presents selected factors in which smoking mothers tend to differ from nonsmoking mothers[10-12] that were not controlled for by Stone and colleagues. Because for most infants and children the mother is the primary caregiver, it is conceivable that children of smoking and nonsmoking mothers may be raised in a very different psychodynamic milieu in ways that may affect their sleeping pattern. How can we, then, claim that it is the intrauterine exposure to smoking that caused sleeping disorders and not the reality of being raised by a smoking mother who may be psychosocially very different from a nonsmoking mother? In 2009, Roza and colleagues summarized their study on the effects of parental smoking by stating

> The statistical association of parental smoking and behavioral problems was strongly confounded by parental characteristics . . . adjustment for these factors accounted entirely for the effect of both maternal and paternal smoking on child behavioral problems.[13(p680)]

In these arguments, there is no attempt to claim that constituents of cigarette smoke cannot cause sleep disorders. In fact, several other recent observational studies found similar associations.[13-15] Yet, it is critically important to realize that many psychosocial characteristics of smoking women may account for the measured differences in their children. Until such time when we identify all major confounders and adjust for them, it will be premature to conclude that sleep disturbances in the offspring are caused by intrauterine exposure to constituents of cigarette smoke.

Because pregnant women will never be randomized to exposure to antidepressants or recreational drugs, high-quality observational investigations, such as those by Oberlander and colleagues and Stone and colleagues, will be critical in distinguishing associations from causation in the field of maternal-fetal toxicology.

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Digging Deeper

Understanding the Biological Mechanisms That Connect Low Socioeconomic Status to Poor Health

THE RELATIONSHIP BETWEEN LOW SOCIOECONOMIC status (SES) and poor health outcomes has been consistently demonstrated across the lifespan.[1,2] In this issue of *Archives of Pediatrics & Adolescent Medicine*, Singh and Evans[3] describe a study that investigated relationships between neighborhood SES and exercise testing in children. The authors found that children from neighborhoods with low SES exercised for shorter duration. Heart rate recovery was also delayed in children from neighborhoods with low SES who had high body mass indexes (calculated as weight in kilograms divided by height in meters squared). These findings are important for a number of reasons.

First, the authors focus on risk factors that emerge early in life and that may have implications for disease later in

life. While substantial literature has documented the effects of SES on disease outcomes, only more recently have researchers begun to investigate potential precursors to disease. For example, complementing the study by Singh and Evans,[3] researchers have documented that low SES is associated with physiological responses and biological risk markers including heightened cardiovascular reactivity to acute stressors,[4] heightened blood pressure in adolescence,[5] and higher levels of metabolic syndrome indicators[6] in children and adolescents. These findings are important because they suggest that, even if chronic illnesses such as cardiovascular disease do not get diagnosed until later in life, social environment factors can still affect the developmental antecedents of disease and, hence, that the period of childhood and adolescence is important to study to understand the origins of the health effects of social environment factors such as SES.

See also page 479

In addition, studying these periods earlier in life provides the opportunity for preventive interventions. By understanding the life stages during which associations of SES with physiological outcomes emerge, one can identify at which points in life social environment factors get embedded biologically. With this type of life course understanding, one can better develop interventions targeted at critical periods and aimed at shifting children onto healthier trajectories before disease develops.

A second strength of Singh and Evans' study is the focus on physiological mechanisms. Such mechanistic work allows researchers to better understand how broad and distal social environment factors such as SES influence the health of an individual person. Through this type of study, researchers can begin to develop plausible models of the specific mechanisms than can transform a social experience into disease-relevant biology.[7] This type of mechanistic work is important to establish convincing explanations of the relevance of social environment factors to medical illnesses.

A third important contribution of the study by Singh and Evans involves the measurement of recovery in addition to reactivity in response to stimuli such as exercise. Researchers have traditionally focused on reactivity—ie, how much of an increase one shows in physiological parameters in response to a stressor or physical stimulus—with the idea that greater acute reactivity responses predict cardiovascular outcomes later in life.[8] However, more recently, researchers have proposed that it may not be the magnitude of the increase in physiological responses during exposure to a stimulus but rather the length of time it takes an individual to recover from that stimulus that is key to predicting later-life cardiovascular response.[9] That is, individuals who take longer to return to baseline may, over the long term, experience an accumulation of elevated physiological responses that could predispose them to diseases later in life.[10] Singh and Evans' finding that heart rate recovery was delayed in children from neighborhoods with low SES and high body mass indexes represents an important contribution to this field by bringing the physiological recovery notion into the SES literature.

Finally, the study by Singh and Evans provides insight into the notion of vulnerable groups. By highlighting that children who were both low in SES and high in body mass index had the most impaired heart rate recovery, the authors have identified a subgroup that may be at greatest risk of poor health outcomes. As this study showed, vulnerability factors may come from a variety of levels, including neighborhood factors (eg, neighborhood SES) as well as individual factors (eg, body mass index). Understanding which combination of factors predicts the greatest risk of disease will allow us to both develop more precise models about risk factors for disease and prioritize interventions to those most in need.

In summary, research such as that of Singh and Evans provides important contributions to our understanding of the social environment's risk factors for disease. Future studies should further investigate the developmental antecedents of disease, the subgroups most vulnerable to disease, and the mechanisms that link the social and physical health worlds, all with the goal of broadening our notions of the important contributors to health and well-being across the lifespan.

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