the American Pediatric Society.[18] The group believed that infants and children, rather than the diseases from which they suffered, were special; this pronouncement profoundly affected the development of pediatrics as a unique medical specialty. From the beginning, pediatrics was a contradiction: a holistic specialty.[18] Because the initial focus of pediatrics was largely preventive, the expertise that pediatricians could claim was in the broad, nebulous area of infant and child management.

To define and legitimize their specialty, pediatricians were forced to demonstrate that the successful rearing of infants and children and the prevention of morbidity and mortality among them required specialized medical knowledge. The earlier writings of William Cadogan, particularly his "Essay Upon the Nursing and Management of Children," clarified the new specialty's belief that, for the sake of the young, their rearing should become the "care of men of sense." He declared, "this business has been too long fatally left to the management of women, who cannot be supposed to have the proper knowledge to fit them for such a task, notwithstanding that they look upon it as their own province."[19]

The eighteenth-century and early nineteenth-century physicians' goal for infant and child health was the "medicalization" of activities that traditionally had been the responsibility of mothers and nursemaids. This too was the goal of late nineteenth-century physicians in their effort to legitimize pediatrics as a medical specialty. To accomplish it, they not only had to argue that infant-rearing and child-rearing required medical expertise, they also had to convince their own profession that such specialized expertise was beyond the purview of the general practitioner or other specialist. To succeed, pediatricians needed to focus their attention on infant and child management, contending that successful execution demanded the utmost in medical skill and training. The emphasis was on infant feeding. As pediatric historian Fielding Garrison observed, it became the central focus of the specialty in the late nineteenth and early twentieth centuries and provided pediatricians with an area in which they could demonstrate their specific scientific expertise.[20]

The incorporation of women's, infant, and child health into the medical profession led the medical profession to take an interest in the Sheppard-Towner Act, which was opposed by conservative members of Congress, who raised the issues of states' rights and fiscal irresponsibility. The Anti-Suffrage Association warned that the bill exerted bureaucratic control over family life. However, the law's most ardent opponents were members of the medical profession.

Arguments from the medical professionals primarily focused on the socialization of medicine and interference in the doctor-patient relationship. However, medical professionals were divided. R. A. Meckel observes, "As late as December 28, 1920, the American Medical Association's (AMA) own Committee on Health and Public Instruction went on record as clearly favoring the bill. Moreover, virtually all women physicians, most prominent pediatricians, and a few leading obstetricians also favored the bill. And, at least according to one historian, the rank and file was largely apathetic."[17]

From 1922 to 1926, medical opposition grew stronger because the original bill was enacted to cover the period through 1924, and its opponents knew they would have a second chance of defeating the legislation. The AMA House of Delegates had become predominantly conservative by 1922 and launched a press campaign against the act. At the same time, the AMA section on the diseases of children declared its support for the act and released the text of its declaration to the press. Enraged, the House of Delegates formally reprimanded the pediatricians and adopted a rule prohibiting sections of the AMA from independently passing resolutions or taking stances on social matters or issues.[21]

Changes in the structure of the medical profession had occurred by 1926 that further decreased support for the Sheppard-Towner Maternity and Infancy Act:

> While well-baby and prenatal stations were once training grounds for physicians, clinical education had moved into the hospital, reflecting little need for this resource. They

thus became places of employment for physicians who held marginal status within the profession. The physicians were primarily women who were directing and providing care in the Sheppard-Towner–funded centers.

The character and scope of practice among physicians also changed. Somewhere in the mid-1920s, physicians began to see more patients in their offices reflecting a shift from sick patients to healthy patients. Therefore, they began to make advanced appointments and give routine examinations, and provide preventive health care and instruction in personal hygiene. Hence, it became the opinion of many strong leaders in the medical profession that the work of the Sheppard-Towner Act provided duplication and an intrusion by the state into private practice.[22]

Thus, the success of the Sheppard-Towner Act in changing the way in which medical care was delivered also contributed to its demise. Other influences, including the decreasing unity in the women's movement, contributed to the eventual repeal of the Sheppard-Towner Act as well:

The women's lobby was weakened after elections of 1924 when women did not vote as a cohesive group. Women who did vote often split their votes in much the same way as men did: most flocked to the Democratic and Republican parties, while some backed candidates from the Progressive and Prohibition parties.

Women shared many economic, political, and social disabilities, but their interests remained divided by class, race, and ethnicity as men's did.

Though women had been active in the political process, they had remained outside the back-room negotiations due to their adoption of a moral stand on deal making in politics. Because of this stand, politicians no longer feared women as active participants in the political process.[4]

These shifts in political involvement played important and deadly roles in the repeal of the Sheppard-Towner Act. In 1924, opponents gained strength from a variety of sources, including a Congress committed to budget-cutting and an administration as devoted to the interests and values of business as any administration in U.S. history.[4]

By 1927, the act had been funded for five years. On June 30, 1927, the Senate agreed on a compromise to extend the act for two years. The act was then repealed on June 30, 1929.[23] The repeal of Sheppard-Towner provided the victory needed to further institutionalize the medicalization of motherhood.

Historical and Social Origins of White Racial Attitudes Toward Black Health

Historically, the poor state of African-American health is partly the product of racial attitudes and policies about this group. Slavery created an image of black people as mere instruments for the economic well-being of slaveholders, thus giving black health no significance outside its relationship to the expansion of white wealth. By the 1870s and 1880s, southern policy promoted legal separation and inequalities in service provision. Acts of racial violence went unaddressed. Lynching reached an all-time high in the South by 1892, with 235 that year.[24,25] Institutionalized racism in the North confined blacks to the worst housing and jobs, which ravaged their health.

To force southern blacks to return to the land, many former slaveholders excluded blacks from city health and welfare services. Some white political leaders admitted that the terrible living conditions of blacks contributed to their high death rates. Others blamed the "inherent weaknesses" of black people as a race. However, the head of one southern city board of health rejected the "race constitutional defects" argument and made it clear that the high rate of black deaths during the period was due to poverty and overcrowding.[26]

One historian notes that "[a] larger number of urban Negroes struggled unceasingly to maintain moderately clean and healthful homes in marginal buildings. But many others, forced into some of the poorest housing the city had to offer, eventually stopped striving or caring for anything save survival."[27] Thus, many began the slow decline into social pathology that would come to characterize much of black urban life in the twentieth century.

Blacks were among the poorest people in the nineteenth-century cities. Poverty and racial discrimination confined free blacks to certain sections of the cities, often exposing them to epidemic diseases such as cholera and smallpox. Most observers and health officers at the time identified poor housing conditions as a "major factor contributing to high [b]lack mortality rates."[27] As early as 1820, observers noticed the relationship between the quality of black housing and its impact on black health when Baltimore officials recognized areas, "tenanted by Negroes, and divided by an alley . . . a collection of huts and filth," as major health hazards. Thirty years later, the City Board of Health condemned the "cellars, wretched hovels" occupied by blacks and characterized them as "replete with all that was calculated to engender disease."[27]

In addition to inhabiting unacceptable housing, blacks held the hardest jobs. The slave system had institutionalized an economic value system based on the productivity of blacks, including black women, and allowing high rates of black infant mortality as an acceptable and necessary cost. The postslavery period, while freeing blacks from the domineering and overworking hand of the slaveholders, brought them the desperate poverty that continued to require high productivity for survival and provision for their families. Most blacks suffered from "nutritionally inadequate diets . . . especially women of childbearing age; and the heavy physical labor required of washer women and domestics all contributed to the lower fertility rates within urban settlements. For poor people, the various hardships related to city life conspired against proper prenatal care and led to miscarriages and spontaneous abortions."[28]

Understandably, black leaders, physicians, and scholars felt the greatest pain for the most helpless victims: black women and children, especially the stillborn. A black physician observed:

As to the still-births: why should we be surprised at the great number of still-births among our women, since they do most of the work that is liable to produce this state of things? They do the cooking, the sweeping, the lifting of heavy pots; they carry the coal, the wood, the water; they carry heavy burdens on their heads; they do heavy washing, make beds, turn heavy mattresses; and climb the stairs several times during the day, while their more favored [w]hite sister is seated in her big armchair, and not allowed to move, even if she wanted to.[28]

W. E. B. Du Bois also observed that urban black poverty and misery culminated in high death rates, listing "poor heredity, neglect of infants, bad dwellings and poor food. . . . The influence of bad sanitary surroundings is strikingly illustrated in the enormous death rate of the fifth ward—the worst Negro slum in the city of Philadelphia and the worst part of the city in respect to sanitation. . . . "[29]

After the Civil War, American writers and scholars began to speculate on the fate of the black race, with its health and economic problems, providing theories that the race would soon disappear and that involvement in the health of black individuals wasted valuable resources. While many whites did not care about or ignored the increasing struggles of blacks, problems grew worse. Du Bois wrote in 1896:

> The most difficult social problem in the matter of Negro health is the peculiar attitude of the nation toward the well-being of the race. There have, for instance, been few other cases in the history of civilized peoples where human suffering has been viewed with such peculiar indifference. Nearly the whole nation seemed delighted with the discredited census of 1870 because it was thought to show that the Negroes were dying off rapidly, and the country would soon be well rid of them. So recently, when attention has been called to the high death rate of this race, there is a disposition among many to conclude that the race is doomed to early extinction; there is little left to do but moralize on inferior species. What the Negro death rate indicates is how far this race is behind the great vigorous, cultivated race about it. It should then act as a spur for increased effort and sound upbuilding, and not as an excuse for passive indifference, or increased discrimination.[29]

Unfortunately, the poor quality of black health did not "act as a spur for increased effort and sound upbuilding" for black health.

In 1906, Du Bois published *The Health and Physique of the Negro American,* in which he described the health status of blacks: "In 1890, the death rate per 1,000 living was 27.4 for the Negro and 19.5 for the white; in 1900, the figures were 25.3 and 17.3 respectively."[30] Mortality among black infants was particularly high: 494.27 of every 1,000 living black children under one year of age in 1890 died as compared with 249.38 whites, with figures altering in 1900 to 371.5 blacks and 158.0 whites.[30] Du Bois summarized:

> In 1900, there were 1,467 babies born in Philadelphia and 25 per cent died before they were one year old. Of every five persons who die in a year, two are children under five years of age. The diseases of cholera infantum, inanition and marasmus, which are simply the doctor's way of saying lack of nourishment and lack of care, cause many unnecessary deaths of children.
>
> The undeniable fact is, then, that in certain diseases the Negroes have a much higher rate than the whites, and especially in consumption, pneumonia and infantile diseases.[30]

He concluded his report by saying that "the high infantile mortality . . . is not a Negro affair, but an index of a social condition."[30]

Black Women and Reform

African-American leaders' aspirations had been raised by the Emancipation Proclamation but then dashed by changes brought on by urbanization, industrialization, and immigration. These changes brought with them continued repression; therefore, scholars like Du Bois, Booker T. Washington, and an extraordinary group of African-American women began to forge a new direction of self-help for their people.

In the last decade of the century, it was clear to African Americans that they must work out their own salvation in a hostile environment and that blacks must be united in their efforts at racial elevation. This emphasis on self-help and solidarity stressed the economic approach, but it was applied to all efforts. The philosophy espoused by Alexander Crummell of separating blacks from the institutions of whites became the method used to emphasize and create self-esteem and racial pride.

This new agenda of self-help and racial solidarity created black businesses and educational institutions and built up the church as an integrated social institution within the black community. Accompanying this emphasis on racial pride came an interest in racial history born out of a need to (1) assert and prove black equality with whites as one means of convincing whites of blacks' worthiness of political and civil rights and (2) give themselves a sense of dignity and pride of race to offset the doctrine of black inferiority espoused by whites.[25] Out of these efforts came a kind of cultural nationalism that brought an unprecedented commitment to the betterment of all blacks from within the black community itself.

The prevailing definitions of black women and of blacks in general during the nineteenth century served as social control mechanisms over them and their communities. However, black women were brought into the forefront of the struggle for black and women's rights by the movement begun by Ida Wells with her antilynching campaign in 1893. Wells was forced to go to England to have her claims against racism acknowledged. "It is idle for men to say that the conditions which Miss Wells describes do not exist," a British editor wrote. "Whites of America may not think so; British Christianity does and all the scurrility of the American press won't alter the facts."[31]

A few years later, black women came together nationally to establish in 1896 the National Association of Colored Women's Clubs (NACW) to begin the process of self-definition and self-valuation that revealed their powers in executive organization and administration of the affairs and needs of their communities. Their work resulted in the establishment of kindergarten classes in the public school, child care programs for working mothers, fund-raising for the establishment of hospitals, and nursing programs, and it provided the community network and infrastructure on which the black community could build.

According to Linda Gordon, the black women's club movement shared common elements with the white women's club movement. The two groups were organized in much the same way. Membership consisted mostly of middle-class educated women; neither group questioned the superiority of middle-class values. High on both organizational agendas were social reform, aid to the poor, and self-actualization. The choice of those served distinguished the missions of the black and white movements.[32] Mary Church Terrel, one of the wealthiest and best educated black women of her time, declared: "Self-

preservation demands that we go among the lowly, illiterate, and even the vicious, to whom we are bound by ties of race and sex . . . to reclaim them."[33]

While education was an important priority of black women in this period, health was equally important. Susan L. Smith put it best when she said, "[I]f historians had considered the organized social welfare activities of African Americans, especially women, they would have seen a significant part of the indigenous roots of southern public health work."[34] The work of Lugenia Burns Hope illustrates this commitment. In 1908, Hope and women of the now Atlanta University Center came together to provide services in all the black community's 16 residential districts in Atlanta, Georgia. This federation, named the Neighborhood House, opened its doors in 1911 as the first black settlement house in the country.[35] Its projects included a survey of the conditions in black neighborhood schools in 1913, which documented the overcrowded and unsanitary conditions of schools, brought pressure on the Atlanta school commissioners, and caused changes by city government in 1914.[35]

Health care was the next issue, addressed by a house-to-house survey of neighborhoods. Tuberculosis was a major problem, and, in 1914, Hope brought officials of the local antituberculosis association together with black leaders to combine efforts toward changing blacks' living conditions. She proposed establishing a clinic to provide direct services and health education, and in 1916, the Neighborhood Union Health Center opened its center free of debt and with sufficient staff to offer a full range of services.[36] The clinic expanded rapidly to become a model for black communities in other southern cities.

In addition, in 1919, Hope began using the clinic to train neighborhood workers in home nursing, child welfare, and care of the mentally infirm, thereby expanding the scope of the clinic programs and helping residents acquire new skills and participate in the self-help movement. Hope and her staff promoted the program by appearing in community churches, schools, and lodge halls. Services were provided where the clients were: playgrounds, homes, and streets. By 1919, "Hope sent 143 visitors into 5400 homes. Their contacts, plus those . . . in schools and other places, reached 45,000 people, nearly three-fourths of Atlanta's [b]lack populations."[37] The strategies used by Hope and her partners were duplicated and transferred to other black communities throughout the South through the NACW Clubs and provided a ready model for health and social welfare work within the black community.[38]

The dominant social forces and decision makers did little to facilitate the approaches developed by African Americans during the early nineteenth century on behalf of their communities. Black middle-class women were dismayed that their white counterparts questioned the morality of black women. Black women's experience under slavery was held against them as a link to shame and degradation.[39,40] The partnership between the middle and lower classes of black women was based on the recognition that their fates were inextricably tied together and that the success of their efforts of social betterment were dependent on each group's contribution, that the real struggle, as Anna Julia Cooper said, was "the painful, patient and silent toil of mothers to gain title to the bodies of their daughters."[40]

Although some black women expressed concern about their own class within the movement, the black clubwomen as a group had a different attitude toward class and the poor than did the society in general, as expressed by the focus of the white women's movement. Black women had learned through the lessons of their own lives that it was opportunity and environment—not circumstances of birth or previous experience—that separated them from the masses.[33] Josephine St. Pierre Ruffin's opening statement at the Boston convention depicted the commitment black clubwomen felt toward all black women when she said the movement was "created not for the sake of fine, cultured women," but for "the thousands of self-sacrificing young women teaching and preaching in lonely southern backwoods, for the noble army of mothers who have given birth to these girls, mothers whose intelligence is only limited by their opportunity to get books."[41] Therefore, the efforts of black women were directed toward providing the same options to their communities that were available in the dominant society. They believed that by providing those options, black communities could respond to all their community's deficiencies, including deficiencies in the health of mothers and infants.

White women of the reform era were mostly unable or unwilling to confront the social inequities created by segregation and racism. They, therefore, prescribed standards of care for mothers and infants that ignored the social context of the time and fragmented the communal approaches fostered by black women of the era.

If the community mobilization and community development fostered by black women could have been included in the definitional process, the result would have been programs that better accommodated the pluralities reflected in America's social system. Further, the approaches favored by black women offered support at the neighborhood level and established a workable system of participatory governance, which, if included in the reform movement, would have increased the integrity and status of American women by helping all of them identify and respect the strength associated with diversity. Collins suggests that black women provided an alternative humanist vision of societal organization.[42] This alternative worldview put forth a communal alternative to medicalization that translated human need into community responsibility, and appreciation of human likeness and a respect for differences, acceptance of race pride as a necessary element to self-help and progress and respect for the rights of those affected to define remedies for their problems as participants in the decisions of policy. Our history tells us that the bias about the "race question" marginalized the efforts of African-American communities to empower themselves and the larger community.

CONCLUSIONS

The evidence on the nature of the problem of infant mortality was produced, evaluated, and disseminated by white women in the reform movement. With the evidence they produced, they defined the tenets of the problem. Driven by two agendas, professionalization and social commitment, their efforts resulted in the exclusion of African-American women and children from equal engagement in the definition of the problem. Differences in community norms, as typified in Figure 1, rendered African-American women "invisible" within the context of decisions

MOVEMENT Participants	DEFINITIONAL Claims	ORGANIZATIONAL BASE	METHODS	PROGRAM FOCUS	GOVERNANCE	SOCIAL PROCESS	OUTCOME OF CLAIMS
MEDICAL REFORM MOVEMENT Medical Profession	Abnormal Maternal Physiology	MEDICAL SOCIETIES Local, County, & State	CLINICAL JUDGMENT Physical Examinations	TREATMENT OF DISEASE	MEDICAL CONTROL	Re-Federalization PUBLIC HEALTH SERVICE	SUSTAINED
PROGRESSIVE CHILD WELFARE MOVEMENT Middle/Upper Class White Women's Clubs & Settlement Houses	Unhealthy Social Environment	CHILDREN'S BUREAU	SCIENTIFIC JUDGMENT •Aggregate Data Collection •Standards of Practice	IDENTIFYING SOCIAL/ ENVIRONMENTAL NEEDS	PROFESSIONAL CONTROL	Federalization SHEPPARD-TOWNER ACT	ABDICATED
NEIGHBORHOOD REFORM MOVEMENT Middle/Lower Class Black Women	Neglect	NEIGHBORHOOD CLUBS	COMMUNITY-BASED SURVEY DATA •Church-Based Missions •Self-Help Groups •Community Organizations	DEPENDENT ON COMMUNITY'S JUDGMENT OF NEEDS	LOCAL CONTROL	COMMUNITY MOBILIZATION	MARGINALIZED

Figure 1. Conceptual distinctions among definers in the process of constructing the problem of infant mortality.

and services affecting the larger society. They were not a part of the national decision-making process.

As well as being unwilling or unable to oppose the prevailing policy of segregation, white women reformers were wed to their methodology. Typified by the categorical and scientifically derived policies of the Children's Bureau, their methods were determined, and restricted, by their worldview, which excluded the community of concerns valued by African-American women. Reform women failed to recognize that, in the words of Martha Minow, perspectives are partial and hence only reflect one view.[43] They could have best served the needs of African Americans by seeking alternative views. By acknowledging the claims of black women, reform women might have developed a different approach to the problem of infant mortality by coming to understand it within the context of the black communal experience, which means within the context of the systemic disadvantage created by segregation and racism. White women involved in welfare reform defined standards of maternal and infant health based on the willingness of women to conform to a set of individualistic middle-class Victorian standards, which assumed poor women had the same values, means, and opportunity as their white middle-class counterparts. African-American women's concept of maternal and child health embraced the larger communal issue of segregation and racism, seen as the filter through which opportunities, services, and influence were provided, or denied, to blacks regardless of their relative poverty or wealth. Limited by their social assumptions, white women in the reform movement did not hear or understand the voices of their black sisters. They were therefore unable to develop more comprehensive models of health and welfare reform in partnership with black women, models that could have included the knowledge that black women had gathered from their experiences within their own community and within the country at large.

Organized medicine benefited not only by the structure that the professional and legislative accomplishments of reform women created but also by the division among different groups of women. Further, the influential role organized medicine had in Congress placed physicians and public health officials in a better bargaining position within the structure than women as a whole. Figure 1 illustrates the social process enacted by participants in each movement and shows how the medical profession's privileged access enabled them to recast the elements of the Sheppard-Towner Act favorable to them and secure their placement under the control of the U.S. Public Health Service. Ultimately, this accomplished for medicine the definition of infant mortality as a physiological problem and sustained it within the realm of medical care and organized medicine.

Black women's claims were built on a communal interpretation of the interrelatedness of all the problems affecting the progress of the race—an aggregation of the whole that liberal individualism or reductionist medical science could not successfully disaggregate. Black women's claims were based on the relationship of health to education, social opportunities, moral character, cultural values, and general social betterment. This set of values continues to have credence within the African-American community and suggests three conclusions: (1) that the traditional reductionist empirical approach to the study of infant mortality restricts the options afforded by other more comprehensive social approaches; (2) that problem definitions can narrow and limit horizons, erect barriers to alternatives, limit solutions, and thereby sustain existing patterns of expertise (and authority); and (3) that taking into account the social and historical context of a problem—the interplay of avowed, disavowed, or marginalized claims—can allow us to design programs that will have a more sustained impact.

The continued disparity of birth outcomes in African-American communities requires looking at this troubling condi-

tion through a new perspective, with broader visions, including in the design of programmatic responses the claims and experiences of those most directly affected by the problem. As history has already demonstrated, they will have better answers than we.

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Community Research: Partnership in Black Communities

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Finding common ground for forming relationships between researchers and a particular community can increase the relevance of research as a resource for setting priorities and developing strategies for change tailored to the interests and needs of the community. In the 1990s, research is expected to increase in U.S. populations that experience a disproportionate burden of disease, disability, and premature death. James Mason, former assistant secretary for health, has argued that redoubling research efforts in minority communities is essential for achieving the Year 2000 Health Objectives.[1] The potential benefit is also greatest in higher-risk populations. Since large proportions of preventable diseases and deaths are caused by factors that can be changed by motivated individuals supported by friends, family, and community, progress toward reaching the Year 2000 Objectives will require greater insight by researchers into the behavior, motivation, and relationships of people at the grassroots level.

Collaboration is especially important when each party has a stake in the design of studies and the orientation and interpretation of research findings—for example, research on the academic achievements of black youth, investigation into drug abuse among black women, and studies on the causes of poverty. The importance of this collaboration also arises from the fact that if those who form hypotheses are from different cultures from those being studied, ambiguity resulting from a limited understanding of local culture can cause a biased interpretation of observed behavior. For example, a study on health care systems in rural Georgia found that the perceptions of the numerically small but powerful upper middle class defined the health problem and thus directed the selection of the intervention.[2] Further, observation of religious worship services, parenting, courtship, youth gang rituals, and other social behavior in ethnic and minority subcultures has resulted in biased interpretations of the meaning of these activities in the lives of those being observed.[3,4] Bias has important implications concerning how well a program will be accepted in a community.

Community insiders are certainly experts in the social meaning of disease and can help researchers identify relevant issues, causal mechanisms, and implementation of acceptable interventions.[3,5,6] Behaviors that appear chaotic and irrational to outsiders may seem rational and normal to the insider.[4] Because the social meaning of events can often be understood best within the context of the history of a community,[7] members of the community are best capable of evaluating and communicating that meaning.

This article explores four potential models for collaborative research in black communities in the United States and their attendant challenges to the community and to the scientific approach. We present the perspective of health educators, research scientists, and community activists who have an interest in community-based research.

Models of Community Research

Throughout this discussion it is important to keep in mind what we mean by "community," in particular the black community. The "black community" as a unit of identity for black Americans is *not* the same as geographic/demographic clusters in which the majority of the people are black. Places demarcated by natural or psychological boundaries serve as a common core of commercial and human service organizations, churches, and schools, and, where the population is all black or predominantly black, are called black communities. However, community can be defined also by shared interests, common fate, social and political history, and cultural affinity. Blacks share a bond to the history, ethos, and institutions that form the heart and soul of the identity known as the black community. Even though black communities are by no means homogeneous geographically, the experience of being black in America has produced sentiments and experiences that have molded a sense of "peoplehood." A sense of peoplehood, formal or infor-

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mal ties to religious and social organizations, and family conceptually define the black community for the majority of African Americans. Researchers seeking community models must consider both the geographic boundaries of the black community and the sense of cultural identity that also unites it.

These black communities may be poor, working class, middle class, upper class, or of mixed socioeconomic status. Their social organization ranges from organized and stable to disorganized and transient. Styles of leadership, patterns of social organization, and internal coherence will vary in accordance with past and present events and traditions within the community and in the broader society.

The challenge for researchers is to design models for collaboration that maximize the potential for community participation. In the first model, the persons the researchers consult for advice and consent are at the periphery of community cultural systems; often they work for human service organizations and usually live outside the community. In such instances, the research is community based but fails to achieve optimal involvement because the people of the community remain unaware of the purpose of the research and have no influence on research design.

In the second research model, researchers identify influential people within community cultural systems (e.g., churches, clubs, fraternal orders, and civic associations) to whom they explain the research design and from whom they request endorsement and cooperation. The researcher retains total control of the project. This model is community based, but the community role is essentially passive.

In the third model, researchers contact influential community leaders to explain the research and to ask for support, advice, and guidance in hiring community people as interviewers, outreach workers, and screeners. These influential people may be invited to serve on an advisory board. This model is community based but not community involved, since community members do not contribute to the design of the research nor do they have a significant role in interpreting findings. This model achieves greater community involvement, but its potential for manipulating communities is greater, as well, because those selected for employment with the project are often trusted and influential members of the local cultural systems.

An example of this third model is a community-based program designed and conducted in 1985 in East Baltimore by the Johns Hopkins medical institution and community leaders. This program was designed to control hypertension and related cardiovascular risk factors in a predominantly black inner-city population. A task force that included representatives from city hall, churches, schools, the city health department, and Johns Hopkins Medical School provided oversight. During the time period of the program, hospitalization for hypertension declined by 30%. Much of the success of the model was attributed to the recruitment and training of community residents as certified health workers skilled in community outreach, counseling, and monitoring blood pressure.[8]

Another example of the third model is a study begun in 1978 by health educators at the University of North Carolina. The goal of this community education project was to increase use of health care resources for control of hypertension and diabetes. The intervention called for training community-selected volunteers as health advocates who would share information with others in their social networks and play a facilitating role in linking individuals with resources. Project staff asked for community input, especially from those in leadership positions in black churches, to help define project objectives. These discussions resulted in expanding the intervention to include rodent control in one neighborhood; in another community, people insisted on changing traffic flow at a dangerous railroad crossing. Two years after beginning the project, an evaluation indicated higher levels of knowledge about the cause and prevention of the targeted diseases. Competency in reaching out to health professionals and others able to help in solving local problems increased. Before the health project, the vast majority of community members had never made demands to local governments or service agencies. The project directors believe that acquisition of skill in self-advocacy will eventually have a greater impact on the overall quality of community life than on the disease-specific conditions that stimulated the intervention.[9–11]

Two potential pitfalls beset this third model. One is the process by which community advisers or partners are selected. They may not represent the community as a whole but only special interests. The second pitfall involves the manipulation of community members who could sanction unethical or unscientific projects, unknowingly. These projects could then escape critical scrutiny and perhaps receive endorsement from local and broader-based black-interest advocacy groups. Poor communities might perceive employment of a few people as justification for research, without assessing the research program's possible benefits and liabilities to the community as a whole.

The fourth model is best suited for research in black communities because it both involves and enables the community. In this model, the researcher seeks community assistance in setting the direction and focus of the research. The problem to be studied is defined and the research design is constructed after the community to be studied is consulted for its definition of the problem, its analysis of contributory factors, and its potential solutions. The research design enfolds the experiences and knowledge of the people who will be studied rather than bypassing them in favor of a research agenda defined solely in relation to scientific protocol. The community thus becomes a collaborator in the research. The community also negotiates, as a collaborator, the goals of the study, the conduct of the study, and the analysis and use of study findings. Community members know beforehand what to expect from their research efforts as collaborators. Therefore, this model becomes not just community based but community involved, as well.

An example of a community-university project exhibiting these characteristics was carried out by Hatch et al. at the University of North Carolina School of Public Health in the early 1980s. The project was funded by the American Heart Association (AHA) to test the benefit of aerobic exercise on cardiovascular fitness in a population of black women. The researchers' primary goal was to understand how to gain sustainable community participation in cardiovascular fitness activities; the participants' primary goals were to feel better and look better. These multiple aims did not conflict, but the dual agenda required more time to conduct the research than research focused solely on the researchers' objective of cardiovascular fitness. The AHA's evaluation followed a biomedical protocol. The researchers were interested in the psychosocial dynamics of gaining participation and sustaining involvement. The measure of success for the participants was their ability to

achieve a reduction in waist size. The participants organized a fashion show at local churches to show off their achievements. The three-year funding ended in 1988, but services initiated through the project have continued through volunteer fitness leaders and an advisory board of community members and health professionals.[12]

Challenges to the Black Community When Forming a Research Partnership

The community must act to protect its own needs and interests in any research collaboration, including selecting a research problem that has social significance for the community, assuring the physical safety of community participants, preventing socially damaging uses of scientific data, and assuring long-term social benefit to the community by establishing a community-based infrastructure to continue interventions.

To implement community-research partnerships, we need strategies for understanding the internal functioning of the microcommunity (local) in the context of its relationship to the broader, macrocommunity (macro) of black people. Identifying uniqueness at the micro level requires insights best acquired when local people are involved in setting the research agenda.

Interests of the researcher and the community are likely to differ. The community is most likely to be attracted to the potential of using research to solve immediate social problems, whereas the researcher and the funding agency seek information for scientific or policy purposes. Investigators who can include community issues in their research agenda will be more likely to find support in the community. In those black neighborhoods that have been of particular interest to public health researchers, most residents are aware of the need to improve their quality of life and of the helping role science can play. However, the community should reject research narrowly focused on scientific issues peripheral to the concerns of the people because scarce resources and the finite energy of leaders should not be used for pursuits that do not directly benefit the community. Ignoring local community concerns is likely to compound the sense of frustration that is all too often the by-product of unrewarded collaboration. A related problem, which both scientists and the local community need to address, is the tendency to design research that fails to look beyond the individual and the local community to the broader issues of social policy, values, and the equitable distribution of public goods and services, such as education, health care, law enforcement, and recreation.

The challenge of protecting study populations has been fairly effectively dealt with by organized science, as represented by the National Institutes of Health, the Centers for Disease Control and Prevention, universities and medical schools, and similar organizations such as hospitals and research institutes. Institutional review boards, human subjects committees, and explicit policies on the part of funding organizations help reduce the potential harmful effects of research, encourage informed consent, recognize confidentiality, and, most recently, require samples that represent the population's characteristics (gender, race, and ethnic origin).

Many members of the black community, however, still distrust scientific research directed at black people. The Tuskegee study is often cited by those who warn against cooperation with researchers. That study of untreated syphilis in black men is the longest nontherapeutic experiment in American medicine.[13] Study participants were intentionally harmed because they were told that they were receiving treatment for "bad blood" but actually were only undergoing diagnostic tests to determine the effects of syphilis.[14] Even now, in some black communities, one third of the population may believe that acquired immunodeficiency syndrome (AIDS) is a form of genocide.[13] In a social context of inequality and neglect, human immunodeficiency virus as a weapon of racial warfare is believable to some people.

Data interpretation is a major source of concern for the black community. Biomedical, social, and behavioral sciences research has been used to support negative stereotypes of racial inferiority and to justify racial separation and social neglect. Examples are Jensen's research on black children,[15] Shockley's work on intelligence,[16] and the Moynihan report on black families.[17] These studies were variously driven by the beliefs that group capacities to learn are predetermined by race, biological endowment is racially determined, failure to thrive is grounded in group pathology, and environmental influences explain few differences between blacks and whites in educational achievement, health status, and income.

Research projects that leave no organizational structure capable of continuing beneficial activities in the community contribute to the community's distrust of the research establishment. For example, when the funding for the Johns Hopkins/East Baltimore program ended, the research staff abruptly withdrew. A year later, the advisory board stopped meeting, and the overall activity declined. There was no community-based organizational structure capable of independently continuing the program after the researchers withdrew. After more years of work to refine the project model with community input, a relationship based on trust between the community and the university is now developing.

In 1986, a potentially effective approach for developing long-term community capacity began when the Henry J. Kaiser Family Foundation encouraged communities across the nation to pursue risk reduction/health promotion projects suited to their interests and special needs.[18,19] Announcements about the program were broadly distributed, resulting in hundreds of requests for support. Planning began on the initiative of microbased or macrobased organizations. However, the foundation required organizations to seek cooperation and participation from other organizations with an identified or potential interest. Kaiser apparently wished to assess the efficacy of community-initiated and community-sponsored health promotion as a means to change national health care priorities. The foundation committed $15 million to the project and invited other foundations to join in supporting the effort. Evaluation of this major effort is still in progress; publication of findings is expected within the next year.

During the start-up phase of these projects, the Kaiser Foundation provided technical assistance to communities in planning and evaluation through regional conferences for potential grantees. Those requiring more sustained input were assisted by community health promoters associated with resource universities located in several regions of the nation.

A current project based on a three-way model of interaction among the funder, the researcher, and the community is testing the theory that misunderstandings among participants about motivations and the expected outcome of a project cause conflict. This model promotes open negotiation among those who hold a stake in the expected outcome as well as joint participa-

tion in design and evaluation (Tony Whitehead, The Cultural Systems Analysis Group of the Department of Anthropology, University of Maryland, College Park, personal communication, 1991).

Thus, collaborative research is empowering and enabling—not simply advisory in nature. In many communities, it will take time to establish trust and build the knowledge base needed for substantive contributions to the scientific design, implementation, and critical interpretation of collaborative research. Fortunately, today a cadre of minority professionals in the social, behavioral, and biomedical sciences expressly monitor and advocate actions in their respective disciplines that affect minority populations. Community attitudes may still be cautious, even toward minority researchers. However, black researchers' links with organizations of the broader black community may help overcome this obstacle.

Challenges to the Scientific Community When Forming a Research Partnership

What challenges to scientific values and methods does community collaboration in research present? One challenge is to determine to what extent the rigor of science must be protected. For example, must science always use an experimental or quasi-experimental design on random samples of populations? How will populations be defined? Are there circumstances when storytelling, ethnography, and qualitative methods enhance science? What are those circumstances and what guidelines can direct their adoption and use?

Scientists generally have perceived the positivist scientific tradition as "value-free"—that is, with sufficient application of scientific rigor (as defined by the scientific elite), neutral knowledge will result (many philosophers of science and sociologists have not shared this perception). Community involvement in, if not control over, the research process could be viewed by scientists as potentially threatening to the neutrality of science. During the 1960s and 1970s, there were a number of philosophical challenges to this value-free approach, particularly in the social sciences,[20] but it has continued to dominate the ideology, if not the practice, of scientific research.

However, by including the community as a coparticipant in the definition of the problem and in the formulation of hypotheses, the researcher can meet the real world, perhaps to the enrichment of science. Conversely, community members' own concerns can best be served by logical hypotheses and "clean" methodology. For example, during the late 1980s, a cluster of childhood cancer cases occurred in a relatively middle-class area of San Francisco. Concerned residents held meetings, inviting scientists, the media, and the public to attend. As a result, community members asked epidemiologists to investigate the cancer cluster. In the process, community residents learned how to measure environmental and social links to childhood cancer. In turn, the epidemiologists could pursue their research.[21] Community instigation of epidemiologic investigations like this one have led to major advances in public health and scientific knowledge.

Adequate protection of the rigor of scientific process may be the heart of the challenge that active community involvement in research poses for scientists. Fundamental characteristics of good science include precisely stated research questions; a clearly identified population and sample within the population; replicable methods, including sampling, measures, and data analysis; and results interpretable within the frame of the existing scientific literature. A community or target population may not always understand or sympathize with scientific aims and methods. For this reason, scientists should explain to the community the rationale for using them through communication within a partnership rather than through cooptation, and, in return, the community must educate the scientist about its concerns.

An unavoidable risk of good science (that is, science in which research questions, data, and methods are not manipulated to produce certain answers) is that the data may be open to a number of interpretations. Communities may have less involvement in and control over the data their involvement has generated than they desire. In some cases, the findings may not be what the community wants to hear. Many highly volatile research questions concern the relationship between race/ethnicity and health outcomes. Some community-instigated epidemiologic investigations did not discover the relationship community members expected to find between an environmental hazard and an outcome.[21] In such cases, the community may be concerned that "no results" could open the door to further contamination. Another problematic situation occurs when the evaluation of a popular intervention shows weak or no results, which could lead to termination of a program or to policies unpopular with the community. Scientists should thus explain clearly the value of negative as well as positive results to the development of general knowledge. Scientists thereby preserve the specific contribution that the scientific method can make to understanding while clearly distinguishing it from the contributions of other disciplines, including health and social policy.

Research and Potential Partnerships in the Community of African-American Women

The fundamental issues that should guide public health research on the lives of contemporary African-American women are the same ones that guide any gender-sensitive, responsive research agenda. African-American women should be characterized by the social and cultural conditions that differentiate their lives: infrastructure inequities, primarily economic and political; the consummate effects of sustained cultural erosion; and relationships of resistance and power, powerlessness, and privilege. These "differentials" form a core of common experience of African-American women in this country. In addition, the shared experience of gender difference as well as racial and cultural difference are present in all aspects of life for African-American women. These differences should be taken into account in public health research.

Much of the shared positive experience of African-American women should be captured in community-involved research. In the traditional black community, women transmit attitudes toward maintaining good health through the telling of the story of their lives and the lives of their ancestors. The cultural meaning of health within the context of the ways African-American women lead their lives, as described by the women themselves, should inform research activity designed to develop appropriate health intervention strategies.

Public health research should move away from the reductionist approach frequently used in social science research to explain the life circumstances of African-American women. This approach often presents a deficit model that emphasizes negative outcomes and adverse risk factors, with no inclusion of

protective factors and no understanding of the meaningful, important factors in African-American culture that contribute to positive health outcomes for many African-American women. African-American women should tell their own stories, sort out their own explanations of what contributed to their adverse health outcomes in collaboration with the scientific community. Discussion should present the problems of women as they perceive them and explore black women's responses and solutions.[22]

Community-involved research is necessary for developing appropriate intervention strategies that include African-American women's holistic approach to the definition of health. This research will provide the information needed to craft and implement socially sensitive, politically powerful, and culturally accountable plans, policies, and programs that ensure the inclusion of African-American women's vision.

Research partnerships in the community should sponsor more female leadership and provide critical mentoring and professional development to support and sustain that leadership. Thus, research can become an effective and innovative way to garner moral, political, and material support for social change.

CONCLUSIONS

Community collaboration with the scientific community provides the opportunity for creative problem-solving. Science as a discipline is currently undergoing dynamic change as diseases such as AIDS set in motion new client-professional relationships.

In this new social and political context, the scientific community stands to gain enhanced access to communities of concern—those most vulnerable to poverty and disease. The opportunity arises for communities and science to work in tandem to ensure a more balanced set of political, social, economic, and cultural priorities, which satisfy the demands of both scientific research and communities at higher risk.

Questions involving science and the community are not mere sources of traditional academic debate. What each does affects human lives with critical consequences for all. The community's greatest impact on science will be to make it more difficult to assume the objectivity of the scientific process. We can now begin a social process wherein a search for scientific answers does not become a justification for exclusion and elitism. The community's role in this process will force us to examine the social and political contexts crucial to formulating whole problems and deriving effective answers.

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Commentary on "Community Research: Partnership in Black Communities"

William W. Dressler, PhD

Dr. Hatch et al. provide a useful, insightful overview of models of collaboration between communities and university-based or agency-based researchers. As they point out, communities and researchers can become engaged in the process of research in several ways. In three of the models they discuss, the community remains essentially a passive participant in the process. Their view of an "active involvement" of the community is clearly one in which the community is a *partner* in the definition of the problem to be investigated, as opposed to only a participant in providing research respondents or even advisors and employees. I sympathize with this perspective. I agree that models of community involvement in which the community complies with the requests of the researcher—no matter how skillfully those requests are provided through advisory boards or networks of community leaders—create the potential for intellectual exploitation that differs primarily in degree, although perhaps not in kind, from the more flagrant past examples of asymmetrical relationships. More active involvement of the community can avoid this manipulation.

At the same time, as Hatch et al. note, carrying out research that "involves and enables" the community provokes difficult issues both in the conduct of scientific research and in the development of collaborative relationships. Specifically, collaborative community research can produce tension between scientists and the society that is the object of their study. In this commentary, I discuss this issue further.

An anecdote can perhaps describe one dimension of this potential tension between the concerns of scientific research and the concerns of the community. My first experience as a researcher working in collaboration with a community-based institution occurred in a health research and advocacy organization in a small city in the northeastern United States. As part of my job, I taught a short course in social scientific research methods to persons working in the organization. During one course, a person hired as a community health advocate asked me to comment on a problem: What would we do if the results of a particular study we were working on did not end as we expected? How could we alter the analyses of the data to demonstrate what we already knew we wanted to show?

This question could charitably be described as ingenuous, or it could be described as motivated by intellectual dishonesty. I was not taken aback by the question, because I understood it to be motivated by a sincere belief that the knowledge required to alter the community for the better was already at hand; all the research could do was, perhaps, demonstrate more elegantly what was already known—icing on the cake, so to speak.

I suspect, however, that some researchers in the scientific community, including those who sit on the review panels that determine the viability of particular projects, believe that community research conducted according to the model of collaboration advocated by Hatch et al. can too easily become an exercise in "proving" what researchers and activists want to prove. Those researchers on panels of federal and private funding agencies want pristine scientific research designs and clear testing of hypotheses, and they perceive the collaborative process as a potential bias.

I think that this real tension stems in fact from a misunderstanding of the process of scientific research at both ends of the researcher-community continuum. That is, many researchers who hold to the model of "pristine science" fail to comprehend the *scientific* value of collaboration with the community, and many community activists fail to understand the *power* inherent in scientific theory and method.

Science is fundamentally a process of "weeding out" false statements about the world. Shorn of philosophical niceties, a scientific hypothesis is simply a guess about the relationship between two phenomena. We are guessing, for example, that psychosocial variables are related to an increased risk of preterm delivery. The logical canons of science are the best—not perfect, but simply the best available—method of determining if those guesses (Popper's conjectures) are true or not (Popper's refutations).[1] Our hypotheses about the world often appear true, but turn out false, because one phenomenon can masquerade as another. The very best examples derive from studies of ethnicity, which have determined that ethnic group membership

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often so completely overlaps with social class that one can be mistaken for the other. By sorting out that confounding much racist thinking about ethnicity has been effectively challenged (if not entirely laid to rest).[2,3]

Scientific researchers have, however, an unfortunate habit of thinking that, because their methods of evaluating hypotheses are relatively so powerful, they need pay attention only to their own concerns, especially their own ways of generating hypotheses. Serendipity does in fact loom large in all accounts of scientific experience, but, for the most part, hypotheses are derived from theory, that set of ideas that describes how the world (or, rather, some small part of it) works. Based on the accumulated insight of many bright people, and the combination of numerous empirical evaluations of that insight, statements describing what is known, not known, and surmised about phenomena can be assembled. This is theory. It is great because in combining the thoughts and observations of many, it challenges common sense and received wisdom.[4]

But we sometimes forget that theory can be incomplete, parochial, and unless stringently evaluated, just plain wrong. So-called "stress theory" is a blatant example. Our notions of what stress is and how it affects people are interesting, but they are also based on a peculiarly Western middle-class idea of personhood. Furthermore, much of stress theory ignores social and historical contexts of human behavior.[5] This deficiency does not vitiate its usefulness as guidance for scientific research, but it does require other ways—systematic ways—of generating hypotheses.

This reasoning is precisely why the researcher needs to collaborate with the community, in the active sense Hatch et al. advocate. People know their own communities, the way their communities work, the constraints and obstacles and resources that influence behavior. In defining the problems to be studied and in identifying the variables to be included in the examination of those problems, the community can help researchers overcome the weakness and parochial nature of their theory. Philosophers of science call this process "the context of discovery."[6] Anthropologists call it "ethnography."[7] Sociologists call it "grounded theory."[8] All of these labels describe a process in which the researcher moves beyond the narrow (but useful) confines of theory and previous research to discover new ways of understanding phenomena, informed by the lived experience of real people.

Collaboration in this sense will also benefit the community. If theory can be parochial, so can the community's understanding of a phenomenon. The researcher's theory and accumulated knowledge can greatly benefit the community's attempts to solve real and pressing problems with limited resources. More importantly, however, the strengths of scientific research in evaluating the truth of statements about the world can be of considerable value to the community. If the community wants to bring its resources to bear on an important problem, then it needs to marshal those resources in the most effective way. If the process has not been specified accurately, then the investment of resources is likely to be ineffective.

An example of a potentially ineffective investment of resources is suggested in my own work on stress and adaptation in the black community, which started with the reasonable notion that members of the extended family in the black community would be the main source of social support to individuals experiencing social stress. This support would, in turn, protect those persons from social stress, so they would be at a lower risk of hypertension or psychological depression. In initial ethnographic studies in the community, I found that many individuals, although strongly committed to their extended family, preferred to seek support from others in times of need. In analyzing data on depression, I discovered that extended kin support had no beneficial effect for the entire sample; however, in reformulating the hypothesis, I found that older persons were indeed protected from the effects of stress on the basis of their extended kin support. But for younger persons, nonfamily support was protective.[9] These findings were then replicated in an independent study of high blood pressure, using a different sample drawn from the same community.[10]

This example emphasizes the problem that could be encountered if careful scientific research were not used to evaluate claims to knowledge. The most sensitive and sophisticated analyses of the black community point to the extended family as a mechanism of personal and social adaptation, and these analyses are correct, in part. But these analyses fail to account for the degree of social change in the community and how that social change has altered community support systems, especially for younger persons. Reasonable investments in a program to strengthen systems of social support, and hence to enhance the health of the community, must take into account this diversity within the community.

In addition, careful scientific work can assist the community by providing power. In our society, knowledge very often translates directly into power. In the process of advocacy, knowledge can persuade those who hold political power that resources should be invested in a particular way. Bolstered by carefully evaluated research findings, a community seeking resources can make a virtually unassailable assertion that it "knows" what the problem is and how to best address it. Such knowledge does not, of course, ensure that a community will succeed in its advocacy. But it does seal off an escape route for those who wish to prevent the access of the community to certain kinds of resources. Put more bluntly, careful scientific research, carried out according to the active model of collaboration Hatch et al. advocate, can be the 2-by-4 to use on the mule of a bureaucracy.

I can summarize my argument about the value of collaborative research by returning to my anecdote. The concern of the community advocate who asked the question was very real, not to be dismissed as either naive or dishonest. But he could be reassured by the fact that careful research, informed by scientific theory and method and grounded in the community, is more likely than any other model for the generation and application of knowledge to arrive at a useful set of truths. His ultimate concern, which was to improve the health and well-being of the minority community, could best be served through this process. Collaboration with social scientific researchers who were also committed to such a process would not subvert that aim; rather, it would help to specify how best to achieve it.

To be sure, this fairly abstract (if not downright lofty) discussion of the value of collaboration between the scientific researcher and the community studied does not address many complicated pragmatic issues. Negotiating such a collaborative relationship can demand research skills, time, and patience perhaps notably lacking in some academic researchers. Similarly, the willingness of the community (and ignoring for the moment the complicated issue of just what "the community" is and how

you find it) to enter into the long-term pact required for high-quality research can oftentimes necessitate a difficult shift in values.

A thorough discussion of these issues is well beyond the scope of this short essay. But I will close with a few brief thoughts. First, not all researchers need to become experts in community work. Several academic disciplines (anthropology and sociology, for example) have made understanding communities their business, and members of a research team from these disciplines can help in the translation (in both directions) between the academy and the community. However, specialists in community research cannot be brought on board simply as academic public relations experts, their job being to sell the research—any research—to a community. Rather, community researchers, conversant in both cultures, can help to negotiate differences.

Second, assuming that all communities are naive about the research process would be a mistake. In fact, in many communities, members are quite knowledgeable about, and sympathetic to, the aims and aspirations of the academic researchers, including the constraints and obstacles they face. "The community" is spoken of as a single entity, but there are many different kinds of communities. In the future, empirical attention should consider the dimensions along which communities vary. This study could prove very helpful in future efforts to understand and to intervene in community health problems.

And third, simply thinking about these issues in a different way, or, perhaps, thinking about them at all, can practically assist the researcher. As Hatch et al. suggest, the researcher should consider how scientific work may both benefit the community in a direct sense and manifest the acumen of community members—a major step in a new direction for collaborative research.

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A Legacy of Distrust: African Americans and Medical Research

Vanessa Northington Gamble, MD, PhD

After the abuses of the Tuskegee Syphilis Study were revealed, the federal government strengthened regulations to protect the subjects of human experimentation. These increased safeguards, however, have not erased many African Americans' fear that they will be abused in the name of medical research. The tenacity of this conviction is understandable if one examines the broader history of race and American medicine. The goals of this short essay are twofold: (1) to place the Tuskegee Syphilis Study within its historical context and (2) to examine how race and racism influence contemporary biomedical research.

A historical analysis of racism and American medicine illuminates the ways in which the profession has been used to support racist social institutions and has, in turn, been influenced by them. Examination of this history demonstrates why so many African Americans mistrust the medical profession and its institutions. As efforts begin to include more African Americans in clinical trials and to develop community-collaborative research programs, this legacy of distrust must be addressed, not dismissed as paranoia or hypersensitivity. The challenge is to understand and confront the historically based realities behind these sentiments.

An understanding of the Tuskegee Syphilis Study and its impact on African Americans is imperative for medical researchers. Although the study is not the only case in which black people have been exploited in the name of medicine, it has come to symbolize such abuse. The history of the study is often used to demonstrate why African Americans should not cooperate with medical researchers. Most recently, its specter has been raised in connection with human immunodeficiency virus prevention programs.

Law professor Patricia A. King warns that the Tuskegee Syphilis Study should serve as a caveat to medical researchers when they analyze racial differences between whites and blacks. She writes that "in a racist society that incorporates beliefs about the inherent inferiority of African Americans in contrast to the superior status of whites, any attention to the question of difference that may exist is likely to be pursued in a manner that burdens rather than benefits African Americans."[1] The premise underlying King's comments is that medicine is not a value-free discipline. Rather, it has reflected and reinforced the beliefs, values, and power dynamics of the wider society. Accordingly, it has been influenced by issues of race and racism. History shows numerous examples of the use of medical beliefs to support the alleged inferiority of black people.

Medical theories, for example, were used to justify the enslavement of Africans. Antebellum physicians contended that black people possessed peculiar physiological and anatomical features that justified their enslavement. This medical distinctiveness, they argued, made Africans not only inferior but inherently suited for slavery. For example, the physicians theorized that Africans had thicker skins, which allowed them to tolerate better the rays of the sun. They also observed, in this case accurately, that black people seemed to be less susceptible than white people to some diseases, such as yellow fever and malaria. Plantation owners took note of these observations and, without qualms, worked slaves in environments such as mosquito-ridden swamps, which they believed detrimental to white people.[2]

Medical theories influenced societal attitudes that held that black people were inferior and inhuman. Such attitudes underscored the use of slaves and free black people as subjects for medical experimentation and demonstration in the antebellum South.[3,4] Although poor whites were also used as subjects, blacks were used far more often. Harriet Martineau, after an 1834 trip to Baltimore, commented that "the bodies of coloured people exclusively are taken for dissection, 'because the whites do not like it, and the coloured people cannot resist.'"[5,4] In 1839 abolitionist Theodore Dwight Weld asserted, "'Public opinion' would tolerate surgical experiments, operations, processes, performed upon [slaves], which it would execrate if performed upon their master or other whites."[6]

Two antebellum experiments, one carried out in Georgia, the other in Alabama, confirm Weld's charge. In the first, Georgia physician Dr. Thomas Hamilton conducted a series of brutal experiments on a slave to test remedies for heatstroke. The sub-

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ject of these investigations was Fed, who had been loaned to Hamilton as repayment for his owner's debt. Fed was forced to strip and sit on a stool on a platform placed in a pit that had been heated to a high temperature. Only his head was above ground. Over a period of two or three weeks, the man was placed in the pit five or six times and given different medications to determine which enabled him best to withstand the heat. Each ordeal ended when Fed fainted and had to be revived. But note that Fed was not the only victim in this experiment; its whole purpose was to make it possible for masters to force slaves to work still longer hours on the hottest of days.[7]

In the second experiment, Dr. J. Marion Sims, the so-called father of modern gynecology, used three Alabama slave women to develop an operation to repair vesico-vaginal fistulas. Between 1845 and 1849, the three slave women on whom Sims operated each underwent up to thirty painful operations. The physician himself described the agony associated with some of the experiments.[8] He wrote, "The first patient I operated on was Lucy. . . . That was before the days of anaesthetics, and the poor girl, on her knees, bore the operation with great heroism and bravery." This operation was not successful, and Sims later attempted to repair the defect by placing a sponge in the bladder. This experiment, too, ended in failure. He noted, "The whole urethra and the neck of the bladder were in a high state of inflammation, which came from the foreign substance. It had to come away, and there was nothing to do but to pull it away by main force. Lucy's agony was extreme. She was much prostrated, and I thought that she was going to die; but by irrigating the parts of the bladder she recovered with great rapidity. . . . " Sims finally did perfect his technique and ultimately repaired the fistulas. Only after his experimentation with the slave women proved successful did the physician attempt the procedure on white women volunteers. He found, however, that they could not, or more accurately, would not, withstand the pain and discomfort that the procedure entailed. The black women had no choice but to endure. They, like Fed, were forced to submit because the state considered them property and denied them the legal right to refuse to participate. This history of medical experimentation on slaves profoundly influenced African-American attitudes toward the medical profession even after the Civil War. In the 1920s, for example, many black people believed that they would be experimented upon if they entered hospitals.[7] Thus, the legacy of distrust preceded the 1932 initiation of the Tuskegee Syphilis Study.

The influence of racism on medicine did not end at Appomattox. The medical and public health journals of the late nineteenth and early twentieth centuries contain many articles that discuss the health problems of African Americans. Many of the discussions focused on syphilis. White physicians maintained that intrinsic racial characteristics such as excessive sexual desire, immorality, and overindulgence caused black people to have high rates of syphilis. As Dr. Thomas W. Murrell noted in 1910, "Morality among these people is almost a joke and only assumed as a matter of convenience or when there is a lack of desire and opportunity for indulgence, and venereal diseases are well-nigh universal."[10] Dr. H. H. Hazen echoed this sentiment: "The negro springs from a southern race, and as such his sexual appetite is strong; all of his environments stimulate this appetite, and as a general rule his emotional type of religion certainly does not decrease it."[11] Physicians also pointed to alleged anatomical differences—large penises and small brains—to explain the disease rates.[12]

White physicians, in the early twentieth century, believed that syphilis was difficult to treat in black patients because they could not be convinced to come in for treatment or, if they did, to follow the treatment regimen. In the words of Dr. Eugene Corson, "this absolute indifference [to treatment] is a characteristic of the negro, not only as regards syphilis, but of all diseases. He is simply concerned with the present moment of suffering, and not always concerned then."[13]

Historian Allan Brandt has argued that these assumptions regarding black people and venereal disease influenced the physicians who initiated the Tuskegee Syphilis Study. He writes: "The premise that blacks, promiscuous and lustful, would not seek or continue treatment, shaped the study. A test of untreated syphilis seemed 'natural' because the USPHS presumed the men would never be treated; the Tuskegee Study made that a self-fulfilling prophecy."[14] The Tuskegee Syphilis Study thus did not occur in a vacuum. It represented the continuing influence of racist thought not only on medical theory but on physicians' perceptions of a group of people and consequently on the treatment, or lack of treatment, individuals would receive.

The United States Public Health Service (USPHS) initiated the study in 1932 to document the natural history of syphilis.[15] The subjects of the investigation were 400 poor black sharecroppers from Macon County, Alabama, with latent syphilis and 200 men without the disease who served as controls. The physicians conducting the study deceived the men, telling them they were being treated for "bad blood." The men, for example, were informed that lumbar punctures were therapeutic, not diagnostic.

As part of the project, however, the USPHS deliberately denied treatment to the men who had syphilis and went to extreme lengths to ensure that they would not receive any. When the Tuskegee Syphilis Study began, the standard therapy for syphilis consisted of painful injections of heavy metal compounds, such as arsenic and bismuth, which had to be administered for up to two years. Although this therapy was less effective than penicillin would later prove to be, in the 1930s every major textbook on syphilis recommended it for the treatment of the disease at all stages. Published medical reports have estimated that between 28 and 100 men died as a result of their syphilis. In exchange for their participation, the men received free meals, free medical examinations, and burial insurance.

The Tuskegee Syphilis Study continued until 1972. Throughout its 40-year history, accounts of the study appeared in prominent medical journals. Thus, the experiment was widely known in medical circles. As late as 1969, a committee from the Centers for Disease Control examined the study and decided to continue it. Three years later, a USPHS worker, who was not a physician, leaked details about it to the press. Media disclosure and the subsequent public outrage led to the termination of the study and ultimately to the National Research Act of 1974. This act, established to protect subjects in human experimentation, mandates institutional review board approval of all federally funded projects with human subjects.

After the study had been exposed, many black people charged that it represented "nothing less than an official, premeditated policy of genocide."[15] This was neither the first nor the last time that the issue of genocide has been raised with

regard to the relationship of African Americans and medical research. It has been associated with the development of birth control programs and with the sickle cell anemia screening programs of the 1970s.[16–18]

Most recently, both genocide and Tuskegee have come up in connection with acquired immunodeficiency virus (AIDS). In September 1990, an article entitled "Is it Genocide?" appeared in *Essence,* a black woman's magazine. The author noted: "As an increasing number of African-Americans continue to sicken and die and as no cure for AIDS has been found some of us are beginning to think the unthinkable: Could AIDS be a virus that was manufactured to erase large numbers of us? Are they trying to kill us with this disease?"[19] In other words, some members of the black community see AIDS as part of a deliberate plot to exterminate African Americans. The views of James Small, a black studies instructor at City College of New York exemplify this position. "Our whole *relationship* to [whites] has been of [their] practicing genocidal conspiratorial behavior on us, from the whole slave encounter up to the Tuskegee Study," Small contends. "People make it sound nice, by saying the Tuskegee 'study', but do you know how many thousands and thousands of our people *died* because of that?"[19]

It would be a mistake to dismiss such ideas as those of a paranoid extremist. In 1990 a survey conducted by the Southern Christian Leadership Conference found that 35% of the 1,056 black church members who responded believed that AIDS was a form of genocide.[20] The legacy of Tuskegee has also influenced the wariness that many African Americans maintain toward needle exchange programs.[21,22]

The Tuskegee Syphilis Study symbolizes for many African Americans the racism that pervades American institutions, including the medical profession. A lasting legacy of the study is African Americans' distrust of medical researchers. Dr. Stephen B. Thomas, director of the Minority Health Research Laboratory at the University of Maryland—College Park, laments, "Although everyone may not know the *specifics* of the Tuskegee experiment, they have enough residual knowledge of it so that they mistrust government-sponsored programs, and this results in a lack of participation in [AIDS] risk-reduction efforts."[19] Alpha Thomas, a Dallas health educator, University Hospital, often confronts the legacy of Tuskegee. She notes that "so many African American people that I work with do not trust hospitals or any of the other community health care service providers because of that Tuskegee Experiment. It is like . . . if they did it then they will do it again."[20]

The strengthening of safeguards and the reforms in research standards that followed the public disclosure of the abuses of the Tuskegee Syphilis Study have been insufficient to change African Americans' historically based fears of medical research. These apprehensions contribute to the low enrollment rate of African Americans in clinical trials.[23] A 1989 study conducted by pharmacologist Craig K. Svensson demonstrated the underrepresentation of African Americans in clinical trials. He reviewed 50 clinical trials for new drugs that had been published in *Clinical Pharmacology and Therapeutics* for the three-year period 1984–1986. He discovered that the percentage of black subjects was less than their percentage in the cities in which the research was conducted and less than their percentage in the general population of the United States. More recent studies confirm this underrepresentation of African Americans in clinical trials for AIDS drugs.[24,25]

Why this underrepresentation of black people? As one physician has put it, "We're battling centuries of mistrust based on historical actions of the very institutions involved."[26] The attitudes and practices of medical researchers towards African Americans also cannot be discounted. Once at a job interview, I was told that black people are not included in clinical studies because "it is a well-known fact that they are noncompliant." Furthermore, in the past, most clinical researchers have used white men as the standard or norm from which to extrapolate data to the rest of the population. Young white men were presumed to be a homogenous population that had fewer confounding factors. Members of minority groups and women were frequently excluded from clinical studies. However, federal guidelines now call for the inclusion of these groups in studies unless a compelling reason exists for their exclusion.

Does it matter that African Americans have been excluded from therapeutic drug trials? In the case of the Tuskegee Syphilis Study, clearly the inclusion of the men in a nontherapeutic experiment was detrimental to their health; today, however, exclusion from a therapeutic one may be harmful. For example, recent studies suggest that there are racial and gender differences in the therapeutic efficacy of some drugs.[25,27,28] In addition, it is crucial to have African Americans participate in clinical and public health studies that examine diseases and conditions that disproportionately affect them.

The researchers associated with the innovative research strategy to examine preterm delivery in African-American women recognize that a historically-based mistrust still influences African Americans' perceptions of biomedical research. They understand that these attitudes represent a significant research obstacle. These researchers have chosen not to cavalierly dismiss this legacy of distrust but to confront it. They have acknowledged that the voices and experiences of African-American women are crucial for the project's success. In a radical departure from traditional scientific studies, the investigators have actively solicited advice about the study from the African-American lay community. Their goal is to develop a collaborative research study that is conducted *with* African-American people, not *on* them. The efforts of these researchers are a significant step in eroding the legacy of distrust that has so profoundly shaped the relationship of African Americans to medicine.

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Psychosocial Measurement: Implications for the Study of Preterm Delivery in Black Women

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THE LIFE STRESS MODEL AND ITS APPLICATION TO PREGNANCY OUTCOME

Black infant mortality rates in the United States today are twice the rates for white infants, an urgent issue that shows no signs of change in the near future.[1,2] Research efforts that have concentrated on traditional risk factors such as smoking, parity, and social class, have been unable to identify definitive explanatory factors. Various research efforts have recently contributed cumulative support for the hypothesis that high levels of exposure to stressors and other psychosocial factors may put women at increased risk for adverse reproductive outcomes, particularly low birthweight and preterm delivery.[3–5] Unfortunately, most studies of stress and pregnancy outcome have not taken advantage of the conceptual depth and recent methodologic developments in life stress research.[6–14] Thus, this summary article has three broad goals: (1) to provide a brief review of the literature linking psychosocial factors to preterm delivery and other adverse reproductive outcomes; (2) to describe the major components of the "life stress paradigm" as they have evolved in research in psychology, psychiatry, sociology, and epidemiology and to provide an overview of the major available measures; and (3) to pose methodological questions that need consideration if we are to apply this paradigm toward understanding the excess rates of black preterm delivery and infant mortality.

In particular, the aim of the article is to identify and separate individual components of the life stress paradigm that may influence pregnancy outcome rather than to highlight measurement approaches that group several of these components together into single indices.[15] As an example, "stressors" (e.g., the occurrence of stressful life events) are described separately from "stress responses" (e.g., symptoms of anxiety or depression). We believe that the delineation of linked aspects of experience and response is critical in understanding the specific psychosocial and physiologic mechanisms through which individual psychosocial factors may affect reproductive outcome and in developing targeted interventions.

To cover the entire field of life stress research in a single summary is an enormous and clearly impossible task. Therefore, our general approach has been to organize and focus discussions of psychosocial constructs and instrumentation so that a reader new to the field of psychosocial research will find a clear presentation of the issues and an overview of commonly used instruments. A reader with knowledge in a specific area will find additional resources to further his or her work. Each section is accompanied by a review table that provides information on the populations in which measures have been developed or used and summarizes their psychometric and administrative characteristics. Instrument characteristics were obtained from a review of the Mental Measurements Yearbook,[16–20] Test Critiques,[21–23] the Educational Testing Service,[24] and the published literature.

Life Stress Model

Our discussion and critique of measures is organized in two sections that cover the broad components of the life stress paradigm: (1) stressors and (2) potential effect modifiers (personal dispositions, psychologic state, and social networks/social support). Understanding interrelationships among these broad psychosocial constructs has been the focus of life stress research for more than two decades; the development of research strategies to test specific models and hypotheses for effects on physical and mental health[6,9,10] offers a solid groundwork for research on pregnancy outcome. Exploration of the relationships of stressors, personal dispositions, psychologic state, and social networks/social support to pregnancy outcomes and their possi-

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ble interactions may provide important etiologic clues to the excess rates of adverse pregnancy outcomes in black women. Figure 1 presents an overall model of how psychosocial factors may act directly or indirectly to affect pregnancy. The potential relationships among stressors, possible effect modifiers, and pregnancy outcome are illustrated in the context of a physiologic substrate that may mediate effects of any or all of these factors on pregnancy outcome, since in this model we assume that all psychosocial factors may influence pregnancy outcome directly, through effects on behavior, or indirectly, through alterations in physiologic state (e.g., neuroendocrine, immunologic responses). The relationships of all risk factors to adverse pregnancy outcome, both psychosocial factors and physiologic responses, are also illustrated within the broader social, environmental, political, and historical context in which women live.

Successful tests of etiologic hypotheses and the development of targeted interventions to address the excess rates of adverse pregnancy outcome among black women depend critically, however, on valid, reliable measures, particularly for black women. Each section of the article provides an overall definition of the psychosocial construct, describes specific measures and common measurement approaches, and offers critiques of these measures' utility for research with black women.

Life Stress and Pregnancy Outcomes

The cumulative evidence from studies of different populations, using varied design and measures, is that psychosocial factors are associated with increased risk of preterm delivery, low birthweight, and other pregnancy outcomes. Tables 1 and 2 outline a review of recent research. We chose studies for review based on the following criteria: (1) the study relief on epidemiologic methods such as case-control, cohort, or intervention designs; (2) pregnancy outcomes were the major outcome of interest; and (3) psychosocial factors were among the major risk factors investigated. Studies are grouped by outcome (preterm delivery/low birthweight and mixed outcomes) and by study design. In this section of the text, we briefly discuss and critique research on psychosocial factors and pregnancy outcome within major constructs of the life stress paradigm as illustrated in Figure 1. Most research to date has focused on life events and on social support, both independently and in relation to life events.

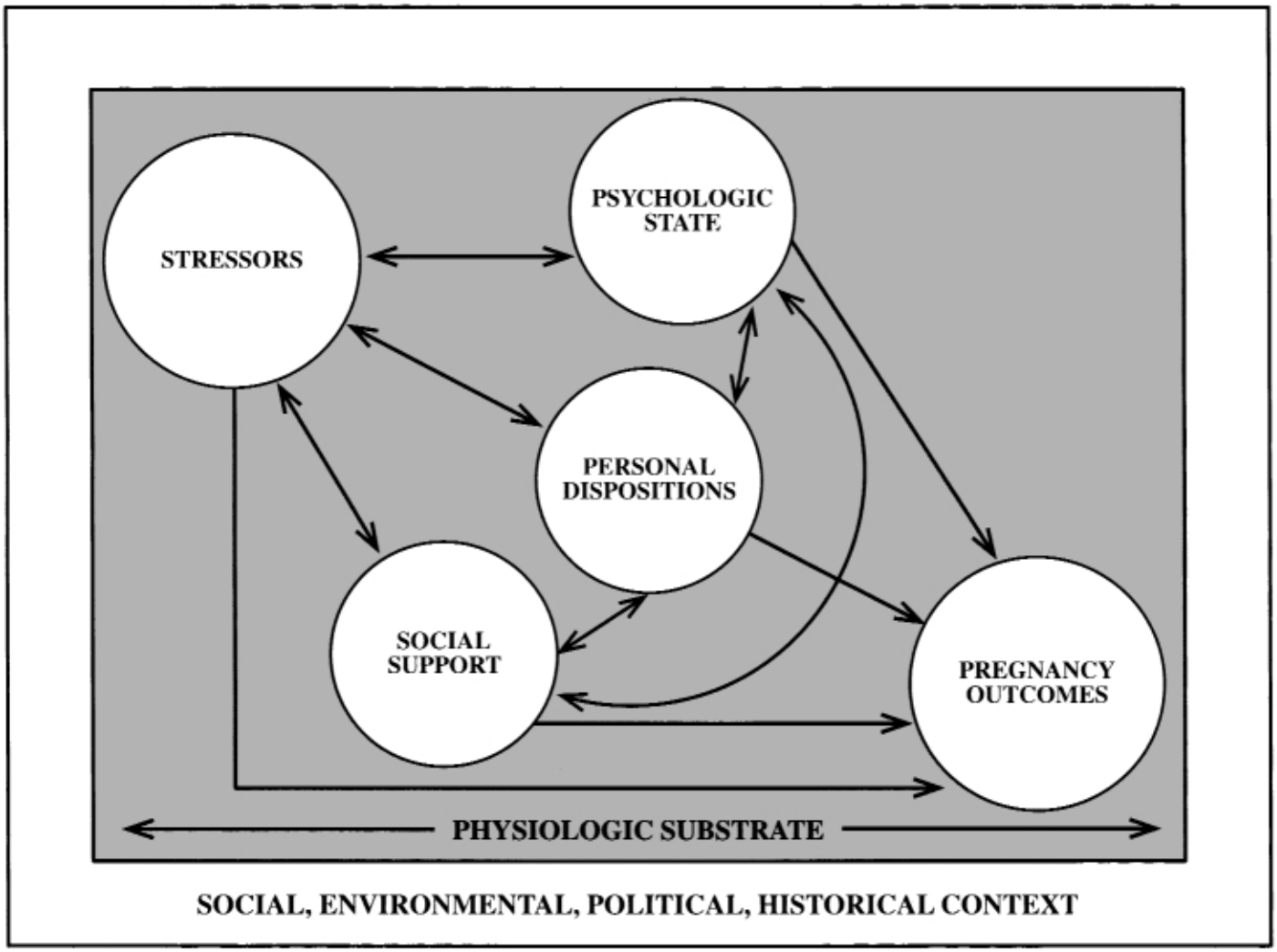

Figure 1. Psychosocial factors and pregnancy outcomes.

Table 1. Selected studies of psychosocial variables and pregnancy outcome: endpoint—preterm delivery (PTD) and low birthweight (LBW)

Reference	Study design, size, and setting	Populations studied			Exposure/psychosocial instruments				Findings
		Age (years)	Socioeconomic status	Race/ethnicity	Life events or stressors	Personal disposition	Psychological state	Social support	
Gunter (1963)[60]	Case-control study Matched 1:1 (age, parity) 40 case = 20 (PTD) control = 20 (term) Tennessee, hospital	Mean case = 21.2 control = 26.6	≥8th grade education	Black 100%	Cobb Life Chart	Thematic Apperception Test (TAT)	Cornell Medical Index	—	Psychosomatic and neuropsychosomatic characteristics and the social or life situation of the mother may be associated with prematurity. Mothers of premature infants (8.3) recalled more stressors in life than mothers of normal infants (4.15).** Various TAT scores demonstrated that mothers of premature infants differed significantly from mothers with normal infants.*
Williams et al. (1975)[225]	Case-control study 46 case = 23 (PTD) control = 23 (term) Washington, hospital	<20 case = 8.6% control = 8.6% 20–29 case = 56.6% control = 74% >30 case = 34.8 control = 17.4%	Head household occupation: White collar case = 69.7% control = 47.9% Blue collar case = 8.6% control = 34.8% Farm case = 4.3% control = 13.0% N/A case = 17.4% control 4.3%	White case = 78.4% control = 91.4% Black case = 13.0% control = 0.0% Other case = 8.6% control = 8.6% 3 of 23 (13%) premature births were to black women	Holmes-Rahe Schedule of Recent Experience (SRE)	—	—	—	Both cases and controls reported increases in life events as they neared delivery. Clusters of life change were not predictive of premature delivery.
Berkowitz and Kasl (1983)[63]	Case-control study 465 case = 166 (PTD) control = 299 (term) Connecticut, hospital	—	—	White case = 69.3% control = 76.3% Black case = 30.7% control = 23.7%	Modified version of Holmes-Rahe Social Readjustment Rating Scale (SRRS) consisting of 27 items	5 questions regarding woman's attitudes towards the pregnancy. Scale internal consistency reliability .83; mean item correlation .49	—	3 questions to assess emotional support received from partner/husband during pregnancy. Scale internal consistency .54; mean item correlation .28	White women with a PTD reported a higher number of life events and expressed a more negative attitude toward the pregnancy than white women with a term delivery. Both white and black women combined with high pregnancy desirability were at an increased risk for a PTD when exposed to high levels of life events (OR white low = .77, high = 1.99; OR black low = .72, high = 2.00; df = 1).*
Mamelle and Munoz (1987)[32]	Case-control study 600 case = 200 control = 400 France, hospital	—	—	—	Elements of fatigue in occupations that constitute possible risk factors for PTD	—	—	—	Two risk factors significantly related to PTD were work on industrial machinery (OR = 1.7; CI = 1.01, 2.9)* and mental stress (OR = 1.54; CI = 1.03, 2.3).*

(*continued*)

Table 1. (*Continued*)

Reference	Study design, size, and setting	Populations studied: Age (years)	Populations studied: Socioeconomic status	Populations studied: Race/ethnicity	Exposure/psychosocial instruments: Life events or stressors	Exposure/psychosocial instruments: Personal disposition	Exposure/psychosocial instruments: Psychological state	Exposure/psychosocial instruments: Social support	Findings
Cepicky and Mandys (1989)[226]	Case-control study matched 1:2 111 case = 40 (widowed) control = 38 (married) control = 33 (unmarried) Czechoslovakia, national survey	Mean case = 24.8 ± 4.3 control 1 = 24.7 ± 5.3 control 2 = 22.8 ± 5.3	—	—	Cases experienced death of spouse during pregnancy.	—	—	—	There was no association between death of spouse during pregnancy and length of pregnancy or birthweight.
Klebanoff et al. (1990)[34]	8,648 case = 4,412 female resident physicians control = 4,236 wives of male resident physicians U.S.A., national survey	≤25 case = 0% control = 6.5% 25–29 case = 54.5% control = 59.5% 30–34 case = 38.3% control = 28.5% ≥35 case = 6.9% control = 5.5%	High SES	White case = 90% control = 93% Black case = 5% control = 2% Asian case = 4% control = 4% Other case = 1% control = 2%	Cases worked as residents during pregnancy (long hours in a stressful occupation).	—	—	—	There was no association between PTD or delivery of an infant small for gestational age and occupational stress.
Burstein et al. (1974)[61]	Cohort study (P) 61 Canada, hospital	Range 17–42 Mean 27.08 ± 5.10	—	—	—	—	Taylor Manifest Anxiety Scale (MAS) Pleshette Pregnancy Anxiety Scale (PAS)	—	No association between anxiety and birthweight was found. The correlations between MAS and age ($r = -.265$)* and PAS and age ($r = -.276$),* gravidity ($r = -.249$),* and parity ($r = -.289$)* demonstrate that anxiety in pregnancy decreases with age and number of pregnancies.
Standley et al. (1979)[57]	Cohort study (P) 73 U.S.A., NICHD	Range 18–36 Mean 24.6	100% married women	White 100%	—	—	Measures of prenatal anxiety: general, physical, fetus, childbirth, childcare, infant feeding, and psychiatric symptoms	—	Correlation between prenatal anxiety and LBW did not reach statistical significance.
Newton et al. (1979)[62]	Cohort study (R) 132 England, hospital	≥37 weeks range 14–38 mean 24.5 ± 4.9 33–36 weeks range 17–36 mean 25.3 ± 4.7 <33 weeks range 18–34 mean 24.4 ± 5.7	Social class ≥37 weeks I&II 20%; III 28%; IV 7%; V 45% 33–36 weeks I 7%; II 14%; III 41%; IV 7%; V 31%	—	Modification of Cochrane-Robertson Life Events Inventory (LEI)	—	—	—	Significantly more major life events occurred in the preterm than the term group,* and the difference between the very preterm and the term groups was highly significant.** The incidence of major life events in the one week prior to the onset of labor was much higher in the preterm groups.

			<33 weeks I&II 7%; III 33%; IV 13%; V 46% (social class not recorded in two preterm pregnancies)							
Mamelle et al. (1984)[31]	Cohort study (R) 1,928 France, hospital	—	All working women	—	Elements of fatigue in occupations that constitute possible risk factors for PTD	—	—	—	After controlling for confounding factors, multiple regression analysis revealed that increasing fatigue scores (0–5) were significantly related to increases in the rate of premature birth (2.3%–11.1%).* Multiple logistic analysis of medical, social, and occupational factors showed significant risk factors for prematurity are fatigue index (reg = .36; *sd* = .08; *t* value 4.56)*; previous premature birth (reg = 1.70; SD = .41; *t* value 4.15)*; and parity (neg effect) (reg = −.50; SD = .19; *t* value −2.59).*
Newton and Hunt (1984)[55]	Cohort Study (P,R) 224 Hospital, United Kingdom	Range 14–38	61% social classes I–III	White 95%	Modification of Cochrane-Robertson Life Events Inventory (LEI)	—	Spielberger State-Trait Anxiety Inventory (STAI)	—	Objective life events were significantly related to PTD*** and LBW (P = .004).
Ramsey et al. (1986)[39]	Cohort study (P,R) 102 Texas, health department	Mean 21.4 ± 4.3	Low income	Hispanic 28%	Holmes-Rahe Schedule of Recent Events (SRE)	—	—	Olson Family, Adaptability and Cohesion Evaluation Scales (FACES) Smilkstein Family Adaptability, Partnership, Growth, Affection and Resolve (APGAR)	Family function and structure were related to birthweight. Women who described their families as disengaged delivered babies weighing less than women who had more cohesive families. Using only significant* variables in a reduced model, money-related stress and family enmeshment continued to be determinants and added 9% in explaining the variance in infant birthweight.
Reeb et al. (1987)[28]	Cohort study (P) 140 Ohio, hospital	Mean 24	Low income	Black 100%	Dohrenwend Psychiatric Epidemiology Research Interview (PERI) Life Events Scale	—	Derogatis' Brief Symptom Inventory (BSI)	Stack Support Scale Smilkstein Family Adaptability, Partnership, Growth, Affection and Resolve (APGAR)	Four factors (low family functioning, stressful events, Quetelet's Index, and cigarette smoking) predicted LBW: 65% sensitivity, 84% specificity, and 42% positive predictive value (PPV). Family functioning, alone, predicted LBW with 65% sensitivity, 64% specificity, 31% PPV.

(*continued*)

Table 1. (*Continued*)

Reference	Study design, size, and setting	Populations studied			Exposure/psychosocial instruments				Findings
		Age (years)	Socioeconomic status	Race/ethnicity	Life events or stressors	Personal disposition	Psychological state	Social support	
Reeb et al. (*continued*)								Olson Family, Adaptability and Cohesion Evaluation Scales—FACES II (truncated version) Hudson's Index of Family Relationships (shortened version)	
Stein et al. (1987)[29]	Cohort study (P) 483 United Kingdom, hospital	Range 16–41 Mean 27.3	61% middle class 39% working class	—	Paykel Interview for Recent Life Events (RLE)	—	Wing Present State Examination	—	Adverse life events and psychiatric state were not associated with LBW or PTD.
Brooke et al. (1989)[227]	Cohort study (P) 1,513 United Kingdom, hospital	Range ≥ 15	88% social classes I–III	White	Paykel Interview for Recent Life Events (RLE)	Eysenck Personality Inventory	Goldberg General Health Questionnaire (GHQ)	Social support was measured but standardized questions were not used.	There was no association between psychological factors, and birthweight after smoking was controlled.
Homer et al. (1990)[56]	Cohort study (P) 786 National Longitudinal Survey of Labor Market Experience	14–21	Mean education = 12 years 16% less than federal poverty level Survey oversamples economically disadvantaged whites	Black 16% Hispanic 7% Survey oversamples blacks, Hispanics	Karacek's job characteristic scoring system	—	—	—	After controlling for job related exertion and other maternal characteristics, there was no association between job-related psychologic stress and PTD, LBW. However, for the 70 women who stated they did not want to work outside and had high stress jobs, their infants' birthweights (3,062 gms) were almost 500 grams less than those of infants born to women with less stressful occupations (3,561 gms) (mean effect = −295 gms).
Klebanoff et al. (1990)[228]	Cohort study (P) 7101 New York, Washington, Oklahoma, Texas, Louisiana, clinical centers	59.1% 20–29	Education level 43.3% < high school 67% < $15,000 yearly income	White 33.6% Hispanic 34.9% Black 31.6%	Work strain including standing for long periods of time, heavy work/exercise, light work/exercise	—	—	—	Heavy work or exercise was not associated with PTD (adjusted OR for ≥4 hrs/day heavy work = 1.04). Prolonged periods of standing were associated with a moderate increased risk of PTD (adjusted OR for ≥8 hrs/day of standing = 1.31).*
McCormick et al. (1990)[41]	Cohort study (P) 458, 388 New York, community clinics and hospital	25% < 19	Education level 56% < high school Low income	Black 100%	Dohrenwend Psychiatric Epidemiology Research Interview (PERI) Life Events Scale	—	Goldberg General Health Questionnaire (GHQ)	Situational social support was measured.	Stress, social support, and mental distress were associated with smoking but not LBW.

Pagel et al. (1990)[26]	Cohort study (P) 100 Washington, hospital	Range 14–38 Mean 26.3 ± 5.3	Education level Mean 14.1 ± 2.2 Mean income $15,000–$20,000	White 78% Asian 8% Black 7% Other 7%	Rand Life Event Index	—	Spielberger State-Trait Anxiety Inventory (STAI)	Smilkstein Family Adaptability, Partnership, Growth, Affection and Resolve (APGAR)	Increasing life events before pregnancy was significantly related to LBW (R^2 = .55, ΔR^2 = .05).**
Lobel et al. (1992)[76]	Cohort study (P) 130 California, hospital clinic	Range 18–42 Mean 27.7 ± 5.0	Education level Mean 10.8 ± 3.3	Latino 64% Black 20% White 12% Other 4%	Cohen Perceived Stress Scale (PSS) Golding Stressful Life Events Measure	—	Spielberger State-Trait Anxiety Inventory (STAI)	—	Stress factor significantly predicts both LBW (R = −.85)** and PTD (R = .30).*
Steer et al. (1992)[229]	Cohort study (P) 712 323 adolescents 389 adults New Jersey, hospital	Adolescents: Range 12–17 Mean 15.00 ± 1.08 Adults: Range 18–29 Mean 21.59 ± 3.36	—	Black 61.8% Puerto Rican 28.7% White 9.6%	—	—	Revised Beck Depression-Inventory (BDI)	—	The risk of a LBW infant and PTD rose 5%–7% for each point the BDI increased in both unadjusted and adjusted models. Clinically depressed adults' (score > 21 BDI) risk of delivering a LBW infant was 3.97 (CI = 3.80, 4.15)*; risk of PTD was 3.39 (CI = 3.34, 3.56).* There was no relationship between PTD and LBW in the adolescents.
Papiernik et al. (1985)[33]	Intervention trial (12 yrs) No control group 16,794 France, provider-based sample	Mean 1971–1974 = 26.2 1975–1978 = 25.7 1979–1982 = 26.0	Education level primary and secondary ≈50% blue collar workers and those not working were married to blue collar workers	French, Turkish	High-risk women encouraged to reduce physical efforts, prescriptions for rest issued	Modify attitudes of pregnant women, including those concerning lifestyle	—	Effect of presence/absence social support on PTD and LBW—social support provided by midwives or placement of family help in the home during pregnancy	Other intervention methods: used scoring system to detect preterm birth, change number and objectives of prenatal care consultations, and use preventive hospitalization. During three periods of four years each, significant declines in preterm birth rates (5.4%, 4.2%, 3.7%)** and LBW (4.6%, 4.0%, 3.8%)** were achieved.
Olds et al. (1986)[38]	Randomized controlled trial 400 intervention = 310 comparison = 90 New York, provider-based sample	Range 14–34 47% < 19	Education level Mean intervention = 11.34 comparison = 11.21 61% social classes IV and V (Hollingshead)	White Nonwhite	Effect of free transportation for regular prenatal and well-child care visits	—	—	Effect of presence/absence social support on PTD and LBW—social support provided by nurse home-visitor during pregnancy through home visits in addition to the provision of free transportation services	Adolescents visited by nurses gave birth to newborns who were an average of 395 g heavier than those of the comparison group.* Women who smoked who were visited by nurses exhibited a 75% reduction in the incidence of PTD* compared to controls.

(*continued*)

Table 1. (*Continued*)

Reference	Study design, size, and setting	Populations studied			Exposure/psychosocial instruments				Findings
		Age (years)	Socioeconomic status	Race/ethnicity	Life events or stressors	Personal disposition	Psychological state	Social support	
Spencer et al. (1989)[52]	Randomized controlled trial 1,227 intervention = 655 comparison = 633 United Kingdom, provider-based sample	Mean intervention = 23.0 comparison = 23.2	Social class IV or V or unemployed intervention = 78% comparison = 75%	Asians excluded	—	—	—	Effect of presence/absence social support on birthweight—social support provided by family worker supervisor during pregnancy through home visits	Availability of family worker service for at-risk pregnant women did not significantly influence either the overall mean birthweight or the percentage of LBW.
Heins et al. (1990)[53]	Randomized controlled trial 1,458 intervention = 728 comparison = 730 U.S.A., provider-based sample	% 20–34 intervention = 64% comparison = 61%	Education level < high school intervention = 63.1% comparison = 61.7% Low SES	Black intervention = 52.2% comparison = 54.5% White intervention = 46.5% comparison = 44.8% Other intervention = 0.7% comparison = 0.1% Unknown intervention = 0.6% comparison = 0.6%	—	—	—	Effect of presence/absence social support on LBW—social support provided by nurse midwives during pregnancy through appointments at clinic	No statistically significant difference in birthweight was observed between the intervention and comparison groups.
Oakley et al. (1990)[42]	Randomized controlled trial 509 intervention = 255 comparison = 254 United Kingdom, provider-based sample	Mean intervention = 27.9 ± 4.9 comparison = 28.1 ± 5.3	Working class intervention = 78% comparison = 75%	White 95% Afro-Caribbean/Asian 5%	—	—	—	Effect of presence/absence social support on LBW—social support provided by midwives through prenatal home visits and telephone calls	Intervention group mothers had a mean birthweight 38 grams higher than comparison group babies (95% CI = 72.6, 146.6).

Bryce et al. (1991)[50]	Randomized controlled trial 1,967 intervention = 983 comparison = 987 Australia, provider-based sample	<30 intervention = 52.7% comparison = 53.1% ≥30 intervention = 45.9% comparison = 44.1%	Social classes Professional intervention = 33% comparison = 34% Clerical intervention = 35% comparison = 35% Manual intervention = 31% comparison = 31% Unknown intervention = .6% comparison = .6%	White intervention = 90.6% comparison = 89.3% Aboriginal intervention = 2.7% comparison = 2.9% Other intervention = 4.8% comparison = 5.9% Unknown intervention = 1.8% comparison = 1.9%	—	—	—	Effect of presence/absence social support on PTD—social support provided by midwives through antenatal home visits and telephone calls	Results provided little evidence for the effectiveness of social support in the prevention of PTD. Observed relative reduction in preterm births in the intervention group was 13.8% (95% CI = 8.2%, 31.5%). The unadjusted odds ratio for preterm birth in the intervention group was 0.84 (95% CI = .65, 1.09).
Villar et al. (1992)[51]	Randomized controlled trial 2,235 intervention = 1,115 comparison = 1,120 Latin America, provider-based sample	Mean intervention = 24.3 ± 6.6 comparison = 24.6 ± 6.6	Education level Mean intervention = 8.4 ± 3.7 comparison = 8.4 ± 3.8 Low income intervention = 55.1% comparison = 54.9%	White intervention = 60.3 comparison = 59.8	—	—	—	Effect of presence/absence social support on PTD—social support provided by trained female social workers and support person through home visits	There was no evidence that the intervention had any significant effect on PTD, LBW, or intrauterine growth retardation. No protective effect of the psychosocial support was detected among women at highest risk.

— = Not reported or not studied.
(R) = retrospective.
(P) = prospective.
*$P < .05$.
**$P < .01$.
***$P < .001$.

Table 2. Selected studies of psychosocial variables and pregnancy outcome: endpoint—mixed outcomes: maternal and infant complications

Reference	Study design, size, and setting	Populations studied: Age (years)	Populations studied: Socioeconomic status	Populations studied: Race/ethnicity	Exposure/psychosocial instruments: Life events or stressors	Exposure/psychosocial instruments: Personal disposition	Exposure/psychosocial instruments: Psychological state	Exposure/psychosocial instruments: Social support	Findings
Nuckolls et al. (1972)[36]	Cohort study (P) 170 Military hospital	<18 4.9% 18–19 33.9% 20–24 55.9% 25–29 4.8% >29 0.3%	Education level 67.3% ≤ 12 yrs Hollingshead's Two Factor Index of Social Class I = 1.2% II = 4.0% III = 16.3% IV = 50.4% V = 28.0%	White 100%	Holmes-Rahe Schedule of Recent Experience (SRE)	Nuckolls TAPPS measures subject's feelings and perceptions concerning herself, her pregnancy, and her overall life situation.	—	Nuckolls TAPPS measures subject's feelings and perceptions about relationships with her husband, extended family, and the community.	Among women with high SRE scores before and during pregnancy, those with high TAPPS scores had one third the complication rate of women with low TAPPS scores. In the absence of high cumulative life change, there was no significant relationship between psychosocial assets and complications.
Gorsuch and Key (1974)[27]	Cohort study (P) 118 Hospital	Range 17–41 Mean 20.9	Low income Marital status married 54% separated 7% divorced 8% single 31%	White 61% Black 39%	Life Change Inventory adapted from Holmes-Rahe Schedule of Recent Experience (SRE)	—	Spielberger State-Trait Anxiety Inventory (STAI)	—	Anxiety during the first trimester and life stress during trimesters two and three were related to a general category of abnormal pregnancy outcomes. Correlation with number of abnormalities during trimesters two and three: major personal injury or illness (r = .24)*; addition of a new family member (r = .27)*; death of spouse (r = .18)*; marriage (r = .21).*
Norbeck and Tilden (1983)[37]	Cohort study (P) 117 U.S.A., medical center	Range 20–37 Mean 26.2 ± 4.21	Education level Range 9–18 Mean 13.8 ± 1.98	White 61% Black 14% Hispanic 11% Filipino 5% Chinese or Japanese 4% Other 5%	Sarason Life Events Scale (LES)	Rosenberg Self-Esteem Scale	Spielberger State-Trait Anxiety Inventory (STAI) Lubin Depression Adjective Check Lists (DACL)	Social Support Questionnaire, part two only Three-item measure of tangible support	Life stress in the year prior to delivery was significantly associated with overall complications* and gestation complications.** Emotional disequilibrium was related to infant complications.**
Smilkstein et al. (1984)[47]	Cohort study (P) 93 Washington, hospital	Mean 25.9%	Students over-represented 21%	—	Holmes-Rahe Schedule of Recent Experience (SRE)	—	—	Smilkstein Family Adaptability, Partnership, Growth, Affection and Resolve (APGAR)	A multiple correlation coefficient 0.33** was obtained, indicating that biomedical risk (b_1 = −0.14), psychosocial risk (Family APGAR) (b_2 = 0.03) and their interaction predicted 11% of the variance in the postpartum complications measure.

Rizzardo et al. (1985)[46]	Cohort study (P) 319 Italy, hospital	Range 16–42 Mean 27.4	Education level Range 5–18 Mean 8.5 Middle-lower class	White	Paykel Interview for Recent Life Events (RLE)	Modification of Andrews Scale of Coping Style	Free anxiety subscale of the Middlesex Hospital Questionnaire Standley Scale for Pregnancy Specific Anxiety	Surtees' Index of Social Support	Variables most predictive of complications were previous pregnancies, social support, general anxiety, and coping style. Together these variables explained 16.7% of the variance.
Molfese et al. (1987)[44]	Cohort study (P) 96 Illinois, obstetric clinics	Range 17–40 Mean 27.2 ± 4.5	Education level 96% > high school Middle to upper income levels	White 89% Nonwhite 5% No information 6%	Dohrenwend Psychiatric Epidemiology Research Interview (PERI) Life Events Scale	Rotter Internal-External Locus of Control	Blau Maternal Attitude Toward Pregnancy Instrument (MAPI)	Vaux Perceived Social Support Inventory	Did not show strong relationship between life event stress and perinatal complications. However, specific pregnancy attitudes, locus of control, and perceived social support played a stronger role in predicting complications. In a regression model for maternal and infant outcome measures, three of the five infant outcome measures (1-min Apgar, 5-min Apgar, birthweight) were predicted by combinations of the life events stress scores and specific intervening variables. Birthweight: life events stress = .12, perceived social support = .79; $F = 4.05$*, R = .33, $R^2 = .11$.
Rizzardo et al. (1988)[54]	Cohort study (P) 109 Italy, hospital	Range 17–40 Mean 26.4	Education level Mean 9 years Middle-lower class	—	—	Modification of Andrews Scale of Coping Style	Spielberger State-Trait Anxiety Inventory (STAI) Standley Scale for Pregnancy Specific Anxiety	Surtees' Index of Social Support	State anxiety in the third month of pregnancy was significantly higher* among women with obstetric complications. None of the other psychosocial variables examined were related to complications.
Norbeck and Anderson (1989)[43]	Cohort study (P) 208 U.S.A., obstetric clinics	Range 18–39	Education level 41% < high school Low SES	Hispanic 37% White 35% Black 28%	Norbeck Revised Life Events Questionnaire (LEQ)	—	Spielberger State-Trait Anxiety Inventory (STAI)	Norbeck Social Support Questionnaire (NSSQ)	For black women, anxiety was predictive of birthweight* in the opposite direction than what was expected. Among white and Hispanic women, there was no relationship between psychosocial variables and birthweight.

(*continued*)

Table 2. (*Continued*)

Reference	Study design, size, and setting	Populations studied: Age (years)	Populations studied: Socioeconomic status	Populations studied: Race/ethnicity	Exposure/psychosocial instruments: Life events or stressors	Exposure/psychosocial instruments: Personal disposition	Exposure/psychosocial instruments: Psychological state	Exposure/psychosocial instruments: Social support	Findings
Williamson et al. (1989)[25]	Cohort study (P) 513 Missouri, family practice	14% < 20 83% 20–34 3% > 35	Education level 20% < high school	White 94%	Holmes-Rahe Social Readjustment Rating Scale (SRRS)	—	—	Berkman Social Network Index	Those women whose life change scores (LCS) increased from the second to the third trimester had a significantly higher rate of poor outcomes (neonatal death, transfer to neonatal intensive care unit, LBW or 5-min Apgar score < 7) than those whose LCS did not increase (9.2% vs. 3.9%, $P = .015$).*
Zuckerman et al. (1989)[40]	Cohort study (P) 1,014 Massachusetts, hospital	34% ≤ 20 49% 21–29 17% ≥ 30	Education level 40% < high school Monthly income 42% < $500	Black (North American) 49% Black (other) 18% Hispanic 18% White 8%	—	—	Center for Epidemiological Studies-Depression (CES-D)	Norbeck Social Support Questionnaire (NSSQ)	Depressive symptoms during pregnancy were associated with increased life stress,*** decreased social support,*** poor weight gain,** and the use of cigarettes,*** alcohol,*** and cocaine.*

— = Not reported or not studied.
(R) = retrospective.
(P) = prospective.
*$P < .05$.
**$P < .01$.
***$P < .001$.

Stressors

Life events. Several studies have found evidence of specific relationships between the occurrence of stressors and pregnancy outcome (Tables 1, 2). A recent, well-conducted cohort study by Williamson et al. (1989) measuring acute stressors at two intervals during pregnancy, 18–22 weeks and 32–36 weeks, found that women who reported increases in level of stress were more likely to have a poor pregnancy outcome than women who did not note changes in levels of stress.[25] Similarly, in a cohort study, Pagel et al. (1990) found a statistically significant inverse relationship between increased life changes and birthweight.[26] Gorsuch and Key (1974) in their cohort study reported that the amount of life change during the six months before delivery was associated with an increased risk of an adverse pregnancy outcome.[27] Moreover, they noted that both anxiety and amount of life change were independent risk factors for adverse pregnancy outcomes. Another recent study by Reeb et al. (1987) conducted among a cohort of low-income black women found four factors that predicted low birthweight: greater number of life events, low family functioning, mean Quetelet's index at weeks 12–16, and cigarette smoking.[28] Stein et al. (1987), however, found no support for the hypothesis that either life events or psychiatric disorders were risk factors for low birthweight and prematurity in their study of a large cohort of women, 30% of whom were working class.[29]

Role strain. Of various role strains, the one most studied in relation to adverse pregnancy outcome is work strain. Saurel-Cubizolles and Kaminski's (1986) review of work during pregnancy provides support for an overall association of employment and adverse pregnancy outcome,[30] although confounding of occupation with social class is problematic. Work strain can be divided into psychosocial and physical components. In two studies of pregnant women, Mamelle et al. (1984)[31] and Mamelle and Munoz (1987)[32] found that mental stress associated with work was associated with a higher risk of preterm delivery; the support for an association with physical exertion is less consistent. The striking results among low-income women of a reduction in rate of preterm delivery in the intervention study of Papiernik et al. (1985) are intriguing, because the intervention combines the possibility of paid antenatal work leave with weekly cervical exams and attention to the early signs and symptoms of labor.[33] However, the recent case-control study by Klebanoff et al. (1990) does not show a relationship between work strain and preterm delivery.[34] Interestingly, the Klebanoff study found no association of work strain with preterm delivery, but women in the higher occupational stress group had a substantially higher rate of preterm labor.[34,35] Thus, this study provides evidence that stressors such as work strain may have differing etiologic importance along the continuum of reproductive outcome. We note that studies of work during pregnancy are subject to the healthy worker effect, rendering comparisons of workers and nonworkers difficult. Housework is not without its own strain; nonworkers are thus not unexposed, and workers may be additionally burdened.

Effect modifiers

Social support—separate exposure. A few studies have examined the direct relationship between social support and pregnancy outcome.[25,28,33,36–42] Cohort studies by Reeb et al. (1987)[28] and Ramsey et al. (1986)[39] and an intervention study by Oakley et al. (1990)[42] reported that family functioning or social support positively affected birthweight, but the cohort study by Williamson et al. (1989)[25] was unable to detect such an association. Two intervention studies by Papiernik et al. (1985)[33] and Olds et al. (1986)[38] detected significant decreases in preterm delivery and low birthweight as a result of the support given by a midwife or nurse during pregnancy. Of the studies that considered interactions, the cohort studies by Nuckolls et al. (1972),[36] Norbeck and Tilden (1983),[37] and Zuckerman et al. (1989)[40] noted key interactions between measurements of stress, anxiety or depression, and social support. Generally, these studies link social support with better pregnancy outcomes.[26,28,33,36–40,42–48] A comprehensive data base of social support in pregnancy has been compiled by Elbourne et al.[49]

Ramsey et al. (1986)[39] studied the contribution of family structure and function to birthweight. Abnormal family function was a strong predictor of poor pregnancy outcome. Financial strain explained 5% of the variance in birthweight, and family enmeshment (family as a stress producer) explained 7% of the variance. Ramsey et al. hypothesized that the family contribution to infant birthweight might be explained in part by poor nutrition and compromised immune function related to poor family functioning. McCormick et al. (1990) in their cohort study investigated the association between smoking, sociodemographic factors, attitudes toward pregnancy, health behaviors, stressful life events, social support, and symptoms of mental distress.[41] Smoking behaviors were associated with social support, stress, and mental health although these variables were not directly related to low birthweight. In a randomized controlled trial, Olds et al. (1986) found that adolescents visited by nurses during pregnancy gave birth to infants who were an average of 395 g heavier than the comparison group.[38] In another intervention study, Oakley et al. (1990) found only a slight increase in birthweight (38 g) for women with a history of low birthweight births and midwife support throughout pregnancy; spontaneous onset of labor, vaginal delivery, and less frequent use of epidural anesthetic were all more common among the intervention group.[42]

Results of intervention studies to provide antenatal support by Bryce et al. (1991),[50] Villar et al. (1992),[51] Spencer et al. (1989),[52] and Heins et al. (1990)[53] have shown little evidence that social support is effective in preventing preterm delivery or low birthweight. However, in the study by Olds et al. (1986), antenatal support was effective in preventing preterm delivery among adolescents.[38] Several study limitations may have influenced results of the Bryce et al. (1991) study.[50] First, because of a lower number of preterm births than expected, only 60% power to detect significant effects was possible. Second, only five midwives were employed to support almost 1,000 women. Finally, midwives were instructed not to provide antenatal care, advice, and information in order to avoid conflicts with information provided by the primary provider. This restriction could have significantly hampered the ability of the midwives to provide meaningful support and of the mothers to understand the role of the midwives in providing it.

Interactions—life events and social support. In a landmark study, Nuckolls et al. (1972) looked at the relationships between stress, psychosocial assets, and outcomes of pregnancy.[36] The cohort study determined the extent to which psychosocial assets were protective of health and the degree to

which multiple life changes were detrimental to health. Nuckolls et al. found no association between life changes and pregnancy complications. However, pregnant women with high life changes scores had fewer complications when psychosocial assets were present. In a cohort study, Smilkstein et al. (1984) studied three types of psychosocial risk (life events, family function, and social support) and their ability to predict pregnancy complications; psychosocial risk was related to both delivery and postpartum complications.[47] This study suggested that psychosocial risk assessment alone and in combination with biomedical risk assessment significantly improved prediction of pregnancy outcome.

Norbeck and Tilden (1983) investigated the relationship between life stress, social support, and emotional disequilibrium.[37] In this cohort study, high life stress and low social support were significantly related to emotional disequilibrium but not related to complications of pregnancy. There is some evidence to suggest that smoking and alcohol use confound these results.[26] In a follow-up cohort study using the same theoretical model, Norbeck and Anderson (1989) studied the psychosocial predictors of pregnancy outcomes.[43] For complications of gestation among blacks, support from the spouse or partner explained 19.5% of the variance in total gestation complications and 9.1% of the variance in gestational age. Higher levels of social support from the pregnant woman's mother also explained an additional 13.7% of the variance in total gestation complications. With the exception of support from friends, which was associated with higher rates of labor complications, higher levels of social support were associated with fewer labor complications. For white women, Norbeck and Anderson found a negative relationship between levels of social support and pregnancy outcome. This suggests that the social network among whites may have reinforced poor health practices.[43] A buffering effect of social support on life stress and pregnancy outcomes was not found. Study results from Pagel et al. (1990) complement these findings.[26]

Molfese et al. (1987), in a cohort study, assessed the modulating effects of psychological and social variables on stress and pregnancy outcome.[44] The results showed that intervening variables such as social support, pregnancy attitudes, and locus of control were stronger predictors of pregnancy outcome than life event stress.

In a cohort study, Rizzardo et al. (1985) studied perinatal complications.[46] They found that social support was among the variables most predictive of complications. Previous pregnancy outcome, social support, general anxiety, and coping style explained 16.7% of the variance. Perinatal complications differed significantly between the groups with and without obstetrical complications when analyzed separately. In addition, they found a negative relationship between social support and complications. Since the number of social contacts does not necessarily reflect the number of supportive relationships, Rizzardo et al. (1985) hypothesized that this effect may be a result of measurement error.[46] In another cohort study, Rizzardo et al. (1988) investigated the link between various psychosocial factors and obstetric complications and found that women with higher anxiety levels had more obstetrical complications.[54] Other psychosocial variables, including pregnancy-specific anxiety, coping style, and social support, did not affect perinatal complications.

In an intervention study to test the effect of a supportive companion specifically on perinatal problems, length of labor, and mother-infant interactions, Sosa et al. (1980) demonstrated that, for women with uncomplicated deliveries, the presence of an attendant during delivery significantly reduced labor length (perinatal complications did not include preterm delivery or low birthweight; therefore, it is not in Table 1).[48] In a follow-up intervention study, Klaus et al. (1986) also found an inverse relationship between level of support and labor length.[45] Klaus et al. suggest that, since higher levels of adrenaline are associated with prolonged labor, the presence of a supportive companion may have decreased catecholamine levels and shortened labor length.[45]

Methodologic limitations. Although the evidence is more than suggestive, several problems make the literature on psychosocial factors and pregnancy outcome difficult to interpret. First, for many studies, results were based on relatively few occurrences of the outcomes of interest.[25,28,29,37,41,55,56] Thus, statistical power is low, and effects that may actually exist could be difficult to detect.

Second, many investigators mixed together heterogeneous outcomes,[5,25,27,37,57] which may account for reported weak associations.[5,58] For example, infants with Apgar scores < 7, low birthweight, anoxia, dysmaturity, severe jaundice, and selected congenital malformations were all grouped together as "abnormal infant outcomes."[27] Even the consideration of apparently similar outcomes related to small infants likely constitutes a heterogeneous group of outcomes. Kaminski et al. (1973) noted that the cutpoint of 2,500 g is not a "biologic limit" for low birthweight and that it combines infants who have low birthweight because of prematurity, those who are small for term, and those with birthweight too low for gestational age.[59]

Third, only a few investigative teams considered potential confounding factors and effect modifiers in their analyses (e.g., age, parity, preexisting medical conditions, access to prenatal care). Many investigators did not account for possible interactions between selected risk factors and the outcome of interest. The small sample sizes would have made such considerations problematic in any case. As an example, no study before 1975 considered effect modification by, or the potential confounding effects of, smoking or alcohol consumption during pregnancy on infant outcome.[27,36,60,61]

Similarly, most studies did not evaluate the possibility for confounding or interaction involving psychosocial variables. Since a woman's ability to manage stressors may depend on her personal disposition, her psychologic state, and the composition and adequacy of her social network to provide support, failure to consider the interaction between these factors in analyses of stress and adverse pregnancy outcomes may have led investigators to miss key causal relationships. All the studies described in Tables 1 and 2 used standardized instruments to measure stressors, but these measures incorporate only limited assessments of exposure to the range of possible stressors. Many studies reviewed did not evaluate either personal disposition or social support and thus were unable to assess whether these factors moderated stress or anxiety. Additionally, in a few studies, reports of prenatal stressors were obtained after delivery and thus may be subject to recall bias, reflecting the stress of an adverse pregnancy outcome rather than accurate reports

of stressors that may have influenced the outcome.[31,39,45,60,62,63]

Finally, the theoretical generalizations about the relationship of psychosocial factors to preterm delivery that can be extracted from the results of these studies ultimately depend on the range of measured constructs, the instrumentation used to measure them, and an understanding of the physiologic mechanisms through which these factors affect pregnancy outcome. The following sections of this summary article discuss the major checklist and interview instruments used in the studies reviewed in Tables 1 and 2 and place them in the context of other available instruments that measure stressors, personal disposition, psychologic state, and social networks/social support. We highlight theoretical and measurement issues that have specific implications for the study of psychosocial risks for preterm delivery in black women.

PSYCHOLOGICAL INSTRUMENTS: METHODOLOGIC CRITIQUE

Stressors

Research on stressors has been broadly focused on two types of stressful experiences: (1) stressful life events (e.g., divorce or job loss) and (2) chronic strains (e.g., work strain). Differences in exposure to such stressors may lie at the heart of the differences in rates of adverse pregnancy outcome between black and white women, modified by characteristics of personal dispositions, psychologic state, and social support. This section summarizes the diversity and characteristics of stressors that can be considered within these broad categories, reviews the major instruments used to measure them, and discusses the adequacy of those instruments to reflect the experiences of black women.

Life events: definition. Stressful life events can be placed in two broad categories: recent stressors, usually considered to be acute experiences within the past year, and remote stressors. By far the majority of research on stressors has concentrated on the health effects of recent stressors, primarily studies of exposure to multiple events over a short time period, usually under a year. Research on recent life events has developed from two related, but different, theoretical grounds for defining what is or is not a "life event," a critical measurement decision. The first conceptualization postulates that "stressful life events" consist of recent experiences that lead to life change, or experiences that require some type of physiologic readjustment or behavioral change in routine, usually measured with checklist inventories of events.[7,64–66] Holmes and Rahe (1967) initiated survey research in the field of life stress with an inquiry into the impact on health of life events that clustered at the time of illness onset and extended the notion of an adaptation response to stressful experimental stimuli to postulate a similar adaptation response to recent life events.[64] The second conceptualization of the nature of stressful life events postulates that life events consist of recent experiences that are likely to arouse strong emotion, regardless of the specific emotion produced, usually measured using open-ended interviews.[9,67,68]

These two formulations of "stressful life events" overlap, yet their differences have important implications for the interpretation of research seeking to understand the relationship between stressors and preterm delivery. As an example, the frequency of events that produce change or events that produce strong emotion may be different between black and white women. The relative frequencies of events that produce change and those that are associated with strong emotion may also be different in pregnancy from those occurring in other periods in the life cycle.

Research on stress and adverse reproductive outcomes, as well as most research on the health effects of stressors, has focused almost exclusively on recent stressors, with little attention to the potential long-term impact of remote stressors, largely because of methodologic problems in reliable recall. However, the few investigations that have considered the effects on mental health of remote stressors, such as early loss or separation from a mother,[9,69] have found strong associations with depression, alcoholism, and panic disorder, and findings on the long-term mental health effects of other traumatic events, such as sexual or physical abuse in childhood, are rapidly growing.[70,71]

Little research is available that describes long-term effects of remote psychosocial stressors on current physical health, however. The mechanisms through which remote stressors may exert effects on current mental and physical well-being are complex, but they should not be ignored in future research that considers the impact of stressors on physical health. Early exposure to stressors may substantially influence (1) the development of personal dispositions such as coping style, concepts of self-efficacy, and locus of control; (2) psychologic state, such as susceptibility to depression or anxiety; (3) characteristics of the social situation, such as the composition of social networks or choice of a partner; and (4) subsequent risk for particular stressors. There is also evidence of differential biologic responses to current stressors based on exposure to remote stressors. Investigation of differential exposure to remote but long-lasting stressors and of the indirect psychosocial and physiologic pathways through which they may exert their effects may be a fruitful avenue in research seeking to understand the excess rates of adverse pregnancy outcome in black women.

Life events: dimensions. Investigators have measured a range of dimensions of events, or intraevent variability, to identify the components of stressors. Dimensions of events that have demonstrated relationships to poor health include the amount of behavioral change associated with the event,[11,64,72] as well as its magnitude, desirability, perceived stressfulness,[72–76] and level of contextual threat.[9] Recent events such as "exit" events (e.g., deaths, job loss),[77] events with a high level of overall life change,[73] and events marking nonnormative role transitions (e.g., getting married, having children, or changing jobs at nonnormative points in the life cycle)[78,79] have all been shown to affect health adversely.

Even if an investigator relied on an ideal list of events composed both of events drawn from universal human experience and of events specific to the social group or population under study,[65] the same stressor is not likely to affect all persons equally. Consequently, the salience of the experience is also an important component of stressors to consider,[80] although it has not been routinely measured in the standard instruments. Individuals may construct an internal hierarchy of their most salient roles or areas of life functioning. Only events that occur in these areas may be deleterious, regardless of the objective characteristics of the event.[81] Little is known about the stability

in time of personal commitment to particular life areas or roles, but one would anticipate that commitment (and therefore salience) is likely to vary at least by such factors as stage in the life cycle, gender, ethnicity, and social class, and thus may be important to consider in assessing black/white differences in pregnancy outcomes.

Life events: measurement. We discuss below instruments that derive from the two conceptualizations of events described earlier: events that lead to behavioral change and events associated with strong emotion. Instruments developed from the first model are usually administered as checklist inventories, used in self-report or interview formats. Instruments from the latter model are usually administered as interviews. Our focus is on those measures used in the studies of stress and pregnancy outcome summarized in Tables 1 and 2.

Checklist inventories. The checklist inventories reviewed below differ in the content of events considered, their inclusion of probes to measure aspects of intraevent variability, and the possibility of confounding of measurement of stressors with potential effect modifiers such as psychologic state. The Schedule of Recent Experiences (SRE)[20,64,82] and Social Readjustment Rating Scale (SRRS)[20,64] rank a list of life experiences in terms of the average amount of life change expected as a consequence of that experience (Table 3). The amount of life change associated with each recent experience can then be summarized to yield a total life change score. As shown in Table 3, the SRE and SRRS have been used extensively in populations that vary in age, socioeconomic status (SES), and race/ethnicity and have been shown to have good test-retest reliability, as does the Interview for Recent Life Events (RLE), an interview scale modified from the SRRS by Paykel et al.[77,83,84] The Life Events Inventory (LEI),[85] which is a modified version of the SRE, measures relative severity of stressors and includes weights derived from groups likely to have particular experiences. Criticism of these instruments has been extensive, however.[7,65,66,86,87] The inclusion of items that are psychological or physical symptoms, not events per se, makes these instruments particularly poor choices for the study of the mechanisms through which stress may be related to adverse pregnancy outcomes. For example, several of the items reflect normal changes of pregnancy (e.g., change in appetite or sleep).

The original approach of Holmes and Rahe emphasized that the occurrence of *any* life change requires readjustment. The Life Events Scale (LES)[75] developed by Sarason et al. allows for separate measurement of positive and negative change associated with each listed event, as well as an overall assessment of life change, and has low to moderate reliability (Table 3). Like all checklists reviewed in Table 3, it does not include equal numbers of presumably positive and negative events. The Psychiatric Epidemiology Research Interview–Life Events (PERI-LE)[65] was developed on an urban sample and has been used in diverse populations (Table 3).[11] In the PERI-LE, life events in seven domains (love and marriage, family matters, health, having children, work, financial matters, legal matters) fall into three classes: (1) events that might be confounded with psychiatric or physical health conditions or caused by the respondent's own actions (dependent events), (2) events consisting only of physical illness and injury, and (3) events that were unquestionably independent of both physical and mental illness (independent events).[6] This categorization provides a clear separation of the measurement of specific types of life events from both physical and mental health outcomes. Negative and positive change associated with events are not assessed in the original instrument; recent modifications include procedures for probes and coding for a range of dimensional characteristics (e.g., control, anticipation, amount of behavioral change).[11] Drawing on characteristics of extreme stressors such as military combat, Dohrenwend et al. have also suggested that simultaneous exposure to a "pathogenic triad" of life events—involving (1) "fateful loss events" (events whose occurrence was likely to be independent of the respondent's actions or psychopathology), (2) physical exhaustion, and (3) disruptions in social networks—might be specifically associated with adverse health outcomes.[11] Their work raises issues for the measurement and investigations of specific combinations of stressors that might be particularly important for black women or particularly important during pregnancy.

Checklist inventories critique. In 1983, Thoits summarized critiques of the content of the checklist inventories of life events that have been derived from a life change perspective and that have specific implication for the use of these measures in research on exposure to stressors in the lives of black women during pregnancy.[66] Acknowledging that no list of events could possibly cover the entire range of life experience, she noted that, in many checklist inventories, however, whole classes of events have been ignored. As Dohrenwend et al. point out, this is a crucial issue since "the decisions we make in the construction of [a] list [of events] will determine the kinds of inferences and generalizations that we can make."[65] First, the SRE and many other checklists (Table 3) omit socially controversial events (abortion, infidelity, sexual abuse). Second, most checklists emphasize the events of young adulthood but do not cover these experiences in depth. For instance, events specific to pregnancy are not routinely included on checklists. Third, most checklists underrepresent or entirely omit events more common to women, to particular race or ethnic groups, or to a particular social class.[86]

In addition, most checklist inventories are heavily loaded with negative events. Although negative events are associated with increased risk of psychological distress, little information exists on the influence of positive events on physical health or on their interaction with negative events. The co-occurrence of positive events may exert a "neutralizing" effect on the relationship of negative events to health outcomes.[73,88,89] In addition, a negative event that signifies the end of a longstanding chronic strain, such as a divorce following a conflictual marital relationship, may bring relief and not distress.[14,90] Positive events may also buffer behavioral and physiologic responses associated with negative events. As with negative events, both absolute levels of exposure to positive events and the relative distribution of positive and negative events likely vary by social class, gender, ethnicity, etc. Additionally, most life events are neither entirely positive nor entirely negative but have both positive and negative characteristics.[72,80] Pregnancy itself is a good example of an event with mixed negative and positive characteristics.

Interviews. The Life Events and Difficulties Schedule (LEDS)[9] uses a detailed but unstructured interview methodology. Interviewers conduct lengthy open-ended interviews, probing for a range of events and the psychologic states leading up to and following each event. Interviewers then use this information to rate systematically 28 contextual scales covering basic characteristics about the events, without taking into consideration the respondent's personal response to the event, a process that

requires extensive interviewer training. The most significant interviewer-rated scale has been a measure of the "threat" or the psychological meaning of the event.[9,10] Other aspects of intraevent variability have been considered in relation to psychopathology by investigators using modifications of the LEDS (e.g., level of danger, loss, uncertainty) (Table 3).[10,91,92]

As noted before, Holmes and Rahe based their theoretical framework for the interpretation of stressors on a physiologic understanding of the consequences of adaptation and change, whereas Brown et al. concentrated on the psychological meaning of life events, rather than physical and behavioral adaptation as such (LEDS, Table 3).[9,10,67] Life events are considered in terms of the strong emotions they might arouse, regardless of the quality of emotion involved. Behavioral change and adaptation are not absent from their formulation but are viewed as important only as they relate to the psychological meaning of an event. Within this formulation, these investigators list 40 types of events, in the following eight categories: (1) changes in a role for the respondent (e.g., changing a job, losing or gaining an opposite sex friend); (2) changes in a role for close relatives of the respondent (e.g., changing a job, losing or gaining an opposite sex friend); (3) major changes in health of the respondent; (4) major changes in health for close relatives of household members; (5) forecasts of change (e.g., respondent told about being rehoused); (6) residence changes and any marked change in the amount of contact with close relatives and household members; (7) valued goal fulfillments or disappointments; and (8) other dramatic events involving the respondent, a close relative, or a family member (e.g., brother being arrested).[10] Thus, the LEDS includes events in a broad range of areas: change in routine activity, changes in a role, or change in the fulfillment of a cherished idea or goal.[10]

Many of the events covered by the LEDS appear on the checklist inventories reviewed earlier, but many other events do not. Specifically, "nonevents" such as forecasts of change (#5) and loss of valued goals or disappointments (#7) are not usually considered on checklist inventories.[23]

Interviews critique. Criticisms of the methodology used by Brown et al. derive from several sources. Although their interview method attempts to separate the occurrence of events from the measurement of mediating factors and the measurement of outcome, it does not entirely succeed.[11,93,94] Measures of contextual threat incorporate measures of mediating factors, especially aspects of the social situation of the respondent (e.g., social support), thus making the understanding of the etiologic role of life events alone impossible. As with the checklists, however, interpretation of causal inference from exposure to health outcome may be problematic. Several of the categories above could include events that were indicators of psychopathology; thus, the occurrence of events would be confounded with psychologic state (i.e., change in amount of contact with family member).

Life events: general methodological concerns. Stress process researchers have begun to examine practical measurement issues that apply to all measures of life events and chronic strains. Accurate assessment of the co-occurrence of events in a period of time, or the "layering" of events, particularly the relative timing of positive and negative events, further complicates the task of making causal inferences about exposure to life events and illness. It seems particularly important for future research on adverse pregnancy outcomes to understand both the timing and status of events at their point of measurement because specific periods of gestation may be more "vulnerable" to stressors than others and the potential buffering effects of positive events may be particularly important.

Researchers who study the cumulative effects of multiple events (e.g., use summary measures of life events reported on checklists or in interviews) must also develop strategies to separate closely related events from each other, since events often occur in a causal, or linked, sequence. For example, "trouble with a boss" may lead to "demotion," "being laid off," ensuing "financial difficulties," and then, hopefully, the "start of a new job." These experiences are distinct life events, but are clearly linked together. Respondents may handle the reporting of linked events on checklists in varying ways. Some respondents may report each event; others may view the process as stemming from only a few events (i.e., financial difficulties may be subsumed under the job loss) and report only part of the sequence. In the extreme case, some events reported on checklists may be redundant—including both "being laid off" and "stopping work for a significant period of time, not retirement."[90] Without clear guidance from the investigator, such variability will go undetected[89] and is particularly likely to be a problem in studies that rely on self-reported versions of life event checklists, rather than assessment through structured interviews. This potential problem is important to consider in research on pregnancy outcomes. The analysis of testable hypotheses—for instance that amount of behavioral change is associated with fetal growth—depends on an accurate assessment of the number of events with behavioral change; decisions about categorizations of linked events is critical.

Each of the instruments described above uses summary measures of exposure to multiple events within a limited time period as the measure of exposure to stress. The use of summary assessments of multiple events as measures of exposure to stress has an inherent problem of causal interpretation. Specific events, by their very nature, may lead to specific outcomes: rape, for example, is more likely to lead to anxiety than depression.[95] The inclusion of such an event on an inventory of stressors unlikely to relate to the specific health outcome under study may attenuate the relationship of exposure to outcome; no information currently exists on whether specific stressors are associated with adverse pregnancy outcomes.

Accurate measures of the relationship of events to health are affected by other methodological problems as well. Life event inventories are either self-administered, in which the respondent checks off recent events from a list, or administered as a list of items in a structured interview, sometimes with additional probes. Respondents may get bored or tired with long lists of events and fall quickly into a "no" response sequence, or they may simply forget to mention experiences that occurred early within the specified period of time. Research on falloff of reporting over time and of reliability supports this assumption.[68,96,97] Use of short lists of life events—each centered around a major life domain such as love and marriage, health, or work and inserted after relevant sections in the interview—can encourage recall of events. Such an approach should also prove less boring and less likely to lead to a "no" response set.[12,90]

Perhaps the most important methodologic concern is that reliance on selected and biased lists of events as measures of exposure to stressors may lead investigators to miss important

Table 3. Instruments used to measure stressors: life events.

Instrument	References	Populations tested			Characteristics of the instrument				Comments (time)
		Age (years)	Socioeconomic status	Race/ethnicity	Validity	Reliability	Number of items	Self-report/ interview	
Schedule of Recent Experience (SRE)[a]	Rahe et al. (1964);[82] Holmes and Rahe (1967);[64] Mitchell (1989)[20] Tables 1 and 2: Nuckolls et al. (1972); Gorsuch and Key (1974); Williams et al. (1975); Smilkstein (1978); Boyce et al. (1985); Ramsey et al. (1986)	≥13 (see validity)	Low-middle income	Whites Blacks Hispanics	Predictive validity: health status and SRE .2–.3, most valid when administered to individuals ages 25–55	Test-retest .78–.83 (2 weeks–5 months)	42	Self-report	Measures frequency of recently occurring stress-producing life events.
Life Events Inventory (LEI)[a]	Cochrane and Robertson (1973)[85] Table 1: Newton et al. (1979); Newton and Hunt (1984)	Adults 14–38	Students, psychiatric patients, psychiatrists, and clinical psychologists UK Class I–III	Whites	Correlation coefficient mean weight: patients vs. psychiatrists = .82; patients vs. students = .74; psychiatrists vs. students = .94. Coefficient of concordance = .89 ($P < .001$) (Spearman)	—	55	Self-report	Modified version of SRE. Measures relative severity of stressors. Includes weights derived from groups likely to have particular experiences.
Social Readjustment Rating Scale (SRRS)[a]	Holmes and Rahe (1967);[64] Mitchell (1989)[20] Tables 1 and 2: Berkowitz and Kasl (1983); Williamson et al. (1989)	Adults	Middle-lower income	Whites Blacks Asians Hispanics	Concurrent validity for contrasted groups correlation coefficient > .90; except whites vs. blacks = .82 (Pearson); Kendall's coefficient of concordance = .477 ($P <$ .0005)	—	43	Self-report group test	Weighted version of SRE.
Interview for Recent Life Events (RLE)[a]	Paykel et al. (1969;[83] Paykel et al. (1980);[84] Paykel (1983)[77] Tables 1 and 2: Rizzardo et al. (1985); Stein et al. (1987); Brooke et al. (1989)	Adults ≥ 15	Various SES	Whites Blacks	—	Test-retest .95 (specific event); test-retest .85 (event's month of occurrence) (6 months)	64	Self-report/ interview	Derived from SRRS, checklist usually administered as an interview. (30 mins–1¼ hours, includes coding time).
Life Events Scale (LES)[a]	Sarason et al. (1978)[75] Table 2: Norbeck and Tilden (1983)	Adults	Students, naval personnel	—	Concurrent validity: with Beck Depression Inventory and total LES score .24 ($P <$ .05); with	Test-retest (5–6 weeks) positive change .19–.53 ($P <$ .001); negative change .56–.88 ($P < .001$)	57	Self-report	Measures life changes and event desirability. Separately assesses positive and negative life

(*continued*)

Table 3. (*Continued*)

Instrument	References	Populations tested: Age (years)	Socioeconomic status	Race/ethnicity	Characteristics of the instrument: Validity	Reliability	Number of items	Self-report/ interview	Comments (time)
Life Events Scale (*continued*)					Locus of Control .32 ($P <$ .02)	(Pearson)			events and rates event impact.
Psychiatric Epidemiology Research Interview (PERI) Life Events Scale[a]	Dohrenwend et al. (1978)[65] Tables 1 and 2: Reeb et al. (1987); Molfese et al. (1987); McCormick et al. (1990)	Adults	All levels	Blacks Whites Hispanics	—	—	102	Self-report/ interview	Measures amount of change experienced as result of life events. Time depends on number of events per respondent and use of additional probes.
Life Events and Difficulties Schedule (LEDS)	Brown and Harris (1978),[9] (1989)[10]	Adults	All levels	Whites	—	—	—	Interview	Includes measure of contextual threat. Requires extensive interviewer training. Modifications of scale developed by Finlay-Jones and Brown (1981)[91]; Miller and Ingham (1983)[92]

— = Not specified in studies reviewed.
[a]Used in studies of pregnant women.

variations in the impact of recent life experiences on health within different social or ethnic groups or between men and women; omission of important events will certainly lead to lower correlations between life events and illness. Surprisingly, the ten years since Thoits's critique has seen little substantive change in the content covered by checklist inventories. The content of an event inventory or interview used to measure exposure to stressors is a crucial starting point in the development of psychosocial research to explain causes of the excess risk of preterm delivery and infant mortality experienced by black women. Without appropriate measurement of the broad construct of stressors, such research is highly likely to miss etiologically important psychosocial risk factors. All of the measures above are likely to omit life experiences that may be more common or more important in the lives of black women. Of the measures of life events reviewed, the PERI and the LEDS offer the broadest framework from which to develop new measures that include additional areas of life experience. The use of qualitative techniques to assess the full construct of stressors that occur in the lives of black women, in context of existing quantitative measures, is an important next step in the development of quantitative measures of stressors with validity for black women.

Chronic strains: definition, dimensions, and measurement. Differences in exposure to chronic strains and their interactions with life events may play a key role in explaining the high rates of poor pregnancy outcome in black women. Chronic strains can be conceptualized in four broad areas: (1) persistent life difficulties or chronically stressful situations that can be considered as corollaries of life events; (2) role strain, both the strain within specific roles and the strain of holding multiple roles; (3) chronic strains that derive from societal responses to a person's characteristics that depict him or her as part of a class of persons, such as racism or sexism; and (4) chronic community-wide strains that may operate at an ecologic level, such as the chronic strain of residence in a high crime area or residence near an environmental threat.[98] A fifth category, frequency of daily hassles such as waiting too long in line or being stuck in traffic, has also been considered as a source of chronic strain.[99] Difficulties of measurement and the substantial problems of the confounding of reports of exposure to hassles with personality dispositions, psychologic state, and social situation make the unique contribution of this type of chronic strain extremely difficult to interpret in relation to health outcomes.[100] We discuss the first four types of chronic strain in the following sections, with particular attention to those areas considered to date in studies of stress and pregnancy outcomes (e.g., work strain). We discuss other types of chronic strain in their potential importance for black women.

Persistent life difficulties. Investigators studying either of the two conceptualizations of the nature of life events have considered the role of persistent life difficulties either as a unique contributor to illness or in combination with acute life events. Persistent life difficulties can be defined as stressors either per-

ceived to be chronic or quantitatively assessed to be chronic, using the duration of the stressor as a measure of chronicity. Because persistent difficulties are often the source of acute life events, the interrelationship of these two sources of life stress is complicated to untangle and presents a challenge for further development of measures.

The measurement of persistent life difficulties is an important component of Brown's conceptualization of the construct of life stress and is an integral part of the LEDS (Table 3). All recent experiences that continue for more than four weeks are not treated as life events but are considered to be ongoing difficulties and treated analytically as a separate source of stress from life events. The impact of persistent difficulties has also been of interest to those investigators studying the importance of life change following life events. Wheaton has developed community-based strategies to enumerate an inclusive inventory of chronic strains.[14,101] Other investigators have used simple measures of the duration and recency of events to separate life events from persistent chronic strains.[12,13,90] The consistently strong relationship identified by these investigators between chronic strain and depressive symptoms provides incentive for further investigations of the role of chronic strain and other adverse health outcomes.

Role strain. Strain inherent in simultaneously inhabiting specific roles and multiple roles has particular importance for women; levels of exposure to multiple role strain is likely to differ by race/ethnicity, social class, and age as well. There is conflicting evidence, however, about whether role strains interact to increase the health effects of stressful life events.[14,78,89,102,103] Differences in role strains between black and white women, both within roles, across roles, in the average number of multiple roles, and in the interaction of role strains with life events, may help to explain the excess rates of preterm delivery and other adverse pregnancy outcomes that persist even among college-educated upper-middle-income black and white women.[104]

Types of role strain include work strain,[105–107] relationship strain (e.g., with a partner or in other important relationships with friends or family members),[65,106] and caretaking strain (e.g., parenting and other forms of interpersonal caretaking, such as that associated with care of a loved one with Alzheimer's disease or AIDS, or the strain of financial responsibility for extended family members).

There have been a number of approaches to the measurement of work strain. Mamelle et al. constructed scores for "occupational fatigue," with subscores for mental strain, environmental strain, physical strain, machine work, and work posture; mental strain was associated with preterm delivery (Table 1).[31] Karasek et al. hypothesized that work strain results from the interaction of job demands, or "task pressures," and job decision latitude, or "potential control over job-related decision making."[107] Indeed, jobs with high demands and low decision latitude were associated with increased risk of heart disease.[107]

Eckenrode and Gore point out that stress may cross over between work and family life, with both positive and negative effects.[108] They note that stress at work may carry over to the family through "a stress contagion process" whereby the worker's distress affects the family. On the other hand, work strain may "serve to mobilize family resources . . . lead[ing] to more cohesiveness among family members."[107] The following components of the stress process that they and their colleagues discussed may be important aspects of role strains and other stressors to consider: (a) the transmission processes by which this crossover occurs; (2) the stress-mediating processes, the mechanisms through which workplace and family stressors affect workers and their families; and (3) the stress moderating processes through which resources buffer stressors and chronic strains. These processes may differ between black and white women; in addition, the types of crossover may differ in the two groups. Thus, qualitative measurement strategies offer a useful approach to further refinement of existing measures of role strain.

Discrimination. A third category of chronic strain results from social responses to characteristics of a person that mark him or her as a member of a class of people, such as the chronic strain of persistent exposure to racism or sexism. A person's characteristics can be mutable or immutable; as income and social standing can change, so discrimination because of class or SES can change. Characteristics of race, ethnicity, disability, sexuality, and gender are, for the most part, immutable and incur strains likely to influence health.[109–111] Stigmatization and discrimination can result in an increased frequency of all types of stressors but can also result in discrimination-specific stressors, such as hate mail, catcalls, lewd remarks, racial epithets, as well as more broad-based structural/environmental/institutional stressors. None of the measures of stressors listed in Table 3 includes discrimination-specific stressors nor provides measures of chronic strain as a result of stigmatization; innovative strategies are needed to describe and measure these sources of stress and strain. This is an especially important area for further instrument development and hypothesis testing, since chronic strain associated with racism has been proposed to account partly for the excess rates of preterm delivery in black women.[112]

Acute and chronic strains in communities. Communities, as well as individuals, can be exposed to acute and chronic stressors. The occurrence of natural or man-made disasters can expose a community to periods of acute or chronic stress.[113–121] These studies suggest that physical and mental health effects vary by several characteristics: (1) the type of disaster (natural, technological, regional conflict), (2) the length of the disaster period (floods may occur over a long period of time, technological disasters such as Three Mile Island may entail long periods of clean-up, regional conflicts may continue for years without resolution, social and ethnic/racial conflicts may last indefinitely), (3) the degree of material loss created by the disaster, and (4) the community's perception of the disaster.

Research on the health impact of stressors is starting to include ecologic indices of exposure to community-wide strains, in addition to the stressors and strains that occur to individuals.[90] It is plausible, although largely untested, that exposure to sources of chronic community strain, such as those associated with environmental disasters or residence in areas of social disaster (e.g., communities in long-standing poverty or with high rates of crime, HIV/AIDS, or violence), might exacerbate the impact of chronic and acute stressors on mental and physical health. Using ecologic indicators of the social well-being of a community, such as divorce rates, mortality rates, employment rates, etc., Linsky et al. have developed indices of "community stressors," analogous to measures of life events for an

individual. These ecologic measures of "community stress" can then be compared across communities.[122] Strategies such as this one and others, such as small area analysis, are needed to measure the impact of exposure to community strain and its interaction with other sources of stress.

Summary. There is a great range of potentially stressful experiences, both acute and chronic, that may affect the lives of women and thereby also affect pregnancy outcome. All measures lack coverage of the range of experiences that may specifically occur to black women, yet several measures have sufficient strength and grounding in previous stress research to be informative and useful. Overall, the instrument development strategy we suggest to address these concerns is to draw on qualitative techniques to assess the broad construct of stressors in the lives of black women, pregnant and nonpregnant, and then to develop new quantitative measures that target experiences most likely to be etiologically important for specific pregnancy outcomes and that incorporate the strengths of currently available measures.

Effect Modifiers

Personal dispositions. In this section, we consider measurement of three aspects of personal dispositions in detail: (1) coping styles, (2) perceived control, and (3) mastery/self-efficacy (Table 4). Personal dispositions are likely to influence a range of responses, from the initial appraisal of an experience as stressful to the final response to manage the stress, and can be conceptualized as the social and psychological assets an individual may draw upon and the enduring emotional and psychological characteristics of an individual that may intervene in the pathway from stress to adverse health outcome.[15] In considering available measures, we evaluate the adequacy of the instrument to include responses that may be of particular importance for black women or of particular importance during pregnancy.

Coping: definition. The first measures of coping were based upon a hierarchial approach that treated coping efforts as defense mechanisms.[123] Folkman and Lazarus criticized this approach because it seemed to be closely tied to outcome and paid less attention to the problem-solving aspects of coping.[124] The transactional approach to the stress process assumes the presence of a person-environment interaction perceived by the individual as taxing or exceeding his or her current resources and threatening well-being.[125] Pearlin and Schooler offer a definition of coping behavior as "behavior that protects people from being psychologically harmed by problematic social experience, behavior that importantly mediates the impact that societies have on their members."[106] Coping is thus "the process through which the individual manages the demands of the person-environment relationship that are appraised as stressful and the emotions they generate."[125] The process by which one arrives at this appraisal of threat has been termed "primary appraisal" and is regarded by some as the first step in the coping process. "Secondary appraisal" involves decisions about action in response to the stressor, such as asking oneself what can be done about the stressful situation.

Coping strategies activated in response to a stressful appraisal thus vary from individual to individual and are closely related to the perceived level of threat or harm posed by a stressful transaction. Lazarus and Folkman postulate that cognitive appraisals of situations of harm or loss in which damage has already occurred are the most stressful, because an individual is unable to initiate behaviors that could mitigate the outcome. Appraisals involving situations of threat of harm or loss that do allow for anticipatory coping are less likely to be stressful. Appraisals of harm, loss, or threat are characterized by negative emotions such as fear, anxiety, or anger.[125] Appraisals involving situations of challenge differ from the other two types of appraisal because they focus on the opportunity for gain or growth and are characterized by positive emotions such as eagerness, excitement, and exhilaration. Coping responses are generally aimed at reducing psychological arousal and are thought to be aspects of learned behavior. Thus, effective coping may act to reduce the physiologic arousal associated with the occurrence of stressors during pregnancy.

Several typologies (for example, those of Folkman and Lazarus, Billings and Moos) have been proposed for categorizing coping behaviors and responses. In general, effective coping responses include maintaining open communication, taking active steps at problem-solving, feeling a sense of control, and using humor. Ineffective coping responses include hostility, self-blame, and avoidance.[126] Some stressful situations may prove impossible for the individual to overcome despite active coping efforts, however, leaving the individual with less desirable coping alternatives such as tolerating, minimizing, accepting, or avoiding the stressful person-environment transaction. Folkman and Lazarus divide coping strategies into two categories, problem-focused and emotion-focused (see Ways of Coping Checklist [WCC], Table 4).[124] Problem-focused coping determines why and to what extent a situation is stressful and assesses the practical options available to deal with it, leading to efforts directed at managing or altering the environment causing the problem. Emotion-focused coping regulates the emotional response to the problem. Billings and Moos proposed a similar conceptual scheme that consists of three categories of coping response: active-cognitive, active-behavioral, and avoidance coping methods.[127] Active-cognitive coping includes attempts to manage one's appraisal of the stressfulness of the situation; active-behavioral coping refers to the attempts to deal directly with the problem and its effects; avoidance coping includes attempts to avoid confronting the problem (see Health and Daily Living Schedule [HDLS] Table 4).

Coping: measurement. The WCC[22,124,128] was developed by researchers in the Stress and Coping Project at the University of California, Berkeley, and addresses many of the criticisms leveled against earlier measurements of its kind (Table 4).[124] Its primary purpose is to identify the specific strategies (both problem-focused and emotion-focused) individuals use in response to a stressful person-environment transaction. Respondents are asked to recall a recent stressful event and then respond on a four-point Likert scale about the frequency with which they used 66 coping responses in dealing with the stressful situation. It is one of the most widely used instruments for measuring coping style. The Coping Resources Inventory for Stress (CRIS) also measures stress coping resources but uses a true/false scale.[129,130] These tests have good validity and reliability for the populations in which they have been evaluated.

A similar instrument to the WCC is the HDLS (Table 4).[20,131] Here too respondents are asked to recall the most

Table 4. Instruments used to measure personal disposition

Instrument	References	Populations tested			Characteristics of the instrument				Comments (time)
		Age (years)	Socioeconomic status	Race/ethnicity	Validity	Reliability	Number of items	Self-report/ interview	
Coping									
Ways of Coping Checklist (WCC)[a]	Folkman and Lazarus (1980);[124] Folkman and Lazarus (1985)[128] (revised); Keyser and Sweetland (1985)[22]	Adults 45–64, College students	Middle income	Whites Blacks	Construct validity supported in multiple studies. Correlation between P/E scales .35–.44	Multiple studies α coefficient range .56–.91. Internal consistency of P and E scales .80–81	68 (original) 66 (revised)	Interview/ self-report	Measures problem-focused and emotion-focused coping strategies. Checklist designed to elicit information about strategies used to deal with specific stressful encounters; yes/no (original), 4-point Likert scale (revised).
Coping Resources Inventory for Stress (CRIS)[a]	Curlette et al. (1988),[129] (1990)[130]	No specific age listed	Various occupations and educational levels	Various races	Content, construct, and criterion validity supported	Internal consistency α coefficient .97–.84. Test-retest .75–.95 (3 scales, 4 weeks)	280	Self-report	Measures stress coping resources using true/false scale. (45–75 minutes)
Health and Daily Living Schedule (HDLS)[a]	Moos et al. (1983);[131] Mitchell (1989)[20]	Youth form: 12–18 Adult form A, B: adults	All levels; youth form most appropriate for middle income youth	—	—	Internal consistency α coefficients .60–.74	Youth form-9 indices (4 pgs); adult form-47 indices (16 pgs)	Self-report/ interview	Measures active cognitive, active behavioral avoidance; problem-focused and emotion-focused responses. Two forms: youth and adult. (30–45 minutes)
Problems of Everyday Life Interview (PEL)[a]	Pearlin (1975),[132] (1980),[133] Pearlin and Radabaugh (1976);[134] Pearlin and Johnson (1977);[135] Pearlin and Schooler (1978);[106] Pearlin and Lieberman (1979);[78] Pearlin et al. (1981)[102]	18–65	Urban Chicago 1972; low and middle income; 58% women	Whites Blacks	—	Test-retest .44 (time not stated)	>100	Interview	Measures potential strains, coping strategies, and resources, and emotional stresses.
John Henryism Active Coping Scale	James et al. (1983);[136] James et al. (1987);[137] James et al. (1992)[138]	21–50	Various SES levels	Whites Blacks	—	Internal consistency Cronbach's α .72–80; black women = .71–.77; black men = .67–.80; white women = .74; white men = .66	12	Self-report	Measures strong behavioral predisposition to cope in an active, effortful manner with the psychosocial stressors of everyday life.

(*continued*)

Table 4. (*Continued*)

Instrument	References	Populations tested: Age (years)	Socioeconomic status	Race/ethnicity	Characteristics of the instrument: Validity	Reliability	Number of items	Self-report/ interview	Comments (time)
Perceived control									
Internal-External Locus of Control Scale[a]	Rotter (1966);[142] Educational Testing Service[24] Table 2: Molfese et al. (1987)	College students, prisoners, 8th–12th graders	All levels	Blacks Whites	Discriminant and interitem validity supported; correlation with Marlowe-Crowne Social Desirability Scale = .07 to −.35	Internal consistency α coefficient .65–.79. Test-retest .60–.83 (1 month)	23	Self-report	Measures individual differences in the generalized belief regarding control of individual destinies.
Internal Control Index (ICI)	Duttweiler (1984);[143] Educational Testing Service[24]	Adults, junior college students	Professional, management, supervisor, semiskilled, unemployed	Blacks Whites	Convergent validity with mirels factor I of Rotter IE scale r = −.385, (P < .0001)	Internal consistency α coefficient .85	28	Self-report	Measures internal locus of control (LOC).
Locus of Control Inventory of Three Achievement Domains	Bradley and Webb (1976);[144] Bradley and Gaa (1977);[145] Bradley and Teeter (1977);[146] Educational Testing Service[24]	13–90	All levels	Blacks Whites	Concurrent validity between three subscales and Crandall's Intellectual Achievement Responsibility scale; intellectual r = .78; social r = .45; physical r = .54; (df = 58, P < .01). Construct validity .38–.49	Kuder-Richardson 20 reliability coefficient for 3 subscales: intellectual r = .53; social r = .54; physical r = .52; total scale r = .75	48	Self-report	Measures LOC orientation in three achievement domains: intellectual, social and physical. Items in each domain measure successful and unsuccessful outcomes.
Multidimensional Locus of Control Scale	Levenson (1973)[147]	Mean = 37	Low-middle class, psychiatric patients	Whites Blacks	Construct validity significant difference between groups tested on powerful others (PO) and chance scales (CS) (P < .01; P < .05); committed versus voluntary patients PO, mean (diff) = 4.4 (P < .03); correlation between PO and CS r = .54 (P < .01)	Kuder-Richardson internal consistency α coefficient: internal scale .67; chance scale = .79; powerful others = .82. Test-retest (5 days); IS .08; CS .78; PO .74	24	Self-report	Modification of Rotter's I-E LOC scale. Measures expectancies of control through internal, powerful others, and chance scales.
Multidimensional Health Locus of Control Scale[a]	Wallston et al. (1978);[148] Wallston and Wallston (1981);[149] Educational Testing Service[24]	≥17, healthy and ill persons	Middle class	Whites	Predictive validity: health status (HS) and IHLC r = .403, P < .001; HS and CHLC r = −.275, P < .01; HS and PHLC no correlation (r = −.055)	Internal consistency α coefficients for all scales .67–.77; forms A and B .83–.86. Test-retest: IHLC .48–.77; CHLC .38–.73; PHLC .46–71	81	Self-report	Measures the individual's belief that health is or is not determined by one's own behavior. Three scales: internal (IHLC), chance (CHLC) and powerful others (PHLC).

(*continued*)

Table 4. (*Continued*)

Instrument	References	Populations tested: Age (years)	Populations tested: Socioeconomic status	Populations tested: Race/ethnicity	Characteristics of the instrument: Validity	Characteristics of the instrument: Reliability	Characteristics of the instrument: Number of items	Characteristics of the instrument: Self-report/ interview	Comments (time)
Mastery/self-efficacy									
Thematic Apperception Test (TAT)[a]	Buros (1965),[16] (1972),[17] (1978);[18] Mitchell (1985)[19] Table 1: Gunter (1963)	≥4	—	Whites Blacks Hispanics	Comparative validity unknown due to open-ended nature of test; concurrent validity for contrasted groups of subjects supported	Comparative reliability unknown due to open-ended nature of test. Average internal consistency .13. Scoring reliability .54–.91. Test-retest .76–.85 (20 yrs)	20 (picture cards)	Self-report	Provides information about content of subjects' cognitive and affective reference frames. Normative data for minorities exists. The Themes Concerning Blacks (TCB) is an adaptation of the TAT. (100 minutes in two sessions one day apart)

— = Not specified in studies reviewed.
[a]Used in studies of pregnant women.

important problem they faced in the past year and to indicate on a three-point scale the frequency with which they used 32 coping responses in dealing with the problem. It has good internal consistency reliability. Billings and Moos investigated the role of coping responses and social resources in mediating the effects of stress on physical and psychological health.[127] Coping responses were measured with a 19-item scale that included the following responses: tried to see positive side, prayed for guidance, considered several alternatives, tried to find out more about the situation, and talked with spouse or other relative about the problem. These responses were categorized according to two formats: (1) active-cognitive, active-behavioral, and avoidance; and (2) problem-focused and emotion-focused responses. If one compares the two more frequently used measures, α levels are slightly higher for the WCC, but the HDLS has fewer items and a shorter Likert scale. These measures have been used with populations of black women; however, the range of probed coping responses may not reflect those black women use.

A number of studies have attempted to identify specific types of coping responses and their efficacy. In the late 1970s, Pearlin and Schooler conducted a population-based study of coping in a large urban setting using the Problems of Everyday Life Interview (PEL)[78,102,106,132–135] (Table 4). The study dealt with responses to common stressors in marriage, parenting, household economics, and occupation. They identified 17 coping factors in the following three categories: responses that modify the situation, responses that function to control the meaning of the problem, and responses that control the impact of the stressful event after it has occurred. Specific types of coping responses measured included self-reliance, advice seeking, controlled reflectiveness, emotional discharge, positive comparisons, negotiation, self-assertion, selective ignoring, optimistic faith, and others, with adequate test-retest reliability. Efficacy of coping (defined as the extent to which coping prevents emotional stress) depended upon matching the coping response to the situation. In addition, the size of the coping repertory was an important mediator; the fewer the available coping responses and personal resources, the more likely one is to experience stress, given a problematic situation.[106]

Sherman James has described a specific type of coping response in black men found to be important for vulnerability to the cardiovascular effects of exposure to stressors. Named after John Henry,[136–138] the black folklore hero who outperformed a steam drill and then died from exhaustion, John Henryism is "a strong behavioral predisposition to cope in an active, effortful manner with the psychosocial stressors of everyday life."[138] In a study of the relationship of coping styles and hypertension, individuals with little education who scored high on the John Henryism Active Coping Scale had higher rates of hypertension.[136] Although this finding was not as strongly supported in a recent study of a middle-class black population,[138] further investigation of this coping response and its relation to hypertension in different populations will be interesting. For black women, who may certainly struggle with discrepancy between their own efforts and social constraints, consideration of John Henryism may help elucidate the process through which psychosocial factors affect adverse pregnancy outcome. Other methods are needed to delineate additional coping responses specific to black women as well as responses specific to different periods in the life cycle, such as pregnancy.

Coping: general methodologic concerns. Several concerns about measurement and interpretation arise from review of these instruments. First, the range of coping strategies represented in these instruments may not accurately reflect the range of those used by both men and women or by adults of differing racial, ethnic, cultural, and social class groups.

Second, researchers have assumed that coping responses

reflect stable response strategies that do not change over time and are consistently applied in all situations. Evidence that coping responses may be situationally specific, with problem-focused efforts applied more often to work situations and emotion-focused strategies applied to health-related stressors, belies this assumption and questions the consistency of coping responses across situations.[124] Both the WCC and the HDLS ask respondents to identify a specific problem and then report their responses to that problem. Respondents may specify widely differing problems that inherently demand differing coping responses. Valid comparison of coping styles across respondents within a study is thus problematic in general, and specifically for research on adverse pregnancy outcome in black women, especially so if black and white women are exposed to different types of stressors. A modification of these measures to include a single hypothetical problem vignette (or several hypothetical vignettes) that all respondents could react to would add a needed level of standardization to these instruments.

Third, respondents may also report responses to problems that they have encountered with varying levels of familiarity. Some respondents choose to report coping responses to a new problem; others may report coping responses to a problem that they have dealt with many times. Likely the specific coping responses and pattern of responses that they report will differ in these two situations; this is important to consider when evaluating aggregate data on coping responses from these two measures. For the vignette standardization modification above, prior experience with a problem similar to that of the vignette should be recorded to identify respondents who were hypothesizing their coping responses for the first time.

Fourth, personal and environmental constraints to coping responses may also be culturally, individually, or societally determined. For example, specific types of responses may be prohibited by an individual's religious affiliation or societal constraints stemming from racism.

Perceived control: definition. Conceptualizations of situational control are closely related to conceptualizations of coping. Coping strategies may depend upon the extent to which the individual perceives the availability of a course of action in which he or she can exert some degree of control over the stressful situation. Averill identified three types of situational control: behavioral, cognitive, and decisional.[139] Behavioral control involves the availability of a response that can influence or modify the objective characteristics of a threatening event. Cognitive control involves the mental processing of threatening material to reduce the stress. Cognitive control is achieved through collecting or appraising information. Decisional control involves the opportunity to decide among various courses of action. Variations on the typology of control have been made by others,[140,141] but common elements reflecting greater situational control involve the ability to collect information about a situation in order to reduce its toxicity, belief in the availability of a response that can modify its adversity, the freedom to exercise one's repertory of responses, and prior experience with similar situations.

Perceived control: measurement. Rotter's Locus of Control Scale[24,142] is perhaps the most widely used to measure internal-external beliefs about situational control, with good discriminant validity and high internal consistency reliability (Table 4). Its sets of paired statements about situational control make it difficult to use in an interview format, but self-report versions seem to work well. Although widely used, the Rotter scale has been criticized.[143] Based on variables thought to be most related to internal locus of control (cognitive processing, autonomy, resistance to influence attempts, delay of gratification, and self-confidence), the Internal Control Index (ICI)[24,143] was developed to address problems with the Rotter scale (Table 4). The ICI is more reliable for measuring internal locus of control in adults than previously developed instruments. There is also evidence of convergent validity.

A more refined measure of locus of control is the Locus of Control Inventory of Three Achievement Domains[24,144–146] (Table 4). This instrument assesses locus of control orientation in three achievement domains: intellectual, social, and physical. By focusing on specific control expectancies based on these domains, more accurate behavioral predications can be made. The scale's convergent validity and reliability are good. Levenson's Multidimensional Locus of Control Scale[147] subdivides external control into two subscales, assessing the role of powerful others and chance, also with good reliability (Table 4). Walleston's Multidimensional Health Locus of Control Scale[24,148,149] is interesting because it allows assessment of locus of control as applied specifically to health (Table 4).

Mastery and perceived self-efficacy: definition. An individual's sense of personal efficacy or mastery is a personality characteristic that affects the choice of coping strategies employed in light of a stressful experience.[150] Other research suggests that the individual's level of self-efficacy in important life domains is related to self-esteem because judgments of competency or self-efficacy contribute to self-esteem.[151] The overlap with self-esteem makes the assessment of self-efficacy difficult to separate from an assessment of psychologic state—particularly affective states such as psychologic distress or depression.

Although mastery and self-efficacy are closely related concepts, they differ along dimensions of effective action and assessment of situational control. Bandura defined self-efficacy in terms of the individual's judgments of how well the individual could execute courses of action required to manage prospective situations.[152] Pearlin and Radabaugh[134] defined mastery as the individual's sense of control over the important circumstances in one's life.[102] Life events and chronic strains may lead to adverse health outcomes when they involve an individual's self-concept, specifically in terms of mastery and self-esteem.[102,150] Persistent role strain may lead to the individual's sense of failure; this lack of success, in turn, may lead to decreased self-esteem and sense of mastery.[102,150]

Perceptions about self-efficacy clearly influence behavior and motivation as well as response to life events and the development of solutions to manage them.[152] Judgments of self-efficacy influence the degree and persistence of individual coping efforts despite difficulties encountered during the process.[152] People who take actions based on misjudgments about personal efficacy may suffer adverse consequences. Therefore, from a functional perspective, the accurate appraisal of one's own capabilities and the realistic appraisal of the possibility of success has considerable influence on the use of coping strategies. In general, people avoid circumstances that they perceive might exceed their coping abilities but may perform with confidence those actions that they perceive themselves capable of handling.[152]

Judgments about self-efficacy are based on four sources of information: inactive attainments, vicarious experiences, verbal persuasion, and physiological state.[152] Inactive attainments are the principal source of efficacy information and are established from mastery experiences. In general, success increases perceived self-efficacy, whereas failure decreases perceived self-efficacy. Failures that occur early in an activity and that do not reflect the level of effort or the effect of external occurrences decrease the individual's perception of self-efficacy. Vicarious experiences, or social comparison, involve the individual's judgments about self-efficacy that are derived from observing the successes and failures of others whom one perceives as like oneself.[9,152] Noticing the successes of others perceived to be similar to oneself can increase self-efficacy in the observer, who then determines his or her own capability of mastering comparable activities. Alternatively, noticing the failures of others who are judged to have extended strong efforts and who are perceived to be similar to oneself decreases self-efficacy.[9] When individuals observe competent models of mastery, they learn skills for managing challenges and difficult situations.[152] Individuals use verbal persuasion to influence others to believe in their capabilities.[152] When the persuasion heightens individual performance and ultimately produces success, skill development is increased and self-efficacy is enhanced. Physiological state serves as a barometer for judging individual capability and self-efficacy.[152] When circumstances are stressful and an individual experiences physiological evidence of the stress, then the individual may perceive vulnerability, his or her ability to perform may be affected, and self-efficacy may be decreased.

Mastery and perceived self-efficacy: measurement. The Thematic Apperception Test (TAT)[16–19] was developed more than 40 years ago and has been used extensively in varying populations, including black subjects and pregnant women (Table 4). Not surprisingly, as a clinical measure, its use varies substantially according to scoring, administration, and interpretation.[17,18] This test is probably best used for investigating personality, for developing individual case studies, and for generating hypotheses that can be tested with other instruments,[18] particularly because the TAT is time-consuming to administer and validity and reliability are difficult to assess because of the open-ended format of the questions (Table 4). In the TAT, the examinee sees 20 pictures and tells a story about each picture. The examiner then assesses the stories in terms of "needs (inner states that can be expressed), press (inner states that involve a perception of some external force acting upon the subject), and thema (condensed themes comprised of need-press combinations that represent motivational trends within the individual as they relate to the identified hero)."[18]

Summary. Overall, the measures of coping, perceived control, and mastery described above have good to excellent reliabilities, are relatively easy to administer, have been used in black populations, and are useful instruments for investigators interested in these personal dispositions. The question of validity, however, is still open. As we discussed earlier in the sections on stressors and coping, for the purposes of research on the role of psychosocial factors and pregnancy outcome among black women, the items on which each of these measures are based may not adequately represent the range of responses specific for black women. The John Henryism Active Coping Scale is a first step in the direction of developing measures that address specific coping strategies; similar approaches would be useful in the areas of perceived control and mastery. A possible strategy for investigators would be to develop new measures that build on the ones reviewed, using qualitative or open-ended approaches to explore additional dimensions of each construct.

Psychologic state. The construct of psychologic state describes an extensive range of emotions, from moods to psychiatric disorders. This section focuses discussion of instrumentation on measures of the symptoms and diagnoses of depression and anxiety for two reasons. First, the available research on psychosocial risk factors and preterm delivery has concentrated primarily on symptoms of anxiety as an obstetric risk factor. Second, the great majority of studies on the impact of stressors have investigated the impact of stressors on depressive symptoms. The latter observation is important because (1) depressive symptoms are a common response to stressors and may be an important part of the pathway from maternal stress to adverse pregnancy outcome and (2) broad generalizations from the life stress literature on the impact of stressors and the mechanisms through which they exert their effects are practically limited to the relationship of stressors to this single aspect of psychologic state. Much further work needs to be done to establish the consistency or diversity of psychological and physical consequences of stressors and the psychological and physiologic mechanisms underlying these relationships.

Instruments to measure psychologic state are usually considered within two categories: measures of psychological symptoms and measures of psychiatric diagnoses. Instruments within each measurement category vary in their structure and mode of administration. Some require trained clinicians as interviewers (e.g., psychiatrists, psychologists, social workers); others can be self-administered or administered by non-clinicians (Table 5). Since texts such as that edited by Thompson review the characteristics of most of the major available instruments in detail (see especially chapters 2, 4, 5, and 6),[153] we present here only a brief review of the major instruments used to assess either symptoms or disorder of depression and anxiety and focus instead on issues of confounding between measures and the implications of confounding for making causal inferences.

Psychologic symptoms: measurement of depression and anxiety. Instruments that measure symptoms of depression usually cover the following symptoms: depressed mood, changes in sleep or appetite, loss of interest in usual activities, changes in level of activity (restlessness or lack of energy), and feelings of worthlessness. Self-report symptom measures such as the Minnesota Multiphasic Personality Inventory (MMPI),[154,155] the Beck Depression Inventory (BDI),[21,156–158] the Eysenck Personality Inventory,[16–19,159–163] the Depression Adjective Check Lists (DACL),[17,164] the Coopersmith Self Esteem Inventories (SEI),[19,165,166] the Derogatis' Brief Symptom Inventory (BSI),[20,167] the Center for Epidemiologic Studies Depression Scale (CES-D),[168–171] and clinical measures such as the Clinical Measurement Package[172] and Hamilton Depression Scale[173] all include items to measure these symptoms (Table 5). These instruments vary in length, in the balance of somatic and psychological symptoms, and in the populations in which they have been used, but they all measure symptoms of depression (Table 5).

It is not clear, however, whether or not they are measuring symptoms of depression in isolation from other types of symp-

Table 5. Instruments used to measure psychologic state

Instrument	References	Populations studied: Age (years)	Populations studied: Socio-economic status	Populations studied: Race/ethnicity	Characteristics of the instrument: Validity	Characteristics of the instrument: Reliability	Characteristics of the instrument: Number of items	Characteristics of the instrument: Self-report/ interview	Comments (time)
Symptoms of depression									
Minnesota Multiphasic Personality Inventory (MMPI)[a]	Hathaway and McKinley (1940),[154] (1943)[155]	Adults ≥16	All levels	International	Nonnormative fixed reference group a limitation—large cultural differences between groups may cause comparability problems with reference group	High intercorrelations among clinical scales. Test-retest low .50s to low .90s	550	Self-report	Most widely used personality inventory. Although developed for clinical diagnosis, widely used/adapted as linear measure of personality traits. Includes measures of anger and hostility.
Beck Depression Inventory (BDI)[a]	Beck et al. (1961);[156] Beck (1970);[157] Beck and Steer (1987) (revised);[158] Keyser and Sweetland (1985)[21] Table 1: Steer et al. (1992)	≥12	—	—	Content-high. Concurrent validity: with psychiatric rating .65–.67, with MMPI D scale .75	Internal consistency correlation coefficient .86. Spearman-Brown coefficient of reliability .93	21	Self-report	Measures presence and degree of depression in adolescents and adults. Useful as screening device. (≤10 minutes)
Eysenck Personality Inventory[a]	Eysenck and Eysenck (1963),[159] (1964),[160] (1968),[161] (1969); [162,163] Buros (1965),[16] (1972),[17] (1978);[18] Mitchell (1985)[19] Table 1: Brooke et al. (1989)	Adults	All levels	Various races	Limited validity evidence; accepted validity based on similarity to Maudsley Personality-Inventory(MPI)	Forms A and B correlation .75–.91. Test-retest .81–.85 (individual scales, 1 year); test-retest .84–.88 (forms A and B, 1 year)	57	Self-report	Revision of MPI. Measures extraversion, neuroticism, and psychoticism, and includes lie scale from MMPI. Revised version of EPI: Eysenck Personality Questionnaire. (10–20 minutes)
Depression Adjective Check Lists (DACL)[a]	Lubin (1965);[164] Buros (1972)[17]	Adults: high school, college, graduate students	Various education levels	—	Concurrent validity with MMPI Depression Scale .58. Interlist correlation .80–.93. Correlation with depression ratings: experts .79; residents .59; self-rating .95	Internal consistency .79–.90. Split-half reliability: Normals .82–.93; patients .86–.93. Parallel form reliability is satisfactory.	32 in lists A-D; 34 in lists E-G.	Self-report	Seven forms (A–G). Lists A–D developed using female subjects. Lists E–G developed using male subjects. Measures affective state. (3–5 minutes)
Coopersmith Self-Esteem Inventories (SEI)[a]	Coopersmith (1967);[165] Johnson et al. (1983);[166] Mitchell (1985)[19]	School form—8–15 Adult form—≥16	—	Whites Blacks Hispanics	Convergent validity with Piers-Harris Children's Self Concept Scale r = .47; Children's Social Desirability Scale r = .63	School form only (5th grade): internal consistency .86; inter-rater .85–.97 Kuder-Richardson 20,	50 School; 8-lie scale; 25 adult	Self-report	Measures self-esteem. Consists of five subscales: lie scale, four scales to assess perceptions of peers, parents, school, and self. Adult and

(*continued*)

Table 5. (*Continued*)

Instrument	References	Populations studied: Age (years)	Populations studied: Socio-economic status	Populations studied: Race/ethnicity	Characteristics of the instrument: Validity	Characteristics of the instrument: Reliability	Characteristics of the instrument: Number of items	Characteristics of the instrument: Self-report/ interview	Comments (time)
Coopersmith Self-Esteem Inventories (*continued*)					($P < .01$ both). Discriminant with CSDS $r = .17$ ($P > .05$)	reliability coefficient .87–.92 (grades 4–8)			school forms available. Developed for women. (10–15 minutes)
Derogatis' Brief Symptom Inventory (BSI)[a]	Derogatis (1977);[167] Mitchell (1989)[20] Table 1: Reeb et al. (1987)	Mean age = 24	Norm groups heavily weighted toward lower SES	Norm groups overrepresent blacks	Concurrent validity with two scales .30–.72 (Wiggins content scales, Tryon cluster scores of MMPI)	Internal consistency Cronbach's α .71–.83. Test-retest .68–.91; alternate form .92–.99	53	Self-report	Brief version of Symptom Checklist-90. Provides multidimensional symptom measurement for psychiatric, medical, and normal individuals. (7–10 minutes)
Center for Epidemiological Studies-Depression (CES-D)[a]	Radloff (1977);[168] Roberts (1980);[169] Vernon et al. (1982);[170] Roberts and Vernon (1983)[171] Table 2: Zuckerman et al. (1989)	Adults	All levels	Whites Blacks Hispanics	—	Internal consistency: >0.88	20	Self-report/ interview	Measures lack of initiative, appetite loss, insomnia, feelings of loneliness. (5 minutes)
Clinical Measurement Package (CMP)[a]	Hudson (1982)[172]	≥12	—	—	Concurrent validity with Beck Depression Inventory .76–.85. Discriminant .52–.92. Comparative discriminant .08–.92. Construct .56–.76 (6 of 9 scales)	Internal consistency α coefficient .89–.98	25 items on 9 scales	Self-report	Measures nine types of tests on feelings about self, sex, and relationships.
Hamilton Depression Rating Scale	Hamilton (1960)[173]	Adults	All levels	All levels	Has been used to discriminate validity for inpatient and outpatient groups; good concurrent validity with psychiatrist's global rating of severity (Thompson [1989])[b]	Interrater reliability α coefficient .87–98	22	Interview	For use by clinicians only. Most commonly used symptom measure for depression in clinical drugs trials.
Symptoms of demoralization									
Psychiatric Epidemiology Research Interview (PERI)[a]	Dohrenwend et al. (1978)[65] Tables 1 and 2: Reeb et al. (1987); Molfese et al. (1987); McCormick et al. (1990)	Adults	All levels	Blacks Whites Hispanics Mexican Americans	Concurrent validity with CES-D: blacks .69; whites .66; Mexican Americans .70	Internal consistency α coefficient: blacks .92; whites .93; Mexican Americans .94. Short scale (27 items) has α = .93	44	Self-report/ interview	Measures symptoms of demoralization, anger, hostility, depression and anxiety (15–20 minutes)

(*continued*)

Table 5. (*Continued*)

Instrument	References	Populations studied: Age (years)	Socio-economic status	Race/ethnicity	Characteristics of the instrument: Validity	Reliability	Number of items	Self-report/ interview	Comments (time)
Well-being									
General Health Questionnaire (GHQ)[a]	Goldberg (1972);[177] Goldberg and Hillier (1979);[178] Buros (1978);[18] Mitchell (1985)[19] Table 1: Brooke et al. (1989); McCormick et al. (1990)	Adolescents; adults 15–74	Various educational and income levels	Blacks Whites Hispanics	Concurrent validity with previous version of GHQ-60 (60 items) .71–.88	Test-retest .51–.90 (6 months)	28	Self-report	Measures the presence of psychological dysfunction of distress. Consists of four subscales: somatic symptoms, anxiety and insomnia, social dysfunction, severe depression. (5 minutes)
Symptoms of anxiety									
Manifest Anxiety Scale (MAS)[a]	Taylor (1953)[179] Table 1: Burstein et al. (1974)	17–42	—	—	Concurrent validity correlation with MMPI .68 Correlation between old and new version .85 (Pearson)	OLD Test-retest .81–89 (3 weeks-17 months) NEW Test-retest .88 (4 weeks)	50	Self-report	Derived from the Minnesota Multiphasic Personality Inventory (MMPI). Developed to select subjects for research in human motivation.
State-Trait Anxiety Inventory (STAI)[a]	Spielberger et al. (1970);[180] Buros (1972),[17] (1978);[18] Mitchell (1985)[19] Tables 1 and 2: Gorsuch and Key (1974); Norbeck and Tilden (1983); Newton and Hunt (1984); Rizzardo et al. (1988); Norbeck and Anderson (1989); Pagel et al. (1990); Lobel et al. (1992)	Adults; high school–college students	Low income	Blacks Whites	Concurrent validity correlation with 3 scales .52–.80 (IPAT Anxiety Scale, Manifest Anxiety Scale, Affective Adjective Check List)	Interitem consistency Kuder-Richardson coefficient .91. Test-retest (1 hour–104 days): state women .16–.31, men .33–.54, trait women .76–.77, men .73–.86. α reliability, state .83–.92, trait .86–.92	40	Self-report	Distinguishes between state anxiety (transitory condition) and trait anxiety (stable condition of anxiety proneness). Norms are separately presented for female, male students. (15–20 minutes)
Diagnosis of depression/anxiety									
Schedule for Affective Disorders and Schizophrenia (SADS)[a]	Endicott and Spitzer (1978)[182]	Adults	All levels	Whites Nonwhites	Predictive validity of SADS with: Katz adjustment scale by relative (KAS-R) = .23–.58; KAS-S2 by subject = .34–.46; SCL-90 by subject = .15–.68	Internal consistency Cronbach's α .47–.97. Joint evaluation of reliability r = .82–.99. Test-retest r = .49–.93 (48 hours–1 week)	—	Interview by clinician	Intended for use with individuals currently experiencing psychopathology. (1.5–2 hours)
Diagnostic Interview Schedule (DIS)[a]	Robins et al. (1981),[183] (1982)[184]	18–65	All levels	Whites Blacks Hispanics	Lay diagnoses confirmed by clinician diagnoses range 53%–95%;	Concordance of diagnoses not affected by age, sex, and type of service	28 plus probes	Interview by nonclinician or clinician	Allows lay interviewers or clinicians to make psychiatric diagnoses according

(*continued*)

Table 5. (*Continued*)

Instrument	References	Populations studied: Age (years)	Socio-economic status	Race/ethnicity	Characteristics of the instrument: Validity	Reliability	Number of items	Self-report/ interview	Comments (time)
Diagnostic Interview Schedule (*continued*)					mean sensitivity .75; mean specificity .94; mean κ .69	(in/out patient) of subject; recency of condition some effect on accuracy; duration of condition no effect on accuracy			to DSM-III, Feighner and Research Diagnostic criteria. Assessment of lifetime diagnosis as well as current diagnosis.
Present State Examination (PSE)[a]	Wing et al. (1974)[185]	Adults	All levels	Whites	Limited formal validity testing; agreement with other diagnostic instruments, better for syndromes and diagnostic classifications than for individual items	Diagnostic sections average κ = .84; test-retest .64 (time not stated). Test-retest agreement = 84% in patients (time not stated)	140 plus probes	Self-report/ Interview	Originally developed for use in patients, also used in community samples. Focuses on current status (symptoms and function one month before interview). Responses are scored by CATEGO computer program on individual symptoms, functioning, syndromes and diagnostic class. (1 hour)
Structured Clinical Interview for DSM-III-R (SCID)	Spitzer and Williams (1988)[186]	Adults	All levels	Whites Blacks Hispanics	—	—	—	Interview by clinician	Designed to enable clinically trained interviewer to make DSM-III-R diagnoses (affective, psychotic, anxiety, substance dependence, abuse, somatoform, eating, adjustment, and personality disorders). Consists of closed and open-ended questions.

— = Not specified in studies reviewed.
[a]Used in studies of pregnant women.
[b]Thompson C, ed. The instruments of psychiatric research. Chichester, New York: Wiley; 1989.

toms. The Psychiatric Epidemiology Research Interview (PERI)[65] is a self-report measure of 17 subscales, ranging from expression of hostility to depressive symptoms (Table 5). When tested in a population study in New York City, eight of these subscales were found to be so highly correlated that they were indistinguishable from each other.[174] These subscales include measures of depression, anxiety, feelings of helplessness, feelings of hopelessness, and low self-esteem; they describe Frank's concept of demoralization.[175] Roberts and Vernon tested the reliability and validity of the PERI scales in a cross-ethnic study of whites, Mexican-Americans, and blacks in Alameda county.[170] They were able to replicate the Dohrenwend findings in all three groups; again, the eight demoralization subscales were highly intercorrelated. In addition, the PERI scales have been compared to the CES-D and the Bradburn Negative Effect Scale.[171,176] The demoralization subscale was highly correlated with each of the other scales, suggesting that all three were measuring the same underlying construct of nonspecific psychological distress, not symptoms of depression in isolation from other psychologic symptoms (e.g., anxiety). Thus, they may be

truly measuring the presence of generalized distress, also measured by the General Health Questionnaire[18,19,177,178] (Table 5), not depressive or anxiety symptoms per se.

The same problem exists for the interpretation of instruments that purport to measure symptoms of anxiety, such as the Manifest Anxiety Scale[179] and the State-Trait Anxiety Inventory[17–19,180] (Table 5). They are also not likely to be measuring symptoms of anxiety in isolation. There are several possible reasons. For example, depression and anxiety may be distinct disorders that commonly occur together, or they may be disorders on the same continuum, not truly distinct entities.[181]

The likelihood that the currently available measures of psychological symptoms of depression or anxiety are actually measuring the same psychological entity has implications for the interpretation of previous research on psychological symptoms and adverse pregnancy outcome. Studies that have used symptom scales to measure only one or the other or that have measured components of depression such as low self-esteem cannot actually determine the specific identity of the psychologic state that was measured. They then cannot offer information on the specific relationships of depression or anxiety to pregnancy outcome. In this case, the most useful criteria for choice of measure are use in other studies with comparable populations and ease of administration. The CES-D, the Beck Depression Inventory, the PERI demoralization scale, the State-Trait Anxiety Inventory, and the BSI have all been used in the studies reviewed in Tables 1 and 2 and have been extensively used in studies of psychological well-being and mental health. However, all are most likely measuring generalized distress and not depression or anxiety in isolation; thus, they are relatively comparable in terms of the underlying content they measure.

Psychiatric diagnoses: measurement. Interest in understanding the relationship of specific psychologic states to adverse pregnancy outcome leads to consideration of the use of diagnostic instruments that identify the presence of anxiety and depressive disorders as an extension of the measures of symptoms, allowing tests of the relationship of clinical levels of disorder to pregnancy outcome. Currently, four major diagnostic instruments are available and in wide use: the Schedule for Affective Disorders and Schizophrenia (SADS),[182] the Diagnostic Interview Schedule (DIS),[183,184] the Present State Examination (PSE),[185] and the Structured Clinical Interview for DSM-IIIR (SCID)[186] (Table 5). These instruments measure the presence of many different psychiatric diagnoses, including depression and anxiety. We discuss each instrument separately. Further specifications can be found in an excellent review by Hasin and Skodol.[187]

The SADS is a semi-structured interview that was developed for use in a multicenter study of the psychobiology of depression (Table 5). It was developed for use with psychiatric patients, although a version has been developed for use with persons not currently experiencing symptoms (SADS-lifetime version); all versions need to be administered by mental health clinicians. The SADS has two sections; part I is an assessment of current disorder, and part II is an assessment of lifetime disorder. Part I reviews symptom levels during the past week, with probes for both the presence of symptoms and level of severity. Symptoms are eligible for diagnostic criteria only if they include impairment in functioning; both definite and probable cases can be diagnosed. Thus, the relationship of SADS diagnoses to risk factors such as social class may be problematic. Interviewers may not be familiar with all social norms for impairment and thus may inflate rates of disorder among individuals from the low end of the socioeconomic spectrum of social class. Interviewers use all available information to make diagnoses, from medical records and informants as well as from the interview with the respondent; consequently it is costly and time-consuming to use.

The DIS (Table 5) was developed for use in the Epidemiologic Catchment Area Studies, a prevalence survey for psychiatric disorders in over 18,000 individuals in five communities in the United States.[183] Unlike the other diagnostic instruments, the DIS was designed to be administered by nonclinicians, following a fully structured interview and a specified probe flow sheet. Through structured probes, the interviewer determines if reported symptoms are due to a mental disorder or to the use of medication, drugs, or alcohol. Using computer algorithms to establish diagnoses using either the Research Diagnostic Criteria or DSM-III-R criteria, the DIS provides information on both lifetime and current prevalence of psychiatric disorders. Although the use of nonclinicians renders the DIS an attractive instrument in terms of cost, there are several problems with its use in diverse samples. A respondent who is likely to ascribe symptoms to physical illness or medication, drugs, or alcohol is likely to fall below diagnostic criteria for most disorders. This may be more likely to happen among individuals with low "psychological-mindedness" or individuals who lack access to medical care, characteristics that are particularly variable across levels of social class, ethnicity, gender, and age.

The PSE was developed in the United Kingdom by Wing et al.[185] for use with International Classification of Diseases (ICD)–8 diagnostic codes. It was subsequently revised to be compatible with ICD-9 codes. The PSE is a semi-structured interview that requires clinicians or trained nonclinicians as interviewers. It reviews the presence of symptoms during the past month, defining levels of "caseness" and "borderline caseness" for depression, anxiety, obsessive-compulsive disorders, and other mixed syndromes. It is substantially different from the other diagnostic instruments because it does not rely on explicit criteria for specific diagnoses. A composite of the PSE and the DIS, the Composite International Diagnostic Interview (CIDI), is being developed to be compatible with ICD-10 codes.[188,189]

The SCID is a new instrument developed by Spitzer et al.[186] to make diagnoses of major psychiatric disorders and personality disorders using DSM-III-R criteria. It requires clinicians as interviewers, although nonclinicians can be trained to use it. Since it is relatively new, little information on its reliability and validity is available.

Summary. Psychologic state is an important component of the life stress paradigm. The ability to mobilize social support and believe one can cope effectively with stressors is highly dependent on psychologic state. There is also evidence to suggest that a range of psychologic states, from symptoms to disorder, are associated with suppressed immune function.[190,191] Future research on risks for preterm delivery should consider carefully the interpretation of symptom measures and possibly consider the use of diagnostic instruments.

Social networks and social support. Of the many personal resources that contribute to an individual's repertoire of coping strategies, sense of self-efficacy, and mastery, social support has been the most extensively studied. Primary attention has been

directed toward research to identify potential buffering and moderating effects of support on the psychological and physical impact of stressful person-environment transactions.[192,193] Networks may differ between black and white women in such characteristics as size or density of relationship. Thus, while evidence suggests that social support moderates the stressful effects of life experiences, these results should be interpreted with caution, taking into account the diverse ways in which social networks and social support have been explained, understood, and operationalized[194–196] and the suitability of these measures to capture the life experience of black women and the specific network structures of pregnancy. The following section discusses measures of social networks, measures of received support derived from measures of social networks, and measures of perceived support.

Social networks and social support: definition. Israel describes characteristics of the social network along three dimensions: structural, interactional, and functional.[197] The structural characteristics of the social network are size (number of contacts) and density (proportion of people who know one another within the network). Interactional characteristics define the nature of the relationships within the social network, such as type of relationship (neighbor, kin, friendship, etc.), reciprocity, stability, intensity, frequency, dispersion, and homogeneity of the network. Functional characteristics describe various aspects of support provided by the social network, such as affective support (moral support, caring, love), instrumental support (tangible aid such as money, food, help with child care), cognitive support (access to information, advice, feedback), maintenance of social identity (validation of shared world view), and social outreach (social contacts and social roles).[197]

Social networks do not always consist of individuals from whom social support is received. Social networks can also be a source of negative social support and supply, poor advice, or reinforcement for bad health behaviors.[198] Consequently, a distinction must be made between the social network and the type of social support received.[199] Some individuals may have a large network of friends and family members but receive little or no social support. Gurley's concept of "social obstruction" takes negative social support one step further. It explores how intentional negative action not only undermines an individual's ability to cope with stress but also may increase stress related to a specific life event.[200] Gurley defines social obstruction as "the degree to which a person's basic social needs are violated through interactions with others. It may also be broadly understood as behaviors which block or delay normal progress through life, and by which people take away from a person those resources needed to proceed on a chosen course of action."[201] As an example, a battered wife may be systematically prevented by her spouse from seeking treatment for injuries; institutionalized racism can be considered as a stressor that creates specific types of social obstructions.

Social support has been explored in both broad and narrow terms.[202] It has been characterized as the overall basis as well as a specific element of social relationships.[202] Viewed as a resource available within a social network, social support can be defined as a transaction between individuals.[199] This transaction may involve emotional concern, instrumental aid, information, or appraisal and may depend on the relationship between network members.

Barrera proposed that social support consists of three dimensions: social embeddedness, perceived social support, and enacted or received social support.[203] Social embeddedness defines the source of relationships within the social network, such as family, church, or civic organizations. Perceived support refers to an individual's perception of the availability and adequacy of supportive others, whereas received support refers to supportive activities engaged in by others in response to a stressful situation.

There is growing evidence that interpersonal support may affect health.[199,204] Israel discusses network characteristics with a high correlation to health and well-being and suggests that the types of support needed in response to stressors differ across subcultures.[205] Berkman's review of the data relating social ties to physical health revealed evidence of complex relationships between network characteristics and mortality and morbidity.[199] For example, illness episodes or pregnancy may stimulate more social contacts and increase one's desire for maintaining these connections. In contrast, a woman's experience of feeling supported may be influenced by her psychological state;[199,206] although an adequate support system may be in place, an individual may perceive a lack of support because of depression or loneliness.

Berkman's review of the association between social networks and illness revealed an inconsistent pattern of results.[199] Some studies have shown an association between number and frequency of network contacts and decreased incidence of death and illness, but other well-controlled studies have not. There is some evidence that the direct effect of social support on physical health is stronger in men, particularly in warding off cardiovascular disease.[199] Characteristics of networks are important to measure for other reasons, since extensive networks may bring burdens as well as support. For example, women seem to be more vulnerable to the mental health effects of stressors, partly because they seem to be more vulnerable to stressors that occur to social network members (probably because women's embeddedness in a broad network is greater than men's).[207]

Social support: measurement. No instruments have been specifically developed for black women. Instruments to measure social support have developed from the differing conceptualizations of support and assessment of networks outlined above. A central strategy of measurement of social support involves simply counting the number of people with whom an individual interacts on a regular basis, that is, number of social relationships.[208] This approach emphasizes the structural characteristics of the social network. Social network measurements derived from this approach are often network size and density. Interactional characteristics describe the nature of the relationships.[197] Functional characteristics of the social network measure the individual's feelings about what is garnered from these relationships (i.e., self esteem, appraisal advice, sense of belongingness, tangible aid, other advice, validation of world view, access to social contacts).[193,197] Most instruments operationalize social support by measuring both structural and functional characteristics of the social network including number of persons; the interrelatedness of the support group; whether the support is provided by family members, friends, or others; the person's satisfaction with the support; and whether the support is actually provided or perceived to be available if needed.[206] The next four social support instruments are designed to assess various

structural and functional characteristics of social support within an individual's broad social network.

The Norbeck Social Support Questionnaire (NSSQ)[24,209,210] was designed to measure multiple dimensions of social support with three main variables and subscales: total functional, total network, and total loss (Table 6). The functional properties of social support are assessed by measuring affect, affirmation, and aid provided as part of supportive transactions. Total network is measured through three network properties: the number of individuals in the network, duration of the relationships, and frequency of contact with network members. The total loss subscale measures recent losses of important relationships; thus, it can also be considered a measure of exposure to a specific class of stressors and is not appropriately categorized as a measure of support. This subscale is interesting since the effects of recent loss (separation or bereavement) can be differentiated from the ongoing effects of small social networks or low perceived support. Although based on network theory that includes not only persons who provide support but also persons who rely on the individual for support,[211] the NSSQ measures only perceived support and not reciprocal support. The NSSQ has been repeatedly tested with good test developers, and evidence for construct and concurrent validity, strong internal consistency, and excellent test-retest reliability have been demonstrated among various ethnic groups.

The Sarason Social Support Questionnaire (SSQ)[212] measures various dimensions of the perceived availability of and satisfaction with the social support (Table 6). Research conducted by Sarason et al. suggests that the SSQ measures two distinct characteristics of social support (perceived network size and satisfaction).[212] In addition, the likelihood of a social desirability response set is low. As can be seen in Table 6, the SSQ has high internal consistency and test-retest reliability.

Procidano and Heller distinguish between friend support and family support in their measure of perceived social support. The Perceived Social Support from Friends and Family Scale[213] categorizes perceived social support according to the source, since the nature of the relationships based on friendship and family differs in fundamental ways (Table 6). Procidano and Heller suggest that different populations may rely on or benefit from one type of relationship more than another, that change through moving (for education or employment) or death may affect each network differently, and that friend relationships are generally shorter in duration than family relationships. The perceived social support for friends (PSS-Fr) and family (PSS-Fa) scales are composed of single factors, and both have high internal consistency and test-retest reliability (Table 6).

The Social Support Questionnaire[196] compiled by Schaefer et al. measures the emotional, tangible, and informational functions of social support (Table 6). Tangible support is measured based on whom an individual could go to for help in nine different situations, ranging from minor (being able to borrow a cup of sugar) to major (needing care following an illness or injury). Informational and emotional support is assessed based on five questions related to the type and nature of support provided. This instrument is a synthesis of two previously developed instruments. The tangible support measure is based on a questionnaire developed by the staff of the Stress and Coping Project, and the informal and emotional support section is taken from the Cohen Dimensions of Social Support Scale.[196] Measures of tangible and emotional support show stability over nine months, and the internal consistency of the informational and emotional support subscales is high (Table 6). The reliability of tangible support, however, is relatively low (Table 6). In a subsequent study, use of this measure was confined to the informational and emotional support section of the instrument.[37]

The next three instruments are designed to measure exclusively various aspects of social support provided by family relationships.

The Family Adaptability, Partnership, Growth, Affection, and Resolve (APGAR)[47,214,215] was developed as a family function test for practical use in clinical practice, although it has wider applications (Tables 1 and 2).[26,28,39] The Family APGAR is designed to be given to members of either nuclear or alternative lifestyle families. It measures a person's satisfaction with the following five aspects of family function: (1) how resources are shared (degree of assistance received when family resources are needed); (2) how decisions are shared (mutuality in family communication and problem solving); (3) how nurturing is shared (degree of freedom to change roles and attain physical and emotional growth and maturation); (4) how emotional experiences are shared (intimacy and emotional interaction); and (5) how time, space, and money are shared (time commitment made to the family by its members). The Family APGAR has been used among various age, SES, and racial/ethnic populations (Table 6). It also has high levels of concurrent validity, internal consistency, and test-retest reliability (Table 6).

The Family Adaptability and Cohesion Evaluation Scales,[23,216] (FACES III) was designed to measure family cohesion (degree of emotional bonding between and among family members) and family adaptability (extent of role and relationship flexibility in response to stress), the two most important dimensions of marital and family interaction (Table 6). Although the cohesive and adaptability scales are independent, the external validity of the constructs is unknown. In order to improve the utility and validity of family assessments, Olsen et al. suggest that FACES III be used in combination with behavioral assessment, clinical interview, or observational assessments of family interaction.[216] In addition, overall perceptions of support may be an average estimate and may actually vary greatly between specific individuals within the family. The use of this version of FACES III must also be limited to families with adolescent children or with no children.

The Family Inventory for Resources of Management (FIRM)[24,217] was developed to assess a family's repertoire of resources. The instrument requires information about which resources a family has, does not have, or has depleted (Table 6). The FIRM integrates elements based on personal resources (financial, education, health, and psychological attributes), family system resources (adaptability and cohesion), and social support (informational, emotional/esteem, network characteristics). Although FIRM was developed using a population suffering from chronic illnesses, the items were designed for use with any family type.[217] The instrument has concurrent validity and relatively high internal consistency (Table 6).

Social network: measurement. Table 6 lists four instruments used to measure various aspects of the social network. The Social Network List,[218] Interview Schedule for Social Interaction (ISSI),[219,220] and the Interpersonal Network Question-

Table 6. Instruments to measure social support

Instrument	References	Populations studied: Age (years)	Populations studied: Socioeconomic status	Populations studied: Race/ethnicity	Characteristics of the instrument: Validity	Characteristics of the instrument: Reliability	Characteristics of the instrument: Number of items	Characteristics of the instrument: Self-report/ interview	Comments (time)
Social support									
Norbeck Social Support Questionnaire (NSSQ)[a]	Norbeck et al. (1981),[209] (1983); [210] Educational Testing Service[24] Table 2: Norbeck and Anderson (1989); Zuckerman et al. (1989)	22–67	Low income	Various ethnic groups tested	Concurrent validity .44–.56; concurrent validity with PRQ (measures social support) .24–.41; construct validity between NSSQ subscales and FIRO-B constructs: inclusion .15–.24; affection .13–.24	Internal consistency α coefficient .89–.98. Test-retest: .85–.92 (1 week); .58–.78 (7 months)	9 items per individual for up to 20 individuals	Self-report	Measures multiple dimensions of social support. Normative data exist. (5–20 minutes)
Sarason Social Support Questionnaire (SSQ)	Sarason et al. (1983)[212]	Undergraduate students	—	—	Interitem correlation .35–.71	Internal consistency α coefficient .97; correlation between SSQ-N and SSQ-S .34. Test-retest (4 weeks): SSQ-N .90; SSQ-S .83	27	Self-report	Measures perceived number of social supports (SSQ-N) and satisfaction with available social supports (SSQ-S). Validity and reliability studies conducted on women.
Perceived Social Support from Friends and from Family Scale (PSS-Fr, Fa)	Procidano and Heller (1983)[213]	Undergraduate students	—	—	Construct validity supported	Internal consistency Cronbach's α .90; PSS-Fr = .88; PSS-Fa = .90. Test-retest .83 (1 month)	35	Self-report	Measures the extent to which an individual perceives that the needs for support are met by friends (PSS-Fr) and family (PSS-Fa).
Social Support Questionnaire[a]	Schaefer et al. (1981)[196] Table 2: Norbeck and Tilden (1983)	45–64; 20–39	All levels	Various races	—	Internal consistency Cronbach's α: info support .81; emotional support .95; tangible support .31. Test-retest (9 months): tangible .56; info .58; emotional .66 ($P < .001$)	—	Interview	Measures tangible, informational, and emotional support from spouse or partner, friends, work or school associates, relatives, and neighbors.
Family Adaptability, Partnership, Growth, Affection and Resolve (APGAR) (Family)[a]	Smilkstein (1978);[214] Smilkstein et al. (1982),[215] (1984)[47] Table 1: Ramsey et al. (1986); Reeb et al. (1987); Pagel et al. (1990)	Children 10–17; adults 18–70	College students, psychiatric outpatients, low income	Whites Blacks Asians Hispanics	Concurrent validity supported, APGAR/Pless-Satterwhite correlation .80; APGAR/ Therapist estimate correlation .64; interitem correlation .31–.53	Internal consistency Cronbach's α .86 (5 response); Cronbach's α .80 (3 response). Test-retest .83 (2 weeks)	5 with 3 or 5 possible responses to each	Self-report	Measures subject's satisfaction with five components of family function. Clinical diagnosis and research applications.

(*continued*)

Table 6. (*Continued*)

Instrument	References	Populations studied: Age (years)	Socioeconomic status	Race/ethnicity	Characteristics of the instrument: Validity	Reliability	Number of items	Self-report/ interview	Comments (time)
Family, Adaptability and Cohesion Evaluation Scales (FACES III)[a]	Olson et al. (1979);[216] Keyser and Sweetland (1988)[23] Table 1: Ramsey et al. (1986); Reeb et al. (1987)	Adults (parents); children ≥12	Low income	Various ethnic groups considered in instrument development	Cohesion and Adaptability Scales are independent (r = .03)	Cohesion α coefficient .75–.77; adaptability α coefficient .58–.63; total .67–.68	20	Self-report	Measures family systems: cohesion adaptability. No normative data available for various ethnic groups. (20–30 minutes)
Family Inventory of Resources for Management (FIRM)	McCubbin et al. (1981);[217] Educational Testing Service[24]	Adults	—	—	Concurrent validity supported in comparison to Family Environmental Scales	Internal consistency Cronbach's α .89; four scales range = .62–.85	68	Self-report	Measures perceived family resources (four scales): (1) esteem and communication, (2) mastery and health, (3) extended family social support, and (4) financial well-being.
Social network									
Social Network List	Stokes (1983)[218]	Mean = 25.2	College students	—	—	Internal consistency α coefficient for satisfaction scale .92	20 person matrix	Self-report/ interview	May be too complex for low literacy group; measures network size, density, relationship (family, friends), and satisfaction with social network.
Interview Schedule for Social Interaction (ISSI)	Henderson et al. (1980),[219] (1981)[220]	≥18	Low, middle, high	Tested only in "Western" cultures	Concurrent validity with Eysenck Personality Inventory Content and construct validity supported	Internal consistency Cronbach's α 37–.81. Test-retest .51–.79 (18 days)	52	Interview	Explores the range and differentiation of primary group relationships. (45 minutes)
Interpersonal Network Questionnaire	Pearson (1987);[221] Educational Testing Service[24]	18–60	Army reservists, middle SES	Whites Blacks	Concurrent validity with Inventory of Socially Supportive Behaviors .31 (P < .05) (Barrera, Sandler & Ramsey [1981][223])	Overall internal consistency α coefficient .79. Test-retest .54–.77 (6 weeks)	30	Self-report	Measures constructs of social networks such as social participation, confident supports, size, and frequency of contact. (30 minutes)
Networking Skills Inventory	Byrum-Robinson and Womeldorff (1990);[222] Educational Testing Service[24]	Adults	Various occupations	—	Content and construct validity supported	Internal consistency α coefficient .73–.86; relevant info .86; track record .77; working relations .73	24	Self-report	Measures networking skills to improve organizational leadership. Contributes to training and development field by providing feedback on organizational networking skills.

— = Not specified in studies reviewed.
[a]Used in studies of pregnant women.

naire[24,221] all measure network size (number of individuals), density (proportion of relationships that exist compared to total possible), and type of relationships (family or friend) within the social network. Some of the instruments like the Social Network List and the ISSI also measure satisfaction with the network and support received. The ISSI makes a distinction between what support is reported to be available and what is reported to be adequate. This comparison, as well as the interview format, permits a much more thorough investigation to be conducted of the range and differentiation of relationships and social support.[219] The ISSI explores the mutuality of relationships by measuring the extent to which the respondent feels liked and accepted by others in addition to duration, intensity, reachability, and reciprocity of the relationship. Aside from the Social Network List, which lacks validity data, these instruments are all valid and reliable (Table 6).

Unlike the above instruments, the Networking Skills Inventory[24,222] was developed to assess behaviors important to effective networking within the context of organizational leadership. This instrument is particularly useful in interventions in the workplace because it provides valid and reliable feedback about networking skills to participants. Application of the instrument beyond the work environment has not yet been explored, although the application of these measures to the measurement of skills such as health advocacy during pregnancy would be interesting.

In a comparison study of instruments designed to measure social support, Sarason et al. explored the similarities and differences between six instruments with reliable psychometric properties, including measures of perceived available social support, social network characteristics, administered support, and family social climate.[206] Instruments reviewed included the SSQ,[212] the Social Network List,[218] the Inventory of Socially Supportive Behaviors,[223] the Family Environment Scale (cohesion subscale),[224] PSS-Fr, Fa,[213] and the ISSI.[219,220]

Summary. In choosing instrumentation for social support measurement to understand the relation of support to pregnancy outcome, we recommend that structural, instrumental, and functional characteristics of the social network be measured, including dimensions of both perceived support and received support. Research demonstrates that the relative importance of functional characteristics of support and structural characteristics depends on the nature of the relationship under study.[192] Problems may arise in the use of the currently available instruments among low literacy, low SES populations.[43] In addition, little information on validity in populations of various racial and ethnic backgrounds is available for many of these measures.

CONCLUSION AND SUMMARY RECOMMENDATIONS

We began this summary of the major constructs of the life stress paradigm with a concern about the relationship of psychosocial factors to the excess rates of infant mortality and preterm delivery among black women. We outlined the major instruments available to measure these constructs and attempted to summarize the general knowledge of measurement issues within each construct. If we now move to draw on these instruments to address our specific concerns, several overall observations are striking. First, there is a dearth of available information on the reliability and validity of most of these measures in studies of black women. Second, most of the studies of psychosocial factors and adverse reproductive outcome reviewed in Tables 1 and 2 have not measured the major components of the life stress paradigm in adequate breadth, which may account for the weak associations found for stress and pregnancy outcome. For instance, only a few of the studies reviewed considered the relationship of personal disposition to adverse outcome, and many did not measure social networks or social support; most importantly, all relied on instruments that inadequately measure exposure to multiple types of stressors (such as Holmes and Rahe's SRE).

Six measurement considerations concerning the evaluation of life stress should be addressed in order to further our understanding of psychosocial risks for adverse reproductive outcome in black women.[98] First, it is crucial to consider the content of the instrument used to measure exposure to stressors. Differential exposure of black women and white women to stressors not usually included in most studies of stress, such as chronic strains, role strains, "non-events," positive events, the lasting effects of remote traumatic stressors, or unique exposure to stressors related specifically to racism, may be partially responsible for differences in rates of adverse reproductive outcome. A second, and related, consideration is the focus of the stressor. If only stressors that occur to the pregnant woman herself are considered, there may be few differences in exposure between these two groups. If the focus is broadened to include stressors that occur to members of her household and social network, however, black women might be found to be at higher risk. The benefits of support from a diverse network may be overshadowed by greater vulnerability to exposure to stressors that occur to members of the network, resulting in increased "network burden." The timing of stressors is a third consideration. The relative timing or "layering" of stressors, both in relation to vulnerable periods in pregnancy and to each other, may also be an important differentiating factor. Fourth, specific characteristics of stressors that vary within each type of stressor need to be considered. Aspects of intraevent variability such as the severity of the stressor, amount of related behavioral change, degree of anticipation, degree of long-term contextual threat, duration, degree of control, salience to the respondent, and type of appraisal (positive, negative) are aspects of stressors crucial to measure to accurately quantify the stressful components of the exposure. Fifth, practical issues of measurement need to be addressed. Methods to ensure that all stressors reported by a respondent are independent of each other and to develop a priori rules for the handling of "linked" events or events that occur in a sequence and methods to elicit a broad range of stressors and to reduce problems of memory failure, respondent boredom, and the likelihood of negative response sets are also important areas that need to be addressed.

Finally, consideration of the mechanisms through which stressors are proposed to exert their effects is critical. Many instruments to measure stressors contain implicit assumptions about the ways in which stressors lead to poor health outcomes (e.g., through behavioral change or psychological meaning). Ideally, assumptions about mechanism should be separated from the measurement of the occurrence of stressful experiences so that specific hypotheses for psychosocial and physiologic mechanisms underlying the association of stressors to reproductive outcome can be tested and understood. Careful attention to possible mediating and moderating factors is important. For

example, if all black women experience racism, what are the possible protective aspects of personal dispositions, social support, psychologic state, or exposure to other stressors that buffer the strain of racism among those women who do not experience adverse pregnancy outcome?

Very little is known about the role of personal dispositions in buffering or exacerbating the effects of stressors on pregnancy outcome. The role of coping responses and aspects of personality such as self-efficacy, mastery, hardiness, type of flexibility of coping style, perceived control over life experiences, or coping strategies that are specific responses to racism such as John Henryism needs to be addressed. First, descriptive information on the relative distribution of these aspects of personal disposition among black and white women would be informative. Second, conceptual interrelationships among these aspects of personal dispositions need clarification. For instance, are measures of mastery and beliefs about self-efficacy and hardiness highly correlated or does each measure unique aspects of personality? Are these interrelationships consistent across race, ethnicity, class, or gender? Are these personal dispositions stable throughout pregnancy, a situation in which locus of control may have an altered meaning? And finally, how are these personal dispositions related to differences in rates of preterm delivery between black women and white women?

Considerations in the measurement of psychologic state are more straightforward. Most studies of psychosocial factors and preterm delivery have relied on measures of symptoms of depression or anxiety. Measures such as the BSI, the Beck Depression Inventory, the CES-D, the State-Trait Anxiety Inventory, Rosenberg's Self-Esteem Scale, and the PERI demoralization scale are likely to be highly correlated with each other and to measure generalized distress rather than symptoms unique to either depression or anxiety. Nonetheless, anxiety and depression are specific psychologic responses to stressors that may be important components of the life stress process to measure because these responses may have an impact on obstetric risk behaviors such as smoking and may behave differently physiologically. The use of reliable, cost-effective diagnostic instruments in addition to measures of symptoms could clarify whether current or past experiences with depressive or anxiety disorders are specifically related to differences between black and white rates of preterm delivery.

Several considerations can also be raised in the measurement of social support. Cohen and Syme outline a series of questions that provide a useful framework to focus further research on social support in general and are particularly useful for understanding differential rates of preterm delivery for black women and white women.[193] Who provides support (e.g., issues of reciprocity, appropriateness, norms)? What type of support is provided (e.g., emotional, instrumental, informational, confidant)? How do characteristics of the receiver determine the provision of support? What is the degree of event-specific support, the timing and duration of support? What are the costs of providing and receiving support and possible interactions among these costs?[193] Although perceived support may be confounded with psychologic state, the relationships among perceived support, adequacy of support, and received support need further clarification. Further understanding of interactions of perceived support with coping responses and appraisal may shed light on risk factors for preterm delivery. For example, do black women who score high on John Henryism develop social networks with different characteristics than women who score low? Is the relationship between perceived and received support different in these two groups of women, and how does it differ by social class?

These substantive considerations need to be explored within both qualitative and quantitative research strategies. An initial research focus on qualitative approaches (such as the development of focus groups within a range of social contexts) seems crucial to provide the best chance for accurate specification of many of the substantive issues and constructs noted above. For example, such an initial approach is essential for the development of comprehensive inventories of stressors that black women and white women may experience differently. Quantitative research on risks for preterm delivery among black women must then be based on clearly specified hypotheses of both psychosocial and physiologic mechanisms and their interactions. The formulation of hypotheses and testing of specific causal models such as those developed in research on life stress and mental health are essential to the search for further understanding of risks for preterm delivery among black women and for effective interventions.

We emphasize the need for prospective attention to two goals for future research. First, because of the scarcity of previous research on psychosocial risks for adverse pregnancy outcome that adequately measures the major components of the life stress paradigm, it is imperative to design multidisciplinary research programs that systematically take a comprehensive approach to the evaluation and understanding of the specification of these constructs in black women. Second, there is demonstrated need for the evaluation and modification of existing measures. Although many instruments available have not been validated in populations of black women, dismissing them entirely would be premature. Modifications can be assessed in the context of qualitative research with both pregnant and nonpregnant women. In order to clarify the stress-disease relationship in black women, there is also a clear need for the development of new measures that can address consequences of stressors such as racism, sexism, and discrimination based on social class. Evaluation and development of psychosocial instruments is costly and time-consuming. Yet without attention to the theoretical, psychometric, and practical issues of appropriate psychosocial instrumentation, we will not be likely to identify specific risks for preterm delivery in black women. Such efforts to test, modify, and develop new instruments will also add substantially to the measurement resources available for understanding the multiple processes through which stressors affect all aspects of health and disease across race, ethnicity, and social class.

We adapted our methodological critique of measurement of stressors from McLean and Link's chapter in the forthcoming text *Stress and Mental Health: Contemporary Issues and Prospects for the Future.*

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Racism, Sexism, and Social Class: Implications for Studies of Health, Disease, and Well-being

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One of the most persistent and pressing public health problems in the United States today remains among the most poorly understood: the excess rates of low birthweight (LBW) and infant mortality among children born to black women.[1–5] Although both the incidence of LBW and the infant mortality rate in the United States have been declining for many decades, in any given year, black women are twice as likely as white women to experience the sorrow and loss of their babies' deaths before they reach one year of age (Figure 1).[6–10]

To explain these trends, researchers have invoked two well-known facts. One—recorded since the advent of collecting vital statistics—is that infant mortality rates, in the aggregate, are higher among poor and less educated women.[11–19] The second is that black women in the United States have persistently endured higher levels of poverty than white women;[6,20] according to the 1990 census, 34% of black women and 11% of white women were living below the poverty line.[20] The usual inference drawn from these facts is that the high rates of LBW and infant mortality among black women are attributable to their high rates of poverty.

Yet closer inspection of the data reveals an unusual and disturbing pattern among black women. Although their rates of LBW and infant mortality do rise as their levels of poverty and education decrease, the gradient is much less steep than that observed among white women (Tables 1 and 2).[4] One consequence is that although rates of infant mortality and LBW are highest among both black and white poor and less educated women, the black/white ratio of infant mortality rates is *lowest* among women who have not completed high school (rate ratio = 1.7) and is *highest* among women with a college education (rate ratio = 2.0).[1] Bluntly stated, black women have problematic birth outcomes regardless of their socioeconomic position, they fare worse than white women at every economic level, and their disadvantage persists even among the most highly educated black women.[2,4,21]

The flip side of this "black paradox" is the "Hispanic paradox," which should be labeled more appropriately as the "Mexican paradox" because it involves birth outcomes among Mexican-American and Mexican-born women.[22–25] Despite comparable sociodemographic factors (Table 3), the LBW and infant mortality rates among Mexican-American and Mexican-born women apparently are lower than among black women, at every economic level and at all levels combined (Tables 1 and 2).[22–26] The reported birth outcomes of Mexican-American women, however, are on a par with those of white women, and even poor and less educated Mexican-American women have low rates of infant mortality.[10,22–26]

To date, these paradoxes of consistently adverse birth outcomes among black women and favorable birth outcomes among Mexican-American and Mexican-born women remain unexplained. Although some evidence suggests that the "Mexican paradox" may be spurious and result from the underascertainment of infant deaths among Mexican-Americans,[10] the fact remains that blacks experience higher rates of infant mortality than whites for all leading causes of death except congenital anomalies.[9] Only a small proportion of excess black infant deaths can be accounted for by the major known risk factors for infant mortality, such as inadequate prenatal care, higher parity, and little education. Knowledge about the causes of these risk factors is also incomplete. Preterm delivery, for example, is the third leading cause of infant mortality, by virtue of being the predominant cause of LBW infants.[9] Very little is known, however, about what triggers preterm delivery.[27,28] Factors identified to date include infections, incompetent cervix, and other maternal conditions related to pregnancy, such as preeclampsia, abruptio placenta, and cocaine use.[27–29] Yet even taking these into account does not explain black/white differences in infant mortality.

Far from being isolated gaps in our knowledge, these paradoxes represent a larger problem: a persistent inability to explain a myriad of racial/ethnic, particularly black/white, differences in health.[7,30–33] One recent national study, for example, found that among people 35 to 54 years old, the overall black mortality rate was 2.3 times higher than the white mor-

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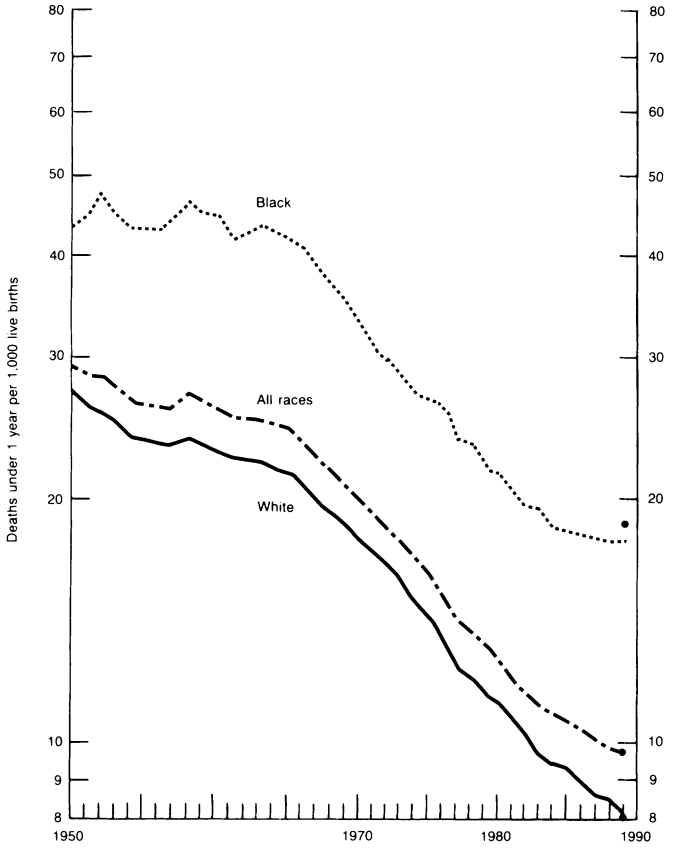


Figure 1. Infant mortality in the United States, 1950–1990. Note: Infant deaths are classified by race of decedent. Since 1989, live births have been classified by the race of the mother; from 1950 through 1988, live births were classified by the race of the child.

Table 1. Infant mortality rate per 1,000 live births, by education and race/ethnicity of mother for infants born in the United States, 1983–1985

	Education				
Race/ethnicity	<12 years	12 years	13–15 years	≥16 years	Years unknown
White, non-Hispanic	13.9	8.9	7.5	6.5	11.5
Black, non-Hispanic	22.4	18.3	16.5	14.4	23.0
Mexican American	10.9	8.2	6.5	8.3	10.5
Puerto Rican American	14.0	11.7	9.4	5.2	33.2
Cuban American	11.8	8.3	8.5	4.8	11.0
Native American	16.8	13.8	11.5	8.1	19.0
Asian American	10.1	9.4	7.2	6.5	11.0

Source: National Center for Health Statistics. Linked birth/infant death files 1983–1985.

tality rate, and only 31% of this excess mortality could be attributed to the six major risk factors linked to excess black mortality (smoking, systolic blood pressure, cholesterol level, body mass index, alcohol intake, and diabetes).[33] Of the remaining 69%, slightly over half (38%) was attributable to family income (presumably operating through pathways other than those represented by the six major risk factors), whereas the rest (31%) remained unexplained—a share as large as that attributed to all the well-known risk factors combined.[33] Additionally, the all-cause mortality rates among black and white people aged 55 to 77 years of age were almost equivalent, despite the greater prevalence of adverse risk factors among black people in this age group.[33] These and other unresolved puzzles suggest that new approaches are needed to identify the

Table 2. Relative risk (and 95% confidence interval) of infant mortality by education and race/ethnicity of mother for infants born in the United States, 1983–1985

	Education				
Race/ethnicity	<12 years	12 years	13–15 years	≥16 years	Years unknown
White, non-Hispanic	1.0 (Reference)	1.0	1.0	1.0	1.0
Black, non-Hispanic	1.60 (1.56, 1.64)	2.04 (1.99, 2.08)	2.18 (2.10, 2.26)	2.20 (2.08, 2.33)	1.98 (1.92, 2.04)
Mexican American	0.79 (0.72, 0.85)	0.92 (0.81, 1.05)	0.88 (0.70, 1.09)	1.27 (0.85, 1.91)	0.91 (0.89, 0.94)
Puerto Rican American	1.01 (0.93, 1.01)	1.31 (1.18, 1.44)	1.25 (1.03, 1.51)	0.80 (0.52, 1.23)	2.83 (2.34, 3.42)
Cuban American	0.85 (0.67, 1.08)	0.94 (0.76, 1.16)	1.14 (0.83, 1.57)	0.74 (0.46, 1.19)	0.96 (0.66, 1.39)
Native American	1.21 (1.11, 1.32)	1.55 (1.41, 1.70)	1.54 (1.30, 1.82)	1.25 (0.83, 1.87)	1.64 (1.45, 1.85)
Asian American	0.73 (0.65, 0.82)	1.05 (0.95, 1.16)	0.96 (0.82, 1.11)	1.00 (0.89, 1.12)	0.95 (0.90, 1.01)

Source: National Center for Health Statistics. Linked birth/infant death files 1983–1985.

Table 3. Sociodemographic profile of women and birthweight distribution by race and ethnicity, United States, 1983–1985 (by percentage)

	White, non-Hispanic	Black, non-Hispanic	Mexican American	Puerto Rican American	Cuban American	Native American	Asian American
Maternal age (years)							
<18	3.4	11.0	7.3	8.8	2.4	8.1	1.4
18–34	90.5	84.4	86.1	85.4	90.1	86.5	86.8
>34	6.1	4.7	6.6	5.8	7.4	5.4	11.8
Marital status							
Married	87.8	39.5	77.5	49.2	83.9	54.1	92.6
Unmarried	12.2	60.5	22.5	50.8	16.1	45.9	7.4
Maternal education (years)							
<12	14.2	28.9	9.5	45.4	20.7	33.9	11.6
12	36.5	36.9	4.4	33.0	35.2	35.0	16.5
13–15	17.4	14.7	1.5	11.2	23.0	13.0	9.7
≥16	15.6	5.9	0.5	4.0	12.5	3.1	18.3
Unknown	16.3	13.6	84.1	6.3	8.6	15.0	43.8
Birthweight (g)							
<500	0.1	0.3	0.1	0.1	0.1	0.1	0.1
500–1499	0.8	2.4	0.9	1.3	0.9	1.0	0.8
1500–2499	4.7	10.1	4.9	7.4	4.8	5.1	5.6
≥2500	94.3	87.1	94.0	91.0	94.1	93.7	93.4

Data source: National Linked Infant Birth-Death Files, 1983–1985.

unknown risk factors and protective factors shaping black/white differences in morbidity and mortality.

One new direction suggested by a small but growing body of epidemiologic research involves investigating whether the everyday realities of racism, sexism, and social class can help explain poorly understood racial/ethnic differences in diseases.[16,34–49] These studies are challenging not only the methods, but also the concepts and conclusions of traditional approaches to analyzing racial/ethnic disparities in health. Although potentially promising, these studies have been hampered by two related problems: the lack of a clear conceptual framework to guide the research and the lack of appropriate measures and methodologies to test the relevant hypotheses. These obstacles demand attention. We need legitimate alternative approaches because conventional strategies have not yet yielded sufficient insights to understand, let alone reduce or prevent, black/white differences in health.

As a step toward developing this alternative research agenda, we examine three issues. First, we summarize and critique the predominant methods employed by U.S. epidemiologists to study how social relations of race, gender, and social class influence health. Rather than provide an exhaustive review of the findings to date, we analyze the conceptual framework underlying this work. We believe it is necessary to consider all three areas, singly and combined, in depth because the rationale for current research strategies on these topics is rarely discussed in the epidemiologic literature, thereby hindering evaluation of the limitations and strengths of the operative concepts, theories, and methodologies. We focus primarily on studies about black/white differences in health (specifically somatic, as opposed to mental, health), in part because these disparities have spurred much of the research regarding racism and health, and in part because the principal public health question motivating this article began with concerns about black/white differences in preterm delivery and has evolved into an examination of the causal factors contributing to preterm delivery among black women. Second, we review additional, as well as newly emerging, approaches to investigating the relationship between racism, sexism, social class, and health. Finally, we propose recommendations for future research. Our perspectives are based on our diverse experiences as white and black public health and social science researchers and advocates: four women born and raised in the United States (one white, three black) and one black South African man now living in the United States.

PREDOMINANT APPROACHES TO STUDYING RACISM, SEXISM, SOCIAL CLASS, AND HEALTH: A SUMMARY OF THE U.S. EPIDEMIOLOGIC LITERATURE

Race, Racism, and Health

Historical background. Studies attempting to determine whether "racism" or "race" is at the root of black/white differences in health in the United States are not new.[50–54] In 1859, Dr. James McCune Smith, the first university-trained black physician in the United States, challenged the prevailing view—one upheld most strenuously by supporters of slavery—that the poorer health status among blacks reflected their innate inferiority.[51] In a novel step for his times, Smith argued that apparently intrinsic traits could be the consequence not of innate factors but of environments that were socially created and inherited from one generation to the next. To prove his point, he compared the prevalence of bone deformities from rickets (a condition he attributed to poor diet) among parents and children in both black families and poor white families. Finding the rates to be nearly equal, Smith concluded that rickets was more prevalent among black families not because they were biolog-

ically "black" but because they were poor and they were poor because they lived in a society that either condemned blacks to slavery in the South or to a marginal existence as the most poorly paid workers in the North.[50]

To Smith, the fundamental point revealed by this and related research was that "race" is a social, not natural, category. Advancing the same type of antiessentialist and population-based thinking that Charles Darwin ushered in through his work *On the Origin of Species* (also published in 1859),[55,56] Smith wrote:

> The fallacy in the argument has consisted in this: the variations in the black race have been arranged together and have been called the type of the race, and as such have been compared with, not the varieties, but the general type of whites, and from this comparison, the illogical conclusion has been adduced that there is a permanent difference between these two races. This argument is about as conclusive as if we were to select all the white men in this city who have grey eyes, and to argue that because the color of their eyes differs from that of the remainder, therefore the two classes belong to different races.[51,p227]

Based on this reasoning, Smith concluded that "the term white is an arbitrary one, when used in contradistinction to black, the latter meaning the colored mixed race now enslaved in the Republic."[51,p233] Just as societal conditions created black/white differences in health, so too they established the very categories of "black" and "white."[50]

Since Smith first articulated these views, a huge and diverse body of work has confirmed (or failed to disprove) his pioneering hypothesis.[56–63] Social and natural scientists now agree racial categories reflect social and ideological conventions, not natural distinctions.[56–67] Although different racial groups may exhibit different physical characteristics (e.g., skin color), groups defined by these traits are highly heterogeneous: genetic differences *between* racial groups amount to only a tiny fraction of genetic differences *within* groups.[56–62,68] Richard Lewontin, a human geneticist, observes that, "for 75 percent of the known human genetic endowment, *all* humans are identical irrespective of their geographical origin."[68,p120] Moreover, for 17 well-studied polymorphic human genes, "85 percent of human genetic diversity is *within* national populations and only 7.5 percent between nations within races and 7.5 percent between major races."[59,p155] In fact, as Lewontin has noted, the more accurate estimate of within-group variation may be as high as 95% because most of the between-group variation stems from the contribution of a few small and relatively isolated populations (e.g., Eskimos and Australian aborigines).[59]

Thus, despite the 1988 definition of race as "persons who are relatively homogeneous with respect to biological inheritance" in *A Dictionary of Epidemiology* (sponsored by the International Epidemiological Association),[69,p88] scientific consensus presently holds this biological understanding of race as, at best, an anachronism.[56–62] Lewontin points out the predominant view: "taxonomic division of the human species into races places a completely disproportionate emphasis on a very small fraction of the total of human diversity."[59,p156] For example, a few diseases have been linked to traits like skin color used to construct racial categories (e.g., malignant melanoma, a type of cancer that primarily affects light-skinned people),[30,70] and also to traits thought to be conditioned by geographic origins (e.g., sickle cell anemia, which may be protective against malaria and which is most common among Mediterranean and African peoples living in regions where malaria has been hyperendemic).[30,58] However, these account for an extremely small fraction of observed racial disparities in morbidity and mortality.[8,30,32] Cooper and David have reported, for example, that in 1977 the age-adjusted death rate in the United States among blacks was 37% higher than among whites, but only 0.3% of the total excess of black deaths could be coded to hemoglobinopathies—that is, to conditions related to sickle-cell anemia, the only known potentially fatal black-linked disease.[32] The accumulated evidence indicates that, for virtually every racial/ethnic group, a handful of genetic diseases seem specifically associated with aspects of their geographic and biological heritage, yet these diseases nonetheless account for only a minute percentage of each group's overall morbidity and even less of their mortality.[30] The fact that we know which race we belong to says more about our society than our biology.[31]

Conceptual framework and definition of terms. Current epidemiologic research regarding racism and health consequently recognizes that the populations categorized as races are not biologically distinct and innately different[31,32,56–62] and does not presume that overall racial disparities in health primarily reflect biologically determined differences in genome or physiology. The combination of overwhelming similarities in genetic constitution and striking differences in health status instead points to an alternative question: what are the possible exposures contributing to these population differences in disease?[71,72] At issue is how health is influenced by injurious social divisions based on race and by cultural differences linked to ethnicity.

Investigators involved in this research uniformly consider the only appropriate measure of race to be self-identification,[66] as opposed to allegedly biological measurements used in some epidemiologic studies, such as skin color[73] or the presence of the Duffy antigen.[74] Individual researchers nonetheless use variable terminology to categorize U.S. racial/ethnic groups (e.g., "Latino" versus "Hispanic").[75–77] To be consistent with national census data and vital records, we employ the terms promulgated by a 1977 directive from the federal government's Office of Management and Budget: "White," "Black," "Asian or Pacific Islander," and "American Indian or Alaskan Native," all of which are considered to be races, plus "Hispanic," which specifies ethnicity only; Hispanics can be of any race (Appendix).[67] Although important ethnic differences exist within each of these racial groups[8,78,79] and also within each of the ethnic subgroups termed "Hispanic,"[23,75,79] we will refer to each racial/ethnic group only in the aggregate (unless we state otherwise).

We define "racism" as an oppressive system of racial relations, justified by ideology, in which one racial group benefits from dominating another and defines itself and others through this domination.[64,65,78–87] Racism involves harmful and degrading beliefs and actions expressed and implemented by both institutions and individuals, as linked to their membership in racially defined groups.[65] At its core, racism is based on four false assumptions:[50,51,53,56,60–62]

1. Humans are naturally divided into biologically distinct and inherently different races.
2. The genetically determined physical attributes conventionally used to identify "race" are inherently linked to other

characteristics (e.g., mental abilities, physiologic functioning) also assumed to be genetically determined.

3. Differences between racial groups (in the realm of culture, behavior, health, and other areas of human life) directly reflect genetically based differences inherent among all individual members of each racial group.

4. Certain races are inherently inferior to other races, whereas others are inherently superior (biologically or culturally).

Translated into daily life, these beliefs and practices both justify and result in vast inequalities in living conditions and opportunities for members of the oppressor and oppressed racial groups.[64,65,79–87] Ranging from blatantly overt to insidiously subtle, the phenomena of racism typically include multiple forms of segregation (e.g., political, occupational, residential), as well as demeaning and often daily insults. Members of the oppressed group are routinely marginalized and patronized by members of the oppressor group. They are suspected of cheating and thievery, suffer rude service at public accommodations and restaurants, encounter hate stares and racial epithets from strangers on the street, and are treated unfairly by law enforcement and other government officials.[64,65,79–87] They face a pervasive threat of violence, ranging from the extremes of hate crimes to the more general fear for one's safety and that of friends and family, particularly those who must live in impoverished areas plagued by drug-related and other violence.[64,65,79–87]

Within the United States, the principal feature distinguishing these oppressor and oppressed racial groups is "color."[64,65,79–87] The origins of this split can be traced back to two linked aspects of U.S. history: the settlement of European colonists in an already inhabited "New World" and their decision to import African slaves (who were easily identifiable) to augment their limited labor force.[50,84,87–90] Reflecting this legacy, current racial antagonisms in the United States principally involve two groups: whites (chiefly persons of European descent) and people of color (persons of African descent, indigenous inhabitants of North America and Mexico, and persons with ancestry traced to immigration from Asia, the Pacific Islands, and the rest of Central and South America).[79,80] Conflicts also have occurred and continue to occur between members of different white ethnic groups (e.g., Anglo-Saxon, Irish, and Italian), between members of different groups of people of color (e.g., blacks, Hispanics, and Asians),[79,91] and, within these latter groups, between subgroups divided by ethnicity or gradations of color.[79] However, it is the day-to-day real differences in life for people on *both* sides of the color line that most sharply distinguish the contours of racism in the United States today.[64,65,79–87]

Predominant approaches to studying racism and health. To date, only a small fraction of epidemiologic research in the United States has investigated the effects of racism on health,[4,21,30,33,37–43,48,49] that is, the health consequences of racial subordination, as opposed to the more traditional public health concern with racial differences in disease. Most of these studies on racism and health have focused on determining whether health outcomes are comparable among members of different racial/ethnic groups at the same socioeconomic level[4,33,37,39,92,93]—the strategy used by Dr. James McCune Smith in 1859.[51]

The implicit hypotheses and logic underlying this approach are as follows:

1. One important manifestation of racism is economic and social discrimination.

2. Because of this discrimination, people of color (and especially blacks) are disproportionately concentrated among the ranks of the poor, the unemployed, or those employed in low-paying (and often hazardous) jobs, as well as among people with limited or no health insurance.

3. Conditions associated with poverty, unemployment, and menial labor affect health adversely and can be aggravated by a lack of preventive and therapeutic health care.

4. Biological similarities between members of different racial groups imply that each will fare equally well or poorly in comparably beneficial or harmful environments.

5. If members of different racial/ethnic groups have similar health outcomes at each socioeconomic level, overall racial/ethnic disparities in health reflect racial/ethnic inequalities in socioeconomic conditions.

To test these hypotheses, this type of research therefore systematically investigates whether social gradients exist for the diseases in question and, if so, whether the adverse outcomes are equally likely among the different racial/ethnic groups at each socioeconomic level. In this approach, questions of race are often implicitly, and at times explicitly, reduced to questions of class.

Stated more technically, these types of studies first examine racial/ethnic differences for the health outcome of interest, accounting for age and gender. In those few cases where no differences exist (e.g., the incidence of colon cancer among blacks and whites),[93] the tacit conclusion is that neither racism nor related socioeconomic factors contribute to explaining the epidemiology of that particular condition. Otherwise, the next step is to adjust for socioeconomic status, alone or in conjunction with other known risk factors for the health outcome under study. If this step reduces or eliminates racial/ethnic differences in disease risk or occurrence, the implication is that socioeconomic factors associated with racial inequalities in living conditions contribute to observed racial/ethnic differences in disease. A corollary is that socioeconomic factors influence the distribution of the disease within each racial/ethnic group.

Research concerned with black/white social class patterns of disease dates back to at least the early 1900s. Examples include studies about overall morbidity and mortality,[33,94,95] congenital malformations,[96] respiratory diseases,[97] cancer,[37,93,98–100] cardiovascular disease,[101,102] reproductive health,[4,5,49] mental health,[103] and access to health care and type of treatment received.[104–106] To adjust for social class, these studies typically use data about the individual study subjects' educational level, family income, and sometimes occupational rank, either separately or combined into a single socioeconomic index (see our discussion of social class measures, pages 94–5). Another variant is to use neighborhood-based measures of socioeconomic level or disorganization.[37,49,93,98–100] These studies have shown that although adjusting for socioeconomic factors often substantially reduces (and at times eliminates) black/white differences for many diverse health outcomes, in many cases these differences in well-being persist even after analyses are adjusted for the standard socioeconomic measures.[30,49,92,93,98–100]

Although not as common, one other accepted approach to studying the effects of racism on health does not assume that black/white health differences can be fully explained by class

alone but instead focuses on the health consequences of structural racial segregation in the labor force.[107–113] This research seeks to determine the extent to which diverse and potentially distinct occupational exposures contribute to each racial/ethnic group's overall burden of illness. The rationale for this approach is that, because of racism, black workers are more likely than white workers to be concentrated in the most hazardous or repetitive and mind-numbing jobs.[84,107–114] thus implying that black and white workers often do not encounter the same types of occupational hazards. Several studies suggest that these occupational conditions—including exposure to occupational toxins, risk of injury, and job-related stressors—may contribute to racial/ethnic differences for a variety of health outcomes.[107–113] However, little research of this type has been conducted, in part because of the relatively small number of black workers employed within specific occupations at any given worksite.[111]

Limitations. In addition to deploring the scarcity of studies, several important criticisms have been raised regarding the predominant epidemiologic approaches to studying the effects of racism on health.[31,32,38,42,48,49,66] These criticisms concern (1) flaws in analyzing the role of social class in black/white and other racial/ethnic differences in health, (2) the absence of measures pertaining to other aspects of racism, (3) the lack of studies addressing diversity among people of color, and (4) the gender-specificity of racism.

Racism and class. Recently, the most common approach to studying racism and health has come under question, that is, the technique of adjusting for social class to determine if observed racial differences in health can be explained by racial differences in social class composition.[32,38,42,48,49] Specifically under scrutiny are the two cardinal assumptions of this methodology: (1) that the socioeconomic conditions of black and white people within each socioeconomic category are at least roughly comparable and (2) that the two groups overlap sufficiently in socioeconomic distribution to permit adjusting for social class. As some critics have begun to note, neither of these assumptions necessarily is true.[48,49,82,91,114–120]

Numerous studies, for example, have established that the "economic return" for the same level of education is lower for blacks than whites (and also for women than men within each racial group). This disparity holds whether economic return is measured in terms of actual salary, nonwage benefits (including health insurance), or occupational status.[48,82,114,116,117,119,120] Additionally, within the same occupational titles, blacks are more likely than whites to be employed in lower-paying and lower-status positions.[48,82,114,116,119,121] As a result of the legacy of residential segregation, not only are black professionals much more likely than their white peers to live in working class and less affluent neighborhoods,[79,82,122–124] but black families below the poverty line are much more likely than white families below the poverty line to be concentrated in impoverished neighborhoods.[49,82,124,125] Furthermore, the black poor on average are much poorer than the white poor. A 1988 national survey found, for example, that among the lowest quintile of the U.S. population defined by family income, 29% of black families versus 9% of white families had no assets or were in debt.[118]

Together, these data suggest that black/white disparities may not be statistically eliminated by adjusting for the standard socioeconomic indicators, especially if the adjustment is conducted with only one measure, as opposed to several. Disparities may remain even for those outcomes where differences are in fact attributable *only* to racial differences in socioeconomic position. The data also raise the possibility of an even more formidable obstacle to analyses dependent upon adjusting for social class—the gross disparity in living conditions among blacks and whites. That is, if no or very little overlap exists in the different strata of the selected socioeconomic indicators, the technique of direct adjustment cannot legitimately be used.[48,49]

A final, albeit limited controversy concerns the legitimacy, from a theoretical standpoint, of adjusting for social class when comparing the health status of blacks and whites. Despite widespread use of this technique, Cooper and David, for example, have argued that "accounting for education, income, etc. in the effort to explain racial differentials represents overcontrol; race is not confounded by the other variables, it is antecedent to them."[32,p113]

Others, however, dispute this logic on several grounds.[31,92,126,127] First, although racism is the primary reason that blacks are disproportionately concentrated in the poorest sectors of the working class and face restricted class mobility, at the macro level, race is not antecedent to class per se.[79,82,84–89,126,128–133] Although the historical intertwining of class and race relations in the United States has resulted in white Americans occupying the most privileged positions in society, more than racism is involved in the creation and continuance of social classes in the United States today. Also causative are macroeconomic forces (both sociopolitical and technological) that shape the structure of the entire U.S. workforce and its position within the world economy.[79,82,84,85,87–89,126,128–133]

Second, despite Cooper's valid observation that "the unadjusted differences are the public health fact of life,"[134,p112] a necessary first step in explaining these differences is to account for the effects of social class on health. We acknowledge that controlling for social class does not address noneconomic aspects of racism, and, for this and other reasons, it may not always statistically "eliminate" socially created black/white differences in health. Failure to consider the role of social class, however, permits naive genetic explanations to remain unchallenged. Consequently, although analyses that conduct this type of adjustment are incomplete if they fail to discuss how racism leads to profoundly different class distributions among blacks and whites, the alternative of not accounting for the role of social class is unacceptable for any studies regarding racial/ethnic differences or any other differences in health status.[31,66,92,126,127]

A useful analogy concerns the standard epidemiologic technique of adjusting for age. Just as this procedure can at times obscure important age-related effects (e.g., a crossover in the age-specific incidence of breast cancer among black and white women),[37] in many more instances it permits concise comparisons of disease rates in selected populations whose age structures differ and for whom age is fundamentally linked to the occurrence of disease. Similarly, although adjusting for social class when comparing racial/ethnic differences in health is inappropriate if conducted before assessing whether effect modification exists (i.e., levels of disease or of risk factors differ among blacks and whites in the same socioeconomic strata), this stipulation does not imply that adjusting for social class is inherently a flawed analytic approach or necessarily reduces black/white differences to solely a question of class. Instead, the technique

of stratifying by, and potentially adjusting for, social class provides a means of determining the extent to which black/white differences in health are mediated by the concentration of blacks in poverty and the working class. If, however, effect modification exists, only stratified results should be presented, as is true for any type of epidemiologic analysis.[135–140]

Other aspects of racism. A second significant limitation of public health research about racism and health is that very few studies have directly addressed the many noneconomic aspects of racism. Although racism clearly has its damaging economic dimensions, other aspects are also likely to be detrimental to people's health.[38–43,64,65,82,87,108,109,120,141–143] These include the psychosocial effects of racial discrimination and oppression (e.g., all forms of racial exclusion and subordination),[38,39,42,65,142] the problems resulting from a lack of access to adequate health care,[34,144] and the physical and psychosocial consequences of residential and occupational segregation and of incarceration.[79,82–84,87,108,109,116,122,124,128,141,145–147]

Of particular concern are the ways in which both subtle and overt forms of racism, both within and across social classes, can invalidate people's sense of self-worth and lead to internalized oppression, that is, the process whereby people of color adopt the oppressor culture's denigrating views and judge both themselves and others in their racial/ethnic group according to these criteria.[64,65,79,82–84,87,142,143] This self-denigration in turn can potentially compromise available social support and renders explicit validation an all-too-rare event.[64,65,79,82–84,87,142,143]

Research in hypertension suggests that everyday aspects of racism adversely affect health. Numerous studies indicate that people's exposure and concomitant response to distressing situations may be an important psychosocial risk factor for elevated blood pressure, through pathways ultimately mediated through physiologic changes.[38–43,148–151] These daily realities of racism could, for instance, act as chronic stressors and block aspirations and support at the individual, family, and community level; shape the content and frequency of the "life events" experienced by black men and women; and limit the range of feasible responses to problems (e.g., it may be dangerous to express anger against someone who can hurt you). Buffers for these negative effects might be the ways in which black men and women have nonetheless garnered relevant social support and developed and maintained affirming identities at the individual and group level.[38–43,82,148–150,152] To date, however, only a handful of studies have begun to develop instruments to assess these aspects of racism and resistance to racism; we will discuss them in the next section of the article (see pages 102–3).

Other adverse health effects may result from yet other aspects of racism. The greater concentration of poor blacks than poor whites in predominantly impoverished neighborhoods suggests that poor blacks may be more likely to reside in unsafe neighborhoods and consequently to suffer more from higher crime rates (including gang-related violence), inadequate housing, and greater exposure to both social distress and environmental contaminants (e.g., lead, toxic waste dumps).[82,120,124,125,145,153–156] Similarly, occupational segregation has often resulted in black workers facing different and typically more severe health and safety hazards than white workers employed in the same industries and being largely excluded from the least harmful and most powerful occupations.[107–110,112,113,145] The alarmingly high percentage of black men linked to the penal system may also be related; on any given day, 26% of black men (as compared with 6% of white men) are incarcerated or under the supervision of the corrections system.[141] A growing body of research also suggests that black Americans receive less aggressive health care than white Americans and are underrepresented in clinical trials.[34,104–106,144,157–161]

The health effects of these diverse aspects of racism, however, have been systematically studied by only a handful of epidemiologic researchers. In recognition of these and related problems, in 1987 the National Institutes of Health (NIH) developed new guidelines that require studies to include meaningful numbers of blacks and other minorities or else justify scientifically why they are not included;[162] the extent to which these guidelines have been implemented, however, remains largely undocumented.

Diversity among people of color. A third important limitation of traditional approaches to studying the effects of racism on health has been the lack of research on the diversity of conditions *among* different subgroups within each racial/ethnic group. As critics have noted,[38,42,78,82,128,163,164] much of the research on black Americans tends to treat them as a single undifferentiated group, rather than as a people internally divided by ethnicity, class, gender, and generation—because of both the great migration of the twentieth century and individuals' coming of age before, during, or after the civil rights movement. Research on the black poor is especially replete with stereotypes,[82,132] as if the "underclass" represented the majority of this group and as if all poor blacks were criminals rather than those often spearheading efforts to rid impoverished black neighborhoods of drug dealers and violence. We discuss examples of research exploring these topics in a subsequent section on emerging approaches to studying the effects of racism on health (see pages 103–4).

Gender-specificity of racism. Last, another important limitation of most current approaches to studying the effects of racism and health has been the lack of attention to the ways in which racism is often gender-specific. In other words, the experience of racism in the United States varies for men and women of color, not in terms of quantity, but rather in its different shaping of their lives, especially as related to gender-specific racist stereotypes.[64,65,84,165–174] To contextualize research about black women thus requires directly addressing their social realities, which not only differ from those of black men and white women but also hold across class lines. To date, however, U.S. epidemiologic research has ignored these differing realities when examining the effects of racism on health.

Gender, Sexism, and Health

Historical background. Investigating the health status of black women also requires considering the relationship between gender, sexism, and health. Studies on the contribution of sexism—not simply sex—to women's and men's patterns of health and disease, however, are a new phenomena.[175,176] Until fairly recently, the predominant assumption has been that women and men have different health profiles because they are distinct biological sexes who differ essentially in their basic natures.[60,177–182] According to this view, women and men have different disease risks not only because of differences in reproductive organs and physiology but also because of biologically determined differences in their social roles, which result in men's and women's exposures to different situations that can benefit or

harm their health.[60,177–182] Stated another way, not only do women get cervical cancer whereas men get prostate cancer, but women allegedly are less likely than men to be employed in hazardous occupations or to engage in risk-taking and health-endangering behaviors, chiefly because women bear and raise children and are inherently more "domestic" and less "aggressive" than men.

Attesting to the tenacious nature of these beliefs, comparable views have existed in virtually every system of ancient classical medicine, whether from Greece,[183–185] Rome,[186,187] China,[188,189] India,[185,190] Africa,[191,192] or the Americas.[193,194] All held that women and men have different constitutions, determined from the moment of conception. These male/female distinctions typically have been embodied in such dualisms as active/passive, intellectual/emotional, and civilized/natural. Projected back even onto human prehistory, in the form of "Man the Hunter" versus "Woman the Gatherer,"[195] these traditional beliefs have been used to explain and to justify persistent sex-based divisions in social tasks, property, power, and even health status.[84,175,180,181,196]

In part because of their longstanding legacy, these ideas have significantly influenced U.S. researchers' investigations of women's and men's health.[175,180] The two general approaches are (1) to study women and men separately or (2) to adjust for sex if both men and women are included in the study sample.[176–179,182,197] Studies focusing on women's health, moreover, typically have been concerned with conditions associated with reproduction (e.g., pregnancy, breast cancer) or diseases influenced by "female" sex hormones (which are biologically active in men, as well, albeit at different levels).[176,179,182,198] When researchers do compare the health experiences of women and men directly, they emphasize contrasting male/female patterns of illness, use of health care, and mortality.[176–179,182,197] One particular goal has been to explain why women live longer than men, despite their apparently greater morbidity;[177–179,197] only within the past few years have investigators recognized that women's extra years are not necessarily healthy.[199]

Outside the field of public health, however, a substantial body of research has examined the relationship between biological sex, as defined by biological characteristics pertaining to the ability to reproduce, and socially defined gender, that is, culture-bound conventions about appropriate roles and behaviors for, as well as relations between, women and men.[60,180,181,200–202] This work has demonstrated that differences between women's and men's social roles and behaviors are not inevitable expressions of biological density but are shaped rather by socially defined gender expectations that vary across both time and culture.[60,180,182,200,201] A common observation is that men's and women's roles differ within virtually every society studied to date[200,201] and that women are more likely than men to have primary responsibility for raising children and performing domestic labor.[200,201] However, roles and behaviors considered appropriate for women in one society may be appropriate only for men in another. For example, women's participation in civic life and shaping of social policy in the male-dominated and elite democracy of ancient Athens would have been unthinkable,[184] but women were highly esteemed leaders within the more inclusive and democratically constituted Iroquois nation.[203]

By separating the concepts of sex and gender and highlighting how knowledge about each has been influenced in part by investigators' often unconscious beliefs and expectations,[180,181,195,196] this new body of research has challenged many accepted axioms about women and men.[60,180,181,195,196,200,201] Far from dismissing the biological or social significance of reproductive differences, this work has suggested new avenues for research on women's and men's health while illuminating how biology alone does not account for the intermeshed but often unequal life circumstances of women and men within and across diverse societies.

Perhaps most importantly, this new research on gender has begun to examine how women's and men's lives, including gender roles and expectations, vary by race/ethnicity and social class within and across cultures.[142,143,181,195,196,204] Particularly, divisions *among* women and *among* men deserve attention. Dr. James McCune Smith would have appreciated this point. When he was debating mainstream medical views about racial differences in health, most physicians believed women were innately more delicate than men.[50,205,206] Such generalizations, however, were meant to apply only to relatively affluent white women and not to the enslaved black women that some of them owned nor to the white working-class immigrant women whose coarse nature they also derided.[50,205] Although these physicians may have agreed that anyone born with a womb belonged to the generic category of female, this type of biologic characteristic evidently was not the sole determinant either of women's nature or of their health.

Conceptual framework and definition of terms. Studies investigating the influence of sexism on health, like those regarding the health consequences of racism, reject the assumptions of biologic determinism. Distinguishing between gender and sex, they instead seek to understand how gender relations affect women's health, particularly diseases that afflict both women and men as well as conditions that occur uniquely among women on account of their link to reproductive organs and physiology (e.g., pregnancy and other reproductive processes, such as menopause, that are not themselves diseases). This orientation is different from the more usual approach of studying sex differences in health status, because the emphasis is on how the social relationships *between* women and men adversely affect women's health.

Reviewing the many facets of sexism, including its profound relationship to heterosexism,[196,207,208] is beyond the scope of this article. We define "sexism" as an oppressive system of gender relations, justified by ideology, premised on the subordination of women by men.[83,84,175,180,181,202,209–211] Like racism, sexism involves harmful and degrading beliefs and actions expressed and implemented in both overt and subtle forms by institutions and by individuals, as linked to their membership in gender-defined groups. Its underlying—and false—assumptions include: [60,83,84,180,181,195,196,202,206,209,211]

1. Men and women are innately different by virtue of their reproductive capacities and are biologically destined to assume different—and allegedly complementary—social and sexual roles; a corollary is that homosexuality is unnatural.
2. The genetically determined reproductive attributes conventionally used to define sex and to identify gender are inherently linked to other characteristics (e.g., mental abilities) that are also genetically determined.
3. Genetically based differences between men and women (in

terms of sexuality, culture, behavior, health, etc.) are inherent among all individual members of each gender group.

4. Men are innately superior to women, both mentally and physically, whereas women are innately more domestic and less aggressive than men.

As expressed in daily life, these beliefs and practices both justify and result in vast inequalities between men's and women's living conditions and opportunities.[83,84,202,209–212] Specifically, men benefit not only from their privileged position in the economy and other major societal institutions (government, religion, the arts and sciences) but also from women's domestic labor at home.[83,84,202,210–212] Another aspect of sexism has been the linkage of male sexuality with power and prerogatives, with women's sexual/social roles often reduced to the categories of "virgin," "mother," or "whore";[83,84,202,210–213] lesbians become an invisible "other."[207,208]

The everyday expression of sexism in women's lives consequently is at once pervasive and rife with contradiction.[214] The sexist dualism of women's inferiority/superiority runs deep: allegedly inferior to men in the public sphere of work, intellect, and civic life, women are simultaneously supposedly superior and cherished in the domestic sphere of care giving, emotional nurturance, and childraising.[83,84,181,196,202,209,211] Even so, women are routinely treated as sex objects and face the daily harassment of street remarks, the fear of rape, and, for some, the threat or memories of sexual abuse and domestic violence.[45,83,84,165,176,181,196,202,209–211,215–218] Moreover, for black women, these and other everyday expressions of sexism, both within and outside the black community, are filtered through presumptions about racial and gender characteristics.[165,171,219–221] For example, the allegedly positive and special characteristics of black women's "strength" has in fact had negative consequences for black women. Examples include the belief of the slave owning society of the early nineteenth century that black women did not require or need the same kinds of care and protection afforded middle-class white women and the contemporary reification of the "strong black mother of the race" of black nationalist philosophies.[221–224]

Additionally, although some women, especially white women, have developed professional careers (often fighting sex discrimination and sexual harassment en route)[210,225] many women—especially black women—have been forced by economic necessity to work outside the home. These women are often segregated in menial, low-paid, dead-end, insecure jobs and have few resources to help them juggle the demands of work and family life.[64,83,84,142,143,210,226,227] Moreover, black career women have testified since the early nineteenth century to the pressures of being "high-achieving" black women. In the nineteenth century, these women described the pressures on black women enrolled in Northern colleges, black women traveling on European antislavery tours, and black women who, with few resources, attempted to begin and maintain schools for black children in Northern states.[228] Black women in the last decade have also written about the difficulties of being the only black woman (often the only black and the only woman) in certain occupations.[222–224,226]

Finally, although many women have found respite in deep emotional connections with others, many also have been socialized to take care of others before they take care of themselves.[45,84,214] For black women, this socialization into woman's role as caretaker of others has often been framed in terms of ensuring the survival of entire groups of people.[223,228] In other words, the caretakers's role and the cultural image of the black woman as everyone's "mammy" have created the expectation that black women are the source of emotional and physical well-being for their own family members as well as for white women, men and children, *and* the black community.[173,229] Conspicuously absent is any caring source for black women themselves.

Predominant approaches to studying sexism and health. Despite considerable theorizing about the relationship between sexism and health (and particularly women's mental health),[142,143,175,206,211,216,230] relatively little empirical research has been conducted on this topic within the United States. Moreover, despite the valid distinction between sex and gender, the standard practice in epidemiologic studies is to ascribe gender on the basis of biological sex and to use the terminology of sex, rather than gender, differences.[176,179] Because our concern is with the effects of sexism—that is, the subordination of women—we will not review the even more limited literature on the effects of male gender roles upon men's health.[204] Instead, we will restrict our discussion to studies examining how traditional female roles and expectations about women may harm women's health, including, but not restricted to, their reproductive health.

Within this small field of work, women's patterns of cigarette smoking and eating habits have received particular attention.[216,231–238] This research has documented how cigarette manufacturers have marketed the concept that cigarette smoking is "sexy" and that women who smoke are glamorous;[231–233] notably, lung cancer recently has surpassed breast cancer as the leading cause of cancer mortality among women in the United States.[239] Investigators have also found that, in a society that continually promotes the idea that only thin women are beautiful,[206,216,236–238] many women smokers considering quitting have often expressed the concern that once they stop smoking, they will become fat.[233,240] Other studies have examined the effects of sexism on women's views of their bodies, particularly with regard to anorexia and bulimia, obesity, and both "crash" and "yo-yo" dieting.[216,234–238,241]

Additional research has begun to examine how traditional views about women's sexuality can pose a serious danger to women's health, at home, at work, and in society at large,[84,171–174,213,216,242–249] Recent research, for example, has examined the historical social construction of stereotypes of black women's sexuality and their effect on the contours of black women's lives, including their psychological and physical health. In the nineteenth century, allegations regarding black women's "hypersexuality" served as a rationale for their rape by white men, including their slavemasters.[248,249] Today, these stereotypes contribute to the types of racism and sexism black women often experience in their everyday work lives, such as racialized types of sexual harassment.[84,171–174] Other epidemiologic studies have documented that women refrain from using contraceptives or from requesting men to use condoms in part because to do so suggests they are "bad girls," simply because they are prepared for nonprocreative sex.[213,242,244,245] Taking this type of initiative may put women in danger of a punishing (even violent) response from men.[213,247] Further research has investigated how the conflation of male sexuality and power has damaged women's health through sexual abuse, rape, and domestic violence.[197,216–218,243,250–252]

Factors associated with "women's work" (both paid employment and unpaid domestic work) have provided yet another focus for studies about the effects of sexism on women's well-being.[145,216,253–255] Two aspects of gender segregation in the workforce have received particular attention: the specific occupational hazards women confront by concentration in a few typically low-paying jobs (e.g., clerical workers, nurses, technicians, teachers, and both domestic and other service workers),[114,145,253–255] and the health consequences of women's multiple roles—that is, among women who work in the paid labor force, the impact of working both in and outside their homes.[177,178,197,256] Very little research has investigated the occupational hazards of housewives, such as falls, injuries, and unregulated exposures to numerous potentially toxic household cleaning agents.[257]

Perhaps the bulk of the work on sexism and women's health, however, has focused on the medical profession's attitudes toward women and toward women's health and well-being.[176,206] Substantial research has documented that physicians are more likely to take men's symptoms more seriously and to discount women's symptoms as psychosomatic complaints.[35,176] Other investigations, often linked to concerns about controlling women's behavior, have examined the medical profession's complicity in "medicalizing" women's social problems.[180,205,206,216] Historical examples include castrating women who were "too aggressive" or performing clitoridectomies upon women who were "too sexual";[205,206] more contemporary illustrations include the high rate of tranquilizers prescribed to women to help them "cope" with oppressive life conditions.[176,197,216,256]

One specific research topic receiving considerable attention concerns how sexism has influenced physicians' treatment of pregnant women.[175,205,206,209,258–263] Most critiques have emphasized the negative consequences of physicians' exclusion of midwives and their disregard for the significance of nonmedical social support (family, friends, social workers) during pregnancy.[205,206,258,262–264] Several also have taken issue with the medical profession's restricted view of reproductive health care as solely the provision of prenatal care and with its equally limited depiction of women's behaviors as the primary determinant of birth outcomes.[205,206,258,262–265] Examples cited include health care providers' tendency to blame pregnant women for not complying with prenatal care or for "recklessly" endangering the well-being of their fetuses (through use of drugs, alcohol, or possible exposure to sexually transmitted diseases, including acquired immunodeficiency syndrome), without first considering the social and economic circumstances of the pregnant women's lives.[247,252,259,260,266] Other criticisms have highlighted how the construct of maternal and child health not only obscures the role of men during pregnancy but also facilitates ignoring the ways in which pregnancy can exacerbate household tensions, even to the point of contributing to domestic violence.[247,252,259,260] As several commentators note, these and related forms of sexism often are compounded for poor women by their treatment as second-class citizens when they attempt to obtain health care from typically underfunded and overburdened public clinics and hospitals.[247,260,265–268] This small but growing body of work suggests that sexism within the medical profession can itself be a risk factor for women's health.

Limitations. One of the notable limitations of public health research, particularly epidemiologic research, on sexism and women's health is its scarcity. Consequently, most of what has been written about the effects of sexism on women's health has relied upon thoughtful interpretation of inadequate and often indirect data concerning women's health.[47,142,143,175,176,182,197,205,206,210,216,269,270] In addition to this glaring deficiency, three other limitations of the predominant approaches to studying sexism and women's health are (1) an emphasis on conditions that affect only women (especially reproductive health), (2) limited attention to the effects of nonviolent forms of everyday sexism, and (3) an underemphasis on diversity of conditions among women, especially those related to race/ethnicity and class.

"Women's health" versus the health of women. Much of the research on women's health, and even on sexism and health, has focused precisely on conditions that affect chiefly or only women, especially reproductive health.[47,142,143,175,176,182,197,205,206,210,216,269,270] Although reproductive studies obviously are important, research regarding aspects of women's health involving diseases that also affect men has been especially underemphasized, if not neglected outright.[35,176,182,197,199,271] Possibly serious consequences include (1) inappropriately assuming that risk factors detected in studies based on men necessarily affect women the same way (e.g., Type A behavior is a stronger predictor of cardiovascular mortality among men than among women),[256] (2) insufficiently studying risk factors more relevant to women than men (e.g., the effect of multiple roles and women's risk of coronary heart disease),[177,178,256] (3) inadequately addressing the effects of comorbidity upon women's overall health and also reproductive health, including the impact of black women's poorer health status upon their adverse birth outcomes,[3,142,143] (4) failing to consider that clinical trials of drugs tested in men may yield different results than those conducted with women (e.g., antidepressants),[176,182,272] and (5) insufficiently examining how physicians' attitudes towards women's health complaints may affect the type of health care women receive.[269]

In recognition of the gaps in knowledge resulting from the limited research on the health of women (including "women's disorders"), in 1986 the NIH promulgated new guidelines regarding the inclusion of women as research subjects in clinical trials and other studies.[199,273] These guidelines, like those for minority health, require investigators either to include meaningful numbers of women as well as men in studies on health conditions that affect both men and women or to justify scientifically why their proposal does not include women.[271,273] For these same reasons, the AMA's Council on Ethical and Judicial Affairs recently has concluded that it may be dangerous, as well as insufficient, not to do research on all aspects of women's health.[35]

Diverse forms of sexism. Another limitation of the current (albeit few) studies that explicitly address the topic of sexism and women's health is that most understandably focus on the health consequences of the most overt forms of harmful and dangerous sexist actions: sexual abuse, rape, and domestic violence, as well as other forms of violence against women.[176,217,218,243,250,251] In contrast, hardly any address the health consequences of routine, everyday sexism.[38,176,274] As in racial discrimination and other forms of racism, however, the psychosocial effects of gender discrimination and oppression are likely to affect health adversely.[38,45,47,83,84,142,143,175,216,236,238]

These include the previously noted problems regarding physicians' treatment of women,[35,175,176,206,209,216,258–263] the physical consequences of women's occupational segregation,[145,216,253] and the ways in which women's gender-based role of taking care of others before they take care of themselves may in itself constitute a risk factor for women's health.[45,83,143,173,216,223,228,229] Some research has observed, for example, that if money for food is limited, women usually feed their children and husbands before they set aside food for themselves.[45] Other studies have suggested that the higher rates of depression among women (especially employed young working-class women with small children) may be linked to conflicts resulting from their particular roles in keeping their families together.[45,275] And, as noted previously, black women may be particularly likely to put the welfare of their families and communities before their own well-being.[173,223,228,229]

Differences among women. A third problem often apparent in research about the effects of sexism on women's health is that women's health and minority health are perceived as two distinct areas—even though a substantial number of women in the United States are clearly women of color.[142,143,214,267,268] Another neglected difference among women is social class, both within and across racial/ethnic groups.[142,143,214,267,268,276,277] Recent sociological research, however, has indicated that the types of sexism women experience may vary with their social class and, as noted before, with their race/ethnicity as well.[64,65,84,142,143,166–171,226,278] Moreover, women's responses to experiencing sexism may also vary by their historical generation (e.g., coming of age before, during, or after the recent second wave of the twentieth-century women's movement).[38,276,277]

Only a small amount of the research to date on the health effects of women's multiple roles has focused on the life conditions of black or white women in either working-class or impoverished single-female-headed households.[142,143,256,267,268] The challenge of working outside the home and managing a household and family, however, may be very different for a white professional woman who is assisted by employed help at home than for a working-class or poor woman; that both should somehow be able to meet all these demands is expressed through different versions of the "superwoman" or "strong black woman" myths.[142,143,173,223,227–229,256,267,268] Failure to consider diversity among women may thus also compromise research on sexism and women's health.

Social Class and Health

Historical background. To understand the health of black women, one must consider the relationship between social class and health. The recognition that poor people have poorer health than affluent people and that people's occupations and social position influence their health is hardly new.[279] It was well documented even in the earliest days of the Western medical tradition.[14,183,185–187] Hippocrates, for example, noted that the laboring poor (both peasants and slaves) could hardly hope to lead healthy lives because they lacked the resources to "live right."[183] Subsequently, Galen, whose works influenced Western medicine for almost two thousand years, explicitly directed his classic work, *Hygiene,* to the rulers and members of the court of imperial Rome:

> As it has been shown that there is a numerous diversity of bodies, so also are there numerous forms of the lives which we lead. It is not therefore possible to administer perfect care of the body in every form of life, but the best that is possible for each, for absolute perfection is not possible in all lives. For the life of many men is involved in the business of their occupation, and it is inevitable that they should be harmed by what they do and that it should be impossible to change it. Some incur such lives from poverty, some from slavery, either descending to them from their parents, or having been taken captive and carried away, which most people consider the only real slavery. But to me it seems that those who through ambition or zeal have chosen some form of life so involved in affairs of business that they can have little leisure for the care of their bodies are also willing slaves to hard masters. So that for these it is impossible to prescribe absolutely perfect care of the body. But whoever is completely free, both by fortune and by choice, for him it is possible to suggest how he may enjoy the most health, suffer the least sickness, and grow old most comfortably.[187,p13]

To Galen, it was clear that only the aristocratic minority could expect to lead healthy lives, while the vast majority lacked the resources to live well.[14,186,187]

Nearly two millennia later, Johann Peter Frank, one of the architects of the early public health movement in Europe, reiterated these points in a powerful and widely circulated speech entitled "The People's Misery: Mother of Diseases," which he delivered in 1790.[11] Urging a new focus on the social causes of disease, Frank declared:

> Every social group has its own types of health and diseases, determined by the mode of living. They are different for the courtiers and noblemen, for the soldiers and scholars. The artisans have various diseases peculiar to them, some of which have been specifically investigated by physicians. The diseases caused by the poverty of the people and by the lack of all goods of life, however, are so exceedingly numerous. . . .[11,p93]

After enumerating the miseries and consequent poor health that afflicted the poor from the moment of birth until their death (including their greater susceptibility to epidemic disease), Frank concluded by urging the rulers of his time to "expel from our provinces the people's misery, most powerful mother of diseases!" He promised that, if they did so, "joy, virtue, patriotism and the former health of the citizens, secured by labor, will be restored."[11,p100]

Within the United States, similar concerns about the links between poverty, poor health, and social disorder have been expressed since the founding of the first colonies.[13,14,280–283] They also played a central role in shaping the growth of the sanitary reform movement during the 1840s[14,280,281,283–285] and the subsequent development of the modern public health movement in the 1880s.[13,14,280,281,283,284] Throughout, a tension has existed between two groups: those who view the poor as the chief cause of their own poverty and poor health (because of supposedly acquired or inherited tendencies to be lazy, depraved, or unintelligent) and those who place the responsibility for poverty and contingent poor health upon the political and economic decisions of government and business (e.g., sanction of low wages, harsh working conditions, and inadequate lodging).[14,280,283–286] From the 1880s to the early 1930s, eugenic explanations of social class differences in health received sub-

stantial support within academia and also overlapped considerably with much of the theorizing about racial/ethnic differences in disease.[53,62,282,283,287–291] Only in the 1930s and 1940s, in reaction to the full-scale application of eugenics by the Nazis, did overtly genetic explanations of social class gradients in health begin to recede into the background.[12,62,288–293]

Despite the overall recognition of the link between social class and health, and in contrast to what occurred in England and other European countries whose public-health movements blossomed at approximately the same time,[14,18,19,293] population-based data on morbidity and mortality in the United States have rarely, if ever, included information on social class.[7,8,126] This omission is not unique to the realm of health. The first U.S. census in 1790, for example, recorded only people's age, sex, and race (and, for blacks, whether free or enslaved).[294,295] Occupation was deliberately omitted, despite James Madison's considerable efforts to include this information, because the U.S. Senate thought data on class-based distinctions would detract from the "common good."[294,p163;295] Not until 1820, when the notion of competing economic interests grew more acceptable, did occupation become a standard item in the U.S. census.[294,295]

The United States remains one of the few industrialized countries that does not include social class data in its national vital statistics.[126,296–300] Although federal agencies occasionally prepare special reports on health characteristics by occupation and industry,[169] national morbidity and mortality data continue to be stratified only by age, race, and sex.[296–299] The question remains: why do U.S. health data, unlike European data, lack information on social class?

Although it is beyond the scope of this article to address the complex reasons underlying these divergent approaches to gathering health data, most explanations focus on differences in demography and ideology.[79,83,84,87,90,130,294,295,300,301] For example, conflicts construed as racial have occurred on U.S. soil since this country's origins as a colony, when white European settlers and their descendants fought against Native Americans and Mexican Americans and also imported Africans as slaves. Reflecting this state of affairs, early U.S. vital statistics documented the health status of the colonial, enslaved, and indigenous populations, thus establishing a framework for collecting health data by race/ethnicity, but not by social class per se.[294,295] By contrast, the population of European countries—until fairly recently—has been chiefly white, albeit divided by national and ethnic differences often alleged to have a biologic basis.[53,56,57,60–63,288,291,292] During the mid-1800s, when Europeans were developing their public health data systems, racial conflicts along "color" lines chiefly involved subjugation of people of color who lived in colonies abroad, whereas domestic divisions often were framed in terms of social class.[14,15,18,19] European public health data thus typically included information on socioeconomic position, ethnicity, and national origin, but rarely on race.[14,15,18,19]

In part because of these reasons, many in the United States, including public health professionals, are accustomed to thinking of the United States as a "classless" society.[80,84,87,90,130,300] Bolstering this view is the belief that social position and mobility in the United States are determined principally by merit and income, in contrast to family origins in the more castelike class structure of many European nations.[87–90] Discomfort in addressing social class in public health research is further compounded by the twin beliefs that science and politics should be kept distinct and that matters of social class and health are political, not scientific.[12,18,50,60,61,181] Even so, many, but certainly not all, epidemiologic studies include at least some partial measures of social class, in recognition of its profound influence on health.[302,303] The predominant approach, however, is to adjust or control for social class, rather than directly study its effects upon health status and disease outcomes.[304,305]

Conceptual framework and definition of terms. Within the United States, the conceptual framework underlying most epidemiologic and social science research regarding social class derives from the Weberian concept of socioeconomic status.[129,130,302,303,306,307] Put simply, Weber's theory holds that individuals' social position, as well as access to and control over societal resources, reflects the interplay of three aspects of social life: occupational class, social status or prestige, and power.[129,130,306–309] Within each of these three arenas, persons can be stratified according to whether they possess more or less of the specified attribute.[129,130,306–309] Although alternative approaches to conceptualizing social class have received considerably more attention in European sociology (e.g., a relational approach to class in which categories are defined by people's relations to each other through the workplace, such as employer versus employee),[130,131,303,307,310,311] they rarely have been used in epidemiologic research in either Europe or the United States.[302,303,312,313] We discuss these approaches in a later section (see pages 106–7).

With the repudiation of eugenic explanations for both the existence of different classes and their diverse health profiles, the two assumptions now guiding most epidemiologic research on class-based inequalities in health (at least within, if not necessarily across, gender and racial/ethnic groups) may be characterized as follows:

1. Membership in the different social classes, as defined by the different factors grouped under the Weberian rubric of socioeconomic status, is not biologically determined, and individuals within the different social classes cannot be distinguished from each other on the basis of genetic factors alone.

2. Differences in health status between social classes consequently result from differences in exposure or resistance to health-damaging conditions, as well as exposure to health-promoting conditions, mediated by people's access to and control over the resources required to live healthy lives. Hypotheses of how social class can affect health include the following ways: shaping who has and who lacks the basic material necessities of life (adequate food, clothing, shelter, sanitation, and health care), who is exposed to—and spared from—a variety of occupational and environmental hazards, and who has and who lacks control over the essential content of daily life (at work, at home, and in the neighborhood).[12,16,44–46,301,304,305,314–319] Also pertinent is the relationship between social class and the different types of behaviors and ways of living that are promoted or denigrated by one's peers and the society at large.[12,16,45,46,301,304,305,314–319]

Although many researchers agree about some of the possible proximate links between social class position and health status, considerable controversy still surrounds explanations of why these class-based differences in health exist.[12,16,45,46,301,304,305,314–319] Echoing earlier arguments, some assert that poor health precipitates poverty;[320,321] others emphasize that the individual habits and "lifestyles" of the poor are the chief determinants of their poor health.[321–324] Still others contend that the same social and economic factors that

produce poverty also create poor health, by outright deprivation and by shaping and constraining the living conditions and health habits of the poor.[16,44–46,300,304,317,321,325] Although much has been written on this topic in the United States,[300,304,321,325] perhaps the clearest and most concise articulation of the differing viewpoints and the evidence supporting or refuting them can be found in the 1980 British study, *Inequalities in Health: The Black Report*[16] and its recent successor volume, *The Health Divide*.[46] Each considers the following four explanations of social inequalities in health: (1) artifact of measurement, (2) natural or social selection, (3) materialist/structural explanations, and (4) cultural/behavioral explanations. Both reports conclude that social inequalities in health are real, that only a small amount can be attributed to social or natural selection, and that material/structural conditions are the fundamental determinant of these inequalities in health, through their direct effects upon health and through their shaping of cultural/behavioral practices that further influence health.[16,46]

One additional question in the debate about social gradients in disease is whether these patterns result primarily from the accumulation of specific exposures to discrete disease-causing agents or from generalized susceptibility to, or compromised defenses against, a wide-ranging set of relatively ubiquitous noxious agents or risk factors,[314,326–329] One corollary is whether research should focus on explaining differences in incidence for specific diseases or on differences in the overall disease distribution or specific clusters of organically unrelated diseases or causes of mortality[304,314,327] (e.g., Cassel's constellation of tuberculosis, alcoholism, schizophrenia, and suicide).[330] Equally questionable is whether the research agenda should focus chiefly on deprivation (i.e., the poorer health status of the poor) or on the full range of inequalities in health (i.e., not only why affluent people have the best health and poor people have the worst health, but also why gradients exist even when material deprivation is not an issue, for example, why junior managers or administrators have worse health than their senior colleagues).[16,46,304,314,327,331,332]

Testing any of these contending hypotheses, however, requires adequate empirical measures of social class, an equally controversial point.[47,129–131,302,303,306,307,310–313] Currently, the three socioeconomic indicators most commonly employed in U.S. epidemiologic research are occupation, education, and family income.[302,303,316] Reflecting different aspects of Weber's schema, they typically are measured in a variety of ways, singly and as combined single indices.[302,303,316] Occupation, for example, generally is grouped by skill, with a fundamental division between manual and nonmanual labor (e.g., "blue-collar" versus "white-collar" workers).[302,303,316] Some epidemiologic studies, however, use "occupational prestige" scales, with occupations ranked in accord with societal perceptions of their status.[302,303,316] Moreover, although most studies record the subjects' current occupation (or last occupation, if retired or unemployed), several have suggested that "usual" occupation may be more appropriate for epidemiologic investigations.[302,303,316,333]

In lieu of occupational class, education has been viewed by some epidemiologists as a more stable indicator of socioeconomic status and thus is more widely used.[302,303,316,334,335] Educational level is also popular because it is easily measured and can be treated as either a continuous variable or as a categorical variable, with cut-points based on credentials (less than a high school education, high school graduate, one to three years of college or vocational school, four or more years of college).[302,303,316,334,335]

In contrast, family income is the least used of the three standard socioeconomic measures, perhaps because many researchers consider income a sensitive topic and may be reluctant to gather the data or may question their accuracy.[302,303,316,334,335] When collected, however, data on family income have been modeled as both continuous and categorical variables (with strata often defined by grouping the data by quartiles or quintiles).[302,303,316,335] Occasionally, researchers have also gathered additional information on family size and age structure to determine the relationship of family income to the federally defined poverty line.[302,313] Currently, a growing body of research suggests that this approach is more meaningful than simply reporting family income, because an income of $10,000 has very different implications for a family of one adult than for a family of one adult and three young children.[336,337]

The composite forms of the three standard socioeconomic indicators also differ considerably.[302,303,306,316] The Hollingshead index, for example, stratifies subjects on the basis of their joint occupational and educational level.[302,303,306,316] Another, the Duncan socioeconomic index, ranks individuals' occupations by virtue of their perceived social status and assesses the average income and education level of men employed in these occupations.[302,303,306,316,338,339] Some epidemiologic studies use factor analysis to collapse a variety of socioeconomic indicators into discrete clusters that can be treated as categorical variables.[302,303,340] Use of composite measures of social class, however, has been discouraged in recent years, because indices often must be disaggregated to determine which aspects of socioeconomic position are most relevant to the disease process under study and also because clusters defined by factor analysis cannot be replicated across studies, limiting comparison of results.[302]

Debate regarding appropriate measures of individuals' social class reaches another dimension in the topic of women and social class and the little-discussed distinction between individual and household class.[47,313,341–352] Most epidemiologic research has employed two entirely different approaches to measuring women's social class.[302,303,313] One is a male-centered strategy in which married women (in the paid labor force or not) are categorized according to their husband's occupational class, whereas unmarried women are classified according to their own class.[302,303,313] The other tactic is individualistic and resembles that used for both single and married men: all women, regardless of their household or marital status, are classified according to their own socioeconomic position, typically as measured by educational level.[302,303,313] If, however, occupational class is used, housewives typically are grouped into one category, regardless of the social class of their partners.[302,303,313]

A final technique used to measure social class involves area-based indicators derived from census data, typically from the tract level.[37,49,98,302,303,313,353–360] Also concerned with occupation, education, and income, these social-area indicators usually characterize census-defined neighborhoods by the percentage of persons employed in blue-collar versus white-collar jobs, the percentage of persons with less than a high school education versus those having four or more years of college, or the percentage of persons at different levels below and

above the poverty line.[37,49,98,302,303,313,353–360] Like their individual-level counterparts, these indicators are treated as single variables or combined via the algorithms of established indices or by factor analysis. In either case, these area-based measures generally are used in three different ways: as proxies for individual-level social class data (usually because the individual data are not included in medical charts, disease registries, or other typical sources of health records), as markers to delineate different socioeconomic regions whose morbidity and mortality rates can then be compared, or as contextual variables.

Predominant approaches to studying social class and health. Much of the ongoing English-language epidemiologic research directly testing hypotheses about the relationship between social class and health has been conducted in Britain, not the United States.[16,46,47,327,361,362] Although exceptions clearly exist,[37, 334,353–357,363,364] the major trend in U.S. research is to control or adjust for social class, using one or more of the methods described previously.

When U.S. researchers have investigated class-based inequalities in health, their most typical approach has been to stratify study subjects' health outcomes by individual-level socioeconomic indicators to determine if any consistent social gradients are apparent (i.e., whether the incidence is inversely related to education).[302,303] A related approach is to compare disease rates among more impoverished versus more affluent neighborhoods.[302,303,353–357] In either case, if gradients are detected, the question then becomes whether known individual-level risk factors—smoking, other health habits, environmental or occupational exposures—could explain the observed differences because of their differential association with social class. Usually this type of question is answered by the researchers' consulting the literature rather than conducting a study to test the hypothesis directly.

Two notable exceptions, however, include the 1987 prospective study of poverty and health in Alameda County, California, by Haan et al.[363] and the 1989 prospective study of educational differentials in U.S. mortality rates by Feldman et al.[364] Both studies documented strong social gradients in overall mortality and disease-specific mortality rates, and both demonstrated that major known individual-level risk factors did not account for these trends, either for overall mortality[363] or for heart disease mortality.[364] The study by Haan et al. compared all-cause mortality rates among adult residents, 35 years and older, in poverty and nonpoverty areas, with follow-up conducted over a nine-year period.[363] They found that the 1.7-fold excess risk of death (adjusted for age, race, and sex) among persons living in poverty areas persisted and remained statistically significant even after the analysis adjusted for the subjects' individual-level socioeconomic position, health practices, social networks, and psychological factors.[363]

Similarly, using data from the 1960 national Matched Records Study and the first National Health and Nutrition Examination Survey Epidemiologic Follow-up Study (conducted between 1971 and 1984), Feldman et al. found that, among white men and women 45 to 64 years of age at baseline, the least educated were approximately twice as likely as the most educated to die from heart disease (a statistically significant difference), even after the analysis adjusted for baseline smoking status, body mass index (BMI), systolic blood pressure, and serum cholesterol level.[364] The excess risk among less educated white men and women 65 to 74 years of age at baseline was 1.4 and 1.5, respectively, and bordered on statistical significance. The authors noted that their results closely paralleled those reported in Ross and Marmot's prospective study, which found that known coronary heart disease risk factors explained only a small portion of the substantially higher rates of heart disease deaths among lower-grade versus higher-grade British male civil servants.[365] Other research conducted in England has likewise found that the usual behavioral risk factors (smoking, diet, exercise) explain relatively little of the observed class-based differences in health.[16,46,361]

An alternative approach to studying class-based differentials in health is implicitly, and, at times explicitly, expressed through research on occupational and environmental health.[107,108,112,145,253–255,366–369] One method is to determine whether persons employed in specific occupations or industries are at excess risk of developing or dying from a variety of health problems.[107,108,112,145,253–255,366–369] The types of exposures studied include physical and chemical agents, ergonomic design, injuries, and psychosocial stressors (Karasek's job stress scale, for example, is based on dimensions of job demands and the latitude to meet them).[370,371] Another method is to investigate whether environmental pollutants can be linked to either individual cases or to population-based incidence rates of particular disease outcomes and also whether these pollutants disproportionately affect certain segments of society—people of color or low-income people who live near a particular worksite or toxic chemical dump.[154,372] However, little research has attempted to determine what portion of ill health among men and women in different social classes—across and within racial/ethnic groups—might be attributable to occupational hazards and environmental exposures.[373]

At a more general level, another strategy to study the links between social class and patterns of health and disease involves assessing correlations between secular trends in the economy (recessions, unemployment) and incidence or mortality rates for numerous health conditions.[367,374,375] Because this research typically is based on aggregate rather than individual-level data, serious questions have been raised about the degree to which its results might be biased by ecologic fallacy.[303,367] Additional objections concern assumptions that underlie the mathematical modeling of the data, including those pertaining to the presumed lag-time between the observed economic events and the subsequent health outcomes.[303,367,375] Recognizing these limitations, other research has begun to explore valid ways of studying the effects of business cycles on health[367] and ways of evaluating the contextual effects of social class and other socioeconomic factors.[49,313,376–378]

However, some of the most thorough and thoughtful U.S. research on the relationship between social class and ill health, at both the micro and macro levels, is not contemporary but instead dates back to investigations of the etiology of pellagra during the 1910s and 1920s by Joseph Goldberger (an epidemiologist), Edgar Sydenstricker (an economist), and others.[12,379–381] Because of the significance of their work, we briefly discuss their approaches in one classic study, "A Study of the Relation of Family Income and Other Economic Factors to Pellagra Incidence in Seven Cotton-Mill Villages of South Carolina in 1916."[379] Their goal was to analyze the complex relationship between the incidence of pellagra, family income, availability of food supply, and diet.

First, to classify the study subjects' economic status, Goldberger et al. obtained data not only on family income but also on the age and number of persons supported by this income, arguing that "it was improper to classify, for example, a family whose half-month income was $40, and was composed of only a man and his wife, with one whose half-month income was also $40, but was composed of a man, his wife, and several dependent children."[379] To address this problem, they employed a scale based on adult male food requirements (adult men and women 16 years of age and older were respectively set as "1" and "0.8," children under 2 at "0.2," those 2 to 5 years at "0.4," etc.). Their economic indicator was then calculated by dividing the total family income by the number of adult male units supported by this income.

The next step was to evaluate the incidence of pellagra among households categorized by this refined measure of economic level. After documenting that the poorest households were at highest risk, the investigators considered three hypotheses to explain this pattern: poor hygiene, different age and sex composition within each economic level, and differences in diet.[379] Goldberger et al. ruled out the first two hypotheses because extensive prior research had failed to link pellagra to any infectious agents, and the relationship between pellagra incidence and economic level persisted even after adjusting for age and sex. Diet was then studied to determine (1) the relationship between household income and food purchases (the poorest households bought relatively more cornmeal and salt pork than the less impoverished households and purchased relatively fewer meats, green vegetables, fresh fruits, dairy products, and canned foods); (2) differences in incidence among households, because of variations in food consumption among family members; and (3) differences in incidence among villages, because some villages with equally low incomes had markedly different rates of pellagra.

Put another way, for Goldberger et al., determining that the risk of pellagra varied by income was not enough. Explaining the actual distribution of the disease that could not be accounted for by income alone was also necessary. In the process of solving this puzzle, they were able to rule out such factors as differences in the villages' sanitary conditions, food prices, and the age and sex distribution of their inhabitants. Instead, they found that the critical issue was the availability of food supplies from both the local market and home production. In addition to villagers' differing in the extent to which they could own gardens and livestock, local company stores (the main source of food) varied in the extent to which they sold fresh vegetables and fruit, and only a few villages had regular meat markets. These differences in turn depended upon (1) whether surrounding agricultural land was devoted chiefly to food or nonfood cash crop production and (2) whether these farmers sold their produce primarily to the villages or to larger towns, a decision affected by such factors as road conditions and the location of railroad stations. Thus, the investigators were able to trace the actual pathways—at the community, household, and individual level—by which socioeconomic factors shaped people's risk of developing pellagra.[379]

Further, Goldberger et al. also established how the endemic, epidemic, and seasonal incidence of pellagra in the Southern United States was inherently linked to the region's dependence on a cash crop economy: when the cotton crop failed or its price decreased on the market, income dropped, diet changed, and pellagra soon was on the rise.[379,380] To both Goldberger and Sydenstricker, population patterns of health and disease would remain incomprehensible if researchers focused only on individual-level causes of disease. They argued that only by considering the social determinants of health could researchers understand the full etiology of disease and thereby set the basis for planning appropriate public health interventions.[12,380,381]

In his subsequent work, Goldberger not only discovered the dietary deficiency responsible for pellagra (an absence of the vitamin niacin), he also demonstrated the efficacy of using niacin-rich dietary supplements (e.g., yeast) to prevent and cure pellagra.[380] Even so, he consistently maintained that the only way pellagra could ever be prevented was by diversification of the Southern economy and amelioration of the plight of its cotton-bound sharecroppers and millworkers.[380] For taking this stance, Goldberger was publicly excoriated by Southern politicians and their appointed public health officials; history, however, proved him right.[31,380]

Limitations. Few of the U.S. studies focusing on (as opposed to merely controlling for) the relationship between social class and health have been as exhaustive or as multifaceted as the pioneering research conducted by Goldberger and Sydenstricker.[12,379,380] Some of the significant limitations of U.S. research are (1) inadequate measures of social class, including inadequate recognition of the different levels of social class (individual, household, and community); (2) an emphasis on objective components of social class (income, type of occupation), with minimal concern for the health consequences of its subjective aspects (e.g., the ability to have a sense of control over one's life); (3) limited attention to the health implications of class origins and mobility, especially as linked to issues of race and gender; and (4) a conspicuous absence of attempts to assess how much known risk factors contribute to observed social inequalities in health. We have already discussed other factors, such as U.S. researchers' reluctance to address issues of social class and health (see page 93).

Measures of social class. The remarkable degree of eclecticism characterizing the choice of socioeconomic measures in U.S. epidemiologic research would most likely never be tolerated for other important risk or preventive factors.[382,p236] Researchers painstakingly ensure that diseases are properly coded in accord with the International Classification of Disease–9 conventions[383] and that biological parameters (e.g., blood pressure, skinfold thickness, assays to determine levels of serum cholesterol or unbound fractions of circulating hormones) are accurately measured. However, most U.S. epidemiologists apparently use whatever measures of social class they consider convenient. They only occasionally consider whether these indicators are comparable across studies or whether they are valid or sufficient for the particular populations being studied.

Yet, as we noted in the discussion of racism and social class, people at the same socioeconomic level but in different racial/ethnic groups do not necessarily have comparable living conditions.[48,82,114,116–119,125] Moreover, most of the conventional measures of occupational class, including occupational rankings and prestige scores, have been defined on the basis of the job content and status of employed white men and thus may not be appropriate for employed white women or employed people of color.[302,306,338,339] Research in the United States, for example, has demonstrated that the Duncan Socio-

economic Index predicts women and men to have the same average occupational prestige, despite women's concentration in lower-paying jobs,[338,339] simply because the index rates white-collar work higher than blue-collar work (even the most poorly paid secretary ranks higher than a well-paid construction worker). These scales also fail to discriminate adequately between women's white-collar and pink-collar, or clerical versus service sector, jobs.[338,339] Similarly, certain occupations with high prestige in the black community—such as postal work—are rated lower on white-defined scales,[82,302,306] thus casting doubts on the utility of these scales for research on health and prestige in black communities.

Also problematic are the diverse ways in which researchers use occupational class measures to assess women's socioeconomic position. Moreover, researchers tend not to consider the effects of these measures on the relationship between health and social class among men. Most research assigns single and married men and single women their own occupational class, whereas married women are sometimes ranked according to their husband's class and at other times according to their own occupational class (including housewife, for women not in the paid labor force).[47,302,303,306] When nearly 60% of married women in the United States work in the paid labor force[384,385] and when at least a third of the women in dual-wage families have jobs at a "higher" occupational ranking than their husbands' or partners' jobs,[306,386] the assumption that married women should automatically be classified according to their husbands' occupational class is increasingly dubious.

These different approaches confuse the distinction between individual occupational class and household social class.[313,341–352] Although an individual's occupational class contributes to his or her household's standard of living, it does not provide the full picture for the growing number of households with more than one adult wage earner.[313,341–352] As we will discuss later (see pages 105–7), research in both the United States and Britain has shown that, at least for women, individual and household class perform differently as predictors or correlates of health outcomes. Individual class is apparently more relevant for outcomes directly linked to working conditions, whereas household class is more appropriate for outcomes associated with overall standard of living.[47,313,345,361,387,388] This distinction further suggests that information on both levels of social class—individual and household—is necessary if social gradients in women's health are to be delineated correctly, and the same may be true for men. Although controversy exists over how best to measure household class,[313,341–352,389] the growing consensus is that it should be set at the highest individual class within the household,[313,348,352] or else should reflect the household's actual, and at times discordant, class structure.[341,344,346,350,389]

Stating that household class must be considered, however, only highlights additional limitations of the predominant approach to measuring social class. The norm for most models of household composition is the nuclear family headed by a married heterosexual couple.[47] This family type, however, excludes nearly one third of households in the United States today.[20,p48] Not only does this restricted notion of "family" omit lesbian and gay households,[207,208] it also fails to comprehend the existence of extended families (e.g., multigenerational families), which constitute an important household type within the black community.[82,116,226,390] Consequently, if household class is to be assessed adequately, the definition of family must include these and other types of families.

Studies that use educational level as the principal measure of socioeconomic position are also affected by concerns about individual versus household class standing. Although some investigators have argued that education provides a more stable and widely applicable measure than occupational class,[302,334,335] an individual's educational level is not necessarily the sole or major determinant of his or her living conditions or way of living.[301–303,311,316] Few epidemiologic studies, however, have assessed the educational levels of both the individual study subjects and those of the other adults who form their households.[302] If the research on women and occupational class is any guide, different relationships between health outcomes and educational level would probably result if both household and individual educational attainment were considered. The relatively common practice of modeling education as a continuous variable is also problematic because it disregards the significance of obtaining credentials.[302,313] Specifically, the difference between 10 and 11 years of education is *not* the same as between 11 and 12 years, since 12 years marks the completion of a high school education.

As one step toward considering household, as opposed to purely individual, measures of social class, some research has used data on family income.[33,302,303,316] Even so, much of this research fails to specify the total number and ages of all persons dependent upon this income, thereby leading to inaccurate assessments of household living conditions.[313,336,337,379] One possible solution to this problem is to use economic gradations based upon family relationship to the poverty line.[302,313] Studies adopting this strategy, however, have often divided persons into broad strata (above poverty line versus below poverty line), thereby ignoring how economic resources differ drastically for persons at less than 50% versus 100% or 101% versus 500% of the poverty line.[336] Moreover, few studies explicitly account for how the current poverty line underestimates the actual level of impoverishment because it inaccurately estimates what portion of a poor family's budget is spent on food.[20,336,391] Although classifying people in accord with the poverty line is a promising strategy, investigators must recognize this measure's flaws and use appropriately defined strata.[302]

Research using economic data to analyze the relationship between social class and health often is beset by another limitation as well: few if any studies gather data on assets other than income.[47,313] Studies in England have shown that owning versus renting a house and owning versus not owning a car are important independent predictors of health status, above and beyond social class position.[47,361] Considering disparities in wealth may also explain black/white differences in living conditions: although the average income of white families is two to three times that of black families, their average net worth is 10 times higher.[118] Similarly, few studies acknowledge that the relative cost of living is higher for poor than affluent households, in part because the poor households must devote a greater portion of their income to obtain necessities (food, rent, fuel, transportation, etc.), and in part because they often must buy on credit, pay inflated prices for staples in neighborhood stores, and purchase money orders because they cannot afford checking accounts.[45,82,116,129]

Consideration of different approaches to measuring individual and household levels of occupational, educational, and economic measures of socioeconomic position highlights an addi-

tional limitation of current approaches to including these types of data in epidemiologic research. It is increasingly well accepted that these types of measures should be used as single variables, not amalgamated into indices or included in factor analysis.[302] Many studies also use only one or another of these measures rather than several, even though each captures only part of people's actual socioeconomic status. The few studies that simultaneously employ data on occupation, education, and economic level[313,363] are the exception, not the rule. Moreover, despite the importance that Weber accorded to power as the third component of his concept of socioeconomic status, few scales have been developed to measure this attribute explicitly,[306–309] and none are used in epidemiologic research.[302,303,312]

Last, even if epidemiologic studies were to use appropriate, multifaceted individual-level and household-level measures of social class, additional evidence indicates that even this range of variables might not explain the effects of social class on health. Class composition and neighborhood living conditions are also important.[49,125,146,153,313,376–378,392] Current research in both the United States and England has documented contextual effects,[49,313,358,376,392] in which the health outcomes of persons with similar individual-level or household-level measures of socioeconomic position vary not only according to geographic region but also by neighborhood conditions.

Contextual analysis, then, may offer public-health researchers an important means to avoid the "individualistic" fallacy,[393] that is, the assumption that individual-level data are sufficient to explain social phenomena, including population patterns of health and disease.[71,313,376,393] The necessity of establishing the context in which persons live was also demonstrated by Goldberger and Sydenstricker's studies of pellagra and other diseases.[379] Even so, only a handful of epidemiologic studies have employed contextual analyses,[49,313,358,376,392] despite their growing use in several other areas of social science research (e.g., education, voting patterns, crime rates).[153,377,378,394–397]

Objective versus subjective aspects of social class. Another limitation of predominant approaches to studying the relationship between social class and health is their exclusive focus on objective measures of class and the material consequences of deprivation. As in the case of racism and sexism, current thinking about psychosocial risk factors for health points to "the hidden injuries of class."[398] Chief among these is the sense of inferiority and inadequacy that pervade the lives of many working-class persons in the United States. In a country often termed the land of opportunity, many believe that individuals who fail to succeed can blame only themselves (except perhaps in times of society-wide economic disasters, like the Depression of the 1930s).[133,146,398–401] Social class and a sense of control over one's life could be associated; some have suggested that lacking this sense of control may be an important psychosocial risk factor.[146,304] The relationship between people's subjective assessment of their social class and their objectively defined position[42,313,342] may also be important, because discrepancies between the two may serve as important indicators of status incongruity,[303,402] a further likely psychosocial risk factor.[42,146,303,326,402] The meaning and implications of being objectively or subjectively classified as middle-class may also vary by race, as attested to by the many black men and women objectively categorized as middle class who nonetheless have poorer health outcomes than comparably classified middle-class white men and women. However, these types of questions have received little attention in the U.S. epidemiologic literature.

Class origins and mobility. Growing recognition of the importance of childhood conditions in influencing adult health outcomes has highlighted another limitation of current research on social class and health: its reliance chiefly on adult indicators of social class.[302,303,305,313] Some diseases, however, may be more linked to childhood class origins than to adult social class position (e.g., long-term sequelae of early childhood exposures to low levels of lead).[403–405]

Research in England, for example, has established that adult height often is an independent predictor of mortality; the hypothesized link is that height serves as an important proxy for childhood living conditions.[305,315,406] One study also found that in 212 areas of England and Wales, adult rates of ischemic heart disease from 1968 to 1971 were highly correlated with the regions' infant mortality rates from 1921 to 1925; childhood nutrition was invoked as a possible explanation.[407] Similarly, a study conducted in Finland by Kaplan and Salonen determined that childhood socioeconomic position was a better predictor of ischemic heart disease in middle-aged men than was their adult socioeconomic position, even after the analysis adjusted for age, cigarette smoking, ratio of high-density to low-density lipoproteins, serum selenium, and adult height.[408]

Psychosocial risk factors related to people's class trajectories—that is, the similarities or contrasts between their childhood class origins and adult class position—also may be crucial. Not only might status incongruity be a problem for persons who are upwardly or downwardly mobile,[326] but a lack of class mobility for persons of working-class origin might itself constitute a negative chronic stressor.[42,146,303,326,402] Conversely, the reinforcement of class privileges for persons whose parents' and whose own adult class positions are at the levels of professionals, business owners, or executives could perhaps be a protective factor. Little epidemiologic research, however, has investigated these hypotheses.

Explaining social inequalities in health. A final deficiency in current work regarding social class and health is the lack of analyses assessing the extent of morbidity and mortality in the United States attributable to being poor, working class, or otherwise of low socioeconomic status. Although the reports issued by the Secretary's Task Force on Black and Minority Health calculated the excess mortality of blacks, Hispanics, Native Americans, and Asian and Pacific Islanders versus that of the white population,[8] no such comparable calculations have been conducted comparing the poor to the affluent or nonsupervisory employees to their employers.[7,373] Even though researchers routinely calculate the population-attributable risk for a variety of diseases and causes of death linked to alcohol or smoking,[373] no such calculations have been conducted for socioeconomic status.

Furthermore, hardly any studies in the United States have attempted to determine whether known risk factors account for observed social inequalities in health status or, more importantly, why. The finding that many known risk factors often do not explain very much of the observed health disparity between social classes, as suggested by Otten et al.,[33] Haan et al.,[363] and Feldman et al.,[364] stands as a stark reminder of how little we know about the major determinants of population patterns of health and disease.

Overall Limitations: Studying Racism, Sexism, Social Class, and Health Combined

Compounding our ignorance is the lack of research attempting to study the conjoint influences of racism, sexism, and social class. Instead, each is usually investigated separately, thus impairing efforts to understand the health of people whose lives cut across these diverse realms of experiences. These limitations are also evident in publications of health statistics and studies that present results only for "men versus women" and "blacks versus whites," when in fact they have data on black and white men and women.[7,409–411] Also problematic is the implicit emphasis on study participants as either objects or victims, in contrast to viewing them as subjects with individual and collective agency, capable of altering the societal conditions that adversely affect their health.[45,83,142,143,216,412]

The urgency of considering both the joint and separate consequences of enduring, as well as resisting, oppression based on race, gender, or class is underscored by the many cited studies suggesting that the complex joint effects cannot easily be parceled into discrete categories of "being black," "being a woman," and "being working class." This research has indicated not only that the toll of racism varies among working-class and professional black men and women,[42,413] but also that the realities and health implications of being working class rather than professional differ for black and white women and men.[48,49,82,114–120] Similarly, issues like "multiple roles" apparently have different meanings and health effects for working-class and professional women,[45,209,256,267] and the types of sexism experienced by women may vary within and across racial/ethnic groups and social classes.[45,64,65,142,143,209,211,267,268] Many studies, however, inappropriately assume that the effects of racism, sexism, and social class are simply additive. Instead, their specific combinations reflect unique historical experience forged by the social realities of life in the United States and should be studied accordingly.

These problems assume a particular salience for studies on the health of black women. One of the difficulties in assessing the social experience of black women lies in the inability of current conceptual models to account for the impact of black women's multiple negative statuses, except by means of such inadequate tools as additive (gender + race + class + age + . . .) or multiplicative models.[166–171,278] Deborah King summarizes this problem:

> Unfortunately, most applications of the concepts of double and triple jeopardy have been overly simplistic in assuming that the relationships among the various discriminations are merely additive . . . [as if] each discrimination has a single, direct, and independent effect on status, wherein the relative contribution of each is readily apparent. This simple incremental process does not represent the nature of black women's oppression but, rather, I would contend, leads to nonproductive assertions that one factor can and should supplant the other.[167,p47]

As an alternative, Gloria Wade-Gayles has described the social space of black women in the United States as a "dark enclosure" within a "narrow space" away from the "larger circle, in which white people, most of them men, experience influence and power."[229,p4] Her model asserts that embeddedness (within the black community) and distance (from the community of people who exercise power) together characterize the social spaces occupied by black women. Wade-Gayles's model thus begins to speak to the complexity of understanding the social realities of multiple statuses. Together with scholarship in other disciplines (e.g., literature, philosophy, history) that is moving away from simple linear models of cause and effect,[116,226,414–416] Wade-Gayles's approach has important implications for building a contextual framework for research on the relationship between black women's social experiences and their health.[170]

The minimal research that simultaneously studies the health effects of racism, sexism, and social class ultimately stands as a sharp indictment of the narrow vision limiting much of the epidemiologic research conducted within the United States today. Although the scope and statistical sophistication of epidemiologic investigations have increased markedly over the years,[135–140,417–420] comparable systemic developments have not occurred in the predominant approaches to studying the effects of racism, sexism, and social class on health.[16,34,35,47,284,304,382] One possible factor contributing to this lack of progress, as we noted above (page 93), may be the reluctance of many researchers to discuss uncomfortable subjects or to tackle issues whose remedies could lie outside the bounds of traditional public health interventions.[284] Whatever the cause, the fact remains that research regarding racism, sexism, social class, and health remains rudimentary and fragmented, a reflection of its position on the outskirts of mainstream epidemiology and the contingent deficit of wide-reaching and active debate within the discipline on the causes of social inequalities in health.

In sum, it is hard to imagine present U.S. epidemiologic studies explicitly testing detailed hypotheses about the social production and political economy of disease, as Goldberger and Sydenstricker once did. Yet without these types of investigations and without the development of alternative conceptual and methodological approaches, epidemiology will fail to meet its basic mandate of explaining current and changing patterns of health and disease in human societies, so as to set the basis for effective prevention strategies.[69,318,419,421]

EMERGING APPROACHES TO STUDYING THE HEALTH EFFECTS OF RACISM, SEXISM, AND SOCIAL CLASS: A REVIEW

Although not yet synthesized into one well-defined paradigm, a small but provocative body of epidemiologic research has begun to develop alternative approaches to studying the health effects of racism, sexism, and social class.[3,16,37–44,46–49,78,104–106,148–150,157,159–164,255,274,313,315,332,345,346,358,361,376,387,388,392,408,422–435] Building upon prior work, these new investigations are seeking to address many of the limitations of the predominant epidemiologic methods. Consequently, they are drawing upon the efforts of numerous disciplines, both within and outside the realm of public health, especially history, sociology, political economy, anthropology, psychology, biology, and the history and philosophy of science.[14,16,31,44,46,47,53,56,60,62,63,135,143,146,174,175,180,181,195,196,200,283,284,287,292,301,317,319,330,380,382,417,420,422,423,425,435–454]

This varied work can be characterized by three fundamental

and interwoven assumptions about the nature of health and disease in human societies:

1. Societal divisions based on race, gender, and class are the expression of social relations, not intrinsic facts of biology. Consequently, social factors, not genetics, primarily explain why people's membership in the groups defined by these social relations can predict their overall health status (apart from diseases involving the reproductive system).

2. The fact that population patterns of health and disease parallel societal divisions based on race, gender, and class implies that these social relations somehow "get into the body" and shape the health of groups on both sides of these social relations, whites as well as blacks and other people of color, men as well as women, and business owners and professionals as well as working-class employees.

3. The responsible mechanisms exist at both the social and biological level (each with their ultimate and proximate causes), and both levels must be studied to understand what creates current and changing population patterns of health and disease. Linking these three assumptions is the core belief that etiologic explanations focusing only on biological mechanisms and ignoring risk differences based on race, gender, and class are as incomplete and as misguided as those focusing only on social factors and ignoring the biological basis of disease.[214,455]

Summing up this view is the important insight elaborated in Geoffrey Rose's eloquent essay, "Sick Individuals and Sick Populations"[71]: causes of individual cases are not necessarily the same as determinants of incidence in populations. Using hypertension as an example, Rose argues that two different questions arise: "why do some individuals have hypertension?" and "why do some populations have much hypertension, whilst in others it is rare?"[71] The former question emphasizes individual susceptibility, whereas the latter shifts the focus to population exposures. The implication is that to understand the epidemiology of any particular disease, as well as the diseases of particular population groups, we need to explore the social patterning of both exposure and susceptibility in our everyday lives, shaped by our intertwined histories as members of a particular society and as biological creatures who grow, develop, interact, and age.[36,214]

Emerging Hypothesis: Exposure, Susceptibility, and the Social Production of Disease

Few investigators engaged in developing this alternative research agenda have written explicitly about how the general precepts mentioned above can be translated into discrete, testable hypotheses.[16,44,71,304,326,332,382,422,425,452,456] For this reason, we briefly describe four important elements of the operative framework informing this emerging work. Each element pertains to different aspects of the relationship between exposure and susceptibility mediated by societal conditions as well as by biological and cultural heritage.

First, this new work posits that the occurrence of disease at both the individual and population level results from a dynamic interplay between exposure and susceptibility. Elucidating the epidemiology of health and disease consequently requires understanding both exposure and susceptibility, separately and in relation to each other. In this alternative view, however, exposure is typically emphasized, because susceptibility in the absence of exposure does not confer added risk of disease. Moreover, the specific events or processes that constitute the exposures or affect susceptibility are deemed extremely varied and can act as specific insults or antagonists for specific diseases, as well as agents capable of affecting the likelihood of developing or resisting a variety of poor health outcomes.

Second, this framework considers how the events and processes integral to both exposure and susceptibility are fundamentally conditioned by history—both by the history of individuals as members of particular societies and also by their histories as biological organisms.[36,214] At issue are not only a society's social relations and its technological level[36,44] but also the organism's ontogeny and phylogeny.[453] Just as certain exposures are unique to certain periods of history (e.g., synthetic petrochemical products were developed after World War II),[447,457] so certain aspects of biological susceptibility are uniquely linked to the embryologic development and subsequent growth and differentiation of diverse tissues within the body. For instance, malignant growths are most likely to develop in the body's most rapidly growing and least differentiated tissues, in part because DNA apparently is most susceptible to carcinogenic initiators during mitosis.[458–461] Consequently, the timing, as well as content, of events and processes pertaining to exposure and susceptibility is crucial.[36,462–464] Rubella acquired in utero, for example, is a far more destructive disease than when it is contracted during childhood,[465] and severe sunburns before puberty are a much greater risk factor for malignant melanoma than adult sunburns.[70]

Third, this orientation views social relations (e.g., of race, gender, and social class) as strongly influencing patterns of both exposure and susceptibility among individuals within the groups defined by these relations at any given time and also over time.[36,382] Even so, the direction of these influences is neither obvious nor uniform. Although morbidity and mortality associated with material deprivation will, by definition, be most extreme among the poor, it is not assumed that these social relations necessarily raise or lower the risk of all diseases within all groups or that the excess prevalence of particular conditions in certain groups is fixed over time. Cardiovascular disease and cancer, for example, were labeled "diseases of affluence" in the earlier part of this century, but they now exact their worse toll among the poor, the working class, and people of color.[7,16,135,373,425]

Fourth, the new research holds that these social relations are determinants of population patterns of health and disease through four principal pathways: (1) by shaping exposure and susceptibility to risk factors, events, and processes; (2) by shaping exposure and susceptibility to protective factors, events, and processes; (3) by shaping access to, and type of, health care received; and (4) by shaping health research and health policy. These pathways are established through the social and economic activities required to maintain and reproduce people's daily existence at the societal and household level, including not only economic and domestic production but also their contingent aspects of consumption and waste.[16,36,44,45,214,301,319,456]

For example, factors that constitute harmful exposures or that exacerbate susceptibility can stem from the interplay of material, psychosocial, and biological conditions (with the latter two often shaped and constrained by material conditions).[16,36,44,45,301,317,319,456] These factors "get into the body" by routes ranging from direct physiologic or genetic damage to physiologic responses to stress mediated by immunologic, hor-

monal, or neurologic mechanisms. Illustrations include exposure to harmful substances or interactions while one is at work, commuting, at home, and in the community; unemployment and underemployment; insufficient income to purchase the necessities of life (adequate housing, clothing, food, and fuel) or to achieve desired social status; inadequate neighborhood food markets, inordinate numbers of liquor stores and gun stores, and high rates of violence and crime; societal promotion of health-damaging behaviors, lifestyles, and stereotypes; social isolation; cultural denigration; feelings of powerlessness and their translation to hopelessness; experience and perception of, as well as response to, unfair treatment (including discrimination and subordination based on race, gender, and class); and finally, biological aging and random or acquired genetic or physiologic defects.[16,36,44,45,125,301,317,319,422,456,466,467]

Similarly, factors that constitute beneficial exposures or that minimize susceptibility likewise can stem from the interplay of material, psychosocial, and biological conditions. Examples include not only being protected (often through social privilege and power) from the above-mentioned harmful situations and conditions but also being buffered by additional social support or a sense of belonging to one's family, community, and society, as well as being aided by specific random or acquired genetic or physiologic resistance factors.[16,36,44,45,125,301,317,319,456]

This alternative framework sets the basis for new types of research on the health effects of racism, sexism, and social class. We describe selected examples of recent investigations exploring these new approaches in the following section.

Emerging Approaches: Studies of Racism and Health

Racism and class. New work questioning the predominant approaches to studying whether social class contributes to black/white differences in health outcomes has begun to consider more explicitly the issue of class-based differences in health outcomes *among* blacks, as opposed to simply between blacks and whites. One study, by Krieger,[37] examined social class and black/white differences in the age-specific incidence of breast cancer. This investigation sought to determine whether social class contributes to the unexplained pattern of higher breast cancer rates among black women than white women younger than 40 years of age but lower rates among black women than white women 40 and older.[37] Contrary to what might be predicted,[36] this study found that the higher overall risk among young black women chiefly reflected elevated rates among young black women from more affluent neighborhoods, as compared to white women from comparable neighborhoods and to black women from working-class neighborhoods. By contrast, the lower overall risk among older black women mainly mirrored the reduced rates among older black women from working-class neighborhoods; rates among black and white women from affluent neighborhoods were comparably high (Table 4). Class-based differences in incidence rates, moreover, were strongest among the black women.[37]

Other preliminary research by Herman on social class and the health status of black and white women has found the association between social class and breast cancer survival to be smaller among black women[48] and has also shown that well-educated and poorly-educated black women are more likely to live in poor neighborhoods than comparably educated white women.[49] Residence in these poor neighborhoods, moreover, increases women's risk of having a low birthweight infant, above and beyond their individual educational status.[49]

The particular value of these examples is that they consider data stratified by social class *within* each racial/ethnic group, instead of only providing results regarding racial/ethnic differences adjusted for social class. This approach permits assessing whether black/white differences vary across class strata and also whether class-based differences within each racial/ethnic group are modified by other characteristics, such as age. Both types of data are thus important, because they provide important clues for understanding how the effects of racism may vary by social class and because they offer useful tests of existing theories of disease causation (i.e., tests of whether these theories account for the joint racial/class patterns of disease occurrence).

Other aspects of racism. In addition to recognizing the profound significance of social class in shaping black/white differences in disease occurrence, several studies are beginning to explore some of the adverse effects of other forms of racism on the health of black Americans, including outright discrimination

Table 4. Social class and black/white differences in breast cancer incidence, by age, San Francisco Bay Area, 1979–1981

Age group and class	Average annual breast cancer incidence[a]		Black/white rate ratio (95% CI)
	Black	White	
<40 years			
Working class	10	9	1.1 (0.7, 1.6)
Nonworking class	18	9	2.0 (1.2, 3.3)
Working class/nonworking class rate ratio (95% CI)	0.57 (0.31, 1.04)	1.04 (0.82, 1.32)	
≥40 years			
Working class	167	213	0.8 (0.7, 0.9)
Nonworking class	245	249	1.0 (0.8, 1.2)
Working class/nonworking class rate ratio (95% CI)	0.68 (0.53, 0.88)	0.86 (0.81, 0.92)	

[a]Age adjusted, per 100,000 women.

Source: Krieger N. Social class and the black/white crossover in the age-specific incidence of breast cancer. Am J Epidemiol 1990;131:804–14. Reprinted by permission.

within each socioeconomic stratum. We discuss examples such as racial discrimination in health care, environmental racism (i.e., the deliberate and disproportionate placement of toxic waste dumps adjacent to or within communities of color), and racism as a risk factor for hypertension. Other research is beginning to investigate protective factors that might assist black Americans in resisting the ways in which racism can erode their health.

Currently, several studies indicate that health providers may discriminate against black Americans by treating them less aggressively than white Americans for a variety of diseases and that this different treatment cannot be accounted for solely by severity of disease or by socioeconomic position.[34] Wenneker and Epstein, for example, found that among patients with ischemic heart disease, even after the analysis controlled for relevant socioeconomic and clinical factors, white patients were still 1.3 times (95% confidence intervals [CI] = 1.1, 1.6) more likely to receive angiography, 1.9 times (95% CI = 1.3, 2.7) more likely undergo coronary bypass surgery, and 1.7 times (95% CI = 0.9, 3.1) more likely to have angioplasty than were black patients.[106] Similarly, Mayer and McWhorter documented that black patients with bladder cancer were 1.8 times (95% CI = 1.3, 2.4) more likely to go untreated than were white patients, even after the analysis took into account age, stage, sex, and tumor histology.[157] Yergan et al. likewise found that neither health status, type of hospital, nor insurance plan could explain why white pneumonia patients received more intensive care than patients of other races.[158] Kjellstrand found that among patients 21 to 45 years of age on long-term dialysis, patients who were not white were half as likely as white patients to receive a kidney transplant, even after adjustment for age and sex.[159] Less directly, Ford et al. have shown that black Americans suffer higher rates of coronary artery disease than white Americans but have lower use rates for both coronary arteriography and coronary by-pass surgery—a disparity that suggests racial bias in access to or the delivery of appropriate health care.[160]

Others have found that black Americans perceive their quality of care to be inferior to that received by white Americans.[105,144] Documented problems include not only numerous examples of physicians' racial and cultural insensitivity[105,143,144,468] but also evidence of inappropriate or coercive practices, such as sterilization abuse among women of color[142,143,216,261] and the greater likelihood of subjecting pregnant women of color to court-ordered obstetrical interventions.[266] Reflecting physicians' biases, a 1987 survey by Bullock and Houston found that 30 of 31 black medical students recounted experiencing racial discrimination in medical school, nearly twice the percentage that reported discrimination in high school and college.[469] Additional accounts suggest that the process of medical education, especially during residency, may be biased by racist attitudes toward the patients (Janet Mitchell, personal communication, 1991). Racial discrimination may also explain why Svensson discovered in a recent review of 50 clinical trials that only 20 included black patients; moreover, nearly a quarter of these 20 studies enrolled a lower percentage of black subjects than were represented in the surrounding population.[161] Echoing these types of findings and concerns, in 1991, Dr. Louis Sullivan—then Secretary of Health and Human Services—observed, "There is clear, demonstrable, undeniable evidence of discrimination in our health care system";[470] he made this statement at a conference regarding the infamous Tuskegee syphilis study in which poor black men with syphilis were followed from the 1930s to 1960s without treatment until they died to determine the "natural" history of the disease.[454]

A different form of health-related racial discrimination also receiving current attention is "environmental racism."[154,155,412] A recent national study by the Commission for Racial Justice of the United Church of Christ, for example, found that, although commercial hazardous waste facilities were almost exclusively located in poor communities, race itself was the key factor in determining which poor neighborhoods were selected as the sites for these facilities.[154] This report also disclosed that three of the five largest commercial hazardous waste landfills in the United States are located in predominantly black and Hispanic communities. Similarly, it documented that uncontrolled toxic waste sites are more likely to be located in neighborhoods where the majority of residents are people of color than in white neighborhoods.[154] The targeting of minority communities for noxious substances has also been documented in other forms, such as research on how cigarette manufacturers in the past few years have begun to focus on minority communities to increase their declining sales.[471]

Hypertension research, however, has presented the most explicit investigations about racism and somatic health. To date, the twofold to threefold higher prevalence of hypertension among black than among white Americans remains largely unexplained, despite numerous studies on the leading suspect risk factors (diet, alcohol, obesity, and their relationship to renal and cardiovascular physiology, as well as possible psychosocial risk and protective factors ranging from anger expression to social support).[134,151] To address this problem, researchers are now investigating the hypothesis that racism itself can be a psychosocial risk factor.[38,40–43,148–150,413,472] In several of these studies, subjects were directly asked questions about their experiences of, and responses to, racial discrimination;[38,40,42,43,149] other studies consider the additional and specific stresses and constraints racism imposes on black Americans.[39–42,148–150] In both cases, the findings imply that the experience of, and reactions to, racism merit further investigation as important psychosocial risk factors capable of adversely affecting health.

One study, for example, used ambulatory monitoring to measure the daytime, evening, and nocturnal blood pressures of black and white women in technical and clerical jobs.[426] Regardless of race, daytime and evening pressures were higher among women who perceived more stress at work than at home, with blood pressures comparable among black and white women in each stress-defined group. Even so, in both stress groups, black women's blood pressures declined less overnight, suggesting that these higher nocturnal pressures could not be attributed simply to differences in perceived stress at work and at home.

As one hypothesis to account for additional sources of stress leading to the excess risk of hypertension among black Americans, Sherman James has developed the concept of "John Henryism."[39–41] The concept invokes the nineteenth-century legend of the black folk hero, John Henry, a railway worker who competed against and defeated a steeldriving machine but then died with his hammer in his hand. James's hypothesis thus focuses on the conflict between people's belief that they can "meet the demands of [their] environment through hard work and deter-

mination" versus the realities of being constrained by limited resources, particularly little education.[39] In his original research, conducted among relatively poor and working class black men in the rural South, James found evidence to support his view that the men at highest risk of hypertension were those who scored high on the "John Henryism Active Coping Scale" but had low levels of education.[39] A subsequent study on a similar population also found elevated blood pressure among black men with high John Henryism scores who felt that being black hindered their job success.[40] A third investigation, expanded to include black women and white men and women, found an excess risk of hypertension among black subjects only (and not the white subjects) who had high John Henryism scores and low socioeconomic status.[41]

Similar hypotheses linking risk of hypertension to conflicts created by disparities between people's aspirations and their chances of achieving them have been investigated by William Dressler.[42,148–150] Among both black women and men in the rural South, Dressler has found, even after adjusting the analyses for age, BMI, socioeconomic status, and educational level, that independent predictors of elevated blood pressure included high levels of such chronic stressors as insufficient income, racial discrimination at work, darker skin color, and high "lifestyle incongruity" scores.[148–150] This last measure evaluates whether people's "lifestyle" exceeds their educational level, with lifestyle measured by ownership of such items as a washing machine or house and also exposure to consumer culture through the print and electronic media.[148–150] To Dressler, the common pathway uniting these diverse risk factors may be the constant frustration experienced by persons seeking to claim a certain social status in the world but persistently denied this status because of low economic resources or outright discrimination.[42,148–150]

Bolstering the view that people's experience of, and reaction to, racist encounters may elevate their blood pressure, Armstead et al. recently conducted an experiment involving 27 black and predominantly female college students.[43] The investigators found that the students' blood pressure rose when they were shown movie excerpts displaying racist incidents but not when they were shown clips featuring angry but nonracial events.[43] The study also reported that over 70% of the women typically held their anger "in" and dealt with racist interactions by ignoring them; the measure of "anger in," moreover, directly correlated with elevated blood pressure.[43]

One additional study by Krieger examined racial and gender discrimination as risk factors for high blood pressure.[38] This investigation, based on telephone interviews with a random sample of 51 black women and 50 white women, 20–80 years of age, who lived in Alameda County, California, found that black women who stated they usually accepted and kept quiet about unfair treatment were over four times more likely to report hypertension than black women who said they took action and talked to others; no clear association existed among the white women. The age-adjusted risk of hypertension among black women who, in response to a list of specified situations (e.g., being made to feel inferior or being discriminated against because of their race or color while they were attending school, applying for a job, working, buying a house, receiving medical care, encountering the police), reported experiencing "zero" such instances was also nearly two to three times higher than the risk among black women who reported one or more such instances. This pattern, moreover, was strongest among women who said they kept quiet in response to unfair treatment. By contrast, no association between gender discrimination and hypertension existed among the white women. Lastly, like Dressler,[42,473] this study found that black women younger than 40 reported more racial and gender discrimination than those older than 40 and that a similar inverse association with age existed among white women for gender discrimination.

One partial explanation of these results, suggested by other research,[474] is that subjective appraisal of stressors may be inversely associated with risk of hypertension: people who can name the source of their problems may be better off than those who are uncomprehending or silent.[38,65] Subjective perception or reporting of discrimination may also be directly associated with membership in particular historical cohorts. Some evidence indicates, for example, that women who came of age during and after the civil rights and women's movements of the 1960s may be more able or willing to identify discrimination than women of prior generations.[42,65,82,276,277] Another possibility is that the association between hypertension and gender discrimination among the black women mainly reflected their experience with racial discrimination (the two discrimination scores were highly correlated). If so, an alternative interpretation might be that questions about gender discrimination may produce more ambiguous answers than questions about racial discrimination.[214] Some women, for example, may share the beliefs linking women's superiority in the domestic sphere with their lesser presence in public life and not find such arrangements oppressive.[83,202,214,261,276,277,475,476] By contrast, no such favorable superior role has ever been granted to black Americans by the dominant culture, except perhaps as musicians, performers, and athletes.[65,82,214]

Finally, a few studies have begun to consider what types of psychosocial resources might enhance black Americans' resistance to the psychological effects of racism and their physiological consequences.[42,413,472] Although limited, these investigations suggest that being involved with black community groups (e.g., black churches),[42,472,477,478] participating in or identifying with the civil rights movement,[42] and understanding the ways in which racism constrains the lives and opportunities of black Americans[413] may each serve as important buffers to the deleterious effects of everyday racism. Other resources may include social support from family members[479] and from self-help organizations, such as the National Black Women's Health Project, that seek to link wellness and empowerment.[143]

Diversity among people of color. Another area of research has begun to counter the conventional treatment of the black population as an undifferentiated racial/ethnic group.[78,163,164,480] As one example, Fruchter et al. recently examined the incidence rates of breast and cervical cancer among four groups of black women in Brooklyn, New York.[163] The women were grouped by place of birth: the United States, English-speaking Caribbean islands, Haiti, and "other" (e.g., Africa, Europe, Canada, Central and South America, and Spanish-speaking Caribbean islands).[163] Besides noting different patterns of unemployment and poverty among these diverse groups, the investigators found that Haitian women were at highest risk of invasive cervical cancer and that American-born women were at highest risk of breast cancer.[163]

Three similar studies have examined the possible link

between variations in birth outcomes and ethnic differences among black women in the United States.[78,164,480] One study reviewed U.S. national linked birth certificate and infant death certificate files for 1983–1984 and found that the incidence of low birthweight was 36% lower among foreign-born than native-born black women.[480] Additionally, after adjustments for age, parity, education, and marital status, the neonatal mortality and postneonatal mortality rates were, respectively, 22% and 24% lower among the foreign-born women.[480] Another study compared U.S.-born and foreign-born black women attending a prenatal clinic for low-income women in Boston in 1984 and found that the foreign-born women generally had better health profiles (including lower use of cigarettes, alcohol, and other drugs), more prenatal care, and better infant birth outcomes than the U.S.-born women—even after the analysis controlled for the effects of gestational age, weight gain during pregnancy, prepregnancy weight-for-height, marital status, maternal age, level of education, number of visits made for prenatal care, and use of cigarettes, alcohol, or cocaine during pregnancy.[164] A subsequent study regarding ethnic variation and maternal risk characteristics among black women in all of Massachusetts similarly discerned important differences among black Americans, Haitians, West Indians, Cape Verdeans, and black Hispanics.[78] The investigation found that black American women were at highest risk of being both teenaged and unmarried mothers and that Cape Verdean women were at greatest risk of having little education.[78]

These data suggest that failure to consider heterogeneity within the black population can potentially lead to flawed disease prevention activities; the same holds true regarding the marked diversity among Hispanics, Asians and Pacific Islanders, and native Americans.[8,79] No studies to date, however, have explicitly examined how the gender-specificity of racism may differentially shape the risk of disease among men and women of color within these diverse racial/ethnic groups.

Emerging Approaches: Studies of Sexism and Health

"Women's health" versus the health of women. Much of the new ferment regarding "women's health" and the health of women is primarily concerned with sexism in both medicine and biomedical research and its implications for women's health.[35,182,199,271,481,482] Of particular concern is the relative absence of research on women's health problems, both apart from and related to their reproductive health.

To address this deficiency, new research is testing pharmaceutical agents in women that previously have been tested only in men (e.g., the use of aspirin to reduce risk of myocardial infarction)[483] and is evaluating whether risk factors, protective factors, and diagnostic tests identified in research conducted upon men perform similarly in women.[177,178,182,481,482] Additional studies are seeking to determine if women have additional risk or protective factors that men lack.[177,178,182,481,482] Although much of the discussion concerns how these diverse drugs and risk or protective factors may be influenced by women's and men's different hormonal profiles, some researchers are investigating how physicians' attitudes towards women may compromise the quality of women's health care.[176–179,182,199,481,482]

Even so, very little of this new research agenda is devoted to examining how gender relations, and specifically the subordination of women by men, influence women's health apart from their interactions with the medical care system. Moreover, although some investigators have recommended that research be expanded to include black, Hispanic, Native American, Asian and Pacific Islander, and poor white women, as opposed to continuing to study primarily white middle-class women,[35,176,199,271,481,482] it remains unclear how this research agenda will account for the different health realities, living conditions, and needs of black women and other women of color.[176]

Diverse forms of sexism. Within the emerging body of research on sexism and women's health, one of the more developed areas concerns possible mistreatment of women by the medical profession. Since the mid-1980s, a growing number of researchers have begun to examine whether the relatively less aggressive medical treatment received by women than by men for a variety of diseases (e.g., cardiovascular disease,[427–429] lung cancer,[430] and kidney failure[159]) can be explained by gender-based differences in the severity of disease. To date, these studies' findings suggest that biomedical factors alone cannot account for these discrepancies; such results imply that physicians may be unjustifiably treating health problems differently among women and men.[35]

For example, in 1987 Tobin et al. reported that men were 6.3 times as likely as women to be referred for cardiac catheterization, even after the analysis controlled for age, previous myocardial infarction, presence of typical and atypical angina, and abnormal exercise tests results.[429] More recently, Ayanian and Epstein found that, compared to women with similar levels of coronary heart disease, men were 20% to 30% more likely to undergo coronary angiography and were 30% to 40% more likely to undergo revascularization.[427] Similarly, Steingart et al. discovered that, although men and women in a national study of postinfarction were equally likely to experience angina and receive antianginal drugs before their index infarction, after this infarction women were half as likely as men to undergo cardiac catheterization (15% versus 27%) and also coronary bypass surgery (6% versus 13%).[428] This 2:1 male:female ratio, moreover, persisted even after the analysis adjusted for relevant covariates.[428] Kjellstrand likewise observed that, among patients 46 to 60 years old who were on long-term dialysis, women were half as likely as men to receive a kidney transplant, even after the analysis controlled for age and race.[159] Finally, in a large case-control study, Wells and Feinstein demonstrated that among persons with comparable symptoms, men were 1.6 times as likely as women to receive a sputum Pap smear to determine if they might have lung cancer.[430] Until adequate studies examine the efficacy of each of these procedures among women as well as men and also investigate factors affecting physicians' clinical decision making, it will be difficult to determine whether these examples of gender-based variation in health care reflect overuse of procedures among men, underuse among women, or appropriate use in both groups.[35] These recent studies, however, suggest that unproven gender-based assumptions rather than scientific evidence may underlie physicians' apparently different approaches to treating women and men.

Other new research regarding women, sexism, and the health care system has begun to explore whether the medicalization of women's reproductive health has adversely affected women's health.[205,206,258,262–264] Recent studies conducted by Oakley et al. in England, for example, have investigated how enhancing

nonmedical social support during pregnancy (provided by social workers) can improve birth outcomes.[262,264] In a randomized controlled trial conducted among 509 predominantly working-class women with a prior history of delivering LBW babies, the nearly 250 infants born to mothers in the intervention group on average weighed approximately 40 g more at birth than did those born to mothers in the control group; the infants and mothers in the intervention group also had significantly better postnatal health.[264]

Outside of medical interventions, however, little epidemiologic research has directly examined how nonviolent forms of sexism (i.e., other than domestic violence, rape, etc.) may affect women's health. Although a handful of U.S. surveys have suggested that sexual harassment at work can undermine women's physical as well as mental health,[225] little if any epidemiologic research has in fact tested this hypothesis.[274] One exception is the previously cited preliminary work on racial and gender discrimination as risk factors for high blood pressure.[38] Also, some recent research in England has begun to explore how the content, not just the empirical number, of women's many roles in holding their families together may constitute a threat to their own health, including issues associated with purchasing and preparing food, obtaining transportation, and being caregivers.[45,432] To date, however, the etiologic question of whether nonviolent forms of everyday sexism pose a hazard to women's health remains largely unasked.[38]

Differences among women. Recently, a "life-cycle" or "life-stage" approach to studying women's health has become increasingly prominent.[176,179,182,197,216] The impetus for highlighting these age-based differences among women stems in part from the growing recognition of how women's health can be affected by biological (including hormonal) changes associated with diverse processes and events in women's reproductive history, such as the passage from prepuberty to puberty to menopause to postmenopause, as well as pregnancy-related events and lactation.[176,179,182,197,216] Although important and necessary, this refined approach at times has erred by reducing women simply to "biological females" and often has not considered how socially determined differences among women—differences based on race/ethnicity and class—can modify not only the social aspects but also the biological timing of these age-based changes.[3,177,180]

Emerging Approaches: Studies of Social Class and Health

New research about social class influences on health is much more prominent in the European than in the U.S. epidemiologic literature.[46,47,306,313,315,327,332,361,362,376,450,484–488] More open to discussing issues of class and health than U.S. researchers (see page 93), investigators in Europe are seeking to explain the full spectrum of current and apparently growing social inequalities in health.[46,331,332,361,362] Many are also addressing the ways in which predominant approaches to measuring social class have hampered investigations of the health of women and of people not in the active labor force.[46,47,313,361,376] In this section, we summarize some of the more recent research from Britain on this topic, as well as examples from the United States.

Overall, this research has demonstrated the importance of investigating the different effects of multiple levels of social class—individual, household, and neighborhood—both independently and in their interactions. Findings from this research indicate that each of these levels can affect health. The data also indicate that studies with individual-level health or disease data that use only neighborhood-based measures of social class (often because no individual-level class data are available) are likely to underestimate the magnitude of class-based inequalities in health detected with individual-level socioeconomic data.[100,313,376]

Measures of social class.

Britain. One new approach to measuring women's class in England recognizes that individual-level and household-level classes represent conceptually distinct indicators of socioeconomic position, each with its own implications for health status.[342–344,351,352,387,389] Also under study are the male-defined categories used in the Registrar General's "I–V" classification itself; empirical analysis, for example, has shown that in the 1971 census, half of all employed women fell into just six (of more than 200) occupational units.[387] This alternative approach thus has opted to move away from single, all-defining social class indicators and is instead examining social gradients in women's health as defined by four characteristics: the woman's individual occupational class, the occupational class of her husband (if she is currently married), housing tenure (owns versus rents her home), and access to private transportation (owns one or more versus no cars).[345,346,361,387,388]

A recent prospective cohort study by Pugh and Moser illustrates the new findings discovered by this emerging body of research.[387] Basing their analysis on a cohort established by taking a 1% sample of the population of England and Wales enumerated in 1971, the investigators examined mortality gradients among women in their working years (ages 15–59 years at baseline) for the period 1976–1981. Of these women, 22% were single, and 73% were married (47% of whom could be assigned an occupational class, while 53% were housewives). Using the standardized mortality ratio (SMR) as their index of mortality (which compares the age-adjusted and sex-adjusted death rate within any given group to that of the nation as a whole), Pugh and Moser established that a much wider range of SMRs could be detected by classifying women simultaneously by their own class, the class of their husbands (if they were married), and home and car ownership, as compared to the range observed by following the conventional approach of classifying single women only on the basis of their own occupation and married women only on the basis of their husband's occupation (Table 5).

The importance of considering the different information provided by individual and household class has also been demonstrated by other research in England. Arber, for example, has shown that social class gradients in the prevalence of long-standing illnesses that limit daily activities are linear for men classified by their own occupational class and for married women likewise classified by their husbands' occupational class but are curvilinear for married women classified by their own occupational class.[488,489] Britten and Heath also have observed that in families in which the women and men have different occupational classes, family size was more closely related to the women's occupational class than their husbands' class.[490] Moreover, Pill and Stott have found that among comparably poor working-class English mothers with young children, beliefs about both causation of and responsibility for illness varied according to whether the women owned or were buying the

Table 5. Standardized mortality rate (SMR) for deaths occurring between 1976–1981 among a 1% sample of women in England and Wales, 15–59 years of age in 1971

Type of women	Lowest SMR		Highest SMR		SMR change (high–low)
	Group	SMR (95% CI)	Group	SMR (95% CI)	
Single women					
Conventional: occupation only	Nonmanual	84 (65, 105)	Manual	160 (122, 203)	76
New approach: occupation plus car	Nonmanual, ≥1 car	69 (47, 98)	Manual, no car	178 (131, 236)	109
Married women with an occupation					
Conventional: husband's occupation	Nonmanual	72 (62, 84)	Manual	96 (87, 106)	24
New approach: own plus husband's occupation, home, car	Both nonmanual, own home, ≥1 car	70 (56, 86)	Both manual rent home, no car	113 (91, 138)	43
"Housewives"					
Conventional: husband's occupation	Nonmanual	71 (60, 83)	Manual	121 (109, 134)	50
New approach: husband's occupation, home, car	Nonmanual, own home, ≥1 car	65 (53, 79)	Manual, rent home, no car	161 (135, 188)	96

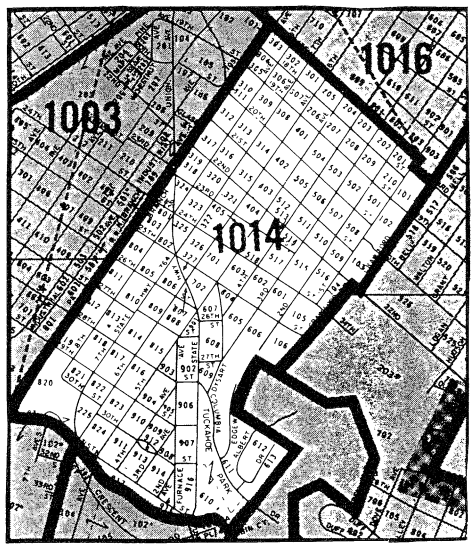
Source: Pugh H, Moser K. Measuring women's mortality differences. In: Roberts H, ed. Women's health counts. London: Routledge, 1990: 93–112. Reprinted by permission.

house where they lived and also with their level of education.[433,434] Even so, all women, regardless of housing tenure or level of education, expressed strong concerns about the constraints imposed by lack of free time.[433,434]

As an alternative solution to the problem of inconsistent and insufficiently informative class measures for women and men, other researchers in England have advocated using area-based indicators, in which individuals are categorized according to the socioeconomic characteristics of their immediate neighborhoods.[358,392,431] In a study of overall mortality, for example, Carstairs and Morris created a deprivation index consisting of the unweighted sum of the percentage of unemployed men, of households with no car, of overcrowded households (1.5 or more persons/room), and of the employed population in low social classes (IV and V).[358] The study found that this index was as sensitive to social gradients in mortality as the traditional individual-level occupational class measure and also had the advantage of being equally applicable to all persons, regardless of age, gender, or employment status.[358] Alexander et al. employed a similar approach in their analysis of mortality gradients among women in Edinburgh and detected comparable social gradients.[431] Using a different social-area indicator (the "ACORN" scale), Morgan and Chinn likewise demonstrated that neighborhood-level socioeconomic data performed as well as the parents' individual-level occupational class data in detecting social gradients in children's health.[392] Moreover, both their study and the one by Carstairs and Morris observed evidence of contextual effects—that is, neighborhood-based social class gradients in health occurred among persons grouped into the traditional class categories on the basis of their individual-level class data.

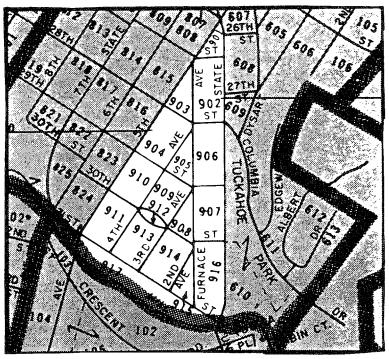
United States. Within the United States, very little research has compared either women's individual versus household class or people's individual-level versus neighborhood-level social class position as predictors or correlates of health status.[302,313,376] One preliminary study by Krieger, however, evaluated the performance of individual-level, household-level, and neighborhood-level (census tract and block-group) measures of social class as predictors of black/white differences in reproductive history among a random sample of 51 black women and 50 white women living in Alameda County, California, in 1987.[313] As indicated in Figure 2, census tracts on average contain 4,000 people, whereas census block-groups (a subdivision of the census tract) on average contain 1,000 people.[313,376] The population in census block-groups tends to be more homogeneous in sociodemographic characteristics than that of census tracts, and the block-group is also the smallest census unit for which adequate sociodemographic data can be obtained, since data at the block level are often suppressed to protect confidentiality.[313,376]

Using these different measures of social class, this study found that, after adjusting the analyses for age, race, and poverty level, household-level, but not individual-level, social class was significantly associated with the women's number of delivered pregnancies, age at first delivered pregnancy, and percentage of pregnancies terminated early (and also yearly income).[313] Additionally, social class indicators from the census-block group better approximated the class effects measured by household-level class than did indicators from the tract level. These findings suggest that, for at least some outcomes, household-level class may be more relevant to health than individual-level class. Reliance upon only individual-level data might thus underestimate the effects of class upon health, a point also stressed by Pugh and Moser in their analysis of SMRs among women cate-



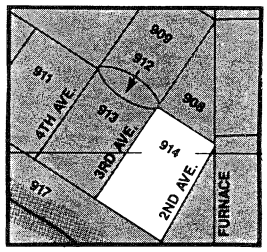
Census Tract (small, homogeneous, relatively permanent area; MSA's are subdivided into census tracts)

Average 4,000



Block Group (BG; subdivision of census tracts or block numbering areas)

Average 1,000



Block (identified throughout the country; always identified with a 3-digit number, and some have an alphabetic suffix)

Average 85

Figure 2. Illustration of the relationship between census tract, census block-group, and census block. Source: U.S. Bureau of the Census. Census '90 basics. Washington, DC: U.S. Government Printing Office; 1990.

gorized by different measures of social class (Table 5). The study also detected evidence of contextual effects: compared with professional women in nonworking class neighborhoods, working-class women from working-class neighborhoods had 1.3 more full-term pregnancies, but working-class women from the professional neighborhoods had only 0.8 more full-term pregnancies.[313] This finding, in turn, implies that, to understand population patterns of health, it is important to examine people's context, not just their personal characteristics.

This study also differed from other U.S. and English research on social class and health in two ways: the measurement of social class and the construction of the household class measure. In contrast to the standard Weberian methods, study subjects were categorized according to a relational approach, which holds that "class" is fundamentally determined not by people's particular occupation or status but by their necessary relations to others through the work process.[131,303,306,307,311] For example, an employer presupposes an employee; one cannot exist without the other. Alternatively, the category "data analyst" provides no information about class position, because someone in this occupational group could be employed on someone else's grant, self-employed, or own a consulting firm.[313] Participants in the study were therefore asked about these aspects of their "usual type of employment" and were then assigned to one of eight mutually exclusive class categories that were in turn combined into three groups: "working-class" (nonprofessional supervisory and nonsupervisory employees), "not working-class" (professional supervisory and nonsupervisory employees; self-employed professionals and nonprofessionals; and those who own a business and employ others), and "other" (not in the paid labor force). Although these categories may not be familiar to U.S. researchers, they directly reflect job control and other social dimensions now thought to contribute to class gradients in disease.[255,303,313,368–371]

The study assigned household class on the basis of the individual-class level of the respondent and, if relevant, of her partner, other head of household, or deceased spouse.[313] Household class equaled "not working-class" if one or more of these persons was "not working class" and otherwise equaled "working-class" (no household contained a respondent who was not in the paid labor force and whose partner or other head of household also was not in the paid labor force). No distinction was made between nonmarried partners and husbands, nor was the gender of the partner ever specified, thereby ensuring that the household class of heterosexual unmarried women, bisexual women, and lesbians living with their partners was treated in the same way as that of heterosexual married women living with their husbands. Among both the black and white women, a higher percentage were identified as "working-class" by the individual versus household class measure (82% versus 65% and 56% versus 43%, respectively). These differences were due to intrahousehold heterogeneity in individual class position: 25% of the 67 respondents individually identified as "working-class" lived in "not working-class" households. Subjects also were characterized by their relationship to the poverty line, their educational level, and three features of their neighborhood conditions: percentage of employed persons in "working-class" occupations, percentage of adults with less than a high school education, and percentage of persons below the poverty line.

A second study, also conducted by Krieger,[376] replicated this preliminary project on a larger scale, by using data from 14,240 black and white men and women who took the Multiphasic Health Check-Up examination in 1985 at the Oakland

facility of the Kaiser Permanente Medical Care Program in Oakland, California. The health outcomes considered were also more diverse and included hypertension, smoking status, height, and number of delivered pregnancies. This investigation again found that estimates of class effects based on census-derived measures approximated those based on individual-level class information. It also detected contextual effects in analyses simultaneously using both individual-level and neighborhood-level social class indicators. Herman observed analogous contextual effects in his study on the effects of neighborhood poverty on pregnancy outcomes among poorly and well-educated black and white women.[49]

Objective versus subjective aspects of social class. To date, little if any epidemiologic research has begun to address whether objective versus subjective measures of social class perform differently as predictors of health status.[302,303] Unpublished results from the preliminary study by Krieger comparing individual, household, and neighborhoods levels of social class, however, found neither subjective measures of social class nor their interactions with objective social class to be associated with the examined reproductive outcomes.[313,382] Alternatively, several sociological investigations have suggested that women's subjective perceptions of social class may be linked to their husband's occupation and that these perceptions of—and aspirations concerning—social class may influence women's choices regarding employment and ways of living[342,398] and thereby perhaps influence health status as well. Provocative research on the "hidden injuries of class," moreover, suggests that discrimination, exclusion, and subordination based on class might produce the same types of psychosocial trauma as racism and sexism.[146,398,432] This hypothesis has yet to be tested in epidemiologic research.

Class origins and mobility. Partly in response to increasing concerns about social inequalities in health, recent British research has sought to determine whether "downward drift" might account for the ill health among the poor—that is, whether poverty is chiefly a cause or effect of ill health.[45,46,361,486] The new findings suggest that the factor of "downward drift" explains only a very small fraction of the health status of the poor.[46,361,491]

Within the United States, however, little epidemiologic research on the health consequences of social class mobility has gone beyond Cassel's classic study on the health effects of class mobility among first-generation and second-generation urban workers in the South.[146,303,326] The much greater rates of morbidity and mortality that Cassel observed among the first-generation workers, despite comparable working conditions, suggested that social disruption itself may be a risk factor for disease. Even less research has focused on the related question of whether childhood or adult social class constitutes the most relevant class variable for specific health outcomes.[302,303] Broman's study, however, found no relationship between social mobility and hypertension among black Americans.[492] Krieger's unpublished results from the preliminary study mentioned above[313,382] indicated that only the women's adult, and not their childhood, social class positions were associated with the examined reproductive outcomes and that this relationship was not modified by their "class trajectory," whether stable, increasing, or declining.

Explaining social inequalities in health. A final new aspect of the emerging research on social class and health is the recognition that the links between social class and health, as mediated by human actions and behaviors, must be *explained*, as opposed to only documented.[16,46,331] To note that the poor have worse health habits than the wealthy is only a first step toward explaining social class gradients in disease; why these differences are associated with social class should be answered.

In England, for example, various investigations are now seeking to understand how material conditions shape so-called lifestyle behaviors.[16,45,46,331,432–434] Hilary Graham, for example, has examined cigarette smoking among working-class women and found that many poor women treat cigarettes as a fixed expense (like rent), as opposed to a contingent expense (like food), because smoking offers these women one of their few opportunities to enjoy a "luxury" and to take some time to themselves so as to have more energy to address their family's needs.[432] Thus, supposedly irresponsible behavior may in fact be the best solution women with limited choices feel they can make to meet the demands of taking care of their family's health, which they often place before their own.[432] One implication of these findings is that prevention programs should take into account the self-reported reasons why people choose seemingly negative health behaviors; these programs should also consider whether these reasons vary by race, gender, and class.

Pill and Stott reach similar conclusions in their recent research about disease causation and responsibility for health among English working-class mothers with children.[433,434] Their studies led them to contrast the realities of these women's lives to the "official definition of the irresponsible individual . . . without gender and free-floating, apart from any social context."[433,p49] Although much concern about "victim blaming" analyses has been expressed in the U.S. public-health literature,[284,300,325,493,494] few epidemiologic studies in the United States have conducted comparable investigations regarding the actual means by which social class gradients in health behaviors—and hence health—are produced. Finally, although some of Robinson's work has begun to examine how periods of economic recession and recovery may be linked to rates of specific types of occupational injuries and illnesses,[367] this topic remains largely unexplored.

RECOMMENDATIONS FOR FUTURE RESEARCH

What are the next steps to further explore how racism, sexism, and social class influence population patterns of health and disease, and particularly the problem of preterm delivery among black women? We present several general and specific recommendations. In every case, however, the starting point of future research must be to determine to what extent observed differences in health based on race, gender, and class can be attributed to accepted risk factors, to the hypothesized risk factors we describe, and to unexplained factors.

General Recommendations

Descriptive data and measures of social class. First, we need more basic descriptive data about the incidence and prevalence of, as well as mortality attributed to, specific types of illness, as stratified by race/ethnicity, gender, and social class combined. To accomplish this end, we will need to develop consistent and better measures of social class appropriate for women and for men in different racial/ethnic groups that take into account the different information offered by individual, household, and neighborhood class. This research should also consider the dif-

ferent meaning and possibly different predictive value of socioeconomic variables that measure "more" versus "less" of particular characteristics, such as income and education, and those that directly address the content of class relations, such as the employer/employee relationship and job demands and control. Additionally, these improved social class measures must be useful for women and men in all life stages (youth, adulthood, and senior years) and in all types of households—whether one is living alone or with roommates; in a heterosexual, lesbian, gay, or bisexual household; or in a nuclear or extended family. This work should also assess the biological relevance of using childhood versus adult social class indicators as correlates or predictors of adult health status and should study the effects of class trajectories on people's health.

Appropriate measures also must be developed to capture the full range of socioeconomic disparities among and between black and white Americans and should consider not only income but all assets. Similar work should develop and refine area-based measures of socioeconomic position. These must take into account the consequences of residential segregation, including the greater concentration of poverty in poor black than in poor white neighborhoods and the greater heterogeneity in class composition in wealthier predominantly black than wealthier predominantly white neighborhoods. Because of these racial disparities in living conditions, research analyzing black/white differences in health in relationship to social class should always present stratified results and should use summary comparisons adjusted for social class only if theoretically justified and empirically warranted.

Contextual analysis. Research should improve the ways both individual-level and neighborhood-level socioeconomic measures are incorporated into epidemiologic analyses. Empirical investigations must evaluate how to assess the realities of socioeconomic position, by using several measures, without resorting to indices or factor analysis. Research also must examine the validity of employing contextual analyses in epidemiologic studies and of using census-based socioeconomic measures to construct population-based incidence or mortality data stratified by social class.[37] Studies should assess the effects of local, regional, and national business cycles on health, emulating the detailed analyses conducted by Goldberger and Sydenstricker.[12,379–381] All of these suggestions may require the extension of current analytic techniques as well as the development of new statistical methods.

Discrimination, oppression, social inequalities and health. Beyond these steps, researchers need to develop and validate new methodologies to elicit the objective and subjective components of discrimination, oppression, and internalized oppression to permit testing these factors' association with risk of poor health. Empirical approaches to quantifying objective evidence of discrimination must be refined in order to assess residential and occupational segregation or pay inequities, for example, and also expanded to assess potentially discriminatory patterns of health care. Specific questionnaires also must be created and validated to assess people's subjective recognition of, attitudes toward, and reactions to unfair treatment, as well as their specific experiences of and responses to, racist, sexist, and class-biased situations. Additionally, measures of significant life stresses must be expanded to include situations such as rape, domestic violence, and other forms of assault, and measures of chronic daily hassles must likewise address the everyday realities of discrimination based on race, gender, and class. One particular goal must be to establish a means of answering the broader question how social inequities resulting from divisions based on race, gender, and class may produce a general state of psychological distress that can, in turn, affect people's hope (or hopelessness) and thus their health behaviors.

More generally, this research must develop empirical methods to assess the effect of societal living conditions, working conditions, and cultural norms on people's ways of living and specific health habits. Such methods may help elucidate why population patterns of health risks and behaviors vary by race/ethnicity, gender, and social class. The research should include not only traditionally defined occupational and environmental hazards but also the health consequences of urban design, urban planning, and social disruption[146,423,425] along with issues associated with incarceration and military service. Investigations must also examine the health effects of interacting with a medical care system whose providers may make biased clinical decisions on the basis of patients' race/ethnicity, gender, and class.

Protective factors. New research needs to focus on how people can best protect their health from being eroded by the deleterious effects of societal divisions based on race, gender, and class. Studies should consider how participating in, or living through, moments of great social change, such as those brought about by the civil rights and women's movements, can affect population risk of disease. These investigations should also explicitly test the hypothesis that people who can acknowledge and verbalize their experiences of discriminatory treatment fare better than those who remain uncomprehending or silent. We need investigation of the potentially protective effects of involvement with groups in which people can openly acknowledge, and perhaps seek healing for, hurts arising from being treated unfairly on account of one's race/ethnicity, gender, or social class (e.g., self-help groups, churches and other religious institutions, advocacy groups, or networks of friends or family). The potentially beneficial and nurturing effects of diverse aspects of people's culture, as shaped by race/ethnicity, gender, and class, merit investigation.

"Why" versus "how" questions of disease causation and prevention. Finally, we must remember that the strongest clues to etiology arise out of variation in disease patterns, not out of uniformity.[71,214] To understand and ultimately prevent inequalities in health associated with social inequalities, we must be guided by the "why" questions of explaining population patterns of disease, not simply the "how" questions regarding the mechanisms of disease causation.[44,71,284,318,418,419,495] For research to set the basis for effective disease prevention policies, it must address the structural determinants of health, not simply factors labeled as individual "lifestyle choices."[284] Continuing merely to catalog individual risk factors from an amorphous "web of causation" no longer can suffice. If our goal is to alter the web rather than merely break its strands, it is time to look for the spider.[382,p244] Because inequalities in health along the divides of class, race, and gender may offer our strongest leads, they should be the focus of our future research effort.[214]

Specific Recommendations for Preterm Delivery

Future research should consider how the relationship of specific biological and physiological responses to stresses associated with racism, sexism, and social class–before, during, and after pregnancy—may adversely affect not only the process of pregnancy but also infants' and mothers' subsequent health.

Researchers should assess events surrounding pregnancy that can affect women's class position (e.g., by leading them to establish a new household or to their being removed from an existing household) as well as modify the content of already existing stressful circumstances, such as by imposing new demands upon women's multiple roles. In every case, all research questions should be investigated among women in all racial/ethnic groups, as stratified by social class.

Before pregnancy. To determine whether pregnancy outcomes are influenced by women's preconception situation, basic descriptive and analytic research needs to be conducted regarding the somatic health and well-being of women prior to their pregnancies. Aspects to be considered include the women's position in their current household and the household in which they grew up (including household demographic and social class composition and also the possibilities of domestic violence and sexual abuse), their situation at work (class position as well as occupational hazards), their community or neighborhood conditions (both socioeconomic and environmental), and the women's sense of themselves in the society at large. The relationship of these factors to women's reproductive decisions must also be investigated, especially concerning their role in determining the occurrence of the index pregnancy, the women's acceptance of—or ambivalence about—this pregnancy, and their decision to terminate the pregnancy or carry the fetus to term.

This research should also evaluate the index pregnancies within the context of each woman's reproductive history (including spontaneous abortions, induced abortions, stillbirths, and livebirths). The effect of the women's perceived and actual reproductive options on their reproductive choices about the index pregnancy likewise must be investigated, especially regarding conditions at home and at work (e.g., potential changes in household composition or the possible effects of pregnancy upon employment status). Also pertinent are the women's perception of, and response to, being treated unfairly, particularly in relation to their race/ethnicity, gender, and social class. The protective effects of cultural heritage must be evaluated, as must the diverse forms of social support the women enjoy before the index pregnancy.

During pregnancy. Additional research must consider events and conditions that, if occurring during pregnancy, can adversely or beneficially affect pregnancy outcomes. All aspects of women's lives must be examined—at home, at work, in the community, and in society overall. The effects of the index pregnancy on women's household composition and stability should be explored, including the pregnancy's influence on women's martial status, as well as the ways in which women's ability to secure prenatal care are shaped by access to adequate health insurance and by their child-care options (i.e., it may be difficult to attend prenatal clinics if no child care is available). The potentially adverse influences of physical and chemical insults (e.g., strenuous work or exposure to occupational or environmental toxins) should be investigated, as should the hormonal, neurologic, and immunological consequences of socially induced stress on pregnancy outcomes. Research should also examine whether pregnancy itself can potentially exacerbate various social stresses (up to and including domestic violence) and whether the psychosocial effects of racism, sexism, and social class as experienced during the pregnancy, including through the provision of prenatal care, can influence pregnancy outcomes.

Future investigations should consider whether nonmedical social support, not just medical prenatal care, can improve women's ability to make informed decisions about being pregnant and about managing their pregnancies and whether such support improves pregnancy outcomes. Specific factors to be considered include nutrition and habitual substance use (tobacco, alcohol, and illicit drugs). The integration of medical care into the woman's broader nonmedical social support and its effects on pregnancy should likewise be examined and evaluated. Similarly, research should determine whether the availability and use of culturally sensitive social services during pregnancy can reduce the impact of psychosocial risks associated with racism, sexism, and social class.

After pregnancy. Studies also need to examine factors that affect the postnatal well-being of the infants, their mothers, and the rest of their families, such as infants' household conditions, the determinants of their mothers' desire and ability to breastfeed (in relation to conditions at work and at home), and both the socioeconomic conditions and specific persons influencing the women's subsequent reproductive choices, including what methods of contraception they use and when they next become pregnant. The effects of continuity versus disruption of medical care also need to be evaluated, especially regarding the women's shift from prenatal to postnatal care.

CONCLUSIONS

Implementing this alternative research agenda will require not only interdisciplinary work but also a greater commitment to involving researchers with integral ties to the communities being studied. To formulate and test hypotheses in the most effective manner, researchers should include representatives of, and health care consumers within, the affected populations at every level of the relevant research projects: conceiving the study, planning its design, recruiting subjects, ensuring participation, and interpreting, as well as disseminating, the results. Above all, future research must be oriented towards providing the basis for effective prevention at both the individual and societal level, which is, after all, both the mandate and justification for epidemiologic research.[69,318,419,421] If we fail to meet these challenges, we will not breach the impasse in our efforts to reduce, if not prevent, social inequalities in health, and our nation will perpetuate the shame and sorrow of black babies dying at twice the rate of white babies, with black families continuing to mourn these deaths as an all too common part of the experience of being black in the United States of America.

APPENDIX

Definition and Rationale for Standard Classification of Race and Ethnicity Developed for Federal Statistics and Administrative Reporting

1. American Indian or Alaskan native: a person having origins in any of the original peoples of North America, and who maintains cultural identification through tribal affiliation or community recognition.
2. Asian or Pacific Islander: a person having origins in any of the original peoples of the Far East, Southeast Asia, the Indian subcontinent, or the Pacific Islands. This area includes, for

example, China, India, Japan, Korea, the Philippine Islands, and Samoa.

3. Black: a person having origins in any of the black racial groups of Africa.

4. Hispanic: a person of Mexican, Puerto Rican, Cuban, Central or South American, or other Spanish culture or origin, regardless of race.

5. White: a person having origins in any of the original peoples of Europe, North Africa, or the Middle East.

These classifications should not be interpreted as being scientific or anthropological in nature, nor should they be viewed as determinants of eligibility for participation in any federal programs. They have been developed in response to needs expressed by both the executive branch and the Congress to provide for the collection and use of compatible, nonduplicated, exchangeable racial and ethnic data by federal agencies.

Source: Wallman KK, Hodgdon J. Race and ethnic standards for federal statistics and administrative reporting. Stat Reporter 1977;July:450–4.

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