JOGNN CLINICAL ISSUES

Psychosocial Pathways to Prematurity: Changing Our Thinking Toward a Lifecourse and Community Approach

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The purpose of this article is to explore the psychosocial antecedents of prematurity. Emphasis is on conceptual areas and supporting literature for (a) the contexts in which prematurity occurs and the diversity of women's experiences; (b) a lifecourse approach to prematurity that highlights allostatic load and the accumulation of trauma and loss in possible prematurity pathways; and (c) diverse psychosocial/biological pathways and mechanisms of prematurity processes. Pathways examining psychosocial and prematurity connections will be explicated, including antecedents and outcomes other than stress proneness and vulnerability. Implications for research are logically derived from a focus on the impact of social context on individual outcomes through multilevel models and methods. Clinical implications are derived from the social contexts, lifecourse, and multiple pathways focus of the article and include increasing social cohesion in communities, population health strategies, particular psychosocial interventions, and attentive listening. *JOGNN, 32,* 650–658; 2003. DOI: 10.1177/0884217503257529

Keywords: Lifecourse—Prematurity—Psychosocial

Accepted: March 2003

Disadvantaged women are more likely to deliver prematurely, suggesting important etiologic connections between psychosocial factors and prematurity. An initial response to psychosocial factors in prematurity was to screen for and attempt to modify individual risk factors. Categories of risk were developed, including predisposing psychosocial or biological circumstances. However, such screening by risk proved to be an inexact science, particularly among primiparous women, as many risk factors were related to reproductive history. Screening for prematurity risk has not led to greater insight into the causes of prematurity or decreases in its rate. By current estimates, only about 40% of premature births are predicted, leaving up to 60% of prematurity in women with low risk scores (Alexander & Keirse, 1990). As early as 1947, Eastman noted that only when the factors underlying prematurity are completely understood can any progress toward prevention be made.

The purpose of this article is to explore the psychosocial antecedents of prematurity. Particular emphasis will be on conceptual areas and supporting literature for the contexts in which prematurity occurs and the diversity of women's experiences, a lifecourse approach to prematurity, and the diverse psychosocial/biological pathways and mechanisms of prematurity processes. Implications for practice will focus on primary prevention and implications for research on multilevel models.

One key to understanding prematurity is to shift our focus to primary prevention. The current model for prematurity prevention is a tertiary one. NICUs, tocolysis, glucocorticoids, and antibiotic prophylaxis are all examples of tertiary measures to reduce the morbidity and mortality associated with biological processes (infectious/inflammatory, vascular, or placental) that are already under way. Secondary prevention involves the screening and identification of women at risk for prematurity. Women who have had previous preterm births, multifetal pregnancies,

or a combination of risk factors are targeted by this secondary prevention model. In contrast, primary prevention of prematurity is only in its infancy. Primary prevention of the multifactorial causes of prematurity involves a wider focus: How do we create the conditions that support healthy women who may become pregnant?

Shifting to a prematurity prevention focus also involves a shift in our time frame. "The evidence that preterm birth is more a chronic process than an acute one suggests that care may be needed days (or years) before contractions begin or membranes rupture, rather than in the hours afterward" (Iams, 1998, p. 55). Framed in that perspective, prematurity is a symptom, "an end product of an incredibly complex process" (Tarkam, 2002).

> We can test for markers, but markers give us little insight into the processes that cause the damage to occur.

Finally, this contextual, upstream focus on prematurity also requires a different way of examining factors in prematurity. Prematurity is multifactorial, not the result of a singular risk factor (Mercer et al., 1996). Furthermore, some risk factors, such as genital infection, cervical length, physical exertion, uterine volume, and placental CRH, are not simply present or absent, but present in varying degrees, their number and relative importance varying for each pregnancy (Iams, 1998).

Pathways

Since the 1980s, when social scientists and epidemiologists began considering psychosocial factors worthy of serious investigation in pregnancy, associations of psychological and social factors with prematurity have been reported. Generally these psychosocial connections to prematurity have been hypothesized via stress pathways (Gennaro & Hennessy, 2003 [this issue]; Pearlin, 1999; Wheaton, 1999), although the associations, especially with stressful life events, have been surprisingly modest (Hoffman & Hatch, 1996). Other pathways linking psychosocial and prematurity connections must now be examined.

Psychosocial Factors: Definitions

Defining *psychosocial* is increasingly complex. There are two large categories of *psychological* and *social* variables associated with prematurity. The psychological group of variables includes, but is not limited to, individual psychological traits such as anxiety, depression, resilience, optimism/fatalism, hostility, mastery/ control/self-efficacy, self-concept, rumination, cognitive style (learned helplessness), intellect, and stress/strain/ coping (Nolen-Hoeksema, Parker, & Larson, 1994; Seligman, 1975; Wells, Hobfoll, & Lavin, 1999). The group of social variables includes both an individual level—social support, employment, socioeconomic status, discrimination (gender, age, race), physical abuse, and power/autonomy/ status/resources (Campbell, Poland, Waller, & Ager, 1992; Curry, Burton, & Fields, 1998; Lobel, Dunkel-Schetter, & Scrimshaw, 1992; Norbeck & Tilden, 1983; McLeod & Nonnemaker, 1999)—and a macro/community social level—poverty/employment, segregation, violence/ conflict/abuse, environmental conditions, constrained opportunity structures, and conditions that undermine the creation of social ties (Lomas, 1998; Misra, O'Campo, & Strobino, 2001). Furthermore, as current research demonstrates, these social variables are more than just the ambient stressors they create, but may also be associated with prematurity through compromised nutrition, poor health behaviors, limited skills, and increased exposure to environmental toxins (Krause, 1999).

Persistent and accumulating evidence links some psychosocial factors, such as socioeconomic status, race, and depression, with prematurity (Cooper et al., 1996), but the associations between other psychosocial variables and prematurity have not been strong and consistent for several reasons. First, psychosocial effects have been narrowly focused on individual-level causal models, rather than multilevel models of the individual within a social context. Second, almost universally, psychosocial factors are measured only during pregnancy, not in the context of the lifecourse. The lifecourse perspective offers a way to conceptualize the accumulated effects of psychosocial variables. Third, psychosocial variables have been assessed singularly or in small clusters of variables (depression, locus of control, anxiety), while ignoring the cumulative effect of multiple clusters of factors to which women are often exposed. Fourth, social support allocated in clinical trials has yielded uniformly negative results (Hodnett, 2000). Many argue, however, that clinical trials are not a true test of social support because needed community support is omitted, as well as effective, intimate personal support that cannot be provided in a clinical trial. Fifth, the most serious limitation of previous psychosocial studies has been the absence of hypothesized causal pathways (Kramer et al., 2001). Bivariate relationships between psychosocial variables and prematurity have been extensively investigated, but specifying the effects of psychosocial variables is far more complex than was initially realized and involves processes in addition to stress (Wheaton, 1999). To better advance research on psychosocial pathways to prematurity, thinking must shift to the contexts of prematurity, a broader lifecourse approach, and include connections between biological and psychosocial factors.

Contexts of Prematurity: Social Mats

In microbiology, organisms organize in communities to perform diverse functions. These communities are described as microbial mats (Teske & Stahl, 2001). When microbes are isolated in test tubes, it is impossible to understand their function. The same is true of individual pregnant women in prenatal care settings, examined out of the context of their larger environment. These larger social and environmental factors are increasingly seen as the context shaping health or illness (Diez Roux, 2001; Lynch, Davey Smith, Kaplan, & House, 2000; Swanson et al., 2002).

The social, economic, and cultural developments of a community and its health are interdependent. Two mechanisms have been suggested to account for these connections: the *neo-material*, that is, the processes that influence resources available to people that subsequently affect their health; for example, public infrastructure, education, health services, transportation, food, housing, and so on; and the psychosocial interpretation, that is, the perceptions people have of their place in the social hierarchy, which leads to feelings of frustration, shame, or relative deprivation, and express themselves through neuroimmunological systems as disease.

Pathways via which unhealthy environments may affect health include higher levels of psychological distress and negative emotions (Seguin, Potvin, St.-Denis, & Loiselle, 1995; Taylor, Repetti, & Seeman, 1997), poorer health behaviors (Adler & Matthews, 1994; Silverman, Raj, Mucci, & Hathaway 2001), violence and environmental effects in particular neighborhoods (Taylor et al., 1997), fewer social resources to buffer stress (Seguin et al., 1995), and physiological/biological changes in the brain and cardiovascular system (Heim et al., 2000; Teicher, 2000). Furthermore, more recent evidence reminds us that the recurrence, persistence, and accumulated effects of these matted contextual stressors most strongly affect health and outcome (Oh, 2001), and the degree of income inequality is a strong predictor of poor health (Wilkinson, 1997).

> Larger social and environmental factors are increasingly seen as the context shaping health or illness.

The connections between psychosocial factors and the course and outcome of pregnancy have been the focus of serious investigation by social scientists and epidemiologists. Their focus mainly has been links between prematurity and individual-level psychosocial variables: stress (see Gennaro & Hennessy, 2003), anxiety, depression, physical abuse, resource loss, and lack of social support (Hoffman & Hatch, 1996; Misra et al., 2001; Turner, Sorenson, & Turner, 2000; Wells et al., 1999).

A Lifecourse Approach to Prematurity

Central to a psychosocial orientation to prematurity is the concept of resources. Families who experience resource loss or have fewer resources to begin with are more vulnerable (Hobfoll, 1991). Parental or sibling death, loss of trust following abuse, periods of hunger, and lack of shelter not only have profound direct effects but also shatter women's fundamental belief that the social fabric of parents, elders, and the community will provide protection. Hobfoll hypothesized that accumulated loss/trauma pathways to poor outcomes may vary according to personal resources such as resilience and optimism. Poor outcomes may not be inherent in external conditions but instead result from a mismatch between external conditions and characteristics of the individual (Aneshensel, 1999).

Two salient links between accumulated loss/trauma and health are emerging. First, biological effects of loss/trauma have been identified (Heim et al., 2000; Teicher, 2000), seen in the hippocampus of the brain and associated with depressive disorders (Brunson, Eghbal-Ahmadi, Bender, Chen, & Baram, 2001). Second, both occurrence of loss/trauma and its lifecourse accumulation affect health outcomes (Alonzo, 2000; Holland et al., 2000; van de Mheen, Stronks, & Mackenbach, 1998). Physiological reactions to chronic conditions are described as an "allostatic load" that results from a breakdown in adaptation on both biological and physiological levels, when conditions are chronic and unremitting (McEwen, 1998). In chronic and intense situations, loss spirals will tend to develop and increase in velocity (Wells et al., 1999).

Studies linking psychosocial factors and prematurity commonly assess abuse, incarcerated family members, minimal support, death of a family member, living alone, or ambivalence about the pregnancy during pregnancy (Sheehan, 1998). Unacknowledged and unmeasured are early life traumas and accumulated losses over the lifecourse. This lifecourse perspective offers a new way to conceptualize the effects of psychosocial factors on prematurity. Early loss/trauma and chronic adversity are no longer only conceptualized as instigating some disorders, for example, depression, but are seen as part of a longitudinal, accumulating chronic process that activates preexisting vulnerabilities and compromises prognoses (Neugebauer, 2001). Wheaton (1999) examined childhood traumas occurring 20-50 years earlier in three large studies and concluded that considering the effect of multiple,

longitudinal, and accumulated variables on each other and on outcomes was not only important but also essential.

Processes by which these accumulated life traumas and losses affect chemical responses and brain activity were first seen in research on the physical and psychological sequelae of abuse/trauma, changes also associated with some prematurity mechanisms (Banyard, 1999; Heim et al., 2000; McCauley et al., 1997; Teicher, 2000). Certainly, cardiovascular reactivity and recovery from accumulated loss/trauma have implications for understanding the biological underpinnings of prematurity, but only a few researchers have explored the interface of accumulated lifecourse psychosocial conditions and biological factors (Holzman et al., 2001).

Diverse Psychosocial/Biological Pathways

The history of prematurity research is littered with singular biological markers such as fetal fibronectin and salivary cortisol and diagnoses such as bacterial vaginosis and short cervical length. These markers and diagnoses describe detectable and treatable medical problems and reveal little about the underlying prematurity process. To prevent prematurity, we must understand why these processes are happening and why body parts or fetal membranes are changed. We can test for markers (if we can identify the correct ones), but markers give us little insight into the processes that cause the damage to occur (Holzman & Paneth, 1998). For prevention, not just prediction, of prematurity, we must propose diverse pathways that outline these complex processes: connections between the psychosocial and biological, as well as the individual and the larger social context over time.

It's Not Just Stress

Psychosocial influences on subsequent prematurity outcomes have generally been hypothesized as stress pathways. In addition to stress, other processes such as discrimination and accumulated loss/trauma must now be considered as factors in prematurity. For example, the following temporal sequence of accumulated loss/trauma might lead to prematurity: (a) Early parent loss promotes educational disruption and lack of coherence and attachment; (b) Sexual abuse by relatives promotes further erosion of trust and brain changes in pituitary-adrenal and autonomic responses; (c) There is high neighborhood exposure to environmental toxins (low-income neighborhoods have higher levels of toxins) and subsequent employment exposure to toxins (people with lower levels of educational attainment take jobs in which exposure to toxins is high); (d) Because of early parental loss, problem-solving and decision-making skills are not learned, leading to poor choices in partners and poor job skills; (e) Lack of finances contributes to poor nutrition; (f) Frequent drug use and STDs result from poor choices and underlying depression. (STDs are associated with a two- to threefold increase in preterm birth.) (Goldenberg, Andrews, Yuan, MacKay, & St. Louis, 1997). Prematurity may result, owing to drug use, poor nutrition, environmental/employment toxins, and the infection/inflammation associated with the reproductive tract.

Although a worst case scenario, this temporal sequence outlines one pathway to a prematurity outcome, with emphasis on variables other than stress and mechanisms other than stress proneness. Sometimes all the variables in this temporal sequence are present; sometimes only a few will be. Other variables may be present or unspecified. Equi- and multifinality remind us that diverse pathways may lead to the same outcome (equifinality), or a singular event or risk factor (drug use or infection) may lead to a range of outcomes (multifinality) in addition to prematurity (von Bertalanffy, 1968). Overall, the prematurity process evolves over time and includes the reciprocal influence of many variables and processes.

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It's Not Just the Individual

The psychosocial origins of prematurity place the individual within a social context. In this context, emerging areas of study are discrimination and inequality as antecedents of prematurity. Krieger (1999) suggested that discrimination for minority women may include a kind of gendered racism and may affect health indirectly, as in differences in treatment by a health care provider; directly at the individual level, as in self-reported discrimination, which may cause endocrine, neurologic, immune, and cardiovascular responses; and directly at the population level, as in residential segregation and poor housing associated with toxic exposures, lack of access to resources, poverty, and political disenfranchisement.

Acute and cumulative exposure to these different aspects of discrimination is a rarely explored pathway in prematurity research, although prematurity rates are nearly double in African American women ("Racial and Ethnic Disparities," 2002). Furthermore, discrimination and its effects have been linked to prematurity pathways of infection/inflammation, maternal vascular disease, and immune function (Krieger, 1999). Future discrimination research must include measurement of exposure (intensi-

ty, frequency, duration) and of susceptibility (responses to and ways of resisting discrimination), and exploration of possible pathways between discrimination exposures and prematurity outcomes. Moving beyond the individual involves widening the lens to include ecological and contextual perspectives on risk (Sameroff, 2000).

It's Not Just Pregnancy

Accumulated losses and traumas beginning in childhood and recurring over the lifecourse have been hypothesized to affect biological and psychosocial outcomes associated with prematurity. Children typically respond to untrustworthy caregiving environments or disruptions in care with lack of emotional regulation and social interaction skills. Cognitive impairments are also often present. Their lives are marked by constrained opportunities, negative social comparisons (I'm dumb), limited life skills, and insecure/anxious attachments (Ainsworth, Blehar, Waters, & Wall, 1978). Children who have repeated experiences of neglect or harm also lack a sense of coherence: "A global orientation that . . . one has a pervasive, enduring . . . feeling of confidence that one's . . . environments are predictable and that . . . things will work out as well as can be expected" (Antonovsky, 1979, p. 123).

A later manifestation of this lack of coherence is seen when survivors of childhood trauma and loss show disproportionate levels of psychological and physical disorders. Early research on precursors of adult depression focused on the effect of parental loss and elevated risk of depression, particularly for women, following undesirable childhood events (Forest, Moen, & Dempster-McClain, 1996). Inability to postpone gratification has also been associated with early deprivation or neglect, which also has implications for behaviors that may lead to prematurity. Resiliency afforded by relationships with significant adults has also been found to be protective against these negative outcomes (Garbarino, Dubrow, Kostelny, & Pardo, 1992).

Pathways of lifecourse loss/trauma leading to psychosocial issues, biological changes, and prematurity are undeveloped but must include components of resources, power, autonomy, status, poverty, prejudice, stigma, and constrained opportunities, as well as individual characteristics (McLeod & Nonnemaker, 1999). Mental health pathways have been extrapolated from these social/individual contexts, but rarely have the biological and physiological been included. Studying psychosocial antecedents of prematurity must involve examination of psychosocial, biological, and physiological processes and their reciprocal influences and pathways over the lifecourse, not just in pregnancy.

Implications for Research

Many conceptual and methodological issues are faced in studying the incredible diversity of women's experiences: enlarging the focus to include the larger social context, attention to lifecourse development, and generalizations in terms of multiple pathways.

Multilevel models are increasingly used in the social sciences to examine the impact of context on individual-level behaviors and outcomes (Teachman & Crowder, 2002). In many situations, individual-level characteristics help to determine contextual location. For example, a depressed person does not attract wide social networks. In addition, some individual-level characteristics used to predict prematurity might themselves be influenced by community conditions; for example, anxiety may be a function of the level of violence in a neighborhood. To further complicate analyses, researchers, then, must assess whether individual characteristics are independent of these contextual effects (Jencks & Mayer, 1990). The clustering of individuals within a larger context presents a potential violation of the assumption of independent observations.

Multilevel techniques avoid these problems by disaggregating the error structure into constituent parts at the individual and contextual levels. Multilevel modeling processes also allow researchers to examine additive effects of contextual conditions on the outcome of interest, in our case, prematurity.

Implications for Practice

Given that the complicated etiological web of prematurity is still being defined and that many of the mechanisms and pathways of prematurity are unknown and unspecified, it is hard to know where to begin. The concepts presented here provide possible direction for interventions: the larger social environment is increasingly seen as the context shaping women's health and prematurity outcomes; prematurity is the outcome of a chronic, complex process involving accumulated factors over the lifecourse; and paths to prematurity are numerous and diverse involving psychosocial and biological factors.

Four interventions logically derived from these concepts are (a) increasing social cohesion in communities, (b) designing population health strategies, (c) designing particular psychosocial interventions for women, and (d) providing attentive listening to individual women. It is the macro power of context as well as the micro power of individual women and changes they make in their lives that will shape new prematurity outcomes.

Increasing Social Cohesion in Communities: Primary Prevention

An appropriate overall strategy would focus on broader social systems and a public health model. Rather than a model that seeks out high-risk individuals (beating the odds), the strategy for prematurity prevention becomes increasing social cohesion and social capital in communities (changing the odds) (Seccombe, 2002).

Social capital, a buzzword of late, refers to resources that come from social relationships within communities, neighborhoods, and families. Specific indicators often include factors such as parental involvement, neighborhood support, membership in groups, levels of trust in fellow citizens, and church attendance. All of these indicators have been associated with health status in both women (Molinari, Ahern, & Hendryx, 1998) and children (Runyan et al., 1998). Furthermore, this "social capital" may be especially important to families and children with fewer financial and educational resources (Runyan et al., 1998). Some argue that income inequality exerts its effect on health only through this social capital variable (Kawachi, Kennedy, Lochner, & Prothrow-Stith, 1997).

Shifting the focus for interventions from individuals to communities and groups includes creating and preserving meeting places, sports leagues, clubs, family and social support services, parent associations, and community centers—all community elements that allow people to come together, exchange views, and develop trust. Nurses practicing primary prevention are involved in the life of their communities, not just isolated in health care (Smith, 2002). Examples of nurses' success in creating social cohesion in communities involve policy and political efforts, as well as organizing school-based clubs for preadolescent girls who are especially vulnerable. Such clubs, although initially not seen as a role for nurses, build individual assets such as self-esteem, confidence, and communication skills, as well as human bonds and community connections: building blocks in the primary prevention of prematurity.

Designing Population Health Strategies

Another public health strategy used successfully in France to decrease prematurity rates involves population health. The French program involved population-based social changes to liberalize work-leave policies in pregnancy, an intensive public education campaign to modify unhealthy behavior, and a commitment to financial support of pregnant women (Heaman, Sprague, & Stewart, 2001). Preterm birth rates declined from 5.4 to 3.7 over the course of 12 years, a reminder that it may require several years for a community-wide prevention effect to be visible in a population.

Population-based health strategies to reduce prematurity in the United States would involve influencing social policies such as enhanced income security, child benefits, family planning, improved maternity and parental leave, and providing community-wide interventions to decrease community violence and substance use. Again, nurses are familiar with lobbying for public policies for women and families and for creating community resources when needed. Recalling Lillian Wald, we know community activism is a legitimate role for all nurses.

Psychosocial Interventions for Women

It is interesting to note that although psychosocial services appear to be an important component of prenatal care for all women, they are surprisingly absent from many prenatal protocols, which focus on the detection and treatment of singular medical problems (Strong, 2000). Prenatal care as currently delivered can do little if anything to prevent preterm births without concurrent emphasis on health behavior and psychosocial issues, factors identified over a decade ago as essential "content" in prenatal visits (Public Health Service Expert Panel on the Content of Prenatal Care, 1989).

Although still limited primarily to pregnancy, psychosocial interventions that have shown promising impact on rates of the low birth weight associated with prematurity include increased levels of supportive communication, home visits, increased feedback, exploration of needed social support, and concentration on relationships that foster self-esteem (Norbeck, DeJoseph, & Smith, 1996; Zimmer-Gembeck, & Helfand, 1996).

Attentive Listening to Individual Women and Their Stories

Some nurses may feel paralyzed to affect social change and wealth distribution. Individual-level interventions are needed as well. Nursing action flows between individual and population health strategies, each shaping possibilities for better prematurity outcomes. Each nurse who listens carefully so that otherwise unrecognized emotional experiences are heard is doing prematurity prevention. Within a lifecourse perspective, listening to women is not only important during pregnancy. The accumulated losses and disconnections of women's lives, pregnant or not, can be addressed in the context of a caring relationship. Caring, listening strategies seem particularly appropriate when impaired attachment is the common antecedent, because women's self-concepts are grounded in experiences of connectedness to others (Gilligan, 1984). Listening for each individual woman's story is important to hear a sense of her history and the context of her life now. "Relational" treatment approaches, framed in the context of women's relationships, equip women with skills needed for managing and maintaining healthy relationships and healthy bodies (Tiedje & Starn, 1996).

Conclusion

Prematurity affects many women and is not restricted to an easily identifiable group or a single risk factor or intervention. Challenges remain in helping the public understand the complex, multifaceted problem of prematurity, and in supporting the primary prevention, population-based, long-term interventions needed for its solution. Health-care providers also must be challenged to

deliver psychosocial-oriented care to all women and children over the lifecourse, so that prematurity can be truly prevented, rather than merely treated.

REFERENCES

Adler, N., & Matthews, M. E. (1994). Health and psychology: Why do some people get sick and some stay well? *Annual Review of Psychology*, *45*, 229-259.

Ainsworth, M., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the strange situation*. Hillsdale, NJ: Lawrence Earlbaum.

Alexander, S., & Keirse, N. C. (1990). Formal risk scoring. In M. Enkin, N. C. Keirse, & I. Chalmers (Eds.), *A guide to effective care in pregnancy and childbirth*. Oxford, UK: Oxford University Press.

Alonzo, A. (2000). The experience of chronic illness and posttraumatic stress disorder: The consequences of cumulative adversity. *Social Science & Medicine*, *50*, 1475-1484.

Aneshensel, C.S. (1999). Outcomes of the stress process. In A.V. Horwita & T.L. Scheid (Eds.), *A handbook for the study of mental health: Social contexts, theories and systems*. (pp. 211-227). Cambridge: Cambridge University Press.

Antonovsky, A. (1979). *Health*, *stress*, *and coping*. San Francisco: Jossey-Bass.

Banyard, V. L. (1999). Childhood maltreatment and the mental health of low-income women. *American Journal of Orthopsychiatry*, *69*(2), 161-171.

Brunson, K. L., Eghbal-Ahmadi, M., Bender, R., Chen, Y., & Baram, T. Z. (2001). Long-term, progressive hippocampal cell loss and dysfunction induced by early-life administration of corticotropin-releasing hormone reproduce the effects of early-life stress. *Proceedings of the National Academy of Sciences of the United States of America*, *98*, 8856-8861.

Campbell, J., Poland, M., Waller, J., & Ager, J. (1992). Correlates of battering during pregnancy. *Research in Nursing & Health*, *15*, 219-226.

Cooper, R. L., Goldenberg, R. L., Elder, N., Swain, M., Norman, G., Ramsey, R., et al. (1996). The preterm prediction study: Maternal stress is associated with spontaneous preterm birth at less than thirty-five weeks' gestation. *American Journal of Obstetrics and Gynecology*, *175*, 1286-1292.

Curry, M. A., Burton, D., & Fields, J. (1998). The prenatal psychosocial profile: A research and clinical tool. *Research in Nursing & Health*, *21*, 211-219.

Diez Roux, A. V. (2001). Investigating neighborhood and area effects on health. *American Journal of Public Health*, *91*(11), 1783-1789.

Eastman, N. J. (1947). Prematurity from the viewpoint of the obstetrician. *American Practitioner*, *1*, 343-352.

Forest, K. B., Moen, P., Dempster-McClain, D. (1996). The effects of childhood family stress on women's depressive symptoms. *Psychology of Women Quarterly*, *20*, 81-100.

Garbarino, J., Dubrow, N., Kostelny, K., & Pardo, C. (1992). *Children in danger: Coping with the consequences of community of violence*. San Francisco: Jossey Bass.

Gilligan, C. (1984). *In a different voice: Psychological theory and women's development*. Cambridge, MA: Harvard University Press.

Goldenberg, R., Andrews, W., Yuan, A., MacKay, T., & St. Louis, M. (1997). Sexually transmitted diseases and adverse outcomes of pregnancy. *Clinics in Perinatology*, *24*, 23-41.

Heaman, M. I., Sprague, A. E., & Stewart, P. J. (2001). Reducing the preterm birth rate: A population health strategy. *Journal of Obstetric, Gynecologic, and Neonatal Nursing*, *30*, 20-29.

Heim, C., Newport, D. J., Heit, S., Graham, Y. P., Wilcox, M., Bonsall, R., et al. (2000). Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *Journal of the American Medical Association*, *284*(5), 592-597.

Hobfoll, S. E. (1991). Traumatic stress: A theory based on rapid loss of resources. *Anxiety Research*, *4*, 187-197.

Hodnett, E. D. (2000). Support from caregivers during at-risk pregnancy (Cochrane Review). In *The Cochrane Library*, *Issue 2*. Oxford: Update Software.

Hoffman, S., & Hatch, M. C. (1996). Stress, social support and pregnancy outcome: A reassessment based on recent research. *Paediatric and Perinatal Epidemiology*, *10*, 380-405.

Holland, P., Berney, L., Blane, D., Davey Smith, G., Gunnell, D. J., & Montgomary, S. M. (2000). Lifecourse accumulation of disadvantage: Childhood health and hazard exposure during adulthood. *Social Science & Medicine*, *50*, 1285-1295.

Holzman, C., Bullen, B., Fisher, R., Paneth, N., Reuss, L., & the Prematurity Study Group. (2001). Pregnancy outcomes and community health: The POUCH study of preterm delivery. *Paediatric and Perinatal Epidemiology*, *15*(Suppl. 2), 136-158.

Holzman, C., & Paneth, N. (1998). Preterm birth: From prediction to prevention (editorial). *American Journal of Public Health*, *88*, 183-184.

Iams, J. (1998). Prevention of preterm birth (editorial). *The New England Journal of Medicine*, *338*(1), 54-56.

Jencks, C., & Mayer, S. (1990). The social consequences of growing up in a poor neighborhood. In L. Lynn & M. McGeary (Eds.), *Inner-city poverty in the United States* (pp. 111-186). Washington, DC: National Academy.

Kawachi, I., Kennedy, B. P., Lochner, K., & Prothrow-Stith, D. (1997). Social capital, income inequality and mortality. *American Journal of Public Health*, *50*, 245-251.

Kramer, M. S., Goulet, L., Lydon, J., Seguin, L., McNamara, H., Dassa, C., et al. (2001). Socio-economic disparities in preterm birth: Causal pathways and mechanisms. *Paediatric and Perinatal Epidemiology*, *15*(Suppl. 2), 104-123.

Krause, N. (1999). Mental disorder in late life: Exploring the influence of stress and socioeconomic status. In C. S. Aneshensel & J. C. Phelan (Eds.), *Handbook of the sociology of mental health* (pp. 183-208). New York: Kluwer Academic/Plenum.

Krieger, N. (1999). Embodying inequality: A review of concepts, measures, and methods for studying health consequences of discrimination. *International Journal of Health Services*, *29*(2), 295-352.

Lobel, M., Dunkel-Schetter, C., & Scrimshaw, S. C. M. (1992). Prenatal maternal stress and prematurity: A prospective study of socioeconomically disadvantaged women. *Health Psychology*, *11*, 32-40.

Lomas, J. (1998). Social capital and health: Implications for public health and epidemiology. *Social Science & Medicine*, 47, 1181-1188.

Lynch, J., Davey Smith, G., Kaplan, G., & House, J. (2000). Income inequality and mortality: Importance to health of individual income, psychosocial environment or material conditions. *British Medical Journal*, *320*, 122-1204.

McCauley, J., Kern, D., Kolodner, K., Dill, L., Schroeder, A., DeChant, H., et al. (1997). Clinical characteristics of women with a history of childhood abuse: Unhealed wounds. *Journal of the American Medical Association*, *277*, 1362-1368.

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine*, *338*(3), 171-179.

McLeod, J. D., & Nonnemaker, J. M. (1999). Social stratification and inequality. In C. S. Aneshensel & J. C. Phelan (Eds.), *Handbook of the sociology of mental health* (pp. 321-344). New York: Kluwer Academic/Plenum.

Meis, P.J., Goldenberg, R.L., Mercer, B.M., Iams, J.D., Moawas, A.H., Miodovnik, M., et al. (1998). The preterm prediction study: Risk factors for indicated preterm births. *American Journal of Obstetrics and Gynecology*, *178*, 562-567.

Misra, D. P., O'Campo, P., & Strobino, D. (2001). Testing a sociomedical model for preterm delivery. *Paediatric & Perinatal Epidemiology*, *15*(2), 110-122.

Molinari, C., Ahern, M., & Hendryx, M. (1998). The relationship of community quality to the health of women and men. *Social Science and Medicine*, *47*, 1113-1120.

Neugebauer, R. (2001). Mending the world's health: World Health Day, 2001. *American Journal of Public Health*, *91*, 551-552.

Nolen-Hoeksema, S., Parker, L. E., & Larson, J. (1994). Ruminative coping with depressed mood following loss. *Journal of Personality and Social Psychology*, *67*, 92-104.

Norbeck, J. S., DeJoseph, J. F., & Smith, R. T. (1996). A randomized trial of an empirically-derived social support intervention to prevent low birthweight among African American women. *Social Science & Medicine*, *43*, 947-954.

Norbeck, J. S., & Tilden, V. P. (1983). Life stress, social support, and emotional disequilibrium in complications of pregnancy: A prospective, multivariate study. *Journal of Health and Social Behavior*, *24*, 30-46.

Oh, H. J. (2001). An exploration of the influence of household poverty spells on mortality risk. *Journal of Marriage and the Family*, *63*, 224-234.

Pearlin, L. I. (1999). The stress process revisited: Reflections on concepts and their interrelationships. In C. S. Aneshensel & J. C. Phelan (Eds.), *Handbook of the sociology of mental health* (pp. 395-415). New York: Kluwer Academic/Plenum.

Public Health Service Expert Panel on the Content of Prenatal Care. (1989). *Caring for our future: The content of prenatal care*. Washington, DC: Public Health Service, Department of Health and Human Services.

Racial and ethnic disparities in infant mortality rates. (2002). *Morbidity and Mortality Weekly Report*, *51*(13), 329-343.

Runyan, D. K., Hunter, W. M., Socolar, R. R. S., Amaya-Jackson, L., English, D., Landsverk, J., et al. (1998). Children who prosper in unfavorable environments: The relationship to social capital. *Pediatrics*, *101*, 12-18.

Sameroff, A. (2000). Ecological perspectives on developmental risk. In J. D. Osofsky & H. E. Fitzgerald (Eds.), *WAIMH Handbook of infant mental health* (pp. 4-33). New York: John Wiley.

Seccombe, K. (2002). "Beating the odds" versus "changing the odds": Poverty, resilience, and family policy. *Journal of Marriage and Family*, *64*, 384-394.

Seguin, L., Potvin, L., St.-Denis, M., & Loiselle, J. (1995). Chronic stressors, social support, and depression during pregnancy. *Obstetrics & Gynecology*, *85*, 583-589.

Seligman, M. E. P. (1975). *Helplessness: On depression, development and death*. San Francisco: W. H. Freeman.

Sheehan, T. J. (1998). Stress and low birth weight: A structural modeling approach using real life stressors. *Social Science & Medicine*, *10*, 1503-1512.

Silverman, J.G., Raj, A., Mucci, L.A., & Hathaway, J.E. (2001). Dating violence against adolescent girls and associated substance use, unhealthy weight control, sexual risk behavior, pregnancy and suicidality. *Journal of the American Medical Association*, 286, 572-579.

Smith, G. (2002, November). *Smith-Kellogg Annual Lectureship: Reducing health disparities*. American Academy of Nursing Annual Conference and Meeting, Naples, FL.

Strong, T. H. (2000). *Expecting trouble: The myth of prenatal care in America*. New York: University Press.

Swanson, J. W., Swartz, M. S., Essock, S. M., Osher, F. C., Wagner, H. R., Goodman, L. A., et al. (2002). The social-environmental context of violent behavior in persons treated for severe mental illness. *American Journal of Public Health*, *92*(9), 1523-1531.

Tarkam, L. (2002, August 6). Too many interventions and too many preemies: A conversation with Charles Lockwood. *The New York Times*, Section F, p. 6.

Taylor, S., Repetti, R., & Seeman, T. (1997). Health psychology: What is an unhealthy environment and how does it get under the skin? *Annual Review of Psychology*, *48*, 411-447.

Teachman, J., & Crowder, K. (2002). Multilevel models in family research: Some conceptual and methodological issues. *Journal of Marriage and Family*, *64*, 280-294.

Teicher, M. H. (2000, December). Wounds that time won't heal: The neurobiology of child abuse. In *Cerebrum: The Dana Forum on Brain Science*, pp. 50-67.

Teske, A., & Stahl, D. A. (2001). Microbial mats and biofilms: Evolution, structure and function of fixed microbial communities. In J. T. Staley & A. L. Reysenbaqck (Eds.), *Biodiversity of microbial life: Foundation of earth's biosphere*. New York: John Wiley.

Tiedje, L. B., & Starn, J. (1996). An intervention model for substance using women. *Image: Journal of Nursing Scholarship*, *28*, 113-118.

Turner, R. J., Sorenson, A. M., & Turner, J. B. (2000). Social contingencies in mental health: A seven-year follow-up

study of teenage mothers. *Journal of Marriage and the Family*, *62*, 777-791.
van de Mheen, H. D., Stronks, K., & Mackenbach, J. P. (1998). A lifecourse perspective on socio-economic in qualities in health: The influence of childhood socio-economic conditions and selection processes. *Sociology of Health & Illness*, *20*, 754-777.
von Bertalanffy, L. (1968). *General system theory*. New York: Braziller.
Wells, J. D., Hobfoll, S. E., & Lavin, J. (1999). When it rains, it pours: The greater impact of resource loss compared to gain on psychological distress. *Personality and Social Psychology Bulletin*, *25*(9), 1172-1182.
Wheaton, B. (1999). Social stress. In C. S. Aneshensel & J. C. Phelan (Eds.), *Handbook of the sociology of mental health* (pp. 277-300). New York: Kluwer Academic/ Plenum.
Wilkinson, R. G. (1997). Heath inequalities: Relative or absolute material standards? *British Medical Journal*, *314*, 591-595.
Zimmer-Gembeck, M. J., & Helfand, M. (1996). Low birthweight in a public prenatal care program: Behavioral and psychosocial risk factors and psychosocial intervention. *Social Science & Medicine*, *43*, 187-197.

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