National Symposium on Family Issues

Nancy S. Landale
Susan M. McHale
Alan Booth
Editors

Families and Child Health

Springer

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Editors
Nancy S. Landale
The Pennsylvania State University
University Park, PA, USA

Susan M. McHale
The Pennsylvania State University
University Park, PA, USA

Alan Booth
The Pennsylvania State University
University Park, PA, USA

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Preface

In the last several decades, scholars from multiple disciplines have turned their attention to understanding the implications of early childhood health for adult health and susceptibility to disease. Whether drawn from biology, medicine, or the social sciences, a growing body of empirical evidence suggests that maternal health before conception, prenatal and perinatal exposures, and conditions in childhood play critical roles in health over the life course. While the broad outlines are clear, scientific understanding of the multiple and interacting influences on child health and their role in later health continues to evolve rapidly. It is clear that health disparities are set in motion very early in life, but many questions remain about the mechanisms through which such disparities emerge and are sustained across the life span.

Young children are highly dependent upon their families, and extensive bodies of scholarship document ways in which family processes and resources influence children's lives. Yet, systematic attention to the role of families in shaping developmental processes that are central to health trajectories is sparse. In this volume, prominent scholars address the family's role in children's health and development, with emphases that range from the intrauterine environment to intra-family processes to larger physical and social contexts that differ by family resources. Their contributions move the field forward toward an integration of family scholarship and cutting-edge research on the developmental origins of health disparities.

The contributions to *Families and Child Health* are based on papers presented at the 19th Annual Penn State Symposium on Family Issues in October 2011. This edited volume is the culmination of two days of stimulating presentations and discussions in four sessions, each of which focused on a different question: (1) To what extent and through what mechanisms does development in the first 1,000 days after conception shape health later in life? (2) How do intra-family processes influence children's physical and psychological health? (3) How does the structural position of children's families influence their health, and what role do early health disparities play in the intergenerational transmission of inequality? (4) What types of social programs and policies are effective in protecting children's physical and mental health?

Each of the first four parts in this volume includes a chapter by a lead author, followed by shorter chapters by scholars from diverse disciplines that extend the

breadth and depth of the discussion. The volume concludes with an integrative chapter that summarizes key themes and overarching conclusions from all of the chapters.

Part I: Bio-social Influences on Early Childhood Health

Knowledge of the biological underpinnings of later health that are set in motion before birth or very early after birth is growing rapidly, as is the understanding of how biological processes interact with social conditions to produce health risks or supports for children. The first four chapters of this volume address biosocial influences on early childhood health and beyond. The first chapter, by David Barker, physician and professor of clinical epidemiology at University of Southampton, UK, and professor of cardiovascular medicine at Oregon Health and Science University, focuses on the developmental origins of chronic adult diseases. Barker assembles evidence in support of the developmental model of disease, which posits that nutrition during fetal life, infancy, and early childhood sets the functioning of key body systems that are linked to chronic disease. In particular, the first 1,000 days of development are considered critical to lifelong health and may even have implications for health in subsequent generations.

The next three chapters expand upon Barker's emphasis on nutrition during the first 1,000 days of life. Christopher Kuzawa, a biological anthropologist at Northwestern University, addresses the puzzling finding that, while fetal nutrition is influenced by the mother's lifelong nutrition, it is influenced minimally by nutritional supplementation during pregnancy. This finding is consistent with an evolutionary framework that stresses the responsiveness of organisms to sustained environmental change, rather than short-term fluctuations, and suggests that interventions should be based on strategies that mimic or promote long-term change in the nutritional environment. Steven Haas, a sociologist and demographer at Penn State University, argues that the developmental origins model of chronic disease needs greater specificity about the prenatal exposures that influence adult chronic disease, the multiple biosocial pathways through which they operate, and the critical periods of developmental plasticity. He also questions the emphasis of the model on the first 1,000 days of life, suggesting that the influence of early life health on adult health may be altered by health inputs and insults that occur across the life course. Nathan Fox, a psychologist at University of Maryland, summarizes the effects of early psychosocial deprivation on the development of institutionalized children in Romania and the effects of an intervention that randomly assigned the children to continued institutional care or enriched foster care. The findings reinforce the conclusion that early deprivation has long-term effects on child development that cannot be reversed; at the same time, the findings support Haas' contention that early health problems may be modified by later inputs. The results of the intervention indicate that if children are removed from inadequate environments when they are very young, some degree of recovery can occur.

Part II: Role of Family Dynamics in Children's Health

Children's family environments after birth are associated with a range of physical and psychological health problems including metabolic syndrome, cardiovascular disease, internalizing symptoms, and externalizing and risk behaviors. Parents can promote child health in their roles as ources of emotional security and attachment, as models for health behaviors, and as engineers of children's everyday environments and experiences (through the provision of opportunities, guidance, knowledge, supervision, and monitoring). Conversely, parents' physical and psychological health problems and problem behaviors, family conflict and discord, and neglect or abuse of children undermine children's well-being in ways that can have long-term health implications. Part II of this volume focuses on the mechanisms through which family experiences get under children's skin, instigating and exacerbating physiological processes that undermine physical and psychological health in childhood and beyond.

The lead chapter by Patrick Davies, a psychologist at the University of Rochester, and colleagues presents an evolutionary reformulation of emotional security theory (EST-R) and outlines how emotional security systems impact physical and mental health through their effects on perceptions of social threats, the stress-response system, and the development of cognitive, emotional, and social skill sets. Davies and colleagues argue that the literature on family processes and children's health has been overly general and not theoretically grounded. The authors provide a framework for increasing the clarity and depth of future research.

Psychologist Barbara Morrongiello and Ph.D. candidate Michael Corbett, both in the Department of Psychology at University of Guelph, Ontario, raise several issues and provide a discussion of extensions of Davies' EST-R model. For example, they point out that having one well-adjusted and emotionally available parent may be sufficient to counteract the negative influence of exposure to parental conflict on child health. Child temperament, gender, developmental stage, and levels of extrafamilial support may be additional moderators of the effects of exposure to adverse family relations. Separate chapters by Dennis Drotar and Douglas Coatsworth, both clinical psychologists (Drotar in the Division of Behavioral Medicine and Clinical Psychology at Cincinnati Children's Hospital and Coatsworth at Penn State), turn to interventions that might be informed by Davies' EST-R model. Drotar argues that an important next step is identifying particular processes through which emotional insecurity affects specific child health risks, behaviors, and outcomes. Greater specificity would allow for the development of targeted intervention strategies. Coatsworth summarizes evidence about the efficacy of family-focused interventions but notes that *how* such interventions work is often unclear. He suggests several ways in which EST-R might inform intervention studies and how intervention studies might help to refine our understanding of how emotional security systems impact children's physical and mental health.

Part III: Link to the Social Environment through Families

Families provide the resources children need for development, but the adequacy of those resources depends on family socioeconomic status (SES). In general, low SES families have fewer social and economic resources and greater stress than do high SES families. Family SES also influences exposure to upstream or meso-level factors (e.g., schools, neighborhoods, health-care systems) that play a role in children's health. Part III of this volume addresses the interactive effects of biological processes, family processes, and the structural position of families on children's health and well-being.

The lead chapter by Nancy Reichman, an economist and professor of pediatrics at Robert Wood Johnson Medical School, and Julien Teitler, a sociologist at Columbia University, summarizes research findings on SES disparities in birth outcomes and childhood health. Disparities in health by SES are present at birth and grow larger as children age, and Reichman and Teitler provide an overview of the types of exposures that may account for this pattern or moderate the influence of SES on children's health. These include family environments, physical neighborhood conditions, environmental toxins, and neighborhood social and economic composition. While these environments largely influence children's health from conception forward, the authors also review evidence that suggests that children's health can be affected by parental exposures before conception, consistent with Barker's thesis about maternal nutrition. The chapter by Marianne Hillemeier, a sociologist and demographer at Penn State, also provides evidence of the importance of women's preconceptional health and exposures to their offspring's health. Drawing on results from a randomized control trial to evaluate the effectiveness of an intervention designed for low-income women, Hillemeier reports that preconception health behavior and attitudes were positively impacted by health-promotion education and social support.

While recognizing the strengths of the Reichman and Teitler chapter, Thomas Glass, a social epidemiologist at Johns Hopkins University, provides a provocative chapter in which he critiques the absence of theory in the literature on social class and health, the lack of clarity in accounts of what social class is and how it shapes health, the overemphasis on individual agency, and the inattention to culture.

Part IV: Impact of Social Policies and Programs on Children's Health

The United States spends more money on health care than any other nation on earth. In the face of its investments, however, our nation continues to experience higher rates of child mortality and chronic health conditions than other developed countries. Furthermore, rates of childhood psychological and behavioral adjustment problems and developmental disabilities are on the rise. Many of these health prob-

lems track into adulthood, creating a burden for families and for the larger society as well as lost opportunities for productive adult lives. Part IV of this volume focuses on social programs and policies aimed at enhancing children's health as well as policies with other aims that unintentionally impact children's health. The chapters consider the challenges inherent in developing, implementing, and evaluating programs and policies for enhancing family supports for children's health.

The lead chapter by social epidemiologist Lisa Berkman and graduate student Emily O'Donnell, both at the Harvard School of Public Health, focuses on work-family policies not explicitly designed to improve the health of children. Based on an ecosocial model of disease and explicit attention to work-family strain, Berkman and O'Donnell argue that family-oriented policies that lack a health-care component may nonetheless influence parental and child health through their impact on parental health behaviors (e.g., smoking, drinking, diet), family economic resources, parental time, and access to social networks and environments. After reviewing evidence linking family and child policies to child health, the chapter reviews challenges researchers face in investigating the causal effects of family policies and the channels through which such policies influence children's health. Patricia O'Campo, an epidemiologist at University of Toronto, follows up on these themes by considering both the macro-social factors that influence extant family policies and work supports and by calling for more evaluation with new methods that synthesize existing evidence regarding the efficacy of complex programs and policies.

Chapters by Paul Chung, a pediatrician at UCLA and RAND, and colleagues and by Rebecca Kilburn, an economist at RAND, explore the impact of policies and programs on particular types of children and specific health conditions. Chung and his colleagues provide a detailed description of the strain experienced by parents of children with special health-care needs (CSHCN) and the importance of family leave policies for the families of such children. Kilburn reviews research indicating that it is not only children's physical health that influences adult health and well-being but also children's psychological and mental health that shapes adult health and achievement and, consequently, should be considered alongside physical health in the development of policies. Kilburn concludes by suggesting that work-family policies may be especially beneficial to children's psychological and mental health, as may be policies that seek to improve parents' human capital (e.g., education) or promote parents' physical or mental health.

Part V: Conclusion

The final chapter is an integrative commentary by Claudia Nau, a postdoctoral trainee at the Johns Hopkins Global Center on Childhood Obesity, and Jessica Heckert, a graduate student in the Department of Human Development and Family Studies at Penn State. Nau and Heckert offer a critical review of the information and questions that are addressed in this volume, including conceptual frameworks, empirical evidence and conclusions, and policy recommendations. They provide an

overview of the challenges to be faced in future research on the role of families in early child health, research that, by extension, is central to understanding health across the life span.

University Park, PA, USA

Nancy S. Landale
Susan M. McHale
Alan Booth

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A lively, interdisciplinary team of scholars from across the Penn State community meets with us annually to generate symposia topics and plans and is available throughout the year for brainstorming and problem solving. We appreciate their enthusiasm, intellectual support, and creative ideas. In the course of selecting speakers, symposium organizers consult with a wide range of people at other universities, at NICHD, and at other organizations in order to identify highly qualified scholars to participate in the symposium. We also sincerely thank Marianne Hillemeier, Cynthia Stifter, Molly Martin, and David Almeida for presiding over symposium sessions.

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Contents

Contributors

David J.P. Barker, Ph.D., M.D. Heart Research Center, Oregon Health and Science University, Portland, OR, USA

Lisa F. Berkman, Ph.D. Harvard Center for Population and Development Studies, Cambridge, MA, USA

Paul J. Chung, M.D., M.S. Department of Pediatrics, David Geffen School of Medicine and Mattel Children's Hospital, University of California, Los Angeles, CA, USA

Department of Health Policy and Management, UCLA Fielding School of Public Health, Los Angeles, CA, USA

RAND Health, The RAND Corporation, Santa Monica, CA, USA

J. Douglas Coatsworth, Ph.D. Department of Human Development and Family Studies, The Pennsylvania State University, University Park, PA, USA

Michael Corbett, M.D. Department of Psychology, University of Guelph, Guelph, ON, Canada

Patrick T. Davies, Ph.D. Department of Clinical and Social Sciences in Psychology, University of Rochester, Rochester, NY, USA

Dennis Drotar, Ph.D. Division of Behavioral Medicine and Clinical Psychology, Cincinnati Children's Hospital, Cincinnati, OH, USA

Nathan A. Fox, Ph.D. Department of Human Development, University of Maryland, College Park, MD, USA

Thomas A. Glass, Ph.D. Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, USA

Steven A. Haas, Ph.D. Department of Sociology and Crime, Law & Justice, Penn State University, University Park, PA, USA

Jessica Heckert, M.S. Department of Human Development and Family Studies, The Pennsylvania State University, University Park, PA, USA

Marianne M. Hillemeier, Ph.D. Department of Health Policy & Administration, The Pennsylvania State University, University Park, PA, USA

M. Rebecca Kilburn, Ph.D. RAND Child Policy, RAND Corporation, Santa Monica, CA, USA

Christopher W. Kuzawa, Ph.D. Department of Anthropology and Institute for Policy Research, Northwestern University, Evanston, IL, USA

Meredith J. Martin, M.A. Department of Clinical and Social Sciences in Psychology, University of Rochester, Rochester, NY, USA

Barbara A. Morrongiello, Ph.D. Department of Psychology, University of Guelph, Guelph, ON, Canada

Claudia Nau, Ph.D. Johns Hopkins Bloomberg School of Public Health, Johns Hopkins Global Center on Childhood Obesity, Baltimore, MD, USA

Charles A. Nelson III, Ph.D. Department of Pediatrics, Boston Children's Hospital/Harvard Medical School, Boston, MA, USA

Patricia O'Campo, Ph.D. Center for Research on Inner City Health, University of Toronto, Toronto, ON, Canada

Emily M. O'Donnell, M.S. Harvard School of Public Health, Boston, MA, USA

Nancy E. Reichman, Ph.D. Department of Pediatrics, Robert Wood Johnson Medical School, New Brunswick, NJ, USA

Mark A. Schuster, M.D., Ph.D. Department of Pediatrics, Harvard Medical School, Boston, MA, USA

Division of General Pediatrics, Department of Medicine, Boston Children's Hospital, Boston, MA, USA

RAND Health, The RAND Corporation, Santa Monica, CA, USA

Melissa L. Sturge-Apple, Ph.D. Department of Clinical and Social Sciences in Psychology, University of Rochester, Rochester, NY, USA

Julien O. Teitler, Ph.D. School of Social Work, Columbia University, New York, NY, USA

Katherine D. Vestal, M.P.H. Division of General Pediatrics, Department of Medicine, Boston Children's Hospital, Boston, MA, USA

Charles H. Zeanah Jr, M.D. Department of Psychiatry and Behavioral Sciences, Tulane University, New Orleans, LA, USA

Part I
Bio-Social Influences on Early Childhood Health

Chapter 1
The Developmental Origins of Chronic Disease

David J.P. Barker

Abstract The search for the causes of chronic adult diseases and the way to prevent them has largely failed. For example, there are now 350 million people around the world who have type 2 diabetes. Hitherto, the search has been guided by a destructive model in which the causes to be identified are adverse environmental influences that act in adult life and accelerate processes associated with normal aging, such as hardening of the arteries and rising blood pressure. This model of causation has had limited success. Cigarette smoking and psychosocial stress go only a small way in explaining why one person lives a short life and another lives to old age. The recent discovery that people who develop coronary heart disease grew differently than other people in the womb and during childhood has led to a new "developmental" model for the disease. During development, adverse influences can permanently change the structure and function of the body, a phenomenon known as "programming." Much of human development is completed during the first 1,000 days after conception—during intrauterine life and infancy. Prevention of chronic disease and an increase in healthy aging require improvement in the nutrition of girls and young women.

The search for the causes of chronic adult diseases, and the way to prevent them, has largely failed. For example, there are now 350 million people around the world who have type 2 diabetes. Hitherto, the search has been guided by a destructive model in which the causes to be identified are adverse environmental influences that act in adult life and accelerate processes associated with normal aging, such as hardening of the arteries and rising blood pressure. This model of causation is based on infectious disease and presupposes that each different disease has a separate cause. It has had limited success. Cigarette smoking and psychosocial stress have been implicated

D.J.P. Barker, Ph.D., M.D. (✉)
Heart Research Center, Oregon Health and Science University,
Portland, OR, USA
e-mail: djpbarker@gmail.com

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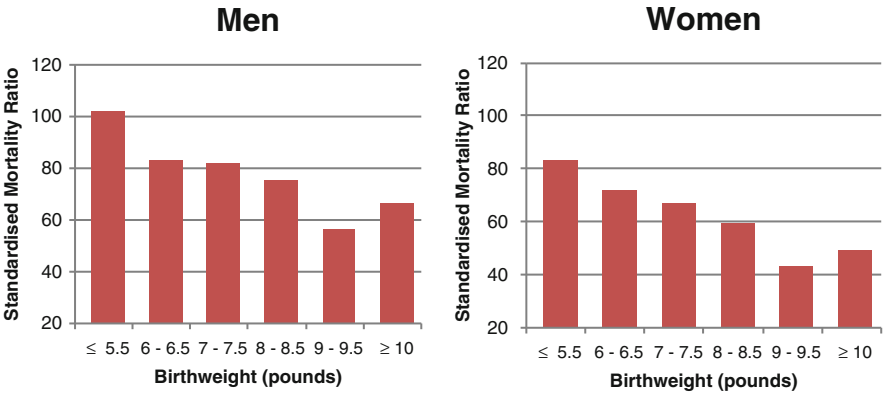

Fig. 1.1 Mortality from coronary heart disease in 15,726 men and women in Hertfordshire

as causes of chronic disease, but these go only a small way to explain why one person lives a short life and another lives to old age. Genes offer another explanation, but the search for these has been expensive and largely fruitless. Genes are unlikely to explain why coronary heart disease which was rare 100 years ago is now the world's most common cause of death.

The recent discovery that people who develop coronary heart disease grew differently to other people during fetal life and childhood has led to a new "developmental" model for the disease (Barker, 1995; Barker, Osmond, Winter, Margetts, & Simmonds, 1989). The model proposes that nutrition during fetal life, infancy, and early childhood establishes functional capacity, metabolic competence, and responses to the later environment by changing gene expression (Jackson, 2000). There is now clear evidence that the pace and pathway of early growth is a major risk factor for the development of chronic disease in adult life.

People who develop cardiovascular disease or type 2 diabetes tended to grow slowly in utero so that their birth weights were toward the lower end of the normal range and they tended to remain small during infancy, the first 2 years after birth (Hales et al., 1991; Osmond, Barker, Winter, Fall, & Simmonds, 1993). After infancy, they gain weight and body mass index rapidly (Barker, Osmond, Forsén, Kajantie, & Eriksson, 2005). These are large effects. If each individual in the Helsinki Birth Cohort had been in the highest third of birth weight and had lowered their standard deviation score for body mass index between ages 3 and 11 years, the incidence of type 2 diabetes would have been halved (Barker, Eriksson, Forsén, & Osmond, 2002). Figure 1.1, based on the original observations in Hertfordshire, UK, shows that the relation between birth weight and later disease is graded (Barker et al., 1989). These findings have been extensively replicated (e.g., Frankel, Elwood, Smith, Sweetnam, & Yarnell, 1996; Stein et al., 1996). They have led to the new developmental model for chronic disease in which the causes to be identified are linked to normal variations in feto-placental and infant development.

Programming

Like other living creatures in their early life, human beings are "plastic" and able to adapt to their environment. The development of the sweat glands provides a simple example of this. All humans have similar numbers of sweat glands at birth, but none of them function. In the first 3 years after birth, a proportion of the glands become functional, depending on the temperature to which the child is exposed: the hotter the conditions, the greater the number of sweat glands that are programmed to function. After 3 years the process is complete, and the number of sweat glands is fixed. Thereafter, the child who has experienced hot conditions will be better equipped to adapt to similar conditions in later life, because people with more functioning sweat glands cool down faster. This brief description encapsulates the essence of developmental plasticity: a critical period when a system is plastic and sensitive to the environment, followed by loss of plasticity and a fixed functional capacity. For most organs and systems, the critical period occurs in utero. There are good reasons why it may be advantageous, in evolutionary terms, for the body to remain plastic during development. It enables the production of phenotypes that are better matched to their environment than would be possible if the same phenotype was produced in all environments.

During development, adverse influences can permanently change the structure and function of the body, a phenomenon known as "programming" (West-Eberhard, 2003). In animals, it is surprisingly easy to produce lifelong changes in the physiology and metabolism of the offspring by minor modifications to the diet of the mother before and during pregnancy (Gluckman & Hanson, 2006; Widdowson & McCance, 1963). Malnutrition and other adverse influences during development permanently alter gene expression. They also lead to slowing of growth, which is why chronic disease is associated with low birth weight.

During development, there are critical periods during which a system or organ has to mature. These periods are brief; they occur at different times for different systems; and for most systems, they occur in utero. Among the major organs and systems, only the brain, liver, and immune system remain plastic after birth. Much of human development is completed during the first 1,000 days after conception—during intrauterine life and infancy. There are three reasons why people who were small at birth and during infancy are more vulnerable to chronic disease. First, they have reduced function in key organs, such as the kidney (Brenner & Chertow, 1993). Second, they have altered settings in their metabolism and hormonal feedback (Phillips, 1996). Third, they are more vulnerable to adverse environmental influences in later life (Barker, Forsén, Uutela, Osmond, & Eriksson, 2001).

The human baby is challenged and does not have sufficient resources to perfect every aspect of its body (Bateson et al., 2004). Instead, it has a hierarchy of priorities. Brain growth is at the top of this hierarchy, and the development of organs such as the kidneys and lungs, which do not function in the womb, are at the bottom. The development of low-priority organs is traded off to protect more important ones.

Compensatory Growth

If the growth of a fetus, infant, or child falters because of malnutrition or other adversity, it has the ability, once the adversity has discontinued, to return to its growth trajectory by accelerated growth. The ability to mount rapid "compensatory" growth following growth faltering is common in animals and familiar to farmers (Metcalfe & Monaghan, 2001). If energy is allocated to rapid growth, the allocation to some other developmental activity must be reduced. In animals, compensatory growth has a wide range of physiological and metabolic costs that include premature death. The costs of compensatory growth are illustrated by its effect on life span. In the Helsinki Birth Cohort, boys who were tall when they entered school lived longer lives (Barker, Kajantie, Osmond, Thornburg, & Eriksson, 2011). But there was a group of boys among whom being tall was associated with a 6-year reduction in life span. These boys were tall because they had undergone rapid compensatory growth.

Fetal Nutrition

Size at birth is the product of the fetus' trajectory of growth, which is set at an early stage in development, and the materno-placental capacity to supply sufficient nutrients to maintain this trajectory. A rapid trajectory of growth increases the fetus's demand for nutrients (Harding, 2001). This demand is greatest late in pregnancy, but the trajectory is thought to be primarily determined by genetic and environmental effects in early gestation. Experiments in animals have shown that alterations in maternal diet around the time of conception can change the fetal growth trajectory (Kwong, Wild, Roberts, Willis, & Fleming, 2000). The sensitivity of the human embryo to its environment is being increasingly recognized with the development of assisted reproductive technology (Walker, Hartwick, & Robinson, 2000). The trajectory of fetal growth is thought to increase with improvements in periconceptional nutrition and is faster in male fetuses. The consequent greater vulnerability of male fetuses to undernutrition may contribute to the shorter lives of men (Eriksson, Kajantie, Osmond, Thornburg, & Barker, 2010).

Maternal Nutrition

The growth of babies has to be constrained by the size of the mother; otherwise, normal birth could not occur. Small women have small babies. In pregnancies resulting from ovum donation, small women have small babies even if the woman donating the egg is large (Brooks, Johnson, Steer, Pawson, & Abdalla, 1995). Babies may

be small because their growth is constrained in this way or because they lack the nutrients for growth. As McCance wrote long ago, "The size attained in utero depends on the services which the mother is able to supply. These are mainly food and accommodation" (McCance, 1962). Since the mother's height or pelvic dimensions are generally not found to be important predictors of the baby's long-term health, research into the developmental origins of disease has focused on the nutrient supply to the baby, while recognizing that other influences, such as hypoxia and stress, also influence fetal growth. This focus on fetal nutrition was endorsed in a recent review (Harding, 2001). In developing countries, many babies are undernourished because their mothers are chronically malnourished. Despite current levels of nutrition in Western countries, the nutrition of many fetuses and infants remains suboptimal because the nutrients available are unbalanced or because their delivery is constrained by maternal metabolism. Around the world, size at birth in relation to gestational age is a marker of fetal nutrition (Harding, 2001).

The graded relation between birth weight and later disease (Fig. 1.1) implies that variations in the supply of food from normal healthy mothers to normal healthy babies have major implications for the long-term health of the babies (Jackson, 2000). A baby does not depend on the mother's diet during pregnancy: that would be too dangerous a strategy. Rather it lives off her stored nutrients and the turnover of protein and fat in her tissues (James, 1997). These are related to her body composition and therefore reflect her lifetime nutrition. A girl is born with all the eggs she will ever have, and the quality of these therefore reflects her mother's nutritional state. Figure 1.2 shows how the critical 1,000 days of development that determine health for life reflect 100 years of nutritional flow.

Studies in Europe and India have shown that high maternal weight and adiposity are associated with the development of insulin deficiency, type 2 diabetes, and coronary heart disease in the offspring (Fall et al., 1998; Forsén et al., 1997, 2000). Gestational diabetes is known to be associated with adverse long-term outcomes in the offspring (Silverman, Purdy, & Metzger, 1996). There is also an increasing body of evidence showing that low maternal weight, body mass index, and skinfold thickness are associated with insulin resistance and raised blood pressure in the offspring (e.g., Ravelli et al., 1998; Shiell et al., 2001). One of the metabolic links between maternal body composition and birth size is amino-acid synthesis. Women with a greater lean body mass have higher rates of synthesis in pregnancy (Duggleby & Jackson, 2001).

Although a mother's diet during pregnancy is not closely linked to the birth weight of her baby, it can program the baby. Follow-up studies of people who were in utero during the wartime famine in Holland have shown that, although the babies' birth weights were little affected, severe maternal caloric restriction at different stages of pregnancy was variously associated with obesity, dyslipidemia, insulin resistance, and coronary heart disease in the offspring (Ravelli et al., 1998). In the Dutch studies, maternal rations with a low protein density were associated with raised blood pressure in the adult offspring (Roseboom et al., 2001). This adds to the findings of studies in Aberdeen and Motherwell, UK, which showed that maternal diets with either a low or a high ratio of animal

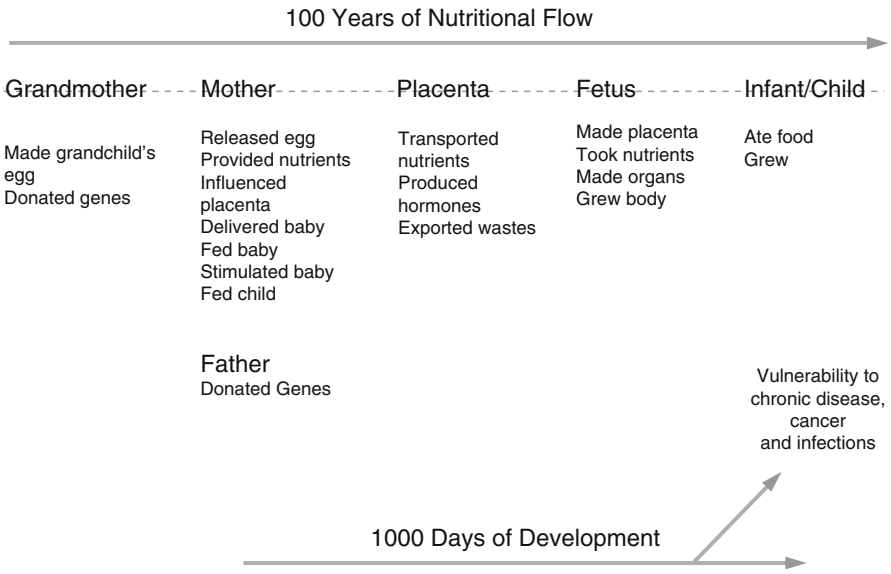

Fig. 1.2 The transgenerational roots of chronic disease

protein to carbohydrates were associated with raised blood pressure in the offspring during adult life (Campbell et al., 1996; Shiell et al., 2001). While it may seem counterintuitive that a high-protein diet should have adverse effects, these findings are consistent with the results of controlled trials of protein supplementation in pregnancy, which show that high-protein intakes are associated with reduced birth weight (Rush, 1989). One possibility is that these adverse effects are a consequence of the metabolic stress imposed on the mother by an unbalanced diet in which high intakes of essential amino acids are not accompanied by the micronutrients required to utilize them.

The Placenta

A baby's birth weight depends not only on the mother's nutrition but also on the placenta's ability to transport nutrients to the baby from its mother. The placenta seems to act as a nutrient sensor, regulating the transfer of nutrients to the fetus according to the mother's ability to deliver them and the demands of the fetus for them (Jansson & Powell, 2007). The weight of the placenta and the size and shape of its surface reflect its ability to transfer nutrients. The shape and size of the placental surface at birth have become a new marker for chronic disease in later life (Burton, Barker, Moffett, & Thornburg, 2010). The predictions of later disease

depend on combinations of the size and shape of the surface and the mother's body size. Particular combinations have been shown to predict coronary heart disease (Eriksson, Kajantie, Thornburg, Osmond, & Barker, 2011), hypertension (Barker, Thornburg, Osmond, Kajantie, & Eriksson, 2010b), chronic heart failure (Barker et al., 2010), and certain forms of cancer (Barker, Thornburg, Osmond, Kajantie, & Eriksson, 2010a). Variations in placental size and shape reflect variations in the normal processes of placental development, including implantation, growth, and compensatory expansion (Burton et al., 2010). These variations are accompanied by variations in nutrient delivery to the fetus.

Conclusion

Under the new developmental model for the origins of chronic disease, the causes to be identified are linked to normal variations in the processes of development that lead to variations in the supply of nutrients to the baby. These variations program the function of a few key systems that are linked to chronic disease: the immune system, antioxidant defenses, inflammatory responses, number and quality of stem cells, neuroendocrine settings, and balance of the autonomic nervous system. There is not a separate cause for each different disease. Rather, one cause can have many different disease manifestations. Which chronic disease originates during development may depend on timing as well as on particular nutrients.

Exploration of the developmental model will illuminate people's differing responses to the environment throughout their lives. As René Dubos wrote long ago, "The effects of the physical and social environments cannot be understood without knowledge of individual history" (Dubos, 1960). The model will also illuminate geographical and secular trends in disease. As the human body has changed over the past 200 years, so have different chronic diseases arisen and then fallen to be replaced by other diseases (Barker, 1989; Floud, Fogel, Harris, & Hong, 2011).

Coronary heart disease, type 2 diabetes, breast cancer, and other chronic diseases are preventable. Their occurrence is not mandated by genes passed down to us through thousands of years of evolution. Chronic diseases are not the inevitable lot of humankind. They are the result of the changing pattern of human development. We could readily prevent them, had we the will to do so. Prevention of chronic disease and an increase in healthy aging require improvement in the nutrition of girls and young women. Many babies in the womb in the Western world today are receiving unbalanced and inadequate diets. Many babies in the developing world are malnourished because their mothers are chronically malnourished. Protecting the nutrition and health of girls and young women should be the cornerstone of public health. Not only will it prevent chronic disease but also it will produce new generations who have better health and well-being through their lives.

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Chapter 2
How Can We Overcome the Biological Inertia of Past Deprivation? Anthropological Perspectives on the Developmental Origins of Adult Health

Christopher W. Kuzawa

Abstract Due in large part to work by David Barker and his colleagues, it is now widely accepted that prenatal nutrition modifies early development, and in so doing, influences adult biology and risk of disease. Much of this research has emphasized the limited capacity of the mother's body to buffer the fetus from stressors which may impair early development and lead to long-term health deficits. Developmental impairment may help explain some of the relationships observed between birth size and adult health. However, many biological responses initiated in utero are not due to damage but instead reflect regulatory changes in the body's metabolic or biological priorities. Some of these developmental sensitivities may have evolved to allow a fetus to use maternal cues to adjust biological settings in anticipation of postnatal environmental conditions. This hypothesis is supported by evidence that fetal nutrition is buffered against short-term fluctuations in maternal intake during pregnancy, while it shows sensitivity to a mother's lifelong nutritional experience. By calibrating fetal nutrition to the mother's average nutritional experiences, maternal metabolism could provide offspring with a reliable estimate of nutritional conditions likely to be experienced in the future. In humans, maternal buffering of fetal nutrition is predicted to limit the deleterious impact of nutritional stress experienced by the mother during pregnancy while also attenuating the health benefits of short-term pregnancy supplements. Designing interventions that mimic sustained improvement in environmental conditions may therefore be needed to optimize health in future generations.

C.W. Kuzawa, Ph.D. (✉)
Department of Anthropology and Institute for Policy Research, Northwestern University, Evanston, IL, USA
e-mail: kuzawa@northwestern.edu

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Introduction

David Barker and his colleagues published seminal studies pointing to links between prenatal nutrition and adult chronic disease (Barker, 1994; Barker, Osmond, Golding, Kuh, & Wadsworth, 1989). Despite initial skepticism of their interpretations of the data among epidemiologists and physicians, more than two decades of research has largely vindicated the model produced by Barker and his colleagues. Birth weight is commonly measured and recorded and as a result has frequently served as a proxy for prenatal nutrition, hormone exposure, and other gestational conditions in much of this work. Studies report that low birth weight not only predicts elevated risk of cardiovascular disease but also leads to deficits in key markers of human capital, such as body size, strength, educational attainment, and even wages. In parallel, experimental work in animal models has established that many developing biological systems are sensitive to conditions in the prenatal and early postnatal environments.

From a policy perspective, the finding that fetal nutrition has long-term effects on important outcomes has heightened interest in the potential benefits to future generations of supplementing the diets of pregnant women. What is less often acknowledged in the developmental origins literature is that pregnancy nutritional interventions typically have modest or even negligible effects on birth outcomes. For instance, in a systematic review of balanced protein-calorie pregnancy supplement trials, Kramer and Kakuma (2003; p. 7) found that protein-energy supplementation has at best very small effects on offspring outcomes such as birth weight. This finding led them to conclude that advising women to increase their nutritional intake "is unlikely to be of major importance." Thus, we are faced with something of a paradox and also a policy dilemma: *fetal* nutrition apparently has large effects on future health, but supplementing *maternal* nutritional intake during pregnancy does not appreciably change fetal nutrition. What might account for this apparent disconnect between what the mother consumes and what the fetus receives?

In this brief commentary, I draw upon principles from my own field of biological anthropology to shed light on the nature of developmental responses to early environments and their likely function. It has been known for more than 50 years that developmental plasticity, or the capacity of developmental biology to be modified in response to environmental change, is an important means by which human populations adapt to changing environments (Lasker, 1969). The literature documenting long-term effects of early environments expands the concept of human developmental plasticity by pointing to likely intergenerational conduits, such as nutrients or hormones passed to offspring across the placenta or via breast milk. Information about the environment, such as the prevailing level of nutritional intake, might thus be passed from the mother to the fetus (Bateson, 2001; Bateson et al., 2004; Gluckman & Hanson, 2005; Kuzawa & Pike, 2005). An evolutionary framework highlights the need to consider the *timescale* of ecological change that these flexible systems are likely built to accommodate. Most nutritional supplementation trials are short-lived and thus mimic transient environmental change. I argue that finding

ways to send intergenerational cues (e.g., nutrients and hormones) that mimic sustained environmental change will be the key to improving long-term development and health outcomes in future generations.

Anthropology and Human Adaptability

To gain insights into effective strategies for modifying early developmental plasticity to improve long-term health, it is helpful to first consider the more fundamental question of why the body modifies its developmental biology in response to early life experiences. As Barker rightly emphasizes in Chap. 1, some of the lingering effects of early experience on adult health simply reflect incomplete buffering of the fetus against nutritional deficit. As one well-documented example, individuals born small often have smaller kidneys with fewer nephrons (Lampl, Kuzawa, & Jeanty, 2002), which has been shown to increase risk for developing hypertension and renal failure in adulthood (Mackenzie & Brenner, 1995). Some long-term effects of early environments are similarly the unintended results of adaptations made by the prenatally stressed fetus to improve its chances of immediate survival. For instance, a fetus that retains more glucose in the bloodstream as a result of having insulin-resistant muscle (e.g., muscle that does not respond to insulin by taking glucose out of the bloodstream) would have a more stable energy supply for its glucose-hungry brain, which is fragile and large relative to the body at this age (Hales & Barker, 1992; Kuzawa, 1998). Although beneficial prior to birth, these same biological adjustments might later elevate risk for developing diabetes, especially if the individual gains excess weight.

Although short-term adaptations and developmental "impairment" help explain some of the long-term health effects of prenatal environments, work identifying the mechanisms underlying developmental programming has also brought to light many examples of early life developmental responses that do not fall into these categories. Take for instance fat deposition. Lower birth weight is associated with *lower* adult BMI, indicating a lower long-term risk of becoming overweight. However, research has found that, as such individuals gain weight, fat is deposited in the visceral depot in the abdomen. Visceral deposition of fat is associated with a number of negative health outcomes. This is because the visceral depot is highly metabolically active as a result of its direct innervation with sympathetic nerve fibers originating in the brain. When the body experiences stress, these sympathetic nerves immediately secrete epinephrine (adrenaline) in visceral fat cells, which stimulates the release of stored fats for use as energy to help the body overcome the stressor or challenge. The free fatty acids that are released by the visceral depot have additional cascading biological repercussions. The very act of mobilizing reserve energy stores sends a signal to the liver that the body is under duress and that glucose should thus be spared for use in more critical functions, such as immunity and brain metabolism. This is achieved by reducing the sensitivity of tissues like muscle and liver to the effects of insulin (insulin resistance) (Kuzawa, 2010). Not only do low birth weight

individuals deposit more fat in this rapidly mobilized depot, but there is also evidence that their fat cells mobilize *more* stored fats when exposed to the same dose of adrenaline, making this stored fuel rapidly available for use (Boiko, 2005).

It should be clear that there is nothing about these biological changes in the fetal growth-restricted individual that reflect either damage or impairment. Rather, these findings are evidence that prenatal nutrition can change the body's regulatory set points: in this example, the body preferentially deposits any excess energy in fat depots that are more rapidly accessible when the body is faced with stress. The fact that lower birth weight individuals also mobilize stored fat resources more rapidly once they experience a stressor shows how these various changes are all components of an integrated shift in the body's functional priorities.

As research has clarified the molecular basis of a growing list of ways that the fetus or young infant modifies development in response to early environmental factors like nutrition or stress, many examples appear to be too complex and functionally integrated to result from incomplete buffering of an early perturbation. Take for instance the recently published findings from an experiment in which maternal separation stress was imposed on mice during the first 2 weeks of postnatal life (Franklin et al., 2010). When researchers looked at chromosomes in the sperm of males exposed to the stressor, there was evidence of modified methylation (an environmentally sensitive chemical process that typically silences gene expression) in the vicinity of several genes. Modified methylation was detected near the genes coding for the cannabinoid receptor and corticotrophin releasing factor receptor, which help regulate the organism's behavioral response to novelty or challenge while also influencing traits such as anxiety. A similar pattern of changed methylation and changes in gene transcription (mRNA) were found in the neurons of female offspring of the stress-exposed males who were mated with non-stressed females, indicating that these experience-induced epigenetic changes were transmitted to offspring via sperm. Early maternal separation increased gene promoter methylation at some loci, while decreasing it at others, pointing to epigenetic changes that were specific to the genetic locus. This cascade of changed epigenetic markings helps perpetuate a similar pattern of gene expression across several generations and thereby modifies offspring's behavioral responses to stressors of the sort that the father experienced early in life. It seems unlikely that this complex sequence of intergenerational changes could simply reflect a form of stress-induced damage or impairment to early developmental biology.

Are Some Fetal Adjustments Made in Anticipation of Postnatal Conditions?

Because some of the biological changes induced by early life cues appear to reflect a change in biological regulation and modified biological priorities, rather than developmental damage, it has been speculated that developmental plasticity might in fact help the fetus prepare for conditions likely to be experienced after birth

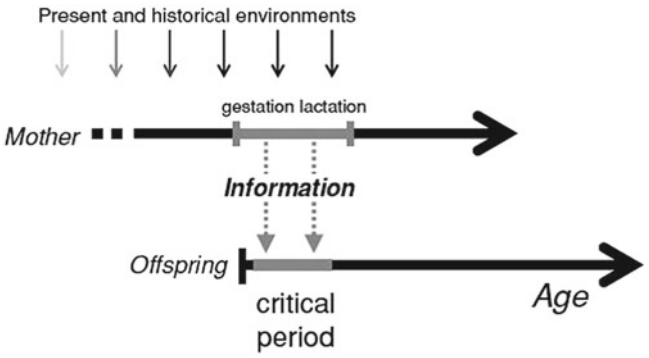

Fig. 2.1 How critical periods facilitate the flow of environmental information between generations. The timing of a critical period determines the capacity for transfer of phenotypic information between generations. Maternal phenotype comes to embody a record of a mother's cumulative environmental experiences, and this information is transferred via nutrients, hormones, and other cues (from Kuzawa & Quinn, 2009, with permission from *Annual Reviews*)

(Bateson, 2001; Gluckman & Hanson, 2005; Kuzawa, 2005) (Fig. 2.1). Some of the adjustments made by the nutritionally stressed fetus in utero, such as a tendency to deposit more abdominal body fat and the glucose-sparing effect of muscle insulin resistance, could provide the advantage of saving scarce glucose for use in more essential functions after birth, like brain metabolism and immunity, if the environment remains nutritionally stressful after birth (Kuzawa, 2010). In addition, other systems that change biological settings in response to early environments, such as stress reactivity (Weaver et al., 2004), immunity (McDade, Rutherford, Adair, & Kuzawa, 2010), and reproductive biology (Kuzawa, McDade, Adair, & Lee, 2010), also suggest a capacity to use early life cues to "fine-tune" how systems function and are regulated. By this reasoning then, nutrition, hormones, and other gestational stimuli experienced by the developing fetus might convey *information* about local ecological conditions, thereby allowing the fetus to adjust priorities in anticipation of these future experiences (Bateson, 2001).

One challenge to the idea of long-term anticipatory adaptation of this sort comes from the fact that humans have long life spans. Because we typically live many decades, any conditions that we experience during a few months of early development, such as gestation or early infancy, may not serve as reliable cues of environments likely to be experienced in adult life (Kuzawa, 2005; Wells, 2003). I have argued that it is precisely the brief and early timing of many of the body's periods of heightened developmental sensitivity that paradoxically could *help* the developing organism overcome the challenge of reliably predicting future conditions (Kuzawa & Quinn, 2009; Kuzawa & Thayer, 2011). The mother's physiology could buffer the fetus against the day-to-day, month-to-month, or seasonal fluctuations in the environment, while passing along more integrative information on average conditions experienced by the mother or grandmother during recent decades. Because the mother's biology and behavior have been modified by her lifetime

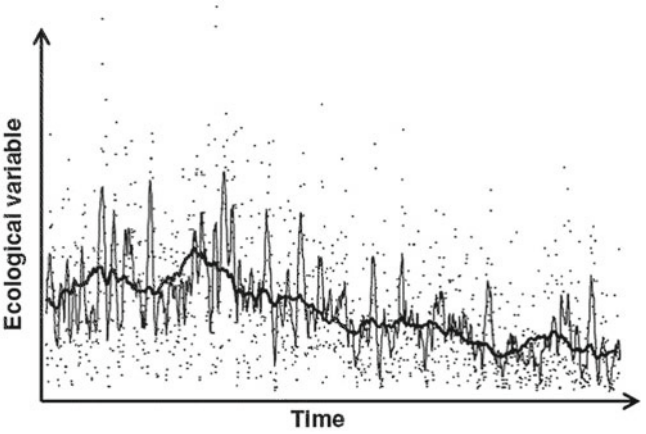

Fig. 2.2 The value of averaging as a way to identify a trend in a noisy signal. The *dots* represent the fluctuating availability of a hypothetical ecological resource, such as nutrition. The two lines are running averages calculated across 10 time units (*thin line*) and 100 time units (*dark line*). As the window of averaging increases, an underlying long-term trend is uncovered. Intergenerational influences of maternal and grandmaternal nutritional history on fetal nutrition may help achieve a similar feat (from Kuzawa & Quinn, 2009, with permission from *Annual Reviews*)

of experiences (including her own gestational environment, and thus the grandmother's experience), the nutrients, hormones, and other resources that she transfers to the fetus in utero or to her infant via breast milk could correlate with the mother's and grandmother's average experiences more than to the vagaries of what she happens to experience during any week or month of gestation itself. If maternal physiology buffered out transient, short-term nutritional variations, while conveying more reliable average information, this could provide a more useful basis for adjusting characteristics such as growth rate, body composition, or nutritional requirements as environmental conditions gradually shift across decades or several generations (Kuzawa, 2005; Kuzawa & Quinn, 2009).

Perhaps the best evidence for such a maternal capacity to convey average, rather than transient, ecological information to the fetus comes from the often negligible effects of a mother's nutrition on the birth weight of her baby (Kuzawa, 2005), as discussed above. Studies generally find that birth weights are lighter in populations in which nutrition has been marginal for multiple generations. Despite this evidence for environmental influence on fetal growth rate and birth size, supplementing the diets of pregnant women generally has minimal effects on the birth weight of offspring. Thus, it appears that maternal *long-term* history in an environment may be an important influence on the resources contributing to offspring growth (and secondarily, on the many traits and functions that are sensitive to and "downstream" of prenatal experience). However, fluctuations in the mother's intake during pregnancy itself—as would result from dietary supplements—have only modest effects on the nutrients that the fetus receives in utero.

This *phenotypic inertia*—reflecting the lingering biological but nongenetic effects of the mother's average experiences in the past—could allow the fetus to track those dynamic features of environments that are relatively stable on the timescale of decades or even several generations (see Kuzawa, 2005, 2008). In this way, the mother's body could pass along biological "memories," allowing developmental adjustments to be made in anticipation of conditions that have dominated in recent generations and, thus, which serve as a best guess of conditions likely to be experienced in the near future (Fig. 2.2).

Speculations on Why Some Nutritional Interventions Fail

Above, I argued that developing organisms will tend to buffer or ignore transient features of their environments but are sensitive to environmental features that are stable over long time periods (i.e., a generation or more). I close by considering the implications of this idea for two research and policy domains: the biomedical use of animal experiments as models for developmental processes in humans and the design of human interventions aimed at improving long-term health.

First, what is "transient" or "stable" for individual organisms is inherently relative. We should expect that adaptively relevant timescales of environmental change for a human will be markedly different from those of a rat or other short-lived species. Thus, humans should be expected to "ignore" environmental changes of the sort to which mice or rats will modify their life trajectory in response. After all, if a rat is born during a stressful season or year, those conditions are likely to predominate during its entire brief life. Because humans will live through hundreds of seasons, what would be an environmental "signal" to a mouse would often simply be "noise" to be buffered out and ignored by a developing human.

This perspective may help explain why individuals in the World War II Dutch Famine Winter cohort, who were exposed in utero to caloric restriction of a similar magnitude as in many animal model experiments, experienced comparably small changes in birth weight followed by more modest long-term effects on metabolism and adult cardiovascular disease risk factors (see Kuzawa & Thayer, 2011). Because biological processes and responses scale with traits like body size and life span, mice or rats can provide a precedent for how programming might work mechanistically in humans but clearly are poor guides to the magnitude of human health impacts of early stressors, which are markedly different across species.

The differences in findings in humans and animals suggest that maternal nutritional stress will generally have reduced negative impacts on offspring biology in humans compared to other species used as experimental models in biomedical research. By the same token, it seems likely that short-term *improvements*, as reflected in the typical design of many interventions, may similarly reap comparably modest long-term benefits. Pregnancy supplementation trials often modify the nutritional ecology of a mother for a period of weeks or months, which represents a change that a human body should be expected to simply buffer. It would not be

advantageous for a human to adjust its strategy *for life* based upon such an abrupt, and therefore likely transient, change in its early experience.

David Barker (Chap. 1) points out that the flow of nutrients that a fetus receives in utero reflects 100 years of matrilineal nutritional experience, which is in general agreement with the evolutionary argument and model outlined here. In an equitable world, knowledge of such long-term effects would motivate the development of policies aimed at improving nutrition of entire populations and optimizing population health over the course of two or three human generations. In the absence of such policies, the principles of biological adaptation and of timescale may lead us to effective shortcuts. To "convince" the biology of future generations to modify developmental trajectories for life—that is, to improve long-term health via interventions—we must aim to design interventions that sustain, or at least mimic, longer timescale environmental change.

How might this goal be achieved? One possibility, which has gained some empirical support (see Kuzawa & Thayer, 2011), is that interventions at several ages will have synergistic effects on offspring outcomes. For instance, the flow of nutrients and hormones across the placenta and in breast milk both appear to influence metabolism, growth, and long-term biological settings in offspring. Might supplementing the mother's diet during pregnancy *and* lactation, sending a signal of consistently improved conditions, have effects that are greater than the sum of their independent parts? Similarly, might the biological effects of favorable nutritional signals conveyed to breast-fed infants via breast milk be enhanced if the mother received supplements during or prior to pregnancy, rather than during lactation alone?

At a minimum, it would seem prudent to envision that the goal of our interventions is not simply to alleviate stress in the present but to find creative ways to mimic cues of sustained environmental change in the recent past (Kuzawa & Thayer, 2011). For we should only expect developing offspring of a species such as ours, with our extended life spans, to aim for a higher developmental trajectory, and to reset biological strategy for life, if they receive signals indicating that nutritional conditions have not only changed for the better but that these improvements are likely here to stay.

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Chapter 3
Developmental Origins of Disease and Health Disparities: Limitations and Future Directions

Steven A. Haas

Abstract The developmental origins of disease (DOD) model seeks to replace the traditional epidemiologic risk-factor model with a perspective focused on the long-term consequences of nutritional resource scarcity during early life and the developmental trade-offs it creates. Research into the developmental origins of adult chronic disease has progressed substantially in recent years. However, a number of critical issues remain unexplored and underdeveloped. This chapter discusses some of those issues while providing an interdisciplinary population health perspective on the future of DOD research, with particular attention paid to health disparities and changes that are needed in health policy and intervention. I argue for research to provide greater specificity of the exposures of interest, a more comprehensive understanding of critical periods, and better theoretical and empirical integration of the developmental origins perspective within the life course and across multiple intergenerational processes.

During roughly a 200-year period between the mid-eighteenth and mid-twentieth centuries, in what are the present Western industrialized areas of the world, there occurred a fundamental transformation in the structure of human disease and mortality (Omran, 1971). This transformation consisted of a long-term shift by which diseases of an infectious nature (i.e., influenza and tuberculosis) were replaced by degenerative and chronic diseases (i.e., cancer, cardiovascular disease, and diabetes) as the major causes of morbidity and mortality. Unlike communicable diseases whose etiology can almost always be connected with a specific pathogenic vector, the etiology of chronic diseases is more complex and multifactorial. While a few chronic diseases can be traced back to one or two pathogenic factors, most chronic diseases are thought to be the result of long-term exposure to a variety of risk factors.

S.A. Haas, Ph.D. (✉)
Department of Sociology and Crime, Law & Justice, Penn State University, University Park, PA, USA
e-mail: sah49@psu.edu

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Modern epidemiology has been successful in identifying some of the more salient risk factors associated with cancer, cardiovascular disease (CVD), and diabetes, including smoking, excessive alcohol consumption, sedentary lifestyle, high-saturated fat/low-fiber diet, and stress. Though these risk factors are no doubt important determinants of chronic disease, they explain only a small fraction of the variability in human morbidity and mortality (Marmot, Rose, Shipley, & Hamilton, 1978). This has led to the search for an alternative to the traditional epidemiologic risk-factor model of chronic disease. One such alternative, the developmental origins of disease model (DOD), gives primacy to developmental processes in the first 1,000 days after conception in shaping susceptibility to disease in later life (Barker, Chap. 1).

The developmental origins of disease model has been the subject of considerable controversy and debate (Gillman, 2002; Joseph & Kramer, 1996; Kuh & Ben-Schlomo, 1997; Rasmussen, 2001). The basic idea behind DOD is that poor maternal nutrition at various critical periods during the development of the fetus has long-term impacts on the risk of CVD, diabetes, and other chronic diseases. Specifically, poor nutrition leads to fetal adaptations, which while channeling resources to the most critical developmental goals, "program" later-life disease by altering the structure and function of important tissues. Thus the DOD model seeks to replace the traditional epidemiologic risk-factor model with a developmental perspective focused on nutritional resource scarcity and the developmental trade-offs it creates (Barker, Chap. 1). While research into the developmental origins of adult chronic disease has progressed substantially in recent years, a number of critical issues remain unexplored and underdeveloped. In this chapter, I discuss some of these issues while providing an interdisciplinary perspective on where DOD research goes from here, with particular attention paid to health disparities and changes that are needed to health policy and intervention.

Greater Specificity of the Exposure

Perhaps the most important limitation of the current DOD literature from a policy-making/intervention perspective is the lack of specificity regarding the exposure(s) of interest and the complex multidimensional biosocial pathways through which they work. Much of the early work on DOD examined the impact of low birth weight on later-life risk of chronic disease and mortality and emphasized intrauterine growth retardation and inadequate maternal nutrition during various periods of gestation (Barker, 1994). In addition, previous discussions of DOD have noted that birth weight itself is a rather poor measure of prenatal exposure (Gillman, 2002). More recent work has moved beyond the early emphasis on the bottom of the birth weight distribution and has instead argued for a graded relationship with outcomes varying across the normal range of human birth weight and maternal nutrition (Barker, Chap. 1). This suggests that the issue transcends global nutritional intake during pregnancy and includes processes that shape maternal health and nutritional

endowments at conception, as well as the transfer of nutrients from mother to fetus during gestation and early infancy via breast milk. The transfer of nutrients from mother to fetus is determined by the shape and size of the placenta (Burton, Barker, Moffett, & Thornburg, 2010). Variation in the placenta is associated with risk of adult chronic disease. For example, placental size and shape have been associated with adult cardiovascular disease (Barker et al., 2010; Barker, Thornburg, Osmond, Kajantie, & Eriksson, 2010; Eriksson, Kajantie, Thornburg, Osmond, & Barker, 2011). However, little is known about the determinants of variability in placental size and shape. Similarly, in regard to maternal nutritional factors, other than the impact of maternal pre-pregnancy weight and folic acid intake, pregnancy weight gain, and gestational diabetes, there is scant information to provide useful policy recommendations about what is most deleterious or beneficial. Outside of the cases of obviously severely undernourished or overweight mothers, or of specific nutrient deficiencies, there is almost no way in which to assess the quality of maternal nutritional endowment to which the newly fertilized embryo will be exposed.

DOD also stresses the importance of compensatory growth. Compensatory growth represents an additional adaptive capacity by which the fetus undergoes accelerated growth following a period of nutritional stress. Though it may improve survival in the near-term, compensatory growth requires a shift of resources away from development in other tissues/organs. Thus, the picture that has emerged is that complex patterns of prenatal nutritional deprivation, adaptation, and growth combined with environmental conditions that spur compensatory growth in early childhood are at the heart of DOD (Barker, Chap. 1). However, the ways in which prenatal growth retardation, followed by compensatory growth, places individuals at risk for various adult chronic diseases are not well characterized. Therefore, even a post hoc examination of individual growth trajectories provides only a limited understanding of the processes involved. In addition, research has begun to show that prenatal growth trajectories vary by sex (Barker, Kajantie, Osmond, Thornburg, & Eriksson, 2011). In other words, at present, the theory can't tell us with much specificity what a healthy soon-to-conceive mother should look like and can provide only a limited picture of what an unhealthy inter-uterine/infant growth trajectory looks like.

Also contributing to the lack of specificity of exposure is the fact that much of what is known about the biological mechanisms underlying the developmental origins of disease is based on animal models (Harding, 2001; Rasmussen, 2001). One mechanism involves the epigenetic processes that regulate gene expression and which may induce disease phenotypes. For example, research in animal models has shown that a protein-restricted diet alters the expression of a number of genes in the lungs, liver, kidneys, and brain, including those associated with macronutrient metabolism, the hypothalamic–pituitary–adrenal axis, and cardiovascular processes (Burdge & Lillycrop, 2010). Human studies using data from the offspring of mothers impacted by the Dutch Hunger Winter in 1944 have demonstrated that in utero exposure to the famine can induce significant differences in the level of DNA methylation relative to unexposed siblings (Heijmans et al., 2008). Similarly, preconception exposure of mothers to famine altered the methylation patterns of genes involved in growth and metabolic disorders (Tobi et al., 2009). Thus, epigenetic

programming is thought to have important impacts on the subsequent development of chronic disease. However, epigenetic research on humans is still in its infancy, and its contribution to explaining adult disease is still to be determined.

Racial and socioeconomic disparities in birth outcomes help illustrate how the DOD's lack of specificity of exposure limits the development of effective policy interventions. Relative to non-Hispanic whites, African-American mothers are significantly more likely to give birth to infants who are preterm and low weight for gestational age (Singh & Yu, 1995). Such disparities are further complicated by large race-ethnic disparities in rates of poverty, socioeconomic environment, family structure, maternal health and nutritional history, age at delivery, and the interactions between these factors. For example, among African-American women, the odds of having a low birth weight or very low birth weight infant rises dramatically with maternal age such that relative to mothers giving birth at age 15, those who are 20 are about one-third more likely to have a very low birth weight baby. Worse still, by age 30, the risk of giving birth to a low birth weight baby is more than twice that for young mothers. For mothers aged 35, the risk of very low birth weight is nearly 3 times higher than it is for young mothers. In addition, the rate at which the risk of poor birth outcomes increases with maternal age varies by maternal SES, with older low SES African-American mothers most at risk (Geronimus, 1996).

The DOD model does not provide much insight into what may be behind such patterns. What is clear is that African-American women are having experiences over the course of their childbearing years that dramatically impact the quality of their birth outcomes, the long-term health of their children, and given that women's ova are developed when they are in utero, the health of their grandchildren as well. Geronimus (1996) has argued that such patterns reflect a process of "weathering" by which African-American women's greater exposure to various social and environmental insults, including socioeconomic deprivation, chronic stress, and racial discrimination, accumulates in the body and reduces their capacity to gestate healthy babies. Much of that exposure happens long after the developmental period emphasized by the DOD. It's unclear what kinds of policy recommendations the DOD model could generate that would eliminate such disparities. This is not to say that the DOD is not useful. Due to the intergenerational processes involved, the developmental origins model may actually be a key to understanding how the weathering process ultimately comes to be embodied in the second and even third generation. If policy interventions are to be designed to eradicate the patterns described above, greater understanding of the DOD exposures and how they interact with other processes over the life course, such as weathering, is necessary.

Better Understanding of Critical Periods

Central to the DOD perspective is the notion of *periods of developmental plasticity*, critical periods of development in utero and in the neonatal period, during which the developing fetus or infant reacts to environmental signals and adapts to meet specific developmental objectives (Gluckman & Hanson, 2004). Gluckman and Hanson

(2004, 2005) propose a generalized evolutionary mechanism to explain DOD. They argue that environmental conditions in utero and the early neonatal period, such as nutritional constraints, act as signals to the fetus of the resource environment it will face in the future. Based on these environmental cues, the fetus undergoes a predictive adaptive response (PAR) to prepare the fetus for the resource environment it should expect (Gluckman & Hanson, 2005). The key argument is that nutritional scarcity during these periods of gestation requires the fetus to make trade-offs between various developmental objectives (e.g., beta cell development in the pancreas, overall growth, brain development). The epigenetic processes discussed above are hypothesized to be the central mechanism. However, the environmental cues that set anatomical and physiological parameters in utero and early infancy may reflect short-term resource fluctuations more than the long-term circumstances that the organism will face. This can result in phenotypic mismatch between the environment that the organism has been epigenetically programmed to expect and the one it ultimately finds itself in (Lucas, Fewtrel, & Cole, 1999; Ozanne & Hales, 2004). That mismatch is at the core of the DOD perspective.

However, from both a theoretical and empirical standpoint, research in this area would benefit greatly from a better understanding of periods of developmental plasticity. A number of important issues remain unresolved or underdeveloped. What constitutes the period of plasticity? What is the timing of onset and duration? Are there multiple discrete or overlapping periods of adaptation? For example, much of the literature focuses on anatomical and physiological adaptations within the first 1,000 days postconception. However, what about adolescence or menopause? Each represents a period of substantial physical change and development as well as emergent social roles and transitions. In addition, very little is known about how the timing and duration of critical periods may vary across physiologic/anatomical systems. For example, are some systems more vulnerable/malleable than others? Similarly, how do the multiple biological pathways active during these critical periods (e.g., stress, epigenetic methylation, nutrition, and exogenous infection) have their impact? How does what is happening in one system or process impact other systems and processes? All of these questions about the complexities of critical periods are important, and answering them is critical to evaluating the utility of the model in explaining human disease and the ultimate goal of developing effective interventions to improve the health of populations.

Better Integration with the Life Course

As discussed above, central to the DOD model is the concept of phenotype-environment mismatch. The postnatal social and physical environment is hypothesized to play a critical role in modulating the impact of induced phenotype. However, despite the importance given by the theory to the subsequent environment, much of the work in the DOD tradition, especially that of Barker and colleagues, has emphasized the very early period of prenatal and neonatal life at the expense of the rest of the life course. It is often argued that what is most important is the first 1,000 days of life,

which set in motion a cascade of events that manifest decades later. A common critique of this thinking is that it has tended to ignore the rest of the life course (Kuh & Ben-Schlomo, 1997). Part of the problem is that research in the DOD tradition has tended to conceptualize the postnatal life course and the social and physical environment experienced after childhood as rather fixed and exogenous. In other words, the social and physical environment are what they are, and the organism has either adopted phenotypes in the first 1,000 days that are conducive to that environment or not. However, research on the social determinants of health would suggest a much more complex picture.

The weathering example, discussed above, highlights the limitation of DOD's inability to fully incorporate other life course processes. Similarly, the paradoxical patterns of race-ethnic differences in health that emerge after infancy and early childhood are also illustrative. For example, Mexican-origin populations in the USA have a birth weight distribution that is conducive to healthy birth outcomes. Only 3.9% of babies of foreign-born Mexican mothers have low birth weight, compared to 5% for US-born Mexicans and 8% for non-Hispanic whites. Conversely, non-Hispanic blacks have a much less advantageous birth weight distribution with 13.1% of babies born to black mothers weighing <2,500 g (Hamilton, Teitler, & Reichman, 2011). However, by age 10, the body mass distribution of Mexican-origin children has shifted dramatically to one that resembles that of black children and one conducive to the development of diabetes and CVD in adulthood (Hamilton et al., 2011). This shift begins as early as age 3 (Kimbro, Brooks-Gunn, & McLanahan, 2007). Something in the lived experience of Mexican-origin and African-American mothers leads them to have very different birth outcomes. Yet, within a few short years, the health profiles of their offspring have converged in ways that have important implications for race-ethnic disparities of adult chronic disease. It is unclear how the DOD perspective and its fairly narrow focus on the first 1,000 days of life can explain this shift. What differences over their preconceptional life course lead Mexican-origin and African-American mothers to have such different birth outcomes yet very quickly lead their children toward the shared trajectories of obesity, diabetes, and cardiovascular disease?

Another example involves the complex interrelationship between health and socioeconomic status over the life course. Health and socioeconomic status interact throughout the life course influencing each other through multiple complex feedback processes. It is clear, for example, that early life health insults have damaging effects on both subsequent health (Blackwell, Hayward, & Crimmins, 2001; Haas, 2007) and socioeconomic attainment (Case, Fertig, & Paxson, 2005; Haas, 2006) and that some of the impact on later-life health operates through selection into lower socioeconomic strata. Similar feedback processes occur between health and other aspects of the social environment such as social network ties (Haas, Schaefer, & Kornienko, 2010; Schaefer, Kornienko, & Fox, 2011).

What these examples illustrate is that the life course matters. The rush to constrain the origins of human disease to the first 1,000 days of life is, at best, misguided. While the parameters that shape health trajectories over the life course may be

shaped in early life, they are not immutable. Rather, those trajectories are very much contingent upon the physical and social environment that the individual experiences. More research is needed to understand how early life health shapes the social context of individuals and how early life influences on adult health are both moderated by subsequent health-related inputs and compounded by further insults.

Better Synthesis of Intergenerational Processes

More than a century ago, Beeton and Pearson (1899, 1901) reported a positive correlation between the life spans of parents and their offspring. Seven decades later, researchers again took up the question of intergenerational transmission of longevity, this time to determine the relative importance of genetic and environmental factors (Herskind, McGue, & Holm, 1996; Vaupel, 1988; Wyshak, 1978). Similarly, over the last 50 years, researchers have explored the pathways underlying the intergenerational reproduction of social class (Blau & Duncan, 1967; Lareau, 2003; Sewell, Haller, & Portes, 1969). There would appear to be important theoretical and empirical connections between these bodies of work. However, until recently, researchers in each of these areas have only engaged each other sporadically.

Human capital theory has long explored the role of health endowments at birth and health insults over the life course as an important determinant of economic outcomes including labor force participation and earnings (Grossman, 1972; Luft, 1975; Mushkin, 1962). In the past decade, there has been greater attention paid to the complex and dynamic connections between health and socioeconomic attainment over the life course and what role each plays in the intergenerational transmission of the other. This line of work has shown that early life health endowments are transmitted across generations (Conley & Bennett, 2000; Currie & Moretti, 2005); early life health plays an important role in both subsequent health (Haas, 2007) and socioeconomic attainment (Case et al., 2005; Haas & Fosse, 2008; Haas, Glymour, & Berkman, 2011); and early life health may be an important mechanism in the intergenerational reproduction of social class (Haas, 2006; Palloni, 2006). It also suggests that SES can act as an important mechanism through which families transmit longevity across generations.

Processes described by the DOD model may help provide a biological mechanism for understanding intergenerational low birth weight and other health outcomes. However, more theoretical and empirical research is needed to better explicate (1) the processes and resource flow that intrinsically link the social/economic life course and the biological/health life course and (2) how both health and socioeconomic status are passed in tandem from one generation to the next. As one cannot understand any species outside of its ecological niche, likewise, a realistic or useful model of human health cannot exist without understanding the social and economic systems within which it is embedded.

Summary

This chapter describes important limitations of the developmental origins of disease model with regard to its ability to inform our understanding of health disparities and provide a useful guide for developing public policy and interventions. It has also suggested some new directions for DOD research in order to address those limitations. Much has been written about the developmental origins of disease perspective, and it has been the subject of considerable scientific debate and controversy. Much of what I have argued above has been covered in one of the many other reviews of the DOD literature (Gillman, 2002; Joseph & Kramer, 1996; Kuh & Ben-Schlomo, 1997; Rasmussen, 2001). Time will tell if the theory will achieve its goal of supplanting the traditional "destructive" model of chronic disease. That will depend largely on whether it addresses concerns raised by critics and whether it provides a useful framework for interventions and policies aimed at improving health and reducing health disparities.

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Chapter 4
The Effects of Early Severe Psychosocial Deprivation on Children's Cognitive and Social Development: Lessons from the Bucharest Early Intervention Project

Nathan A. Fox, Charles A. Nelson III, and Charles H. Zeanah Jr

Abstract Developmental psychologists have long been interested in understanding the effects of early experience on brain and behavior. Much of the research in this area has been with rodents and nonhuman primates. There are, however, situations in which infants and young children are exposed to severe psychosocial deprivation that are amenable to the study of early experience. Infants raised in institutions represent such a group. Indeed, the institutionalization of infants and young children is a worldwide problem, and the study of the effects of this early experience on child development, as well as potential interventions for children in these situations, is of public health importance. This chapter presents an overview of one such study: the Bucharest Early Intervention Project (BEIP). One hundred thirty-six infants and young children living in institutions in Romania were randomized either to be placed in foster family care contexts (foster care group) or to remain in the institutions in which they lived (care as usual group). These children were assessed at 30, 42, 54, and 96 months of age across a broad set of domains. This chapter presents data on two such domains: IQ and attachment. Findings from the BEIP suggest that institutionalization is detrimental to the cognitive and social development of children and that infants and young children living in such situations should be placed as early as possible into family contexts.

N.A. Fox, Ph.D. (✉)
Department of Human Development, University of Maryland
College Park, MD, USA
e-mail: fox@umd.edu

C.A. Nelson III, Ph.D.
Department of Pediatrics, Boston Children's Hospital/Harvard Medical School,
Boston, MA, USA

C.H. Zeanah Jr, M.D.
Department of Psychiatry and Behavioral Sciences, Tulane University,
New Orleans, LA, USA

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Developmental psychologists have, for many years, argued that experiences early in life can have a profound effect upon the course of subsequent development. Studies of human infants demonstrate that learning takes place from a very early age and sets the course for trajectories of either adaptive or maladaptive behavior. Recent evidence suggests that there are certain periods during early development when experiences have a more significant effect than others. These periods, called sensitive periods, are thought of as windows of opportunity during which certain types of experience have a foundational effect upon the development of skills or competencies (Fox, Levitt, & Nelson, 2010; Zeanah, Gunnar, McCall, Kreppner, & Fox, 2011).

Progress in understanding the effects of early experience on development has been facilitated by advances in neuroscience that describe the pattern of brain development during the early months and years of life and the role that experience has in shaping development. A number of neuroscientists (Huttenlocher & Dabholkar, 1997; Granger, Tekaia, Le Sourd, Rakic, & Bourgeois, 1995) have illustrated changes in synaptic density, increases in neural connections, and the subsequent pruning or decreases in synaptic number that occurred during the postnatal period. This blooming and pruning occurs in different brain areas at different times, particularly during postnatal development. These changes occur early in postnatal life in sensory and perceptual regions, while they occur later in areas of the brain involved in higher cognition.

The data supporting the effects of early experience on development have primarily been from rodents and nonhuman primates. Because of obvious ethical considerations, it is not possible to randomly assign infants to conditions of significant deprivation, and so research has relied on situations, medical and environmental, that provide the contexts in which the effects of early experience can be examined. For example, Maurer, Lewis, Brent, and Levin (1999) examined the effects of visual deprivation in infants born with bilateral cataracts. Maurer and colleagues followed infants of a range of age at which they had surgery to remove those cataracts in order to determine the presence and extent of the effects of early deprivation. Similarly, Neville and Bavellier (1998) studied infants born deaf with regard to their language development. Schorr, Fox, van Wassenhove and Knudsen (2005) examined the effects of age of acquiring a cochlear implant in young children born deaf on their abilities to integrate auditory and visual information. In all of these cases, identifying populations of children who are deprived because of a medical or genetic condition of early experience provides researchers with a window into the effects of deprivation and timing of remediation on behavioral outcomes. Examination of the effects of more general psychosocial deprivation and neglect in human infants has until recently been missing from the research field. However, children who are institutionalized in early infancy represent a “natural” condition in which examination of the effects of early experience can be studied.

Institutionalization of Children

UNICEF estimates that as many as 8,000,000 children may be currently living in institutions worldwide. Children are placed in institutions for various reasons including poverty of the family, age of the mother, societal proscriptions about abortions, being orphaned as a result of natural disasters (e.g., the earthquake in Haiti), or parental illness and death (e.g., the AIDS epidemic in Africa). Often, countries are faced with an immediate problem of what to do with orphaned and abandoned children. Countries with few resources or those with little history or cultural frame for domestic adoptions often find that establishing institutions may be the path of least resistance in dealing with these problems. Institutions for young children, however, are not simply the product of twentieth-century poverty or natural or man-made disasters (Walker et al., 2011).

There is a long history of placement of children in institutions because of poverty and abandonment. Much more recently, in the twentieth century and particularly after World War II, there was a movement in Western psychology and psychiatry to identify these environments as having a deleterious effect upon the cognitive and social development of children. Bowlby (1952) and Goldfarb (1945) were among those who highlighted the negative effects of institutions on children's development. As child psychology began to consider environmental influences on child development in 1950s, a renewed interest in the effects of early experience gained prominence. Studies by Dennis and Najarian (1957) and by Tizard and Rees (1974) pointed to the negative consequences of institutionalization in infancy. As a result of the pioneering work of a child psychiatrist, Renee Spitz, the effects of early separation of the young infant from the caregiver were brought to public attention by showing films of children living in institutions (Spitz, 1945).

The research presented in this chapter is drawn from the Bucharest Early Intervention Project. That project was designed to examine the effects of early psychosocial deprivation on social and cognitive development among young children living in Romanian institutions. The project was also the first randomized controlled trial to examine the efficacy of foster care in remediating the negative effects of early institutionalization. A large cohort of infants and young children, living in institutions in Bucharest Romania, were screened for genetic anomalies and gross sensory or physical handicaps. A final sample of 136 infants and young children were selected and assessed while they lived in their institutions. Subsequent to that, the sample was randomized to either remain in the institution (care as usual or CAUG) or be placed into a foster care family home (foster care group or FCG). All the children were assessed at 42, 54, and 96 months of age across a wide range of domains (see Zeanah et al., 2003). In this chapter, we review findings regarding IQ and attachment. For those interested in the research papers from the study, a website with downloadable papers is available (http://www.beip.org).

Context of the Study

In 1966, Nicolai Ceausescu, then the leader of Romania, decided that he could increase the status and power of his country if he could increase its industrial productivity. Ceausescu believed the way to accomplish this goal was to increase the population of workers. He outlawed all contraception, forbid abortions, and taxed families who produced fewer than five children. The birthrate in Romania increased dramatically. Families were forced to have children they could not afford to care for, and poverty became even more widespread. As child abandonment increased, Ceausescu expanded a network of institutions into which families placed infants for whom they could not afford to care. Rather than stigmatizing such families, abandonment was implicitly encouraged under communism because the state was believed to be more effective in raising productive children than were poor families. As a result, large numbers of children were placed very early in their lives in institutions. Importantly, the majority of these children were not orphaned in the conventional sense; rather, they were abandoned and were therefore "social orphans." Moreover, many parents did not give up their legal rights to the child and some even visited their children in the institution at periodic intervals.

In 1989, Ceausescu was overthrown and then executed in a coup. The Western media gained access to the country and revealed the horrific conditions under which many children were being raised. Life in an institution was characterized by deprivation and regimentation. Young children spent their time languishing in cribs and, in general, were not stimulated in any conventional sense of the word. There was little access to caregivers (the ratio of caregivers to children might be 1:15), and the quality of caregiving was poor in most cases (e.g., most caregivers were poorly educated and had no training in child development). Because of the unfavorable caregiver–child ratio, there was a high level of regimentation (e.g., children went to the bathroom at the same time, went to bed at the same time, were dressed similarly, and were fed on schedule in an impersonal manner).

Not surprisingly, as families in the West learned of the plight of these children and began to adopt them in large numbers, they became quickly overwhelmed with the special needs of these children. Many were very small for their age and had significant cognitive and language delays, behavior problems (particularly hyperactivity), and, perhaps most noticeable, problems in forming and maintaining relationships with significant others.

The Bucharest Early Intervention Project

The BEIP began in 1999. It was initiated by three scientists, Charles Nelson (currently at Children's Hospital Boston and Harvard), Charles Zeanah (Tulane University), and Nathan Fox (University of Maryland). The project was an outgrowth of discussions among these three investigators who were also members of a research network (Early Experience and Brain Development) that was funded by

the Macarthur Foundation. Indeed, the Macarthur Foundation provided the funding for first 5 years of the project. The project was located in Bucharest Romania and recruited infants who were then living in institutions in that city. The study began with comprehensive baseline assessments of 136 institutionalized children and their caregiving environments prior to randomization. These children were selected from a larger group of children (>180) who had been screened for obvious neurological or genetic anomalies and had been found to have no obvious chromosomal or major handicapping condition (e.g., cerebral palsy or fetal alcohol syndrome).

Once the baseline assessment was completed, half the children in the study were randomly assigned either to foster care created specifically for the study or to care as usual, meaning continued institutional care. The average age at foster care placement was 22 months (range = 6–31 months). Following randomization, all children were seen for follow-up assessments at 30, 42, 54, and 96 months. The development of children in foster care was compared to the development of the children randomized to remain in the institutions and to a group of typically developing Romanian children, recruited from pediatric clinics. (They had never been institutionalized; $N = 72$.) Children were assessed comprehensively with measures that included physical growth, cognitive function, social–emotional development, attachment, problem behaviors and psychiatric symptomatology, language development, caregiving environment, genetics, and brain functioning. In this chapter we review findings from two areas of study: cognitive development (IQ) and attachment. In all instances, we examined both the effects of early experience (intervention effects) and sensitive periods (timing of intervention).

In analyzing data, we employed an intent-to-treat approach (Montori and Guyatt 2001). Thus, even though a child may have left his or her initial group assignment for any of the reasons described, we analyzed results as if the child were still in his/her original group. For example, if a child originally assigned to remain in the institution was placed in government foster care or if a child originally assigned to our intervention/foster care group was reunited with his/her biological family, we still considered each of these children as members of their original groups. Examining the group differences this way actually biases the effects away from our hypotheses.

Cognitive Development (IQ)

At baseline 30 and 42 months of age, the Bayley Scales of Infant Development were administered. When children were 54 months of age, the Wechsler Preschool Primary Scales of Intelligence (WPPSI-R) was administered. Finally, at 96 months of age (8 years), the Wechsler Intelligence Scale for Children or WISC-IV (Wechsler, 2003) was given. The data from the baseline assessment showed that children reared in institutions showed greatly reduced intellectual performance compared to children raised with their biological families. At baseline, the mean Bayley Mental Developmental Index (MDI) scores were 66 for children in the institutionalized group and 103 for children in the community group (Smyke et al., 2007).

At follow-up, we addressed two questions: Was the intervention effective and was there an effect of age at placement? In terms of the first question, as predicted, children in the foster care group experienced significant gains in cognitive function compared to children in the institutional group. For example, at 42 months, the mean developmental quotient (DQ) was 77 for children in the institutionalized group (IG), compared to 86 for children in the foster care group (FCG) and 103 for children in the never institutionalized group (NIG). At 54 months, mean intelligence quotient (IQ) was 73 for children in the IG, compared to 81 for children in the FCG and 109 for children in the NIG (Nelson et al., 2007).

We also found timing effects with regard to age at placement in foster care. For example, at 42 and 54 months, respectively, the DQ/IQ of children placed in foster care before 18 months was 94/85, those placed between 18 and 24 months 89/87, those placed between 24 and 30 months 80/78, and those placed after 30 months of age 80/72 (for discussion, see Nelson et al., 2007).

The next assessment point was when children were 8 years of age (Fox, Almas, Degnan, Nelson, & Zeanah, 2011). Using the WISC-IV, subdomain scores in verbal comprehension, perceptual reasoning, working memory, and processing speed were attained, and from these a full-scale IQ score was computed. It should be noted that there was a great deal of mobility in the subject populations of both the CAUG and the FCG children over the period of time between the last assessment at age 54 months and the one when children were 96 months of age. Only 15 children of the original 68 remained institutionalized when they were 8 years old, and only 31 of the original 68 children assigned to our foster care intervention remained in these homes when they were 8.

Nevertheless, we analyzed the IQ data with an intent-to-treat approach and found that the children originally assigned to our foster care intervention had higher scores on the verbal subscale compared to children originally assigned to the CAUG. There was also a marginally significant effect ($p = 0.07$) for full-scale IQ with the FCG group scoring higher than the CAUG. There were, however, no timing effects. Interestingly, there were effects of continuity of placement on IQ. Specifically, children who remained in the BEIP foster care homes over the 7- to 8-year period scored higher on full-scale IQ, verbal comprehension, and processing speed compared to children who left those homes (e.g., were reunited with their biological parents) or any of the CAUG (Fox et al., 2011).

The data suggests that even using a conservative intent-to-treat analysis, children assigned to the BEIP intervention exhibited better IQ scores compared to those assigned to CAUG. It also appears that the caregiving environments of the intervention were associated with maintenance of these scores over time.

Attachment

There is substantial literature on the effects of institutionalization on children's socioemotional behavior, all studies completed on children who have been adopted or placed into families after a history of early deprivation (Chisholm, 1998;

O'Connor, Rutter, & The English and Romanian Adoptees Study Team, 2000, 2003). The caregiving environment in institutions is one of routine but also one of instability in caregivers. Caregivers rotated shifts often and had little to no education in child development, nor was there any motivation to form attachment with individual children. Thus, problems with attachment and indiscriminate behavior are expected in this less than typical caregiving environment.

We assessed the quality of the child's relationship with his or her caregivers using the Ainsworth Strange Situation Procedure (SSP) (Ainsworth, Blehar, Waters, & Wall, 1978). This moderately stressful procedure assesses the child's balance between exploratory behavior and proximity-seeking behavior from the parent/caregiver rather than the stranger. Based upon the child's behavior directed towards the stranger and the familiar attachment figure, trained coders classify the child's attachment as secure or one of various forms of insecure. Prior to randomization, all children who were either 11 months or older (both in chronological age and mental age) were tested. Results revealed that the majority of infants did not display behaviors that could be described as reflecting an attachment relationship with the caregiver (Zeanah, Smyke, Koga, Carlson, & The Bucharest Early Intervention Project Core Group, 2005). Indeed, many displayed atypical and unusual responses in this testing context. Over 65% of the institutionalized children had a disorganized attachment with their favorite caregiver, and another 13% had so little attachment behavior that they could not even be classified. In contrast, only 22% of the children living with their families had disorganized attachments to their mothers. More importantly, 100% of the attachments of the community children were rated as being fully developed, but only 3% of the institutionalized children were rated as having fully formed attachments.

Given the profundity of these effects at baseline, we wanted to know how much placing children with families in BEIP foster homes enhanced the quality of their attachments. At 42 months, the preschool version of the Strange Situation Procedure was administered and coded using a preschool coding scheme. All children were seen with their primary caregivers, mothers, foster mothers, or institutional caregivers (Smyke, Zeanah, Fox, Nelson, & Guthrie, 2010).

In brief, the foster care intervention favorably impacted attachment relationships. At 42 months, nearly 50% of children placed in foster care demonstrated secure attachments as compared to only 18% of children in the institutionalized group. As a point of comparison, of children living with their parents in the community, ~65% were securely attached.

Summary

In virtually every domain examined in the BEIP, children living in institutions suffer from delays or disorders. Importantly, since by age 96 months only 15 children were still living in an institution and since almost all of the analyses adopted a strict intent-to-treat approach, these findings may actually underestimate the toll early institutionalization takes on the course of a child's development.

As discouraging as the news is regarding the effects of early institutionalization, the foster care program instituted by BEIP was very efficacious in ameliorating many of the disorders and delays that result from early institutionalization. Thus, improvements were found in virtually every domain examined. Two qualifiers to this general pattern of results are necessary. First, in many domains there is a relation between how old the child was when placed in foster care and the degree of recovery; as a rule, the earlier the better, and after ~24 months of age, recovery may be limited. Second, in virtually *no* domain did foster care children obtain the same level of performance as never institutionalized children (NIG). Thus, for example, although 54-month-old children assigned to foster care before 18 months have IQs that are 25+ points higher than those assigned to the institutionalized group and 96-month-old children assigned to foster care have higher full-scale IQ scores than those who were taken out later, these children still test below children who never spent time in an institution.

Overall, our findings emphasize the substantially deleterious effects of deprived early environments and the importance of ameliorating those environments as soon as possible.

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Part II
Role of Family Dynamics in Children's Health

Chapter 5
Family Discord and Child Health: An Emotional Security Formulation

Patrick T. Davies, Melissa L. Sturge-Apple, and Meredith J. Martin

Abstract Although the impact of family discord on children's coping and adjustment difficulties is well established, continued progress in understanding how and why family processes affect children's mental and physical health will require efforts to formulate theoretically guided hypotheses with greater novelty and pinpoint accuracy. The goal of this chapter is to illustrate how an evolutionary framework of children's emotional security in family relationships may increase depth and precision in interpreting the existing findings and serve as a broad heuristic for future research. After elaborating on how children's sense of security in the interparental relationship is distinct in its nature and function from parent–child attachment security, the chapter addresses how each type of security is proposed to be a product of a distinct set of experiences in the family. In turn, each type of security sets in motion developmental cascades that help to account for why insecurity in family relationships is associated with child problems. We next describe the moderating conditions that may serve as sources of heterogeneity in children's pathways to health and illness. In concluding, we outline future directions that may increase precision and novelty in advancing models of family process and child health within an evolutionary framework.

Understanding how family relationships impact children's mental and physical health is of pivotal public concern as family dynamics play a key role in shaping children's developmental trajectories (Repetti, Robles, & Reynolds, 2011;

P.T. Davies, Ph.D. (✉) • M.L. Sturge-Apple, Ph.D. • M.J. Martin, M.A.
Department of Clinical and Social Sciences in Psychology,
University of Rochester, Rochester, NY, USA
e-mail: patrick.davies@rochester.edu

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Repetti, Taylor, & Seeman, 2002). As other contributors to this volume have cogently highlighted (e.g., Reichman & Teitler, Chap. 9; Morrongiello, Chap. 6; Berkman & O'Donnell, Chap. 12), children's mental and physical functioning hinges, in part, on parents' multiple roles as socialization figures, particularly acting as models for the adoption of healthy and risky behaviors, managing child behavior (e.g., monitoring and enforcing guidelines of conduct), and directing resources for achieving specific socialization goals (e.g., injury prevention, promotion of peer relationships). In offering a complementary focus to these important themes, our chapter addresses how the affective quality of family relationships and children's emotional adaptation to characteristics of the family environment may further elucidate pathways between family risk and child health outcomes.

The value of focusing on emotional processes in the intergenerational transmission of risky and healthy behaviors is well established in the family discord literature (Mead, Beauchaine, & Shannon, 2010; Repetti et al., 2002, 2011). Whereas harmonious, cooperative, and warm relationships in the family are associated with healthy forms of mental and physical functioning, greater family discord, as expressed through unresolved anger, aggression, and emotional disengagement in parent–child and interparental relationships, increases children's vulnerability to psychological and physical difficulties (Cummings & Davies, 2010; Repetti et al., 2011). Moreover, differences between children in their exposure to family harmony and discord forecast a remarkable array of outcomes, including peer and romantic relationship quality, sleep patterns, poor cognitive functioning, substance use, and multiple forms of psychopathology. Having documented the multiplicity of relationships between family and child functioning, we are now faced with the new challenge of understanding how and why emotional processes in the family affect children's mental and physical health. Toward this goal, our overarching objective is to offer a process-oriented model for understanding children's pathways of adjustment within the context of multiple risk factors, different domains of affectively processing and responding to stress, and specific forms of mental and physical health outcomes.

To aid in organizing the array of complex findings, this chapter will review the literature on family discord and child health through the lens of an evolutionary reformulation of emotional security theory (EST-R; Davies & Sturge-Apple, 2007; Davies & Woitach, 2008). According to EST-R, children's concerns about emotional security in family relationships elucidate pathways between family characteristics and children's health. In Fig. 5.1, we offer a graphical depiction of four of the primary components of the theory. Following the ordering of the numbered pathways, the first part of the chapter elaborates on our assumption that children develop a sense of security in the context of interparental conflict that is related but distinct in its nature and substance from that experienced in parent–child relationships (see path 1 of Fig. 5.1). In the next section, we posit that each type of security is proposed to be a product of a distinct set of experiences within the family (i.e., path 2). As individual differences in children's security emerge, they are further proposed to set in motion a cascade of psychological and biological processes that ultimately impact children's mental and physical health (i.e., path 3). Therefore, we will

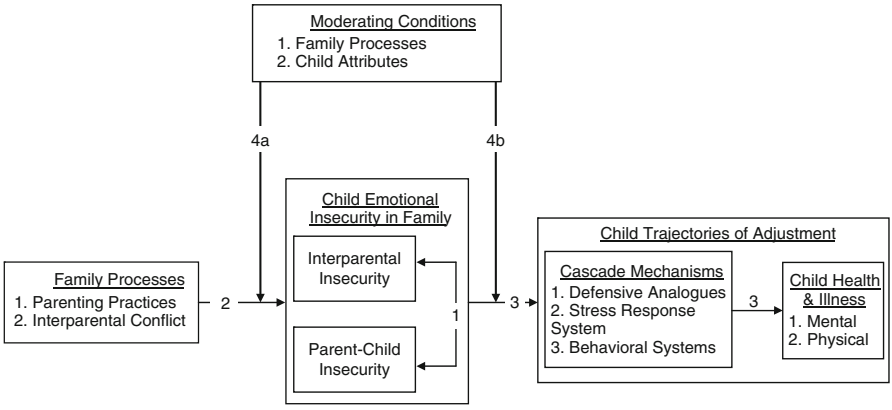

Fig. 5.1 A graphical depiction of the impact of family processes on children's emotional insecurity in the family and their trajectories of adjustment within the reformulated emotional security theory

proceed to describe how the integration of cascade mechanisms of physiological, behavioral, and cognitive systems into emotional security theory may produce mutually informative advances in our understanding of children's response processes and outcomes in risky family environments. However, it is also important to note that children who experience similar patterns of family adversity or insecurity in family relationships often evidence considerable variability in their coping and adjustment. Thus, as depicted by paths 4a and 4b, we will next elaborate on the moderating conditions that may be sources of heterogeneity in children's pathways to health and illness. In concluding the chapter, we address key future implications and directions of EST-R for family models of child health.

The Nature and Composition of the Emotional Security Systems

EST-R specifically postulates that maintaining a sense of protection, safety, and security is a prominent goal for children within the emotion-laden context of the family. A primary thesis is that experiences with aggression, emotional detachment, and unresolved antagonism in the family unit increase children's vulnerability to mental and physical problems by undermining their ability to preserve a sense of security within various family relationships. The significance of emotional security for children's development has a long history in attachment theory (e.g., Bowlby, 1969). Consistent with attachment theory, children's emotional security can be enhanced or undermined by the quality of their parent–child relationships. Thus, the erosion of children's confidence in their parents as sources of protection and support within the attachment relationship accounts for a notable part of the deleterious

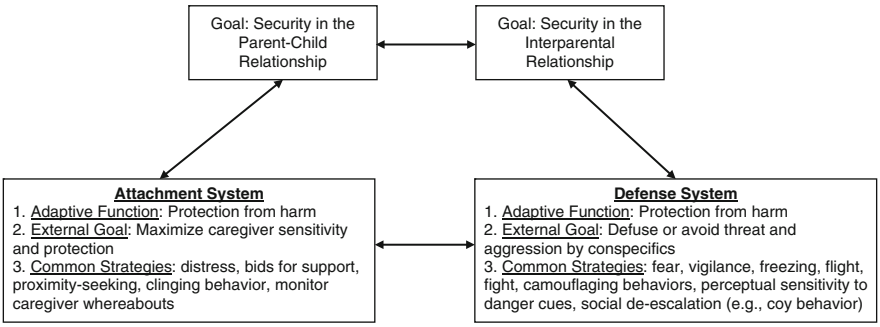

Fig. 5.2 A visual representation of the two ethological modules underlying children's sense of emotional security in the interparental and parent–child relationships

effects of parenting difficulties (e.g., unresponsiveness, intrusiveness, low warmth) on children's psychological adjustment. Although both theories share the assumption that differences in children's abilities to utilize the parent–child relationship as a source of security have important implications for their long-term health, EST-R departs from traditional attachment theory in arguing that *the maintenance of security is a salient goal in other family relationships* as well. Therefore, growing up in discordant homes is theorized to undermine children's emotional security in both the parent–child and interparental relationships. Moreover, children's security in each relationship is proposed to be distinctly organized by two different control systems: the attachment and social defense systems.

Figure 5.2 depicts the two ethological modules underlying children's security processes. Each behavioral system or ethological module is theorized to consist of distinct integrated sets of psychological, biological, and behavioral processes that support the overarching goal of "felt" security in the face of specific family threats (i.e., interparental, parent–child) (Davies & Sturge-Apple, 2007). According to evolutionary accounts, these systems evolved because they increased survival and reproductive potential by addressing recurring problems in the ancestral environment. As dynamic, goal-corrected systems, each is able to flexibly enlist multiple behavioral strategies to achieve felt security in response to differing ecological demands. Although the social defense and attachment systems share the broad adaptive function of protection from harm, they are distinguished from one another based on distinct external goals, response strategies, and conditions that increase their saliency as organizers of behavior.

Within the parent–child relationship, the psychological goal of felt security is primarily achieved through the attachment system. The central behavioral goal of the attachment system is to maximize the sensitivity and protection of caregivers in times of distress or threat (Bowlby, 1969). Throughout evolutionary history, recurring conspecific aggression resulted in adaptive value being placed on affect and behaviors that increased the child's proximity to and protection from a primary caregiver, particularly in the context of unfamiliar adults (Maestripieri, 2003).

Children have access to a wide repertoire of attachment behaviors to achieve felt security, including distress expressions, bids for comfort, proximity-seeking, and monitoring of attachment figures (Ainsworth, Blehar, Waters, & Wall, 1978). How children organize these behaviors to increase accessibility of the caregiver in times of distress forms the primary parameters for deciphering children's ability to successfully utilize parents as sources of protection and support. Thus, although systems for assessing children's emotional security in the parent–child relationship may vary from categorical (i.e., insecure versus secure; Ainsworth et al.; Weinfeld, Sroufe, Egeland, & Carlson, 1999) to continuous (i.e., degrees) schemes (Cummings, 1990; Fraley & Spieker, 2003), common to both of these approaches is the assessment of the child's confidence in the caregiver as a source of protection in stressful or challenging contexts.

In contrast, children's concerns about security in the face of interparental conflict largely reflect the operation of the social defense system (Davies & Sturge-Apple, 2007). According to evolutionary theory, the high cost of conflict between members of a social group is theorized to have put selective pressure on the development of a behavioral system capable of efficiently identifying and responding to social signals indicative of potential threat (e.g., yelling, dominant posturing). Thus, the social defense system evolved to minimize threat posed by members of family and social networks (Boyer & Bergstrom, 2011). Because relationship difficulties between parents can have threatening implications for children either directly (e.g., exposure to hostility) or through the implications of interparental conflict for the structure and stability of the family, the social defense system and its behavioral goal of protecting oneself from interpersonal harm are posited to organize children's response patterns to interparental conflict. As a result, preserving a sense of security in the interparental relationship involves a distinct pattern of protective strategies or action tendencies characterized by fear, freezing, fight and flight behaviors, camouflaging activities, social de-escalation strategies (e.g., comforting, pacifying parents), and heightened perceptual sensitivity to the threatening stimuli (e.g., angry facial expressions, yelling) accompanying interparental conflict. Thus, with respect to individual differences, difficulties in achieving security in the interparental relationship are typically defined as prolonged, intense bouts of fearful distress, avoidance, and/or involvement and negative appraisals or representations of the family in response to conflicts between parents.

Research provides initial support for the distinction between children's strategies for responding to interparental conflict and attachment distress. Based on maternal daily diaries, children's behavioral responses to interparental conflict revealed higher percentages of general distress and fear (40.8%) and attempts to intervene (e.g., comfort, distract) (10.1%) than bids for contact and comfort (3.1%) (Cummings, Zahn-Waxler, & Radke-Yarrow, 1981). Parental diary reports of pre- and early adolescent responses to interparental conflict demonstrated similarly high proportions of fear, flight, and intervention behaviors (Garcia O'Hearn, Margolin, & John, 1997). In contrast, empirical analyses of child behaviors in contexts that activate the attachment system (e.g., child separation, exposure to unfamiliar adults or objects) indicate that children commonly respond with verbal bids for support, comfort-

seeking, and displays of distress directed toward the parent (Ainsworth et al., 1978). In addition, research indicates that individual differences in security across the interparental and parent–child relationships are relatively distinct from one another. For example, latent variable analyses indicate that social defense responses reflecting insecurity in the interparental relationship (e.g., fear, involvement, perceptual sensitivity to the implications of threat) and attachment behaviors within the parent–child relationship (e.g., use of parents as a secure base, representations of availability of parent in times of need) cohere into distinct latent constructs of interparental insecurity and parent–child insecurity (Davies, Harold, Goeke-Morey, & Cummings, 2002).

Although EST-R's ethological approach highlights the distinctive properties of interparental and parent–child security, we also propose that the two systems share some overlap. As illustrated by the bidirectional arrows in Fig. 5.2, the attachment and defense systems do not operate in isolation from one another. Both modules are conjoined by the overarching goal of felt security and the common adaptive function of protection from harm. At a neurobiological level, each system is organized, in part, by the behavioral inhibition system (Gray, 1987; Gray & McNaughton, 2000), a network of neurobiological circuits supporting sensitivity to punishment and inhibition of approach behaviors. Thus, the social defense and attachment systems may often draw on a common subset of behavioral strategies (e.g., distress expressions) to achieve their distinctive behavioral goals. For example, crying is indicated in both the attachment and interparental contexts (Ainsworth et al., 1978; Cummings & El-Sheikh, 1991). The attachment and social defense systems may also share a common set of behavioral strategies by virtue of their similar role in inhibiting various "approach" behaviors (e.g., approaching novelty, social initiations, prosocial caretaking). Consistent with this premise, reductions in exploration, play, and sociability are theorized to signify heightened concerns about security in both the parent–child and interparental relationships (e.g., Cassidy, 2008; Cummings & Davies, 2010). Based on these considerations, it is not surprising to find that security in the interparental relationship and the parent-child relationship are correlated. For example, children's internal representations of the threatening consequences posed by interparental conflict share only a moderate degree of overlap ($r^2 = 0.17$) with their attachment representations of parental availability and responsiveness in times of distress (Sturge-Apple, Davies, Winter, Cummings, & Schermerhorn, 2008).

Family Precursors of Insecurity

Delineating the substance of the interparental and parent–child security systems is only a first step toward demonstrating the utility of EST-R in advancing family risk models. As illustrated by path 2 in Fig. 5.1, the potential value of adopting an evolutionary lens is rooted in its capacity to discriminate family correlates and origins of individual differences in security in the interparental and parent–child

relationship. Accordingly, we now turn to address how the distinction between social defense and attachment systems may provide a blueprint for understanding the precursors of interparental and parent–child security.

Family Precursors of Interparental Insecurity

Although stressors within multiple relationships can have a lasting influence on children's security, evolutionary accounts underscore the characteristic ways in which children utilize defense strategies within particular relationships are likely to emerge, in large part, from their histories of experiences within that same relationship. Therefore, relationship dynamics between parents are theorized to hold primacy in shaping stable individual differences in child security in the interparental relationship. As the cornerstone of many families, the interparental relationship is pivotal in defining how social stress and danger are managed in the family. Emotional qualities of the interparental relationship specifically determine whether challenges in the family are effectively encapsulated and transformed into benign family conditions or proliferate and broaden into significant threats to the safety and security of the children. Thus, individual differences in children's security in interactions between parents are likely based on their earlier histories of exposure to interparental interactions. Supporting this prediction, previous studies have shown that parental difficulties in managing and resolving disputes with one another predict children's signs of insecurity in the interparental relationship even after inclusion of other family relationship processes. For example, studies have revealed that interparental conflict is a significant predictor of concurrent levels of and subsequent increases in children's insecurity in the interparental relationship in multivariate models that also incorporate parenting, family, and child attributes as covariates (e.g., Davies, Cummings, & Winter, 2004; Davies, Martin, & Cicchetti 2012; Davies, Sturge-Apple, Winter, Cummings, & Farrell, 2006).

Toward the goal of identifying the interparental conflict dimensions that are particularly potent in engendering insecurity, evolutionary models conceptualize the social defense system as an encapsulated module responding selectively to a delimited set of contextual stimuli. These stimuli are theorized to be comparable in form to recurring cues presaging significant threats to survival throughout human evolution (e.g., angry expressions, loud noises, aggression, dominant posturing) (Davies & Sturge-Apple, 2007; Ohman & Mineka, 2001). As the function of the social defense system is to defuse conspecific threats, children's social defense strategies in the context of interparental conflict should selectively reflect their sensitivity to histories of exposure to these same interpersonal threat cues displayed by parents in their disagreements (Davies & Sturge-Apple; Hofmann, Heinrichs, & Moscovitch, 2004). Because diminished displays of mutual happiness, support, and cohesiveness are far less reliable as signals of danger if they are not accompanied by hostile threat cues, these parameters of constructive conflict are hypothesized to carry minimal weight in signifying threat and organizing children's social defense strategies

(Ohman & Mineka). Therefore, EST-R places significantly greater weight on destructive conflict characterized by unresolved hostility, aggression, and intense anger as etiological agents versus constructive or benign conflict tactics (see bolded descriptors in Fig. 5.2; Davies & Sturge-Apple; Davies & Woitach, 2008).

Empirical tests of this hypothesis are relatively rare, but existing research offers some promising support. For example, Fosco and Grych (2007) reported that children's exposure to destructive interparental conflict was the only significant predictor of their appraisals of threat within a multivariate model that also examined parental positive emotional expressiveness as a predictor. As a more direct test, findings from two separate studies consistently identified children's exposure to hostile, aggressive conflicts as the only significant predictor of their insecurity in the interparental relationship in analyses that considered constructive conflict, child characteristics, parent–child relationship dynamics, family-level processes, and socioeconomic factors as predictors (Davies, Martin, & Cicchetti, 2012). Replication of this pathway across the two studies was especially striking in light of the variation in measures (i.e., multiple informant versus multiple method), methodological designs (i.e., concurrent versus longitudinal), the developmental period of the children (i.e., preschoolers versus adolescents), and the nature of risk in the samples (i.e., socioeconomically disadvantaged versus working, middle class).

In further highlighting the utility of differentiating among forms of destructive conflict, EST-R proposes that the strength of interparental dysphoria and disengagement as predictors of children's emotional insecurity should fall in the intermediate range between the negligible impact of constructive conflict and the potent effects of hostile conflicts. On the one hand, dysphoria and disengagement signify abandonment, rejection, and interpersonal struggle within the social hierarchy (Gilbert, 2001). Therefore, repeated exposure to these conflict properties is more likely to undermine children's security in the interparental relationship than variations in constructive conflict by signaling the potential for threat (Dixon, 1998). Yet, on the other hand, evidence supports the notion that there is a more potent biological proneness to respond defensively to conspecific anger and fear (Ohman & Mineka, 2001). Thus, it follows that exposure to parental hostile and fearful displays during conflict represents a more imminent threat than disengaged or dysphoric conflict tactics. Supporting these hypotheses, interparental hostility and disengagement were each uniquely associated with domains of children's insecure response to interparental conflict (i.e., behavioral and subjective indices of distress, negative representations) in concurrent and prospective analyses (Davies, Sturge-Apple, Winter, Cummings & Farrell, 2006).

Family Precursors of Individual Differences in Parent–Child Attachment Security

As the attachment system is exquisitely designed to increase accessibility to caregivers in times of distress, children's histories of successfully procuring supportive resources from primary caregivers are theorized to be a primary determinant of

individual differences in parent–child security. Thus, in accord with attachment theory, EST-R accepts the thesis that displays of sensitivity, warmth, and availability by caregivers foster children's appraisals of caregiver accessibility. The end result is the very efficient operation of the attachment system characterized overtly by patterns of behavior reflecting confident, direct bids for support that effectively reduce fear and distress. In contrast, prolonged experiences with harsh, inconsistent, or diminished levels of caregiver availability are key processes that undermine children's ability to reliably use parents as a safe haven or base of security (Belsky & Fearon, 2008).

However, in revisiting the function of attachment, researchers have called for greater specificity by proposing that parental responsiveness in contexts of child distress and support-seeking is a more central antecedent to attachment than general characterizations of availability and warmth (Goldberg, Grusec, & Jenkins, 1999; Thompson, 1997, 1998). Because the external goal of the attachment relationship is to maximize caregiving behaviors in the context of distress, differences between children in their attachment security are most likely to emerge from parental cues detected by children when they are upset or troubled. Thus, some elements of broad "style" characterizations of support may be particularly relevant to the attachment (i.e., ability to use caregiver to allay distress), whereas other dimensions of support (i.e., enjoyment in recreational activities) may be relevant to domains of functioning (e.g., affiliation) that are not tied to security systems (e.g., Marvin & Britner, 2008). Supporting this thesis, maternal sensitivity to infant distress at 6 months was a significant predictor of infant attachment security at 15 months even after taking into account the nonsignificant role of maternal sensitivity to children in benign situations as a predictor in the analyses (McElwaine & Booth-LaForce, 2006).

Attention to the specific function of each insecure strategy organized by the attachment system can further refine an understanding of family antecedents of parent–child insecurity.

Although natural selection likely equipped children with many ways of coping with inaccessible attachment figures, specific stimuli and cues in the caregiving environment may engender distinctive fitness-promoting strategies. Within the attachment literature, studies have repeatedly distinguished between two specific types of strategies based on whether they serve to deactivate or hyperactivate the natural output of the attachment system. Children who adopt avoidant attachment systems deactivate or dampen natural tendencies to overtly express negative affect, seek support, and process attachment-relevant information. In contrast, children with resistant or ambivalent patterns of attachment adopt hyperactivating approaches that serve to amplify and inflate overt distress, dependency, and the processing of attachment cues (Cassidy, 2008; Kobak, Cole, Ferenz-Gillies, Fleming, & Gamble, 1993). Deactivation or avoidance is specifically regarded as an adaptive strategy for limiting exposure to the negative consequences of repeatedly approaching chronically inaccessible, rejecting caregivers. Conversely, hyperactivation of the attachment system may be a functional strategy for eliciting more reliable responsiveness and sensitivity from a caregiver who is inconsistent in supporting the child's needs (Cassidy).

Common Family Precursors of Types of Security

Each type of security is hypothesized to share common precursors. Etiology of disorganized and disoriented (D) forms of insecure attachment exemplifies the potential commonalities in the predictors of interparental and parent–child security. According to many attachment frameworks, freezing, bizarre, aimless, controlling, and contradictory behaviors of children with D attachment patterns reflect significant breakdowns in the coherency of children's strategies of relating to caregivers. Although D patterns may reflect insecure attachment strategies, our theory interprets "D" behaviors as organized and adaptive action tendencies of the social defense system rather than the disorganized, maladaptive products of the attachment system. That is, D attachment patterns may reflect that activation of the social defense system and its goal of defusing conspecific threat as an organizer of behavior (Crittenden, 2008). Consequently, behaviors within the D pattern reflect the conjoint operation of both the attachment and the social defense systems. For example, children's contradictory behaviors characterized by simultaneous or successive approach and avoidance displays can be interpreted as reflecting a virtual "draw" between attachment and defense systems. Likewise, freezing and inhibition of overt distress expressions may specifically reflect self-protective camouflaging efforts that may serve the organized function of reducing children's salience as targets of oppressive adult dominants (Davies & Sturge-Apple, 2007). Thus, although children have a hardwired, phylogenetic tendency to approach the attachment figure, the D strategy signifies that the attachment relationship is simultaneously a source of threat worthy of activation of social defense strategies.

On the basis of this analysis, a key question is as follows: What family experiences engender in children both a lack confidence in their parents as protective figures and appraisals of parents as sources of threat? Arousal of fear in response to signals of interpersonal threat is specifically proposed to elicit social defense system functioning. Exposure to highly frightening (e.g., violence, hostile-intrusiveness, abuse) and frightened (e.g., helpless, fearful behaviors, role reversal) displays by attachment figures is likely to simultaneously signal the potential for interpersonal threat and the unlikelihood that the caregiver is able to offer support (Main & Hesse, 1990). Therefore, frightening and frightened caregiver behaviors are presumed to be particularly strong precursors of D attachment patterns. Moreover, self-protective strategies of the social defense system operate in multiple interpersonal conditions. Witnessing high levels of frightening (e.g., hostile, aggressive), vulnerable (e.g., distressing, fearful), or volatile (e.g., emotionally labile) parental behaviors during interparental conflict is likely to amplify children's social defense responses to caregivers in subsequent parent–child interactions, particularly in stressful contexts. In support of these predictions, children exhibit disproportionately higher rates of disorganized attachment relationships in highly adverse family environments characterized by maltreatment, severe parent psychopathology, and interparental hostility and violence (Cyr, Euser, Bakermans-Kranenburg, & van IJzendoorn, 2010; Owen & Cox, 1997; Zeanah et al., 1999).

As components of the exosystem in ecological frameworks of development (Conger & Donnellan, 2007; Lynch & Cicchetti, 2002; Martin et al., 2010), distal community characteristics are important to consider in the identification of the common origins of family precursors of child security. Extrafamilial sources of adversity in the form of stressful work climates, socioeconomic impoverishment, neighborhood violence, and diminished access to social support networks are posited to indirectly amplify children's concerns about security in the family by undermining the stability and safety of the family unit (e.g., Berkowitz, 2003; Lynch & Cicchetti). Conversely, access to resource-rich environments characterized by family-friendly social policies, social support, neighborhood cohesion, and balanced work environments may strengthen family relationships that serve as a primary basis for children's safety and security (Berkman and O'Donnell, Chap. 12; Reichman & Teitler, Chap. 9). In support of these predictions, studies have identified diminished parental emotional functioning and mental health as explanatory mechanisms in associations between adult exposure to community violence and economic pressures and greater discord and turmoil in interparental and parent–child relationships (Conger & Donnellan; Martin et al.). By the same token, it is important to note that the child's firsthand experiences in the exosystem may directly undermine security in family relationships. For example, children's exposure to neighborhood violence may be so overwhelming that it directly weakens the parent–child attachment system by shaking children's confidence in parents to protect them from danger (see Lynch & Cicchetti).

Mental and Physical Health Sequela of Children's Emotional Security

The developmental value of emotional security as a mediator of family emotional processes is further reflected in its ability to predict a wide array of mental and physical health outcomes. Cross-sectional and longitudinal studies demonstrate that measures of children's attachment security in the parent–child relationship are positively associated with social competence (e.g., Raikes & Thompson, 2008), self-concept (e.g., Cassidy, 2008; Doyle, Markiewicz, Brendgen, Lieberman, & Voss, 2000), emotion regulation (e.g., Kerns, Abraham, Schlegelmilch, & Morgan, 2007; Kochanska, 2001), academic and intellectual functioning (e.g., Moss & St. Laurent, 2001), physical health (e.g., Bosmans, Goossens, & Braet, 2009), sleep quality (e.g., Keller & El-Sheikh, 2011), and substance use (e.g., Schindler et al., 2005) and negatively associated with the incidence of mental health problems in the form of both internalizing (e.g., Warren, Emde, & Sroufe, 2000) and externalizing (e.g., Sroufe, Egeland, Carlson, & Collins, 2005) difficulties. Research tracing the developmental implications of insecurity in the interparental relationship has identified a similar set of sequela, including externalizing and internalizing symptoms (e.g., Cummings, Schermerhorn, Davies, Goeke-Morey, & Cummings, 2006), academic and peer difficulties (e.g., Davies, Woitach, Winter, & Cummings, 2008;

Sturge-Apple et al., 2008), sleep and physical health problems (e.g., El-Sheikh, Buckhalt, Cummings, & Keller, 2007; Keller & El-Sheikh, 2011), and emotion dysregulation and dependency (Davies, Manning & Cicchetti, in press).

The laundry list of outcomes associated with security is impressive in scope. In fact, in his literature review on attachment, Thompson (2008) concluded that the range of outcomes was so expansive, "One might wonder whether there is anything with which attachment security is *not* associated (p. 348)." However, the cost of simply cataloguing the outcomes of types of insecurity is a loss of precision, making it difficult to deduce anything other than this: inherently, positive coping and negative responses of children will respectively beget healthy and unhealthy outcomes. Making sense of the array of outcomes hinges on charting how insecurity in parent–child and interparental relationships produces a developmental cascade of processes that ultimately develop into broader, trait-like patterns of psychological and physical functioning. In this section, we address how EST-R may provide some promising leads in identifying potential mechanisms. As path 3 of Fig. 5.1 shows, we specifically focus on how security systems may be associated with variability in various child outcomes by altering (a) their use of defensive analogues in subsequent interpersonal settings, (b) the physiological components in the stress response system (SRS; Del Giudice, Ellis, & Shirtcliff, 2011), and (c) the operation of other behavioral or ethological systems that organize approach goals.

Defensive Analogues

Lawful variation in processing broader social threats is theorized to underlie associations between the two types of insecurity in the family and children's mental health trajectories. Difficulties in defusing threat in the interparental relationship and accessing caregivers as sources of protection from harm are both proposed to increase the salience of survival or self-protective strategies across multiple interpersonal contexts (Davies, Winter, & Cicchetti, 2006). As a result, children who experience insecurity in family relationships are likely to become increasingly proficient in processing threat and recruiting the resources necessary to guard against its consequences. A primary thesis of attachment theory is that children develop models for filtering, interpreting, and responding to stressors in close relationships that they later use as guides in novel or challenging settings to simplify, evaluate, and adapt to social experiences (Bowlby, 1973; Cassidy, 2008). Expanding this assumption, Johnston, Roseby and Kuehnle (2009) noted that children from discordant homes develop negative scripts consisting of highly automatic rules for responding to parent–child and interparental relationships (Pollack & Tolley-Schell, 2004). Thus, whereas benign or secure scripts in the family are likely to engender greater diversity and flexibility in ways of approaching new social contexts, insecure family scripts are proposed to be more reflexively and intransigently applied to an array of new contexts.

As blueprints for scanning new, potentially threatening social scenes for old dangers (Johnston et al., 2009; Davies et al., 2006), these cognitive-affective analogues are proposed to increasingly coalesce into patterns of mental health difficulties characterized by internalizing and externalizing symptoms. For example, insecurity in family relationships may increase children's vulnerability to depression, anxiety, and withdrawal by predisposing children to experience hypervigilance and submissiveness in social contexts (e.g., school, peers, friends) outside the family (Granot & Mayseless, 2011; Luebbe, Bell, Allwood, Swenson, & Early, 2010; Prinstein, Cheah, & Guyer, 2005). Likewise, threat analogues consisting of selective encoding of threat, malevolent attributions of others' behaviors, and the generation of aggressive solutions to provocative extrafamilial situations may mediate pathways between the security and children's aggression and conduct problems (Dodge, 2006; Granot & Mayseless, 2011).

Empirical findings are consistent with many of these predictions. For example, parent–child attachment security has been associated with more benign and flexible patterns of children's emotional processing and responses to challenging peer problems (Cassidy, Kirsh, Scolton, & Parke, 1996; Granot & Mayseless, 2011). In turn, these social information-processing patterns predict greater social competence and lower risk for internalizing and externalizing symptoms in both cross-sectional and longitudinal designs (Crick & Dodge, 1994; Dodge, 2006). Complementing this work, hostile ways of processing peer transgressions have also been shown to mediate associations between children's insecure representations of the interparental relationship and increases in their school maladjustment over a 1-year period (Bascoe, Davies, Sturge-Apple, & Cummings, 2009). Importantly, this pathway remained robust even after considering children's insecure representations of parent–child relationships, their propensities to experience negative emotion, and their socioeconomic background as predictors in the analyses. Pathways from insecurity to specific patterns of mental health may also vary as a function of the pattern of insecurity. Granot and Mayseless reported that children with hyperactivating (i.e., ambivalent) attachment representations were particularly likely to respond to peer situations by exaggerating distress expressions that may pave the way for internalizing symptoms (Luebbe et al., 2010). In contrast, children with disorganized attachment representations were most inclined to process stressful peer situations in aggressogenic ways (Dodge, 2006).

Stress Response System

Children's emotional insecurity in the family is proposed to impact mental and physical health partly through the adaptive restructuring of the stress response system (SRS) and its evolved biological function of coordinating physiological responses to environmental challenges that had fitness-relevant consequences in ancestral environments (e.g., Ellis, Jackson, & Boyce, 2006; Repetti et al., 2011). This process is supported by the neuroendocrine system including the hypothalamic–pituitary–adrenal

axis (HPA), the sympathetic nervous system (SNS), and the parasympathetic nervous system (PNS). The parasympathetic nervous system serves as the regulator of the autonomic branch through shifting physiological resources toward restorative, homeostatic functioning and reducing physiological arousal (Porges, 1995). However, under conditions of threat, the PNS deactivates, allowing heightened SNS if needed. As such, the SNS primes the body for fight-or-flight responses in the face of threat by increasing cardiac output, oxygen flow, and blood glucose levels (Porges, 2006). Through its role as the third wave of responding (i.e., Gunnar & Vazquez, 2006), the HPA axis and its end product of cortisol prime homeostatic defense mechanisms through the mobilization of energy (e.g., glucose, oxygen) and modulating the processing, encoding, and memory consolidation of emotionally significant events. Although extensive cross-regulation among the three components of the stress response system exists via a central control center located in higher order limbic structures (e.g., Gold & Chrousos, 2002), different systems can assume dominance within the response hierarchy. For example, if the SNS is effective in dealing with environmental threats, the SNS may supersede, and thus limit, the activation of the HPA axis (Del Giudice, Ellis and Shirtcliff 2011).

Allostasis and allostatic load are primary concepts in SRS models. Allostasis refers to the process by which biological "set points" in homeostasis are altered to meet the challenging dynamics of the environment in order to preserve the internal integrity of the organism. Thus, optimal physiological adaptation involves efficient activation of the SRS, including the ability to disengage upon the termination of the arousal-provoking stimulus to conserve psychobiological resources and return to homeostatic functioning. However, in highlighting the operation of allostatic load, successive cycles of allostasis engendered by repeated exposure to environmental adversity are theorized to result in wear and tear on the body that undermine the integrity of multiple domains of functioning. Hyperarousal and hypoarousal are two of the common patterns of allostatic load (McEwen & Wingfield, 2003). Hyperarousal is characterized by extensive amplification of biological set points in response to repeated stressors characterized by heightened activity and dampened recovery of the SRS. Conversely, a hypoarousal pattern represents a downregulated response such that physiological systems evidence substantially dampened activation to environmental stress and challenge.

Research has documented the deleterious impact of allostatic load on the SRS across a variety of outcomes including emotion regulation, social development, mental health difficulties, immune suppression, increased risk for diabetes, cytokine production, and neurotoxic effects (e.g., Danese, Pariante, Caspi, Taylor, & Poulton, 2007; McEwen, 1998; Repetti et al., 2002, 2011; Sapolsky, 2000; Turnbull & Rivier, 1999). Much less is known about how children's security in family relationships is coupled with changes in the SRS and, ultimately, allostatic load. However, a handful of studies conducted have yielded some empirical clues for understanding the physiological underpinnings of emotional insecurity in the interparental relationship. For example, as a central indicator of emotional insecurity, both observational and questionnaire assessments of children's distress responses to interparental conflict were consistently associated with elevated cortisol reactivity even estimating

child hostility and involvement and demographic characteristics as covariates (e.g., Davies, Sturge-Apple, Cicchetti, & Cummings, 2008). In a subsequent study, children's fearful reactivity to interparental conflict was associated with lower levels of resting cardiac marker of SNS (Davies, Sturge-Apple, Cicchetti, Manning, & Zale, 2009). More work is needed to draw definitive conclusions, but the emerging pattern of findings is consistent with theorized linkages between inhibited, fearful behavioral patterns and a physiological profile characterized by high HPA reactivity to stressors and dampened SNS functioning (Ellis et al., 2006).

A somewhat larger literature exists on linkages between attachment security and SRS reactivity. In reflecting the two most consistent patterns of findings, attachment security has been associated with an efficient cortisol response when the attachment system is activated (e.g., Gunnar, Brodersen, Nachmias, Buss, & Rugatuso, 1996), while disorganized attachment is linked with hyperarousal pattern of reactivity (Hertsgaard, Gunnar, Erikson, & Nachmias, 1995; Spangler & Grossman, 1993). Notably, these findings are consistent with the associations between hypercortisolism and insecurity in interparental relationship and broader theoretical arguments that elevated HPA activity is designed to cope with interpersonal events that are perceived highly stressful and uncontrollable (Dienstbier, 1989; Mendes, Blascovich, Hunter, Lickel, & Jost, 2007). Thus, couched within our evolutionary perspective, we tentatively propose that coupling of the HPA system and a fearful, "disorganized" pattern of behaviors across parent–child and interparental contexts is rooted in the common, underlying operation of the social defense system.

Other biobehavioral findings on attachment are more difficult to interpret at this early stage of research. For example, the same studies linking disorganized attachment with elevated HPA activity yielded conflicting findings on whether children with avoidant attachments exhibit a pattern of hypo- or hyperarousal (Hertsgaard et al., 1995; Spangler & Grossman, 1993). Furthermore, the scant empirical work on attachment and children's PNS and SNS reactivity has not produced a readily interpretable body of findings. For example, two studies have reported no links between attachment and PNS and SNS activity (Oosterman & Schuengel, 2007; Stevenson-Hinde & Marshall, 1999), whereas another study revealed higher PNS withdrawal in insecure-avoidant infants only (Hill-Soderlund et al., 2008). More work is needed to better clarify the ontogenetic trajectory of the SRS in response to children's emotional security in family context.

Behavioral or Ethological Systems of Approach

Stable individual differences in the operation of the security systems are also proposed to impact the development of competencies and mental health outcomes by affecting the development of cognitive, emotional, and social skill sets that promote fitness. In offering a broad overview of this general cascade process, resource depletion models propose that regulation of responding in one domain or system of functioning is likely to impair subsequent functioning in other domains by depleting a

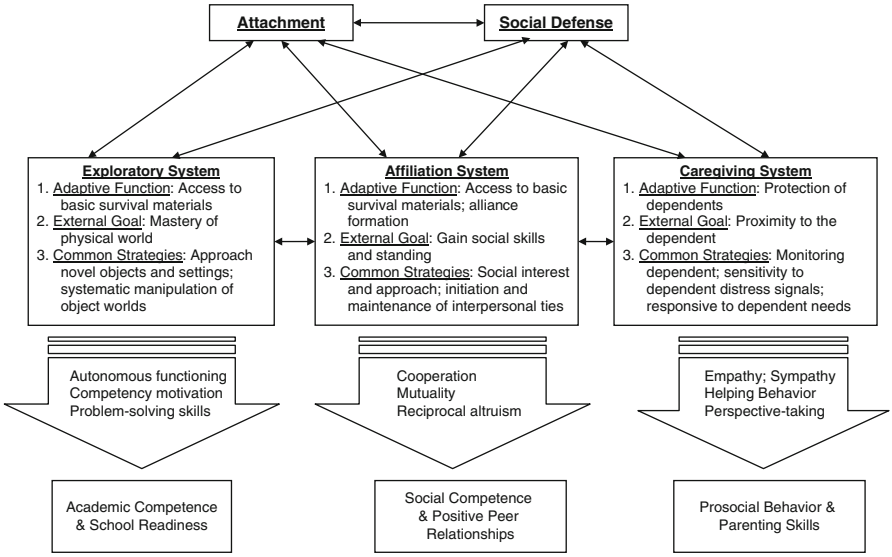

Fig. 5.3 A model illustrating how the social defense and attachment systems impact children's competence in multiple domains by altering the operation of ethological systems that organize approach motives and behaviors

shared reservoir of psychobiological resources (e.g., Baumeister, Vohs, & Tice, 2007). Accordingly, prolonged concerns for security would be expected to tip the balanced allocation of psychobiological resources toward investing in immediate personal safety at the cost of equally investing in mastery of the physical and social environment. Conversely, the efficient operation of the attachment and social defense systems is proposed to afford children more opportunities to devote efforts toward developing specific social and intellectual competencies (Davies et al., 2006; Ford, 2009).

In offering further direction in identifying specific trajectories of development, EST-R posits that the defensive nature of security systems is particularly likely to inhibit approach behaviors organized by other ethological systems. As Fig. 5.3 shows, exploration, affiliation, and caregiving are three particularly salient systems during the childhood years with different external goals and strategies. First, security systems may indirectly shape trajectories of intellectual and academic competence by affecting the functioning of the exploratory system and its goal of mastering the physical world. Greater efficiency in the operation of security systems (i.e., greater security) specifically paves the way for the successful working of the exploratory system as evidenced by intrinsic motivation and behavioral efforts to approach, manipulate, and understand the workings of the physical world (Bernier, Carlson, & Whipple, 2010; Davies, Manning, & Cicchetti, in press; Sroufe, 2005). Over time, greater engagement in the physical world is proposed to promote autonomous functioning, resourceful and flexible problem-solving (e.g., executive

function, attention), and perceived efficacy in academic and intellectual contexts (Blair & Diamond, 2008; Davies, Woitach et al., 2008).

Second, children who are proficient in preserving safety in the family are theorized to have greater opportunities to organize the affiliative system goal of achieving access to survival materials and social standing through the formation and maintenance of cooperative alliances (Irons & Gilbert, 2005; Markiewicz, Doyle, & Brendgen, 2001). The affiliative system is specifically designed to lubricate and sustain social interactions through the regulation of affect expressions (e.g., warmth, trust), behavioral displays (e.g., smiling, touch), shared attention (e.g., turn-taking), and active listening (e.g., eye contact) (Davies & Sturge-Apple, 2007; Depue & Morrone-Strupinsky, 2005; Furman, 1999). Acquiring and refining these skills engenders broader patterns of companionship, cooperation, mutualism, and reciprocal altruism that are proposed to be key building blocks for social competence and harmonious, mutually beneficial peer relationships (Deater-Deckard & Petrill, 2004; Lindsey, Cremeens, & Caldera, 2009).

Third, although the caregiving system is still relatively undeveloped during childhood and adolescence, its adaptive function in protecting dependents requires the development of sensitivity and responsiveness to others' needs early in the life span. Security is theorized to provide the basis for the elaboration of caregiving strategies by arming children with affect-regulation tools and supporting their attunement to their social environment. In childhood, the enactment of care in the form of empathy, perspective-taking, and prosocial (helping) behavior hinges on successfully regulating intense distress responses to witnessing anguish and pain in others (Eisenberg & Eggum, 2009). Provided that children's own security needs do not predominate, the development of close relationships (e.g., best friendships, romantic affiliations) during adolescence and early adulthood offer further opportunities to practice and refine the caregiving strategies that are ultimately critical to developing competent parenting and prosocial skills (Davies, Sturge-Apple, Woitach, & Cummings, 2009). Thus, analysis of the interplay between security in family relationships and the operation of the caregiving system may be a key mechanism underlying the intergenerational transmission of parenting, attachment, and family relationship processes (George & Solomon, 2008).

Sources of Heterogeneity in Pathways of Security

The modest to moderate magnitude of mediational pathways among family relationship processes, children's security in family relationships, and their trajectories of adjustment highlights the considerable variability between children in the sequela of family processes. For example, many, if not most, children exposed to highly adverse family relationships (e.g., violence) do not experience clinically significant levels of psychopathology at any one time (Hughes, Graham-Bermann, & Gruber, 2001). Likewise, children raised in supportive family contexts exhibit a wide range of both healthy and problematic outcomes (e.g., Cummings, Davies, & Campbell, 2000).

Because individual development is regarded as operating within an open system (Davies et al., 2004), the developmental pathways set in motion by emotional relationships in the family are proposed to lawfully vary as a function of children's prior and current experiences and their own attributes, resources, and histories of adaptation. As depicted in paths 4a and 4b in Fig. 5.1, these conditions may directly alter mediational pathways of emotional security in their role as moderators. Developmental psychopathology frameworks provide useful distinctions between two basic classes of moderators: vulnerability and protective factors. As specific types of moderators that amplify family risk processes, the search for potentiating or vulnerability factors effectively answers the question of who is *most* at risk. Conversely, in diluting the impact of family risk processes and potentially enhancing functioning, protective factors help to address the question of who is resilient, especially in the face of adversity. For the sake of illustration, we selectively review work on two promising classes of potential moderators: family system dynamics and child attributes.

Family System Dynamics

Building a family-wide model of emotional security necessitates extending beyond the study of dyadic family relationships by capturing processes in the broader family unit. In integrating family systems theory within an ethological approach (Davies & Sturge-Apple, 2007), EST-R specifically proposes that children will adopt distinct ways of addressing security concerns based on (a) the patterning and form of antagonism across members of the family unit; (b) the structure and hierarchy of power; and (c) differences in relationship boundaries, defined as implicit rules for transmitting resources and emotions across family subsystems (also see the Future Directions section). From the standpoint of EST-R, discerning the nature and locus of moderating effects are key objectives. Whereas determining the nature of the moderating effect consists of distinguishing protective and potentiating factors, identifying the locus involves testing whether these family dynamics alter the pathways between family processes and children's security (path 4a), children's security and their health outcomes (path 4b), or both.

On the one hand, exposure to threats in interparental or parent–child relationships may assume a more benign meaning for children in a broader context of close relationships and well-defined family boundaries that permit children access to support without incurring substantial psychological costs (e.g., loss of autonomy). Though rarely tested, initial studies highlight the importance of documenting the nature and locus of the moderating effects of family harmony and support. For example, Davies and colleagues (2002) reported that family cohesion did not moderate the link between marital conflict and children's insecurity but did serve as a protective factor in associations between insecurity and their maladjustment. Interpreted in our framework, cohesion in the context of conflict may reduce children's insecurity by thwarting the adoption of defensive approaches (i.e., analogues) in new social relationships and challenges, reducing SRS and allostatic load processes and preserving resources for

allocation to behavioral systems (e.g., affiliation, exploratory) dedicated to mastery of the social and physical worlds.

On the other hand, the deleterious implications of parent–child or interparental difficulties for children may be amplified by family processes that reflect ill-defined power hierarchies and instability in relationships. For example, family instability, which signifies degree to which families fail to provide continuity, cohesiveness, and stability for children, has been shown to potentiate associations between marital conflict and children's insecurity (Davies et al., 2002). Further distinguishing between specific types of family boundary disturbances, Davies and colleagues (2004) found that elevated interparental conflict had different implications for children's concerns about security depending on whether the larger family unit was characterized by overly rigid, disengaged relationships or highly enmeshed, intrusive relationships. Although children from both families exhibited greater insecurity in the interparental relationship, concerns about insecurity were more pervasive in enmeshed families than they were in disengaged families.

Child Attributes

Consistent with person-by-environment interaction models, prevailing evolutionary conceptualizations propose that constitutional differences across individuals persist because they confer distinct survival benefits in specific, recurring ecological niches. For example, differential susceptibility theory proposes that children's reactivity to environmental conditions conveys different developmental advantages and costs depending on the balance of resources and adversity in the family (Belsky, 2005; Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007). Thus, when family contexts are characterized by higher levels of discord, children who exhibit low levels of sensitivity evidence better psychological and physical adjustment relative to highly sensitive children. Conversely, in highly supportive environments, children who are highly reactive to environmental stimuli have a much greater capacity to thrive relative to their less sensitive counterparts (see Belsky & Pluess, 2009).

Interpreted in EST-R, these assumptions raise the possibility that pathways among family processes, children's security in the family, and their healthy functioning may be particularly pronounced for children who exhibit high levels of genetic and temperamental sensitivity to environmental cues. In light of the earlier evidence that children's concerns about insecurity are more sensitively attuned to variations in antagonism than cooperation (Davies et al., 2012), it is possible that the negative impact of interparental conflict on children's emotional insecurity in the interparental relationship may incrementally increase for children who exhibit higher levels of biological or temperamental sensitivity. In a similar vein, highly sensitive children may also exhibit a greater proneness to develop disorganized patterns of parent–child attachment when exposed to frightening or frightened caregivers. By the same token, the parent–child attachment system is theorized to be more sensitive to variations in supportive caregiving environments than the social defense system.

Therefore, highly sensitive children may better capitalize on the resources in supportive and responsive caregiving environments and develop more secure parent–child relationships than children who are less sensitive.

Although no studies have explored differential susceptibility with respect to interparental conflict and children's emotional security, molecular genetics research has tested the plausibility of genetic markers of susceptibility in models of child attachment. For example, the dopamine receptor D4 gene (DRD4) is regarded as a central moderator in differential sensitivity models by virtue of its putative role in shaping the attentional, motivational, and reward circuits in the dopaminergic system (Robbins & Everitt, 1999). Bakermans-Kranenburg and van IJzendoorn (2006) found that children with the DRD4 7-repeat allele (associated with lower dopamine efficiency) were more susceptible to attachment disorganization in the context of compromised maternal parenting but less susceptible when mothers were more sensitive. Implicating DRD4 as a moderator of attachment security and developmental outcomes, Bakermans-Kranenburg and van IJzendoorn (2011) reported that securely attached children with the DRD4 7-repeat allele evidenced greater prosocial behavior than those who did not. Research on child temperamental characteristics also conforms to differential susceptibility models of security. Children with more reactive temperaments displayed the highest gains in attachment security in interventions designed to improve maternal caregiving behaviors (Cassidy, Woodhouse, Sherman, Stupica, & Lejuez, 2011; Velderman, Bakermans-Kranenburg, Juffer, & van IJzendoorn, 2006).

Future Directions and Implications

In looking to the future, we selectively address three primary directions in adopting an evolutionary analysis of children's security in models of family functioning and child health.

Form and Function in Defense

A central advantage of evolutionary approaches for psychological frameworks is the focus on both the form and function of behavior. Many existing psychological conceptualizations attend to the form or structural properties of behaviors in defining concepts that are pivotal to understanding how the family impacts child functioning. For example, negative affect may be defined as intense, prolonged displays of distress through facial expressions or gestures in a specific context. However, problems emerge in the conceptual step of clarifying the role of structurally defined constructs in understanding pathways among family and child functioning. With a sole focus on the form of the behavior, psychological meaning is imposed onto the target constructs based on their intrinsic positive (i.e., strengths) or negative (i.e., deficits or impairments) attributes.

As a result, theory has been geared toward cataloguing the family risk factors and unhealthy outcomes associated with the inherently negative (e.g., destructive impairments) coping processes in the family and the positive precursors and healthy sequela of inherently positive (e.g., constructive competencies) ways of responding to family difficulties.

Evolutionary approaches posit that this way of thinking does not accurately reflect the function of children's patterns of responding to the family climate. Rather, the aim is to identify distinct patterns of action tendencies (i.e., form) on the basis of how they serve as adaptive solutions for regulating the relationship between the child and the environment in a way that successfully defuses different types of threat (i.e., function). Accordingly, a key direction is to refine and test pattern-based schemes that distinguish between children's unique profiles of social defense based on a more balanced analysis of both their form and function. The merits of this approach are already evident in our analysis of how hyperactivating (e.g., resistant) and deactivating (e.g., avoidant) strategies of attachment are designed to achieve the adaptive function of protection from harm within specific family niches (Cassidy, 2008; Hilburn-Cobb, 2004). However, future advances in attachment research may be facilitated by the systematic identification of self-protective strategies of attachment (e.g., diversity of strategies in the D attachment category) (Crittenden, Kozlowska, & Landini, 2010; Crittenden & Newman, 2010).

Moreover, very little work has been conducted in applying a functional approach to identifying qualitatively different profiles of insecurity in the interparental relationship (Davies & Sturge-Apple, 2007). However, our analysis of the literature on interpersonal threat suggests that four patterns may be evident based on their distinct functions: (a) secure strategy, reflecting the efficient operation of social defense permitting a balanced distribution of resources to other goals; (b) mobilizing strategy, characterized by efforts to defuse or avoid threat in a manner that sustains heightened arousal to social threat without sacrificing opportunities to access interpersonal resources; (c) dominant strategy, expressed through vigilance to threat in a form designed to directly defeat it through aggression or intimidating posturing; and (d) demobilizing strategy, serving to reduce salience or "lay low" as a way to limit the likelihood of drawing the attention or ire of aggressive dominants (Davies & Sturge-Apple, 2007; Gilbert, 2001). Because evolutionary models suggest that each pattern of security in the interparental relationship is associated with specific configurations of family antecedents and developmental consequences, pattern-based approaches may aid in more precisely identifying pathways between family characteristics, children's patterns of insecurity, and their psychological adjustment. For example, the lay-low strategy of a demobilizing response to interparental conflict is theorized to emerge in ecologies containing high levels of frightening parental behaviors, family violence, and power hierarchies organized by intimidation, while the dominant strategy may be an adaptive solution to contending with parental displays of vulnerability (e.g., depression, anxiety), disengagement, and collapses in the power hierarchy (Davies & Sturge-Apple).

Adaptation, Competencies, and Resources

Delineating the sequela of children's insecurity in the family can also be facilitated by considering the unique developmental advantages and costs associated with specific strategies for coping with stressful events (Del Giudice et al. 2011; Ellis et al., 2006; Korte, Koolhaas, Wingfield, & McEwen, 2005). Even strategies that appear to be inherently negative based on a surface analysis of their structural characteristics likely confer some adaptive benefits. For example, by virtue of its impact on other behavioral systems, hyperactivation of security systems (e.g., mobilizing strategies in the interparental relationship, ambivalent parent–child attachments) may predict some degree of impairment in social skills, peer acceptance, problem-solving skills, and prosocial orientations. However, in spite of its developmental disadvantages, underlying motives to maintain affiliative ties are proposed to confer a unique portfolio of strengths characterized by moderate levels of communion, social interest, and openness to intimacy. Likewise, although children who exhibit security in family relationships may enjoy a higher likelihood of forging healthy, competent trajectories, the developmental landscape may not be uniformly rosy. For example, security may be achieved at the cost of developing proficient systems for detecting threat. Thus, under some conditions (e.g., shifts in interpersonal ecologies such as new peer networks), security may increase the likelihood of victimization.

At a broader level, it is important to note that our treatment of family and child functioning is not intended to exhaustively address all family pathways and mechanisms. As with any mid-level theory, EST-R is designed to partially, as opposed to fully, explain associations between family and child functioning. Given our emphasis on identifying the role of defense and protection, an important future direction is to broaden the conceptual scope to address how dimensions of family resources may alter some of the behavioral systems organizing approach behaviors. For example, parental abilities to coordinate mutuality, synchrony, and dyadic responsiveness in exchanges may lay the groundwork of the acquisition of social skills and peer adjustment (Lindsey et al., 2009). Likewise, although our proposal that destructive conflict cumulatively supersedes the impact of constructive conflict on children's security processes, constructive conflict is theorized to signify increased accessibility to interpersonal resources that are manifested in the experience of positive emotionality and the greater salience of affiliative goals (Davies et al., 2012). As an outgrowth of this process, children witnessing higher levels of cooperation, warmth, and resolution may be better positioned to internalize interpersonal values and rules, acquire interpersonal skills and standing, and master social environments.

Developmental Processes

Finally, examining the security systems within developmental frameworks is a necessary step in further refining evolutionary theories on children's adaptation to family contexts. The virtues of a developmental perspective are evident in the study of

life histories in evolutionary frameworks and its goal of understanding the origins, nature, and consequences of how organisms dedicate energy to fitness goals (i.e., security, affiliation) across their life span (Crittenden et al., 2010; Del Giudice et al. 2011). For example, in addressing the nature of how organisms allocate resources to parent–child and interparental security systems over time, significant gaps remain in understanding how the form and function of each security system in the family changes over development (Cummings & Davies, 2010; Mayseless, 2005; Thompson & Raikes, 2003). Supporting heterotypic continuity, under some conditions, developmental processes produce changes in the form or expression of security without altering the fundamental meaning or function. In other developmental circumstances, changes in measurable signs of security may be manifestations of changes in function. For example, in applying the dynamic-maturational model of attachment across broad periods of the life span, Crittenden and colleagues have illustrated how maturational processes may, in some cases, alter the way the same protective strategy is expressed (i.e., same function in a different form) and, in other cases, may trigger increasing differentiation of strategies that reflect new forms with different functions (Crittenden, 1992; Crittenden et al.).

A developmental focus may also help to identify potential shifts in constellations of risk and protective factors and sequela of security across developmental periods. Neurobiological changes, normative experiential transitions, and their interplay may amplify or dilute the magnitude of pathways between family processes, children's security, and their health. Exposure to stress and adversity during these key periods may slow or disrupt the development of sensitive brain regions, becoming a preexisting risk factor for later stress-related disorders or disease (Lupien, McEwen, Gunnar, & Heim, 2009; Shonkoff, Boyce, & McEwen, 2009). Crittenden and Dallos (2009) pinpointed four key sensitive periods for understanding the etiology and implications of individual differences in attachment security: (a) the 2-year-old stage, (b) the transition to school, (c) puberty, and (d) the transition to parenthood. In focusing on neurobiological distinctions across development, Del Giudice et al. 2011 proposed that plasticity is particularly pronounced around periods that mark significant neurobiological shifts, including (a) toddlerhood (2–3 years of age), (b) childhood (6–7 years of age), (c) early adolescence (11–12 years of age), and (d) adulthood. Prospective data is still needed to systematically test whether the origins, correlates, and sequela of emotional security vary across developmental periods.

Conclusion

In closing, continued progress in understanding how and why family processes affect children's mental and physical health will require more systematic efforts to formulate and test theoretically guided hypotheses with greater novelty and pinpoint accuracy (Richters, 1997). Without the use of theory as a guide, we are running the risk of falling into the trap of organizing the complex findings on family

processes in the form of broad, imprecise, and relatively obvious conclusions. Take-home messages indicating that toxic family environments increase children's vulnerability to impairments in health and illness by engendering inherently negative response styles are reaching a point of diminished returns. Similarly, conclusions that positive and resource-rich environments promote healthy outcomes by fostering positive adaptation to environments offer little in the way of novelty. Our goal in this chapter was to illustrate how an evolutionary framework of children's emotional security in family relationships may offer some headway increasing depth and precision in interpreting the existing findings, generating novel hypotheses, and serving as a broad heuristic for future research.

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Chapter 6
Family Influences on Children's Mental and Physical Health: Some Contributions of and Challenges to the Emotional Security Theory

Barbara A. Morrongiello and Michael Corbett

Abstract In their chapter, *Family Discord and Child Health*: *An Emotional Security Formulation*, Davies, Sturge-Apple, and Martin (Chap. 5) link children's mental health outcomes to emotional and communication patterns expressed between parents. In this chapter, we discuss, from a clinical/interventionist perspective, several unique contributions made by emotion security theory that help to explain the impact family discord has on children's mental health. We then place this theory within a broader context of research on child health outcomes and discuss some challenges for this theory by exploring the role of compensatory parent influences, differential susceptibility to risk exposure in children, socioemotional development, bidirectional parent–child influences, and extrafamilial influences on children's mental health. In discussing these challenges, we consider findings from research on factors that influence unintentional childhood injury risk.

The contribution of family processes to children's mental and physical health is well documented. In the mental health domain, for example, family factors have been linked to mood and behavioral disorders. Depression, anxiety, aggression, antisocial behavior, and conduct disorder have been associated with a variety of family processes including parent–child dynamics, parenting style, family cohesiveness, and divorce (Granic & Patterson, 2006; Oyserman, Bybee, & Mowbray, 2002; Strohschein, 2005; Vostanis et al., 2006). With regard to physical health, families have been shown to impact children's adjustment to disease (Kazak, 1989), adherence with medical treatments (Fiese & Wamboldt, 2000), and the adoption of risk and health behaviors (Christensen & Morrongiello, 1997; Morrongiello & Bradley,

B.A. Morrongiello, Ph.D. (✉) • M. Corbett, M.A.
Department of Psychology, University of Guelph,
Guelph, ON, Canada
e-mail: bmorrong@uoguelph.ca

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1997; Morrongiello, Corbett, & Bellissimo, 2008). Indeed, in a comprehensive review of research focused on characteristics of family systems and physical health, Reppetti, Taylor, and Seeman (2002) found that emotional and communication patterns expressed within families may be linked to later risk for chronic disease and early mortality in adulthood due to earlier stress on children's physiological and neuroendocrine systems. Children in these families also evidenced diminished ability to seek social support and develop other effective coping strategies for managing stress, as well as showing increased vulnerability for behavior problems and substance abuse (e.g., smoking, alcohol, drugs, and sexual promiscuity). These immediate and lifelong risks are presumably interrelated through common biological and psychosocial pathways extending from the family system.

Importantly, recognition of the interrelatedness of marital, sibling, and parent–child relationships and the significance of these relationships for both children's mental and physical health has fostered greater emphasis on family system processes not only in research but also in practice (Anderson, Brackett, Ho, & Laffel, 2000; Campbell & Patterson, 1995; Fiese & Sameroff, 1989; Johnson, 2000; Kazak, Rourke, & Crump, 2003; Seagull, 2000). In this chapter, we consider some select findings that highlight the important role that families play in impacting developmental trajectories for children's mental and physical health. In the first section we comment on several unique contributions of an evolutionary reformulation of emotion security theory (EST-R) developed by Davies and colleagues (see Davies, Sturge-Apple, & Martin, Chap. 5) to explain the impact family discord has on children's mental health. We then place this theory within a broader context of research on child physical health outcomes and discuss some challenges for this theory. In discussing these challenges, we consider findings from our own research on factors that influence unintentional childhood injury risk.

Unique Contributions of the Theory

In their chapter, *Family Discord and Child Health: An Emotional Security Formulation*, Davies et al. (Chap. 5) draw on evidence linking children's mental health outcomes to the emotional and communication patterns expressed between parents and to which children are exposed. There are a number of unique aspects of EST-R for which Davies et al. (Chap. 5) should be commended. First, their emphasis on the importance of examining the *parent–parent* relationship fills a void in a literature that has focused predominantly on the parent–child relationship in studies of children's social–emotional development. Historically, numerous studies have called attention to the fact that exposure to interparental aggression often produced negative social–emotional effects on child witnesses (Kitzmann, Gaylord, Holt, & Kenny, 2003). Davies' research, however, focuses more intently on the nature of aggressive parent–parent exchanges in order to identify those features of the

interaction that produce negative effects on children. This research has helped differentiate the nature and scope of parent–parent interactions that are concerning from those that are not. For example, elaborating different forms of destructive conflict and identifying those that will likely do the most harm is a substantive contribution that aids in strategic planning of therapeutic interventions.

Second, their longitudinal research allows them to focus on the antecedent *processes* and *mechanisms* within the family system. This approach provides an important foundation for understanding the varied and complex pathways to negative child outcomes within the family context. Process-level accounts that focus on the relational context and heterogeneity of developmental phenomenon have proven highly effective frameworks for both understanding and preventing negative child outcomes (Granic & Patterson, 2006). Longitudinal research examining developmental trajectories under different family and parent relationship conditions provides unique and critical evidence on which to make decisions about the nature, scope, and timing of interventions designed to reduce the risk of negative child outcomes. Moreover, it is often the case that the psychopathology that appears in children is linked to antecedent factors that may seem unrelated. For example, current parenting issues are often rooted in much more distal factors, including the intergenerational transmission of ineffective parenting strategies (Avakame, 1998; Doumas, Margolin, & John, 1994). Hence, elucidating how risk factors within families are dynamically related over time and the processes by which they lead to poor child outcomes provides critical knowledge for planning effective and efficient interventions with families at risk.

Finally, the adaptation of an evolutionary framework to facilitate our understanding of the *form* and *function* of child security processes and how these impact children's health is novel, and this framework successfully integrates many diverse findings. Too often in child health research, and health research more generally, theory is underutilized as a tool for explaining why people behave in health-compromising ways or how negative health consequences are actually realized (see Gielen & Sleet, 2003; Glanz & Rimer, 1997 for further discussion). Understanding the functionality of behaviors is particularly important in clinical interventions aimed at changing these behaviors (Ensor & Hughes, 2005; Ferguson, 2008). Davies et al.'s (Chap. 5) theory, therefore, serves to remind us of the importance of understanding behavior within a functionality framework.

Extending and Elaborating the Model: Some Challenges to Address

Although the model outlined by Davies et al. (2011) (Chap. 5) makes an important contribution to the field of child psychopathology, there is evidence that challenges some of the theoretical assumptions and that suggests directions for future research to further test and potentially expand on the model.

Compensatory Parent Influences

According to Davies et al. (Chap. 5), the parent–parent relationship has a major impact on child adjustment. However, a broader review of the pediatric health psychology literature reveals that often the best predictor of child adjustment is maternal adjustment and mental health (Coplan, Arbeau, & Armer, 2008; DeMaso et al., 1991; Lipani & Walker, 2006; Wallander & Varni, 1998). When mothers are doing well, their children usually do well in managing stress, coping with illness, and general life adjustment. There are multiple sources of relationships that can impact feelings of security for a child within a family. Thus, research is needed to explore how individual parent well-being interacts with parent–parent relationship quality to impact child outcomes. We know from our own research on children's perceptions of parents' safety practices, for example, that young children are well aware of differences between parents in safety attitudes and risk practices (Morrongiello, Corbett, & Bellissimo, 2008). Hence, parents individually, as well as collectively, impact children's belief systems and behaviors. Can one well-adjusted and emotionally competent parent, therefore, communicate sufficient security for the child to counteract the potential negative effects of exposure to parent–parent conflicts?

Accounting for Differential Susceptibility

A common finding in the developmental literature is that not all children who are exposed to adversity experience adjustment difficulties. Greater specification of how differential susceptibility to negative effects of parent–parent conflict arises within the Davies et al. (Chap. 5) model is needed. According to the model, repeated exposure to parent–parent conflicts undermines the child's security in the interparental relationship. This can lead to emotional difficulties for the child. With repeated exposure there is greater development of a negative schema, and increased vigilance of conflict presumably emerges. However, not all children within a family experience clinically significant levels of distress even in highly dysfunctional/conflicted families. For example, in a review of literature on the impact of exposure to domestic violence on children, it was noted that ~33 % of children did not experience adjustment difficulties (Holt, Buckley, & Whelan, 2008). Thus, the impact of parent–parent conflict on the child is seemingly moderated by other factors. Within Davies et al.'s model (Chap. 5), one likely moderator that merits further study is the child's *repeated emotional reactions* to parent–parent and parent–child conflicts. It would be informative to tease apart the main effects of the moment to moment emotional reaction to conflict as compared with a child's felt sense of insecurity in the parent–parent relationship. It is likely that these two effects are related in important ways, but each also may account for unique variance in children's long-term adjustment.

Another possible moderator relates to child personality or temperament. For example, dispositionally highly anxious children may be more likely to react to

conflict with greater aversive negative emotion and stress than less anxious children (e.g., Gazelle, 2006; Waters, Neumann, Henry, Craske, & Ornitz, 2008), and these reactions may not diminish but may actually increase with repeated exposure. Furthermore, anxious children may be more likely to develop avoidant coping strategies in the face of repeated conflict as a way of reducing high levels of aversive emotion (e.g., Wilson & Hughes, 2011), whereas less anxious children may be more inclined to attempt to intervene in the conflict. Thus, within the emotional security framework, the child's emotional reactivity may help to explain unique variance in the *disoriented* attachment behaviors that cannot be explained by the characteristics of the conflict alone. Hence, an objectively frightening conflict is likely to engender differential emotional reactions depending on the child, and these reactions are likely to have a moderating effect between the type of conflict and the behavior elicited by it. Moreover, these conflict–reaction–behavior patterns are very likely to change with repeated exposure to interparent conflicts.

Additionally, if one is focused on relational influences on child health and mental well-being, then another likely moderator is child gender. One meta-analysis, for example, found negative psychosocial outcomes for preschool-aged girls in response to witnessing domestic violence, but not in boys of the same age (Kitzmann et al., 2003). How would such difference arise within the emotional security theory? The critical point here is that we need to better understand how *differential susceptibility* arises and operates within this model, and further research on moderators and mediators is necessary to more fully explain this phenomenon. Understanding the complexity of factors and processes by which some children evidence resilience in the face of adverse family-based experiences, while others suffer poor outcomes, is critical to the development of effective prevention and treatment interventions (Egeland, Carlson, & Sroufe, 1993). In the child injury risk research, for example, boys are much more likely than girls to engage in risk taking and experience injuries (Morrongiello & Dawber, 2004; Morrongiello & Hogg, 2004; Morrongiello & Rennie, 1998). Identifying the moderating factors that predict sex differences in risk taking (i.e., boys rate injury vulnerability and potential injury severity lower than girls and attribute injuries to bad luck more so than their own behaviors) points to the need for different interventions to reduce risk taking and prevent injury to boys compared with girls (Morrongiello, 1997; Morrongiello & Rennie, 1998). Hence, identifying key moderators can inform intervention planning in important ways.

Social–Emotional Development

Another challenge to the model that raises a number of questions is that there seems to be very little emphasis on the role of child development as a major factor driving the process of emotional security formation, attachment, and child adjustment. How do children's emerging competencies in understanding relationships and emotions, and regulating their own emotions, impact these risk processes? Can developments in emotional intelligence, for example, counteract effects of exposure

to interparent conflict? Processing of emotional information and emotional self-regulation skills are critical for making sense of the interparental conflict to which one is exposed. These skills develop as children age and there can be considerable variability in these developments at a given age (Kopp, 1989; Thompson, 1989). So, is the impact of parent–parent conflict dependent on child age and emotional intelligence? We need to understand if there are qualitative differences in impact as a function of child age.

Extending this logic, it would seem that children who are poorer at understanding negative emotions and conflict within relationships would be at greater risk for poor outcomes in response to exposure to interparent conflict. Does this mean there is a *critical period* for negative impact of exposure to interparent conflict, with younger children possibly at greater risk than older children? At the least, it would seem to suggest that there will be qualitatively *different* impacts of interparent conflict for children at different ages, depending on the child's level of emotion regulation and intelligence. The importance of emotion regulation for child health outcomes has also been noted in research on child risk taking. Children who show poorer emotion regulation skills engage in greater risk taking (Morrongiello, McArthur, Kane, & Bell, 2012), as do adolescents (Cooper, Wood, Orcutt, & Albino, 2003; Hessler, Hopko, Bare, Lejuez, & Robinson, 2010; Lightfoot, 1997). Hence, expanding the model to account for developmental changes in emotion regulation would seem to be an important consideration.

Bidirectional Parent–Child Influences

There is a great deal of evidence now in the socialization literature that *bidirectional* processes operate within families such that parenting influences children's behavior and, conversely, how children behave and react feeds back to effect changes in parenting (Kuczynski, 2003; Kuczynski, Marshall, & Schell, 1997; Patterson & Fisher, 2002). How are these processes to be accounted for by Davies et al.'s model (Chap. 5)? To understand how child development shapes health outcomes, it is critical to understand how children both respond to and shape their social environments and relationships. What is implicated in health outcomes research is not centered in child characteristics *or* social/environmental influences but rather in the bidirectional and dynamic interplay of these diverse factors.

Bidirectional processes become increasingly important as children age and become more autonomous and agentic in their thinking and behavior. Parents of teens realize quickly that they are losing power and control and need to allow for give/take bidirectional processes in order to keep the lines of communication open. For example, for many years it was *assumed* that knowledge parents' gained about their child's activities was based on their monitoring of the child; in other words, a parent-to-child directional process was assumed. However, research by Stattin and Kerr (2000) showed that this knowledge arose mostly by adolescent disclosure, and the quality of parent–child relationship impacted this disclosure process.

Thus, what was long assumed to be a unidirectional process that gives rise to parent knowledge is in reality a bidirectional one that arises from a dynamic give and take relationship that teens and parents share.

Bidirectional parent–child processes also have been noted to play an important role in understanding children's unintentional injury risk. Boys experience more injuries and engage in more risk taking than girls (Borse et al., 2008; Morrongiello, 1997; Morrongiello & Dawber, 2004; Morrongiello & Hogg, 2004; Morrongiello & Rennie, 1998; Schwebel & Gaines, 2007), and considering the parent–child relationship has provided important insights into reasons for this. Research has demonstrated that girls are more likely than boys to disclose to parents when they have done something risky and experienced a minor injury when playing with peers. By virtue of talking with parents, girls then provide parents many more opportunities for discussing safety and risk than do boys. Hence, these *teachable moments* for girls arise based on child disclosure. Sex differences in disclosure arise, in part, because parent reactions differ to boys' versus girls' disclosure about risk taking and minor injuries. Girls' disclosure engenders reactions of concern and disappointment, whereas boys are often met with anger and frustration (Morrongiello, Zdzieborski, & Normand, 2010). Thus, there exist reciprocal influences between child expectations about parent emotional reactions and parent emotional reactions to children's disclosure. These bidirectional effects result in differential experiences for boys and girls that contribute to boys having greater risk of injury than girls.

Similarly, a number of studies have shown that children's risk-taking behaviors are in part determined by what they think their parents will allow them to do and how they expect parents to react (Morrongiello & Dawber, 2004; Schwebel & Bounds, 2003). Hence, children often show greater risk taking with fathers than mothers, because they believe fathers are more tolerant of these behaviors than mothers. Reciprocally, parental safety practices are heavily influenced by child factors and thus operate differently for different children within the same family. Research has demonstrated, for example, that parents implement safety practices based on their beliefs about their children's personality and anticipated reaction to their safety practices (Morrongiello & Kiriakou, 2004; Morrongiello & Lasenby-Lessard, 2007). They implement fewer precautions, for example, for children they believe are high in inhibitory control and who can be trusted on their own to follow safety rules. Interactions between child behavioral attributes and parent supervision differentially predict injury outcomes (Morrongiello, Klemencic, & Corbett, 2008). For example, attentive supervision can compensate for some child behavioral attributes (e.g., low in inhibitory control) but not others (e.g., high in sensation seeking). Thus, within the family context, child injury risk is determined by interactions of child characteristics and parenting behavior, and these factors show bidirectional influences; neither on their own would adequately explain children's risk of injury. Based on these diverse findings, therefore, expanding Davies et al.'s theory (Chap. 5) to account for bidirectional influences would seem an essential step and one that is likely to reveal age-dependent variation in the risk processes that link exposure to interparental conflict with child adjustment outcomes.

Extrafamilial Influences

Finally, when evaluating influences on children's health, it is also important to take into account broader levels of influence. Could positive extrafamilial relationships protect against any of the negative impacts of destructive parent–parent conflict? Findings in the resiliency literature suggest that extrafamilial and extended family relationships can provide much needed social support that can counteract potentially negative family-based experiences of children (see Armstrong, Birnie-Lefcovitch, & Ungar, 2005). Similarly, peer support has been shown to predict adjustment of children to cancer and other physical health illnesses (Varni, Katz, Colegrove, & Dolgin, 1994; Varni, Setoguchi, Rappaport, & Talbot, 1992). How would such extrafamilial processes be accounted for in the Davies et al. (Chap. 5) theory?

In the parenting and risk-taking literature, there is a great deal of evidence indicating that the age or developmental stage of a child determines the relational context to which they are most exposed, and this has implications for injury risk (Agran et al., 2003; Rivara, Calonge, & Thompson, 1989; Scheidt et al., 1995). For example, toddlers and preschool-aged children are most often injured at home where parents can have a great deal of influence on these outcomes (Morrongiello, 2005; Morrongiello, Ondejko, & Littlejohn, 2004a, b), whereas elementary school children are most often injured outside of the home while alone or in the company of peers (Shannon, Brashaw, Lewis, & Feldman, 1992). Children at these ages are making risk decisions in a completely different socioemotional context wherein parents exert very little influence. Indeed, many have argued that the peer context is one of the greatest risk factors for injury at these ages (Sandels, 1977; Wilson, Baker, Teret, Shock, & Garbarino, 1991). In a series of studies we have shown that the extent to which children are successfully persuaded by peers to engage in risk behaviors depends on the value they place on the relationship. The more they value the relationship, the more importance they place on what the peer is saying and the more they want to share in the experience with the peer—all of which results in them engaging in the risk behavior that the peer is encouraging (Morrongiello & Dawber, 2004).

Clearly, there are powerful influences at work in extrafamilial contexts that can lead children and adolescents to, quite literally, risk their lives. It seems highly unlikely that relationships that can have such a powerful influence on behavior would not have an influence on emotional development. In fact, a number of authors have shown that emotion is a critical component driving risk-taking decisions, especially in adolescence (Lightfoot, 1997). With regard to EST-R, these diverse findings suggest that it may be critical to consider the role of different relational contexts (e.g., peers, teachers, other family members, coaches) in determining security systems and attachment styles, as these contexts may be more important determinants of interpersonal development at older ages than are familial relations. Indeed these extrafamilial relationships may represent important confounds to the model. The only way to determine their relative influence compared to the familial context

may be to include them in the model and test for the unique effects of multiple relational contexts on adjustment outcomes.

Conclusion

Understanding the mechanisms and processes by which risk factors lead to poor child adjustment is essential for prevention planning and intervening with high-risk families. EST-R (Davies et al., Chap. 5) applies an evolutionary framework and emphasizes the importance of the parent–parent relationship for children's emotional adjustment. Although the theory advances our understanding of how repeated exposure to parent–parent conflict produces long-term negative impact on children's social–emotional well-being, this chapter discusses some substantive challenges to the theory and suggests future directions for research to address these issues and further enhance the theory.

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Chapter 7
Emotional Insecurity and Child Health: Implications and Future Directions

Dennis Drotar

Abstract This chapter describes the research and clinical implications of Davies, Sturge-Apple, and Martin's (Chap. 5) emotional security formulation (EST-R) for child health. Research priorities include the following: (1) identify key features of family discord that affect emotional security and child health outcomes, (2) clarify the specific processes by which emotional insecurity affects child health risks and outcomes, (3) clarify the timing and impact of family discord and emotional insecurity on age-specific developmental competencies that affect child health outcomes, (4) identify key and protective factors that influence the impact of family discord on child health, and (5) clarify the implications for preventive interventions that address specific risks associated with emotional insecurity for various child health outcomes. A range of intervention strategies can be targeted to the functional consequences of family discord on the social defense, stress, or behavioral systems.

Introduction

Davies, Sturge-Apple, and Martin's (Chap. 5) emotional security formulation (EST-R) is a comprehensive and creative integration of the critical processes by which family discord can affect child health. The purpose of this chapter is to describe the research and clinical implications of this theory for child health with a specific emphasis on the long-term psychological and health outcomes for pediatric chronic illness.

D. Drotar, Ph.D. (✉)
Division of Behavioral Medicine and Clinical Psychology, Cincinnati Children's Hospital, Cincinnati, OH, USA
e-mail: dennis.drotar@cchmc.org

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Public Health Significance of the Impact of Family Discord on the Health Risks and Outcomes Experienced by Children, Adolescents, and Young Adults

Family risk factors such as family and marital discord have a salient influence on a wide range of child health risks and clinically significant health outcomes. These include conditions with high prevalence and impact on health such as overweight and obesity, the onset and course of chronic pediatric illnesses such as type 1 and type 2 diabetes, symptom control of chronic illnesses such as asthma, nonadherence to preventative health behaviors and prescribed medical treatment in pediatric chronic illness, substance abuse, and accidental and non-accidental injury (Danese, Pariante, Caspi, Taylor, & Poulton, 2007; Drotar, 2000; Felitti et al., 1998; Repetti, Robles, & Reynolds, 2011). The public health significance of the wide range of health problems that can be influenced by family discord stems from the following issues: Such problems begin in childhood and adolescence and last a lifetime, pose significant risk to long-term health and development, are very burdensome to families, and are costly to society.

It is important to recognize that such health problems are very difficult to prevent or ameliorate once they have been set in motion in what can be a cumulative escalation of risk. For this reason, prevention and/or amelioration of such highly prevalent, clinically significant health outcomes represent a critical opportunity. Comprehensive empirically supported models that link critical family influences such as discord with child health outcomes can inform such prevention efforts. Davies et al.'s (Chap. 5) emotional security formulation, which identifies the critical mechanisms by which family discord shapes important health outcomes, can inform the design of interventions by (1) identifying novel targets of preventive interventions that may have more powerful effects than currently available methods, (2) informing selection of the most optimal timing of preventive interventions to reduce negative health outcomes during the course of development, and (3) suggesting innovative and effective methods of prevention and treatment to ameliorate and prevent deleterious health outcomes associated with family discord.

Family Influences on Child Health: The Contribution of Emotional Insecurity

The emotional security model (EST-R) developed by Davies et al. (Chap. 5) underscores several key roles of parents as socialization figures in the development of children's health outcomes: For example, parents are models for adopting and sustaining optimal health outcomes or, alternatively, problematic health outcomes, during the course of the child's life span. Parents also serve as primary managers of their children's behavior by monitoring and enforcing guidelines for behaviors that can enhance or disrupt children's health. Finally, parents direct and control family

resources (e.g., time, attention, reinforcement) that facilitate critical socialization goals for children's health promotion.

One critical contribution of Davies et al. (Chap. 5) is the identification of emotional insecurity as a key mediator of the impact of family discord on child mental and physical health. Children's experiences with aggression, emotional detachment, and unresolved antagonism in the family can enhance their vulnerability to a range of mental and physical problems by undermining the child's sense of security in family relationships. In turn, emotional insecurity has multifaceted effects on the following systems, each of which has both short- and long-term potential effects on child health: (1) social defense and attachment systems, (2) stress response system, and (3) behavioral system, which involves the allocation of family resources to child health (Davies, Cummings, & Winter, 2004; Davies, Sturge-Apple, Cicchetti, & Cummings, 2008).

Implications for Future Research and Clinical Intervention

The emotional insecurity theoretical formulation and supporting data have demonstrated promising findings on children's mental health (Davies et al., Chap. 5) and relevant implications for mental health intervention (Davies, Winter, & Cicchetti, 2006). What is not understood as well are the potential implications of family discord triggered by emotional insecurity for research and interventions to improve children's health. To address these unmet needs, implications are now described.

Research priorities that will extend the findings based on the emotional insecurity formulation include the following: (1) identify key features (e.g., individual differences in the type and duration of exposure) of family discord that affect emotional insecurity (e.g., child health outcomes), (2) clarify and elaborate the specific processes by which emotional insecurity affects specific health risks and outcomes, (3) clarify the timing and impact of family discord and security on age-specific developmental competencies that affect child health outcomes, (4) identify key risk and protective factors that influence the impact of family discord and emotional insecurity on child health, and (5) clarify the implications for preventive interventions that address specific risks associated with emotional insecurity for various child health outcomes.

Identify Key Features of Family Discord That Affect Emotional Insecurity and Child Health Outcomes

Family discord is a highly complex, multifaceted variable. There are substantial individual differences in family discord and profiles of associated risk and protective factors (Davies et al., 2004). For this reason, the critical features of family discord that influence the development of emotional insecurity and ultimately affect children's health risk and long-term health outcomes need to be understood. For example, the

type, intensity, and duration of family discord would be expected to impact the level and chronicity of insecurity experienced by the child but is not well understood.

A substantial body of research has also indicated that factors associated with family discord such as the level of threat experienced from the discord, child abuse, and/or neglect can influence the development of emotional insecurity and eventually impact health (Felitti et al., 1998). Moreover, the nature of the child's involvement in family discord should also be considered in understanding the impact of family discord on child health and eventually in designing interventions to prevent the negative consequences. For example, a child's experience of marital violence is a specific aspect of family discord that has implications for the development of mental health problems such as posttraumatic stress disorder which has wide-ranging implications on children's mental and physical health (Schwartz & Perry, 1994).

Elaborate the Processes by Which Family Discord and Emotional Insecurity Affect Specific Child Health Outcomes

Davies et al. (Chap. 5) have described salient pathways by which family discord and emotional insecurity affect children's psychological outcomes. However, the specific pathways and mediators that are most influential (and hence are potential targets of preventive intervention) would be expected to vary as a function of the specific health risk or outcome. Health risks and outcomes are heterogeneous in their causal pathways, and emotional insecurity might be expected to influence them in different ways. For example, the impact of family discord on allostatic load (McEwen, 1998) might be expected to be most powerful in the development of symptoms that can be triggered by stress and emotional insecurity in chronic health conditions such as asthma (Sandberg et al., 2000). On the other hand, the negative impact of family discord on such processes as caregivers' communication regarding the child's management of chronic illness, monitoring the child's medical treatment management, and/or development of family routines would be expected to have the most powerful influences on the child's capacity for adaptive self-management and adherence to medical treatment (Modi et al.,2012).

Clarify Timing and Impact of Family Discord and Emotional Insecurity on Developmental Competencies That Affect Long-Term Child Health Outcomes

The timing of the impact of family discord and emotional insecurity on specific proximal risks versus long-term health outcomes is another important but unanswered question. Stressors such as family discord have ongoing cumulative and

cascading effects that exert differential influences at various times in development, such as during key developmental transitions (e.g., onset of adolescence) (Repetti et al., 2011). Identification of the ways in which family discord and emotional insecurity affect the critical processes that are clear precursors for clinically significant health outcomes at key times in development will inform both the timing and specific targets of preventive interventions. Such data are particularly important because once established, the psychological and health risks of emotional insecurity appear to have increasingly powerful or cascading effects on subsequent health outcomes (Repetti et al., 2011).

Identify Key Risk and Protective Processes That Influence the Impact of Family Discord and Emotional Insecurity on Child Health Outcomes

Family discord is one of a host of potential family influences on emotional insecurity and child health outcomes. Family risk and protective factors and processes which heighten or lessen the effects of family discord on child health need to be considered in developing targeted interventions to reduce the risk for negative outcomes. For example, in families marked by discord, parental modeling of negative health behaviors and inconsistent support and guidance to develop and sustain positive health behaviors are likely to increase the risk for problematic health outcomes among children with chronic illness (Ellis et al., 2007). On the other hand, the development of family cohesion and routines, support from parents and siblings, and strategies of reinforcing positive health behaviors by parents are likely to buffer the impact of family discord and emotional insecurity on negative child outcomes (Fiese, Wamboldt & Anbar, 2005). Individual differences in children's psychophysiologic reactivity to stresses such as family discord are also expected to influence children's health and adaptation to chronic illness (Treadwell, Alkon, Guirolo & Boyce, 2010).

Implications for Developing Preventative Strategies of Intervention for Children's Health Outcomes

Interventions for children who experience family discord focused on family processes, including interparental conflict and parenting practices, have shown positive effects on children's psychological outcomes (Taylor & Biglan, 1998; Webster-Stratton, Reid, & Hammond, 2004). Davies et al.'s (Chap. 5) emotional security model has identified several critical functional consequences of family discord learned by children and parents that may be important targets of intervention. For example, family discord sets in motion processes that disrupt the social defense, stress, and/or behavioral systems and threaten physical and mental health.

As shown in the following case vignette, the above processes and systems may provide effective targets for interventions that are designed to ameliorate or prevent the negative consequences of marital discord on children's health: *Johnny is a 16-year-old with severe asthma whose symptoms have become increasingly frequent and difficult to manage. His physician has learned that some of his symptoms are triggered by emotional insecurity including anxiety and worry about his parents' chronic marital problems. The management of his asthma has also been complicated by his nonadherence to his daily preventer medication and inconsistent use of his rescue medication in response to his symptoms. His parents' monitoring of his medication use is inconsistent and they disagree about how to help him manage his asthma. His symptoms have become progressively worse and resulted in multiple emergency room visits and hospitalizations. The medical team has become increasingly concerned about Johnny and wonder about the best way to help him.*

Johnny's multifaceted set of family stressors and problems in chronic illness management are shared by many children and adolescents (Modi et al., 2012). Davies et al.'s (Chap. 5) emotional insecurity formulation suggests that various interventions to improve Johnny's symptoms and long-term health can be targeted to address key mediating processes in several domains. One such area is parent–child insecurity. For example, family-centered intervention might be focused on Johnny's relationship with his parents in order to help Johnny manage his anxiety about his parent's relationship and help his parents appreciate the impact of their marital problems on his symptoms. The eventual goal of the intervention would be to reduce the likelihood that emotional insecurity would trigger Johnny's asthma symptoms.

A second potential target for intervention that might be very helpful to Johnny concerns the stress response system. Johnny's recurrent activation of his stress response system in response to his parents' conflict may trigger asthma symptoms, which can in turn impact his long-term health and well-being. Empirically validated intervention strategies such as stress management (McQuaid & Nassau, 1999) may be an effective intervention to help Johnny manage his stress response more adaptively.

Finally, interventions directed toward the behavioral system in order to promote Johnny's resilience to the health risks and outcomes posed by family discord and emotional insecurity might also prove to be useful in helping him manage his asthma more effectively. Empirically supported interventions can be directed toward enhancing family problem-solving skills (Wysocki et al., 2006) and increasing the consistency of his parents' monitoring and supporting his adherence to treatment (Ellis et al., 2007).

Future Directions

Davies et al.'s (Chap. 5) creative work challenges us to develop and extend the emotional security theoretical formulation and relevant research. Several theories (Hobfoll, 1988; Muraven & Baumeister, 2000) have converged on the principle of

resource allocation emphasizing that families and individuals have a finite number of resources to devote to psychosocial adaptation in response to day to day or extraordinary stress. Increased demands on resources such as those posed by family discord can overwhelm the system and trigger maladaptive strategies that can result in negative short- and long-term consequences on physical and mental health. Research that focuses on understanding how family-level resources affect the individual processes that are the key functional consequences of emotional insecurity described by Davies et al. (Chap. 5) would be a useful research direction. Another useful future research direction would be to delineate how the specific functional consequences of emotional insecurity (e.g., social defense, stress response, and behavioral systems) affect specific child health behaviors, risks, and outcomes. Finally, the development and evaluation of interventions designed to ameliorate or prevent children's health risks and negative long-term health outcomes by targeting the social defense, stress response, or behavioral systems (Davies et al., Chap. 5) would have important clinical and public health implications.

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Chapter 8
Could Emotional Security Theory Help Advance Family-Focused Preventive Interventions?

J. Douglas Coatsworth

Abstract In the following commentary, I examine the reformulated emotional security theory (EST-R) through the lens of a family-oriented prevention scientist. Over the past three decades, prevention science has accumulated an impressive body of findings that demonstrate the efficacy of family-focused preventive interventions for improving parenting and family functioning, while also decreasing child and adolescent mental, emotional, and behavioral problems. The following commentary begins with a brief overview of what we know about family-focused preventive interventions, highlights a few critical elements that we don't know, and then discusses how EST-R might help bridge that knowledge gap.

Could Emotional Security Theory Help Advance Family-Focused Preventive Interventions?

One of the goals of the preceding chapter and the series of commentaries was to focus on what we know about the mechanisms through which family experiences "get under children's skin" to influence their physical, psychological, and behavioral health. A comprehensive and complex process-oriented model, a reformulation of emotional security theory (EST-R; Davies, Sturge-Apple, & Martin, Chap. 5), was presented as one model that can increase the depth and precision by which we understand the linkages between risky family environments and children's health and well-being. As Kurt Lewin (1951, p. 169) noted, "There is nothing more practical as a good theory." Valid behavioral theories possess immense power to accelerate the development of novel empirical studies for understanding children's

J.D. Coatsworth, Ph.D. (✉)
Department of Human Development and Family Studies, The Pennsylvania State University, University Park, PA, USA
e-mail: jdc15@psu.edu

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developmental trajectories, and it appears that EST-R may fit this description. Lewin has also been credited with saying, "If you want to truly understand something, try to change it." It is with "change" as the frame of reference and with the backdrop of family-focused preventive interventions that I will address some issues raised by the presentation of EST-R. In particular, I examine whether EST-R can help us understand more completely how family-focused preventive interventions "get under the skin" of children and influence their health behavior. In doing so, I begin with a brief overview of what we do know about family-focused preventive interventions and then turn to briefly examining what we don't know before critically examining how EST-R might help fill some of those knowledge gaps.

Family-Focused Preventive Interventions: What We Know and What We Don't Know

By family-focused preventive interventions, I am referring to interventions that are designed specifically to work directly with one or more family members to influence family-level processes that are hypothesized to reduce risk for or increase protection against youth mental, emotional, or behavioral problems. Over the past three decades, the number of these kinds of interventions has increased substantially. One reason for this increase is conceptual: the family is one of the primary contexts for socialization of youth and, thus, is of great developmental significance. Theoretically, decreasing family risk or increasing family protection could produce early, powerful, and long-lasting effects on child development. Empirically, an extensive body of research has accrued that documents powerful family risk and protective factors for a variety of childhood and youth mental, emotional, behavioral problems (Conger & Elder, 1994; Dishion, Capaldi, & Yoerger, 1999; Hawkins, Catalano, & Miller, 1992; O'Connell, Boat, & Warner, 2009). Family-level risk and protective factors predict to broadband indicators of troubled development, such as internalizing and externalizing, and also to specific problem outcomes including adolescent substance use (e.g., Brook, Brook, Gordon, & Whiteman, 1990), risky sex (e.g., Perrino, González-Soldevilla, Pantin, & Szapocznik, 2000), and delinquency or conduct problems (Derzon & Lipsey, 2000; Loeber & Stouthamer-Loeber, 1986).

Two important broad areas of family risk for youth functioning are the discipline and supervision strategies that parents use to reinforce and shape appropriate youth behaviors. Youth in families that use discipline practices characterized by unclear rules and inconsistent enforcement are at elevated risk for problem outcomes (Dishion & Loeber, 1985). Similarly, harsh discipline including corporal punishment is linked to externalizing problems and substance use (Deater-Deckard & Dodge, 1997; Dishion & Loeber, 1985). In contrast, when parents use "inductive discipline," such as identifying problems and describing possible consequences, supplying rationales and explanations for discipline, and using noncoercive discipline, youth are more likely to develop prosocial competence and less likely to develop externalizing behaviors (Krevans & Gibbs, 1996) or depression

(Natsuaki et al., 2007). Poor parental knowledge, supervision and monitoring, an aspect of child management, is strongly associated with adolescent problem behaviors (Dishion, Capaldi, Spraklin, & Li., 1995; Laird, Pettit, Bates, & Dodge, 2003) and is central to models for the development and prevention of adolescent problem behavior. Parental monitoring is also a central explanatory variable for youth risky sexual behavior (Miller, Forehand, & Kotchik, 1999; Sagrestano & Paikoff, 1997; Tinsley, Lees, & Sumartojo, 2004).

The affective quality of parent–youth relationships is also an important predictor of developmental outcome. Warm and responsive parents build emotionally and instrumentally supportive parent–child relationships characterized by relatively high levels of positive affect and low levels of negative affect (Simons et al., 2006). Parental involvement, characterized by high levels of positive, nurturing, and supportive interactions and low levels of hostile and negative interactions, protects youth's adaptive development even in the face of stressful life circumstances (Conger & Conger, 2002; Masten & Coatsworth, 1998). Likewise, good parent–adolescent communication is protective. When parents clearly communicate their expectations about youth risk behaviors, youth tend to internalize the communicated norms (Whitaker & Miller, 2000) and have more positive developmental trajectories (Wills, Gibbons, Gerrard, Murray, & Brody, 2003).

Over a similar time frame, prevention science has accumulated empirical evidence, often from randomized clinical trials, that family-focused interventions can produce the desired effects. One way we know that family-focused interventions work is through published reviews, which have tended to conclude moderate effects for family-focused interventions (e.g., Kumpfer & Alvarado, 2003; Lochman & van den Steenhoven, 2002). A second way we know family-focused prevention works is because the best of these programs are compiled onto lists which are published or made publically available on websites. For example, Blueprints for Violence (http://www.colorado.edu/cspv/blueprints/index.html), a project of the Center for the Study and Prevention of Violence at the University of Colorado, has developed a list of 11 model programs and 17 promising programs that meet a strict scientific standard of effectiveness. Of these high-quality programs, approximately half aim to change family-level risk and protective factors in some way. Similarly, the Substance Abuse and Mental Health Services Administration (SAMHSA) has developed the National Registry of Evidence-based Programs and Practices (NREPP), a searchable database containing over 200 mental health and substance abuse interventions (http://nrepp.samhsa.gov/Search.aspx). All interventions in the database are rated by reviewers in an effort to help community agencies select scientifically defensible interventions. In contrast to the Blueprints for Violence list, NREPP currently does not specifically endorse the interventions in the database as meeting rigorous scientific standards of evidence. The original intention of the list, however, was to vet prevention programs as model, effective, or promising according to the level of evidence they demonstrated. The 150 "legacy" interventions that were identified in these categories are still available on the website. Family-focused interventions are well represented among these legacy interventions.

A third way that we know that family-focused prevention works is through meta-analyses. A series of recent meta-analyses have taught us that as a whole, family-based prevention and parenting programs designed to reduce substance use, prevent internalizing and externalizing behaviors, or promote positive development have, on average, produced medium-size effects (Durlak et al., 2007; Kaminski, Valle, Filene, & Boyle, 2008; Sweet & Applebaum, 2004; Tobler & Kumpfer, 2000). In addition, Kaminski and colleagues (2008) found that those interventions that taught parents about having positive interactions with their youth, emotional communication, effective discipline strategies such as time out and consistent responding, and included opportunities to practice with their children all showed more positive effects.

The accumulating evidence indicates that interventions can change common parenting practices such as discouraging negative behaviors, supporting positive behaviors, providing advice and information, and being aware of child and youth activities. Moreover, these interventions influence the positive affective quality of parent–child relationships. They also have effects on a variety of child and youth problems with high social and individual costs. These outcomes are typically seen in indicators of youth adjustment including broad spectrum problems like internalizing and externalizing problems, mental health problems, substance use, delinquency, and high-risk sexual behavior. Although evaluations of family-based prevention programs tend to focus on reducing the likelihood of negative developmental outcomes, there is substantial evidence indicating that these interventions influence child and youth abilities to successfully meet developmental tasks such as social–emotional competencies, well-being, social inclusion, or school readiness and performance. Evidence indicates that these interventions are effective across childhood (early childhood, middle childhood, and adolescence) and across life transitions (transition to parenthood, transition to school, and transitions in family structure) and have lasting effects over long periods of time (Sandler, Schoenfelder, Wolchik, & McKinnon, 2011).

Despite this impressive knowledge base, we know much less about *how* these interventions produce their effects. One of the defining features of effective family-based preventive interventions and other behavior change interventions is a strong grounding in theory. Well-implemented preventive interventions conducted in a randomized control trial can yield an experimental test of a developmental theory and can provide strong evidence regarding whether processes hypothesized by the theory are associated with or lead to specific child or youth developmental outcomes. Most family interventions draw from a variety of general theoretical formulations about family functioning and parenting, such as attachment theory, social learning theory, theories of family risk and resilience, or the social development model. These general theories are quite broad in their description of factors and processes and obviously cannot be tested as a whole in an intervention trial. So these broad theories are used to identify a "small theory" (Lipsey, 1993), sometimes also called a conceptual or etiological theory, which describes the problem one wishes to address (e.g., youth substance use) and a select number of specific risk or protective factors that are conceptually linked with the outcomes of interest. Interventions are designed to change those risk and protective factors that are believed to have a causal relation with the outcome. Theoretical, empirical, and practical reasons are used to select the targeted factors.

Theoretically, specific risk or protective factors might be targeted because they are the most interesting in a test of the theory. Empirically, one factor might show a stronger relationship to the outcome than another, so targeting it, rather than the other, ostensibly would produce a stronger intervention effect. Practically, the intervention has to be able to change the factor. Small theories about interventions are not always articulated despite the value to program design and evaluation.

Small-theory models tend to be more global and behavioral and tell us less about the detailed psychological process that is likely taking place to achieve the given outcomes. While a small theory is extremely helpful in guiding analyses of the intervention through such techniques as mediation analyses, it generally leaves us at the level of risk factor and still does not fully explain the *process*, or *processes*, by which the intervention has its effect. Mediation analyses guided by a small theory indicate that changes in one construct, positive parenting, for example, are involved in youth outcomes, externalizing behavior, but do not clearly specify how a change in positive parenting would lead to a change in level of externalizing behavior or prevent externalizing behavior from occurring. As an example, positive parenting, which is an indicator of the quality of the parent–child relationship, is a commonly targeted risk or protective factor in family-focused interventions. Interventions use a variety of activities designed to maintain or enhance the quality of the relationship by encouraging parents to spend more time with their children in positive and fun activities, help parents change their cognitive sets about their child's behavior so that they are more accepting, and generally try to generate a more positive affective tone in the relationship. Positive parenting has been shown to mediate the effects of a family-focused intervention for bereaved children on mental health outcomes for girls (Tein, Sandler, Ayers, & Wolchik, 2006). While, useful in a broad sense, this kind of analysis can tell us that changing the quality of a parent–child relationship can reduce the likelihood of later problem behaviors, it does not tell us how the quality of the relationship gets under the skin of the youth to produce its protective effect. Some evidence suggests that self-system beliefs such as self-esteem, coping efficacy, and fear of abandonment mediate the relationship between relationship quality and youth behavior (Wolchik, Tien, Sandler, & Ayers, 2006), so what would be required is another level of measurement to assess this additional intervening factor to more closely approximate the process of change. What seems to be needed are mid-level theories that articulate detailed processes that help us better understand how family-focused preventive interventions get under the skin of the youth to influence their development. This is where EST-R and other detailed process theories of family functioning can be applied to preventive interventions in an effective way.

How Might EST-R Help Us Understand the Effects of Preventive Interventions?

EST-R provides a level of analyses of critical family processes, and children's experiences that are not typically applied in preventive interventions, yet, would be useful to inform and refine family-focused preventive interventions. I focus my

attention in this commentary on four main points from EST-R: (1) its complexity and specificity, (2) the importance of interparental relationships, (3) hypothesized moderating conditions, and (4) cascade mechanisms.

With the reformulation of emotional security theory, Davies and colleagues (Chap. 5) have proposed a theory to help organize the complex findings that link family processes of parenting practices and interparental conflict with children's emotional responses and with children's long-term mental and physical health adjustment trajectories under varying (moderating) conditions. As already noted, the theory appears to be pitched at a mid-level which provides a conceptual advantage over the nearly untestable broad developmental theories and the highly restrictive small theories often used as foundations for preventive interventions. EST-R allows for generating hypotheses at a level of specificity that can more precisely describe processes by which risk conditions "get under the skin" and influence health and well-being. By extension, it would be possible to articulate with greater precision how an intervention might work by influencing those processes. For example, an intervention might seek to reduce interparental conflict in an effort to prevent later externalizing or internalizing behaviors. A small theory might simply note that reductions in conflict produced by an intervention relate to lower levels of children's problems. A more specific articulation of the process might include a complex sequence of steps such as how changes in conflict relate to changes in children's felt security in the parent–child relationship, which then influences children's willingness to approach novel objects and settings, thereby influencing development of basic skills related to social or academic competence. What is articulated is a causal sequence that more clearly approximates a process of change. Within EST-R, the added complexity of a broader family systems view and the hypothesized dual systems of attachment and social defense that are activated under different threat conditions within the family is unique. Specifically, it focuses on relationships in which the child participates directly (attachment relationship) and on relationships in which the child does not participate directly (interparental relationship) but which can influence the child's affective response of distress and fear. This complexity can be a double-edged sword, however, because it brings with it significant issues in measurement and timing of measurement and can also make it very difficult to precisely specify whether one or more mechanisms are operating to produce the effect. Carefully chosen comparison interventions or control groups are highly valued in studies attempting to isolate one mechanism over another.

Related to the issue of complexity is the emphasis within EST-R on the interparental relationship beyond the parent–child relationship. Relatively few family-centered prevention programs focus specifically on parent-to-parent interactions. In part, this may be because most interventions are based on models of parenting, in which there is one "actor," a mother or father, and typically just one child. This may also be guided by practical reasons. Preventive interventions for families are notoriously poorly attended with usually <50 % of recruited families actually participating, and in some cases the rates are closer to 10 %. So practitioners and researchers typically acknowledge that one parent attending is better than none, and that person is usually

"mom." Fathers tend to participate less often in interventions and research studies (Duhig, Phares, & Birkeland, 2002) and this is problematic because they seem to have a unique role in the development of child problem behavior (Bögels, 2011). So in many cases, like the developmental literature on parenting, prevention science is building a research base on the efficacy of family-based interventions for mothers.

Some family-centered interventions, such as interventions designed to improve the coparenting relationship, do focus on enhancing the interparental relationship as a primary goal of the intervention. Effects of coparenting interventions, such as Family Foundations (Feinberg & Kan, 2008), are effective in altering couple relations, parent well-being, parenting quality, and child outcomes. Results from this study and other coparenting studies indicate that targeting the coparenting relationship at the transition to parenthood may be an effective way to promote parenting quality and child adjustment. This kind of intervention also provides an opportunity to test EST-R experimentally. It is reasonable to assume that Family Foundations is an intervention that could influence those family processes and/or moderating conditions that theoretically will influence children's emotional security and subsequent adjustment. While the empirical evidence supporting the emotional security theory is strong, overlaying that theory onto an intervention that has been demonstrated to change critical elements of the theoretical model would provide even stronger evidence for the theory.

There are several novel brief interventions that focus on changing levels of parent conflict that could also provide information about the validity of EST-R. One intervention Happy Couples and Happy Kids (Cummings et al., 2008) has as a theoretical foundation emotional security theory, although not the reformulated version. The intervention program targets knowledge of constructive and destructive conflict but emphasizes the use of constructive conflict resolution strategies—a point not emphasized in EST-R. The intervention was found to have significant effects on increasing knowledge of the effects of marital conflict on families, behavioral improvements in constructive conflict, and lowered destructive conflicts. The intervention is promising, but must be interpreted with caution because the trial was conducted with a very small sample size. Nevertheless, it seems that this intervention that was predicated in part on an earlier iteration of the proposed theory would be an ideal intervention to implement as a rigorous test of EST hypotheses.

Another advantage of EST-R for prevention science is its acknowledgement of and attention to potential moderating conditions. The important point here for prevention science is that intervention effects are likely to be moderated by a variety of individual or family characteristics and that acknowledging these directly in a conceptual theory of the problem and intervention can facilitate clearer hypotheses and analyses. Moderating effects have generally been given less attention in the intervention literature and almost always are examined post hoc. That is, researchers do not specify the conditions under which their interventions will have stronger or weaker effects before the fact; rather, it is typical that these moderators are discovered after an initial intent to treat analysis fails to indicate an intervention effect. While this may be informative, it obviously capitalizes on chance and a priori specification of the moderating conditions is preferred.

An additional strength of EST-R that, if applied to conceptual models in prevention science, could provide additional insight into how prevention programs operate is the notion of developmental cascades of processes that lead to broader trait-like patterns of psychological and physical functioning. This kind of sequential developmental model is rarely tested in prevention programs, although most implicit conceptual theories would acknowledge that changes in the targeted risk or protective factors would not have direct relations with youth outcomes. Sandler and colleagues (2011) point out that although several family-focused programs demonstrate long-term effects (1–20 years) on youth outcomes, little is known about how improvements in parenting measured immediately after the intervention (or 6 months or 1 year later) carry forth to produce (or sustain) behavioral change years later. These processes are likely to be both socially reinforcing processes, as suggested by the social learning model, and psychological, affective, and biological neurobiological processes within the child that she "takes with her" into other social contexts. EST-R appears to be ahead of the curve in articulating how these cascading processes might operate.

To review, we know that prevention programs work for families, but we have less information about how they work. Moreover, intervention effect sizes are typically in the moderate range, suggesting substantial room for improvement. Improvement could be enhanced and accelerated by using more precise mid-level theories, such as EST-R, to propose and to test specific hypotheses about processes by which family (and intervention) gets under the skin of children to influence child and youth adjustment. In turn, information gained from the intervention study can be used to refine the etiological theory. More theoretical models informing our interventions will expand our science and provide new knowledge. Although I have touched on only a few of the features of EST-R, emphasis on interparental processes, cascading mechanisms, and moderating conditions, the theory appears to be one that could inform and enhance family-focused preventive interventions.

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Part III
Link to the Social Environment Through Families

Chapter 9
Lifecourse Exposures and Socioeconomic Disparities in Child Health

Nancy E. Reichman and Julien O. Teitler

Abstract This chapter focuses on what is known about socioeconomic disparities (SES) in child health and different types of exposures during childhood, in utero, and from the previous generation that may contribute to those disparities. SES disparities in child health appear to be substantial, begin at very young ages, persist, and compound over the lifecourse. Few types of exposures specifically during childhood appear to be promising explanations for observed linkages between SES and child health, although neighborhood and city characteristics, environmental toxins, parent behaviors, and children's epigenetic responses to exposures deserve more scrutiny. An increasing number of studies point to the importance of in utero and intergenerational exposures, suggesting that health disparities emerge earlier than previously thought. Maternal nutrition (in utero, prior to conception, and during the mother's own fetal development and childhood) is a promising area of research for understanding SES disparities in child health, as are prenatal and preconceptional exposures to toxins through occupations and residential environments. For significant advances to be made in understanding health disparities among both children and adults, we need a better conceptualization of the process leading to health disparities, more knowledge about determinants of health, and better conceptualization and measurement of health, particularly among children.

A great deal of research reveals large socioeconomic disparities in health among adults in the United States, United Kingdom, and most other developed countries (Adler & Rehkopf, 2008; Mackenbach et al., 2008), including those characterized

N.E. Reichman, Ph.D. (✉)
Department of Pediatrics, Robert Wood Johnson Medical School,
New Brunswick, NJ, USA
e-mail: reichman@princeton.edu

J.O. Teitler, Ph.D.
School of Social Work, Columbia University,
New York, NY, USA

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by relatively generous welfare states (Mackenbach et al.). Health appears to be associated with social hierarchies even in nonhuman species (Sapolsky, 2005), suggesting that health disparities are a ubiquitous characteristic of social groups. Much less is known about socioeconomic disparities in health specifically among children; as such, it is difficult to pinpoint when during the lifecourse the disparities emerge. This chapter focuses on (1) what is known about socioeconomic disparities in child health; (2) exposures during childhood, in utero, and from the previous generation that may contribute to socioeconomic disparities in child health; (3) feedback loops from child health to family socioeconomic status (SES); and (4) important unanswered questions vis-à-vis socioeconomic disparities in child health.

Theories from economics, sociology, and epidemiology suggest that socioeconomic disparities in child health exist early in the lifecourse and may increase with age. In economics, health is considered a capital good; individuals are born with varying health stocks, which increase through health-related investments and depreciate over time. Sociological theories of health disparities focus on differential access to resources. Prominent explanations in the epidemiologic literature focus on stress resulting from the internalization of status positions.

According to the economic theory of health production (Grossman, 1972), investments in health (market purchases of health "inputs," such as medical care, as well as time allocated to health-related behaviors) are a function of an individual's stock of human capital, particularly income and education. Income yields the ability to purchase health inputs and reduce harmful exposures. Education allows individuals to select better health inputs and environments given their income and to more efficiently utilize fixed inputs. Children cannot invest in their own health, so that function is relegated primarily to parents (and to the state). Parents' human capital influences both the initial health stock of their children (e.g., by shaping prenatal behaviors such as taking vitamins and refraining from cigarette smoking) and subsequent health investments during infancy and childhood. As children age, their own education and cognitive ability may also come into play.

The Grossman theory suggests that socioeconomic disparities in child health will exist at birth and compound with age because children from poorer families will have (1) poorer health endowments at birth, which may adversely affect their health trajectories; (2) lower levels of health investment and higher levels of harmful exposures throughout childhood; and (3) lower levels of investment in their own human capital, reducing opportunity costs of engaging in health-compromising behaviors such as risky sexual behavior and illicit drug use as those opportunities present themselves.

Link and Phelan (1995) put forth the "fundamental causes" theory that health disparities emerge from unequal access not only to material resources but also to social resources including knowledge, power, prestige, and connections (social capital) that can be drawn upon to prevent and treat diseases. As evidence that resources matter, they pointed out that high-status individuals have much more consistent advantages vis-à-vis preventable and treatable diseases than for diseases that have

no known or avoidable causes (Phelan, Link, Diez-Roux, Kawachi, & Levin, 2004). With its focus on prevention and treatment of preventable and curable diseases, the fundamental causes theory should apply as much to child health as adult health and would not in its general formulation predict widening health disparities by age. Risks of all preventable illness or disease should be lower for higher status than for lower status individuals, throughout the lifecourse.

According to social psychological theory developed in the epidemiological literature, health disparities emanate from stresses associated with occupying subordinate positions within social hierarchies. As evidence of the importance of relative social position (as opposed to absolute resources), Marmot and Wilkinson (2006) pointed to large gradients in mortality by occupational grade among male British civil servants exposed to very similar environments. The primary mechanism through which stress is thought to result in ill health involves neuroendocrine pathways. Specifically, experiencing acute stress from occupying a subordinate position leads to increased flow of adrenaline and blood pressure, which—when chronic—suppresses cardiovascular health and immune function. The longer one experiences acute stress, the more severe the impact on health. Stress might also affect health indirectly by inducing health-compromising behaviors such as cigarette smoking or alcohol or drug abuse. This theory is consistent with widening disparities in health over the lifecourse.

By extension, each of the three theories discussed above would suggest that racism, segregation, and discrimination would produce racial disparities in health—through access to resources, control over one's environment, or stress.

What We Know About SES Disparities in Child Health

National figures on socioeconomic disparities in health have not routinely been available for the USA. Yet, interest in amassing documentation on health disparities is emerging, as evidenced by the recent release of the first periodic health disparities and inequalities report by the Centers for Disease Control and Prevention (CDC) (2011). Figures are even rarer for children than they are for adults. (The CDC report focuses almost entirely on adults.) A relatively sparse patchwork of reports and studies from the USA, England, and Canada suggests that substantial child health disparities by socioeconomic status do exist. However, this body of work is fragmented and incomplete, focusing on a limited set of health measures and ages and operationalizing SES different ways based on education, income, or occupation. (See Adler & Newman, 2002, for a good discussion of the various measures of SES in the context of SES disparities in health.) As such, it is difficult—if not impossible—to pinpoint when during the lifecourse socioeconomic disparities emerge. In this section, we present data and summarize existing research on SES patterns in infant and child health in the United States.

SES Disparities in Birth Outcomes

We begin by examining SES disparities in infant health at birth using measures based on birth weight and gestational age. Birth weight is a widely used and much-studied marker of infant health. It is well measured, reliably recorded, readily available from vital statistics files and many other data sets, and used for international comparisons (Reichman, 2005). It is often categorized as very low (<1,500 g or about 3.3 lb), low (<2,500 g or about 5.5 lb), or normal (>2,500 g or about 5.5 lb). Births can also be characterized by gestational age as very preterm (<32 weeks), preterm (<37 weeks), or term (37 weeks or more). Babies considered small for gestational age, who are growth retarded, typically fall under the 10th percentile in sex-specific birth weight for gestational age. All low birth weight babies are preterm or growth retarded (or both), and virtually all very low birth weight babies are preterm.

Babies born in the United States are more likely to be low birth weight than those born in almost every other developed country (Reichman, 2005). Low birth weight is the second leading cause of infant mortality in the USA, after birth defects, and surviving infants are at high risk for debilitating medical conditions, including cerebral palsy, mental retardation, respiratory distress syndrome (RDS), bronchopulmonary dysplasia (BPD), retinopathy of prematurity (ROP), and deafness. RDS and BPD can lead to feeding difficulty, recurrent respiratory infections, asthma, and growth delay. ROP, a disorder caused by abnormal growth of blood vessels in the eye, can lead to blindness. Almost without exception, the prevalence of these disabling conditions increases as birth weight decreases.

US natality data reveal large disparities in birth weight and gestational age outcomes by maternal education, the only available measure of SES in birth records. Figure 9.1 presents rates of low birth weight (LBW), very low birth weight (VLBW), preterm birth (PTB), and very preterm birth (VPTB) in 2008, the most recent year of data available, for mothers with 12–15 years of completed education and for those with 16+ years of education. While the rates of LBW and PTB are higher for the former than the latter (8.6% as compared to 7.0% for LBW and 12.8% as compared to 10.3% for PTB), the disparities for VLBW and VPTB—measures associated with particularly high risk of subsequent morbidity—are even more dramatic. Compared to mothers with 16+ years of education, the less educated mothers are 36% more likely to have infants who are VLBW (1.5%, as compared to 1.1%) and 50% more likely to have infants who are very preterm (2.1%, as compared to 1.4%). Women with less than 12 years of education are at the highest risk of adverse birth outcomes (not shown), but maternal age is so confounded with education for that group that comparisons of SES differences based on education are likely to be misleading.

These socioeconomic disparities in birth outcomes, which are unadjusted for confounding factors, are highly generalizable according to a recent systematic review by Blumenshine, Egerter, Barclay, Cubbin, and Braveman (2010). The vast majority (93) of the 106 English language articles on SES disparities in Organization for Economic Co-operation and Development countries published from 1999 to 2007 reported a significant association between SES (measured a variety of ways) and birth outcomes, including measures of growth retardation. The studies that did

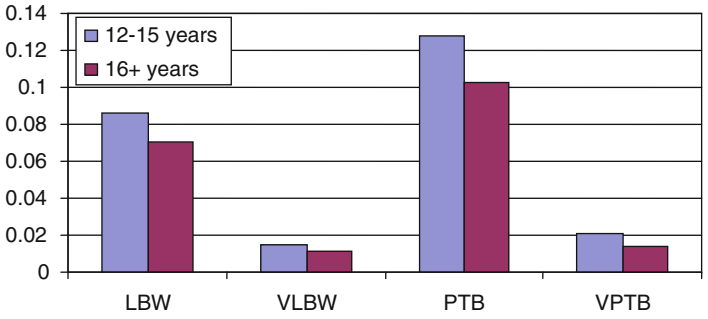

Fig. 9.1 Rates of Low Birthweight (LBW), Very Low Birthweight (VLBW), Preterm Birth (PTB), and Very Preterm Birth (VPTB) by Maternal Education, United States, 2008. Source: Computed online from VitalStats, National Vital Statistics Data System, United States Department of Health and Human Services

not find significant associations had small or nonrepresentative samples or controlled for SES-related behaviors or biomedical variables that may lie along the causal pathway between SES and birth outcomes. Complicating the picture a bit, Finch (2003) found the relationship between income and birth outcomes to be complex, with possible thresholds after which income may cease to be beneficial.

Racial and Ethnic Disparities in Birth Outcomes

In the USA, there are large and glaring health disparities by race. Figure 9.2, which shows rates of LBW, VLBW, PTB, and VPTB in 2008 for non-Hispanic black and non-Hispanic white mothers, makes clear that racial disparities in health are apparent at the starting gate (birth). The other racial groups reported by the National Center for Health Statistics for 2008 had rates of low birth weight close to that of whites—7.4% among American Indians or Alaska Natives and 8.2% among Asians and Pacific Islanders (Hamilton, Martin, & Ventura, 2010, Table 8). The pattern of racial disparities in these measures mirrors that of educational disparities. While the rate of LBW of blacks is almost double that of whites (13.7%, as compared to 7.2%) and the rate of PTB of blacks is 1.6 times that of whites (17.5%, as compared to 11.1%), the disparities for VLBW and VPTB—much more severe birth outcomes—are even larger. Compared to non-Hispanic white mothers, non-Hispanic black mothers are 2.5 times more likely to have infants who are VLBW (3.0%, as compared to 1.2%) and 2.4 times more likely to have infants who are very preterm (3.8%, as compared to 1.6%).

Because race and SES are strongly intertwined in the USA, it is difficult to disentangle health disparities along the two dimensions. Although studies have not been successful at explaining black/white disparities in LBW with available measures of SES, other studies have found that, in disadvantaged samples, black/white disparities in LBW are much smaller than those in the overall population

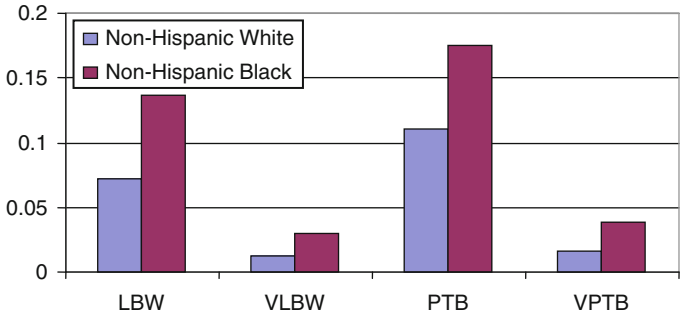

Fig. 9.2 Rates of Low Birthweight (LBW), Very Low Birthweight (VLBW), Preterm Birth (PTB), and Very Preterm Birth (VPTB) by Maternal Race, United States, 2008. Source: Computed online from VitalStats, National Vital Statistics Data System

(Reichman, Hamilton, Hummer, & Padilla, 2008), suggesting that there are complex interactive effects between SES and race.

Birth outcomes also vary among women of different ancestral origins. Hispanic mothers have outcomes on par with those of non-Hispanic whites (the rate of LBW for Hispanics was 7.0% in 2008), but within that broad group rates differ widely. Women of Cuban and Mexican descent had rates of 7.8% and 6.5%, respectively, while Puerto Rican mothers had a rate of 9.9% (figures calculated in VitalStats). The unexpectedly low rate among Mexican origin women, given their socioeconomic disadvantages, has been referred to as an *epidemiologic* or *Hispanic paradox* and is thought to reflect cultural differences in diet, stress, or lifestyle (Markides & Coreil, 1986). Complicating the picture, birth outcomes are more favorable for foreign-born mothers than for native-born mothers of the same race or ethnicity (e.g., Landale, Oropesa, & Gorman, 1999), but appear to vary by duration of residence in the USA in a curvilinear pattern, with low birth weight decreasing over the first few years here and then increasing thereafter (Teitler, Hutto, & Reichman, 2012). It is not known how much of the immigrant birth outcome advantage is due to cultural differences in behaviors and lifestyle as opposed to selective migration. Finally, it is not clear that ethnic and immigrant birth outcome advantages lead to subsequent health advantages, as recent studies have found that Mexican-American children appear to be less healthy than their non-Hispanic white counterparts (Hamilton, Teitler, & Reichman, 2011; Padilla, Hamilton, & Hummer, 2009). The bottom line is that the relationships between ethnicity, SES, and health are extremely complex.

SES Disparities in Child Health Beyond Early Infancy

A recent set of findings as part of a broader study of SES disparities in health in the USA provides some contemporary national figures for children (Braveman, Cubbin, Egerter, Williams, & Pamuk, 2010). The authors looked at three child health

Table 9.1 Percentages of children in United States with select health conditions by household income tercile, 1999–2006

	Low	Mid	High
Diabetes			
12–17	0.7	0.5	0.6
Obese			
4–11	14.2	10.3	7.5
12–17	17.9	15.1	11.5
Low HDL			
12–17	17.2	15.9	15.9
High cholesterol ratio			
12–17	6.6	6.4	4.7
Hypertension			
12–17	0.8	0.6	<0.1
Asthma—ever diagnosed			
0–3	12.4	6.4	7.2
4–11	15.6	15.1	11.8
12–17	18.7	16.8	19.5

Source: National Health and Nutrition Examination Survey, compiled by Melissa Martinson of Princeton University

outcomes (in addition to two behavioral measures related to child health) from recent nationally representative surveys: (1) infant mortality (death before age 1 year), (2) respondent-assessed health status (based on a 5-point scale ranging from excellent to poor) for children less than 18 years old, and (3) respondent assessments of any activity limitation due to chronic disease for children less than 18. They compared all three outcomes by education and the last two by family income. With only one exception (activity limitations by education), they found consistent evidence of SES gradients, with better health outcomes at increased levels of education and income.

In Table 9.1, we present nationally representative statistics on several indicators of poor child health by household income tercile (compiled by Melissa Martinson of Princeton University) from the National Health and Nutrition Examination Survey. Obesity, hypertension, diabetes, low high-density lipoprotein (HDL) cholesterol, and high cholesterol ratio are based on physical examinations and/or laboratory reports, rather than on more subjective self-reports. These measures also have the advantage of capturing health risks that may not have yet resulted in morbidity. The asthma measure is based on parental report of the child ever having been diagnosed by a medical professional. See Martinson, Teitler, and Reichman (2011) for detailed descriptions of the various measures.

An income gradient exists across all measures other than diabetes and asthma. In the case of asthma, it is possible that more consistent preventive care among high-SES families results in greater rates of diagnosis, suppressing observed SES gradients.

Children's Health Trajectories

Case, Lubotsky, and Paxson (2002) found a widening SES gradient in health as children age, using several large, nationally representative data sets from the USA and relying primarily on subjective assessments of children's overall health by mothers. Several health-related behaviors—regular bedtime, seat belt use, regular source of care, well-child care, parents' body mass index (a proxy for diet), mother's well care, and smoking in the home—accounted for a small portion of the increasing income gradient in children's overall health status with age, but most of the increase remained unexplained. The authors found less convincing evidence of increasing income gradients by age when using measures of individual conditions, suggesting that overall health status is more relevant than the presence of specific conditions or that SES differences in subjective assessments play a role. We similarly did not find evidence of increased gradients by age for obesity and asthma (the two specific indicators reported across age ranges) in Table 9.1 above.

Currie and Stabile (2003) replicated the cross-sectional results of Case et al. (2002), using a sample of Canadian children (from the National Survey of Children and Youth), who had universal health insurance. Exploiting the longitudinal nature of their data, they tested two hypotheses about mechanisms underlying the steepening SES gradient in child health (measured several different ways) with age: (1) low-SES children are less able than higher SES children to recover from a given health shock, so the negative effects of health shocks persist and accumulate over time; and (2) low-SES children recover from health shocks similarly to high-SES children, but are subject to a greater number of shocks. The authors found evidence that the cross-sectional relationship between children's health, family income, and age arises primarily because low-income children are subject to a greater number of health shocks than higher income children. A recent study using panel data from the US found evidence for both mechanisms (Condliffe & Link, 2008).

There is some evidence of a widening SES gradient in child health with age in Australia and the UK, both of which provide universal health care. Khanam, Nghiem, and Connelly (2009) found that the widening gradient is evident in Australia and that the pattern can largely be explained by maternal physical (self-reported) and mental health. Currie, Shields, and Wheatley Price (2007) and Propper, Rigg, and Burgess (2007) did not find evidence of a widening SES gradient in child health with age in the UK; however, Case, Lee, and Paxson (2008) reanalyzed the data from the Currie et al. (2007) study and did find a widening gradient, although it was less steep than that in the USA.

The theory and empirical evidence reviewed thus far suggest that poor children's health disadvantages exist from birth and compound over time. Since children do not make their own health investment decisions, the observed patterns suggest that exposures to health-compromising influences or environments are at play. Murasko (2008) found evidence that the cumulative effect of income on health appears to be strongest prior to adolescence, supporting the argument that SES disparities and

trajectories in child health do not reflect children's own health-related behaviors. Very little is known about how exposures produce or mediate the effects of SES on health, particularly among children. As Evans and Kantrowitz (2002) explain, links have been established between environmental exposures (including pollutants, toxins, noise, and crowding, as well as housing, school, work, and neighborhood environments) and health, and between SES and health, but the extent to which exposures explain SES disparities in health has rarely been investigated.

Exposures by Which SES Might Shape Child Health Disparities

In this section, we focus on exposures that may play a role in shaping SES disparities in child health. Relevant exposures include features of physical, social, family, and policy environments. We do not say much about policy environments, the focus of Berkman and O'Donnell (Chap. 12), but it is important to point out that policies targeted at low-income individuals or families that are shown to be effective could potentially reduce SES disparities in child health. We also do not go into detail about family exposures involving parent–child interactions and psychological processes as those are covered by Davies and colleagues (Chap. 5). Exposures can be pathways through which SES affects health or, in some cases, determinants of SES (e.g., segregation can affect access to education and employment opportunities). SES associations with exposures can be complex and change over time; for example, higher SES used to be associated with increased prevalence of smoking (leading to higher mortality from lung cancer), but since the late 1960s, higher SES has been associated with lower rates of smoking and lung cancer (see Link, 2008).

This section presents a broad overview of the different types of exposures—in childhood, in utero, and intergenerational—that may mediate or interact with (or affect) SES to produce child health disparities. It paints with broad strokes, emphasizing breadth over depth and offering salient and contemporary examples rather than a detailed review of each topic. The conclusions are general and speculative—based on observed associations between exposures and child (usually infant) health and between exposures and SES—because, as far as we know, there is almost no existing literature on the role of exposures in shaping SES disparities in child health.

Childhood Exposures

Physical Neighborhood Conditions

Two key aspects of physical neighborhood conditions are particularly salient in the neighborhood and health literature—physical disorder and the built environment. Physical disorder refers to signs of neighborhood neglect or deterioration, including

litter, graffiti, boarded-up buildings, and broken glass. The built environment reflects land use (the mix of commercial, residential, industrial buildings), "natural" features (trees, gardens, parks), and architecture and urban design (benches, building aesthetics, bicycle lanes, etc.). Physical disorder is thought to affect health (1) by discouraging walking and other outdoor activities and (2) by signaling—correctly or not—that a neighborhood is dangerous, leading parents to keep their children indoors where they may be less physically active and more exposed to toxins such as secondhand smoke (Miles, Panton, Jang, & Haymes, 2008; Molnar, Gortmaker, Bull, & Buka, 2004). Features of the built environment that encourage walking and physical activity may favorably affect health by reducing body mass index and insulin resistance. Availability of healthy food and health-care facilities may increase health by encouraging good diets and regular use of health care (per the Grossman model, access would lower the cost of these health inputs).

Studies have revealed associations between neighborhood physical disorder and mortality, cardiovascular disease, and homicide (Cohen, Farley, & Mason, 2003), physical activity (Hoehner, Brennan Ramirez, Elliott, Handy, & Brownson, 2005), obesity (Stafford et al., 2007), and asthma (Cagney & Browning, 2004). Other studies have uncovered associations between the built environment and health. Denser commercial activity and land use mix, in particular the presence of grocery stores and restaurants, as well as proximity to transit stops, are thought to encourage pedestrian activity and thereby reduce overweight, cardiovascular disease, and diabetes (Frank et al., 2006; Mobley et al., 2006). A recent review article indicates that neighborhood residents who have better access to supermarkets and limited access to convenience stores have healthier diets and lower levels of obesity (Larson, Story, & Nelson, 2009). A subsequent study found, more specifically, that availability of recommended foods in stores within one mile of residence is associated with improved quality of diet (Franco et al., 2009). Proximity to health-care providers has been linked to improved use of pediatric health care in urban areas regardless of health insurance status (Currie & Reagan, 2003), and increased care may improve health. Associations have been found between greenery in neighborhoods—trees, window boxes, sidewalk planters, front gardens, and parks—and physical activity (de Vries, Bakker, van Mechelen, & Hopman-Rock, 2007), obesity (Liu, Wilson, Qi, & Ying, 2007), and childhood asthma (Lovasi, Quinn, Neckerman, Perzanowski, & Rundle, 2008). Aesthetically pleasing architecture and pedestrian facilities (e.g., streetlights, traffic control devices, and benches) are thought to promote health by encouraging walking, though evidence of associations with health are not abundant.

Physical disorder and built environments vary substantially by socioeconomic status (Neckerman et al., 2009), which is not surprising given the large extent of residential segregation by income and race in the USA. Thus, these aspects of neighborhoods could potentially mediate and exacerbate socioeconomic disparities in health. However, persistent methodological challenges, particularly with respect to residential selection, make it very difficult to assess the extent to which this may be the case (Manski, 1993; Oakes, 2004).

Environmental Toxins

The CDC reported on 148 chemicals that individuals in the USA are exposed to through air, water, food, soil, dust, and other media (such as consumer products) and that are known or suspected to have serious health effects (USDHHS, 2005). These include metals, tobacco smoke, solvents, and pesticides. Certain exposures to environmental contaminants result from proximity to toxic sources, such as landfills. Others, such as ambient air pollution, involve exposures that cover large geographic areas. An Environmental Protection Agency (EPA) report (2003) describes linkages between levels of many environmental contaminants in air, water, food, and soil and childhood illnesses including asthma. Buildings, especially housing, represent another source of environmental health risk to children. In particular, asbestos, lead paint, rodents, dust mites, lack of heat, and mold all have been linked to adverse adult and child health outcomes (World Health Organization [WHO], 2006). The hypothesized biological mechanisms depend on the specific type of exposure and outcome. Although there are exceptions, poor individuals are more likely than non-poor individuals to be exposed to most environmental hazards (WHO, 2009). As such, exposures to environmental toxins may plausibly shape SES disparities in child health, though the extent to which they do is an open question.

Neighborhood Social and Compositional Environments

Direct links from neighborhood social organization to health have rarely been demonstrated. However, a number of studies point to associations between neighborhood social interactions or "collective efficacy" and health behaviors, including crime, delinquency, and risky behaviors such as early or unprotected sex (see review article by Sampson, Morenoff, & Gannon-Rowley, 2002).

A few studies have investigated whether racial and ethnic composition of a neighborhood is associated with infant health, potential pathways being lower access to health resources in high minority concentration areas, race-based targeting of tobacco or fast-food advertising, segregation-induced stress, neighborhood social cohesion, and social support. With the exception of Roberts (1997), which found negative associations between the concentration of blacks in census block groups and birthweight, most studies have found no evidence that neighborhood racial concentration affects birth outcomes (Buka, Brennan, Rich-Edwards, Raudenbush, & Earls, 2003; Morenoff, 2003; Reichman, Teitler, & Hamilton, 2009). The Reichman, Teitler, and Hamilton (2009) study found that city-level racial composition is associated with birthweight among infants of unmarried black mothers, controlling for census tract-level racial composition. This finding is consistent with research by Ellen (2000), who argued that city-level segregation, in particular racial centralization and uneven distribution across neighborhoods, may be more salient than neighborhood attributes for some health outcomes. Studies of ethnic concentration, usually confounded with immigrant concentration, are mixed. Eschbach, Ostir, Patel, Markides, and Goodwin (2004), for example, found some protective effects of living in high immigrant

neighborhoods, but Osypuk, Diez Roux, Hadley, and Kandula (2009) found inconsistencies in effects across health outcomes.

The extent to which neighborhood social and compositional factors explain socioeconomic disparities in health is not known, but we are doubtful that they account for a substantial portion of the disparities, given the moderate effect sizes reported in the empirical literature. The effects of city-level compositional factors may be more important.

Family Environments

Parents make many decisions that affect their children's health such as whether and how long to breastfeed; how often, when, and where to obtain medical care; types and quantity of food provided; level of supervision; children's activities that promote exercise; and safety and cleanliness of the home environment. All of these factors have been linked to child health, albeit in a very fragmented and disjointed literature. For example, breastfeeding is associated with decreased incidence and/or severity of a wide range of infectious diseases and appears to be protective against many other health conditions, including sudden infant death syndrome, diabetes, obesity, hypercholesterolemia, asthma, and neurodevelopmental delays (American Academy of Pediatrics, 2005). Children who are exposed to secondhand tobacco smoke are at increased risk for sudden infant death syndrome, acute respiratory infections, asthma, and ear infections (USDHHS, 2006).

A consistent concern in research investigating associations between health-related parenting behaviors and child health outcomes is the extent to which observed associations reflect unobserved confounding effects of SES, making it difficult to establish clear causal links. However, the flip side of this dilemma is that parent behaviors represent potential sources of SES disparities in child health. Another complication is that not all of these behaviors are clearly beneficial or detrimental to children's health. For example, as indicated earlier, unsanitary home conditions including the presence of mold, rodents, and dust mites are associated with poor child health. However, exposure to excessively sanitary environments during infancy may lead to a deregulated immune system. This theory has been termed the *hygiene hypothesis* and is invoked as an explanation for why some countries with highly advanced economies (e.g., the USA and Japan) fare worse than some economically developing countries in asthma prevalence and other health outcomes (McDade, Rutherford, Adair, & Kuzawa, 2010; Yazdanbakhsh, Kremsner, & Van Ree, 2002).

Regardless of the strength of the causal links and the complexities of certain of the associations, it is generally accepted that good food and nutrition, regular health care, activities that encourage exercise, and clean and safe homes are beneficial for children's health. According to the economic theory of health production discussed earlier, poor parents would invest less in these inputs both because they face greater income constraints than their nonpoor counterparts and because they are likely to be less educated (and therefore have less health-related knowledge and/or less efficiency at using it). For the most part, the lower levels of investment would have adverse

effects on their children's health. In addition, psychological theories suggest that confronting severe income constraints could have indirect effects on child health by increasing the parents' stress and/or substance abuse, which may adversely affect their parenting behaviors.

Health care is a particularly expensive health input. The American Academy of Pediatrics (AAP) recommends eight routine visits to a pediatrician in the first year alone, with a set schedule of assessments over the course of childhood for various developmental milestones, screenings, and immunizations (AAP, 2008). Despite substantial expansions in public health insurance eligibility for poor children under the State Children's Health Insurance Program, many poor children are uninsured for health care for a number of reasons including lack of eligibility, parent's lack of knowledge about eligibility criteria, and burdensome application processes (Hughes & Ng, 2003). Furthermore, having insurance does not guarantee access to care, as parents may find it difficult to find providers who will accept Medicaid and may be deterred by transportation and scheduling difficulties.

Case and Paxson (2002) argued that parent behaviors are important determinants of child health and should receive more attention as potential determinants of SES disparities. The material presented by Davies and colleagues (Chap. 5) suggests that parent–child interactions could shape disparities as well. The potential roles of parent–child relationships and other aspects of family environments, such as parental relationships, living arrangements, and transitions, are potentially important but have not, to our knowledge, been explored in the context of SES disparities in child health.

Epigenetic Processes

Child health can be affected by parenting behavior or other exposures via gene expression. Specifically, exposure to harsh environments (e.g., child abuse) may affect DNA methylation, which affects gene expression, altering stress reactivity and potentially one's risk of physical and mental illness. Though most epigenetic studies are based on animal models, evidence is now suggestive of similar processes among adults (McGowan et al., 2009). There is also evidence of epigenetic transmission of parenting behaviors themselves, potentially leading to a perpetuation of health advantages and disadvantages across generations. See Maestripieri, Lindell, Ayala, Gold, and Higley (2005) for evidence of intergenerational transmission of parental abuse among rhesus macaques; Champagne (2008) for applicability to humans; and Davies et al., (Chap. 1) for much more in-depth explanation of parenting effects on infants and children.

In Utero Exposures

A child's in utero exposures are a function of the mother's fertility decisions/timing (parental age and birth spacing), prenatal health behaviors, physical environments, and stress during the gestational period; all overlaid against a backdrop of her

prepregnancy health status, which has been shown to be important in predicting birth outcomes. In fact, one study found that several prepregnancy risk factors (underweight as characterized by body mass index (BMI), history of chronic hypertension, a measure of physical function, cigarette smoking, and having been LBW herself) were as important as pregnancy-specific factors (including gestational hypertension and smoking) in explaining a woman's risk of preterm delivery (Haas et al., 2005). Another prospective study that surveyed mothers even before they became pregnant found that maternal BMI had positive associations with birthweight and fetal growth, controlling for prenatal variables (smoking, vitamins, weight gain) and sociodemographics (Weisman et al., 2011).

In utero exposures could potentially affect fetal development directly (e.g., environment toxins could cross the placenta), through changes in the mother's health status during pregnancy (e.g., weight gain, development of hypertension), which may affect the birth outcome or affect child health down the road through other pathways, or through epigenetic processes, which may affect predisposition to illness and disease. We briefly review the different sources of in utero exposures, focusing on their potential contribution to SES disparities in child health.

Fertility Decisions/Timing

Parental Age

In the USA in 2008, the rate of low birth weight among teenage mothers was 26% higher than that among mothers aged 20–29 (9.8%, as compared to 7.8%). The rate among the youngest teens—those 15 and younger—was 12.4%, higher than that for all other age groups except 45–54 (figures computed from VitalStats). Teen mothers' birth weight disadvantage has several explanations. A pregnant teenager who is still growing may compete with the fetus for nutrients, and pregnancy within 2 years after menarche increases the risk for preterm delivery (Fraser, Brockert, & Ward, 1995). Adolescent childbearing is strongly related to low SES (Singh, Darroch, Frost, & Study Team, 2001). Because teen birth is associated with both low SES and adverse birth outcomes, it could potentially contribute to SES disparities in child health. However, a finding by Geronimus (1992, 1996) that young maternal age is not always a risk factor for adverse birth outcomes in socioeconomically disadvantaged populations makes this hypothesis less straightforward.

On the other end of the age spectrum, women who give birth in their late 30s or older are also at increased risk for having low birth weight babies. Biological risks include older ova and a greater likelihood of medical risk factors such as hypertension (Goldenberg & Klerman, 1995). Paternal age has also been identified as an independent risk factor for low birth weight; that is, paternal age is positively associated with low birth weight controlling for maternal age and numerous confounders (Reichman & Teitler, 2006). The mechanisms have not been established but may have to do with biological effects of aging. However, because both maternal and paternal ages are positively associated with SES, advanced parental age cannot explain SES disparities in child health.

Birth Spacing

A recent meta-analysis indicates that inter-pregnancy intervals shorter than 18 months and longer than 59 months are associated with increased likelihood of adverse perinatal outcomes including preterm birth, low birth weight, and small for gestational age (Conde-Agudelo, Rosas-Bermúdez, & Kafury-Goeta, 2006). A very recent study found that short inter-pregnancy intervals are associated with autism among offspring and that the association was not mediated by low birth weight or preterm birth, suggesting that there may be other pathways (Cheslack-Postava, Liu, & Bearman, 2011). Proposed mechanisms by which short inter-pregnancy intervals lead to adverse infant outcomes involve maternal nutritional depletion, particularly folate (Conde-Agudelo et al.). Low SES is a strong correlate of unintended pregnancy, which is associated with short inter-pregnancy intervals (Finer & Henshaw, 2006). As such, it is plausible that short inter-pregnancy intervals play a role in shaping SES disparities in child health.

Prenatal Health Behaviors

Prenatal Care

Contrary to received wisdom, a fair amount of methodologically rigorous research on the effectiveness of prenatal care has found very modest or no effects of early or adequate prenatal care on birth outcomes (birth weight, LBW, abnormal infant health conditions) (see Reichman, Corman, Noonan, & Dave, 2009). Although it is difficult for studies to capture the quality of care received and prenatal care may help certain individuals a great deal, it appears that standard prenatal care is "too little too late" to improve birth outcomes at the population level. Some recent studies have taken a broader view of the potential of prenatal care, recognizing that providing pregnant women with health information and counseling (e.g., about nutrition and the risks of substance use)—a typical component of prenatal care—may improve health behaviors and health after the child is born. There is some evidence that prenatal services improve maternal postpartum health behaviors. One randomized controlled study found that prenatal breastfeeding education and counseling increased rates of breastfeeding among urban black low-income women (Kistin, Benton, Rao, & Sullivan, 1990), and another recent study using econometric techniques to address potential omitted variables bias found that first trimester prenatal care decreases maternal postpartum smoking, increases the use of well-child health care, and appears to increase breastfeeding (Reichman, Corman, Noonan, & Schwartz-Soicher, 2010). However, another recent study found no associations between prenatal care and several measures of child health at age 5 (Noonan, Reichman, Corman, & Schwartz-Soicher, in press). Thus, although late prenatal care is strongly associated with low education and having a Medicaid birth (proxy for poverty) (Reichman et al.) and negatively associated with some health-promoting parenting behaviors, it unlikely plays a significant role in shaping SES disparities in child health.

Nutrition

A recent comprehensive and critical review by Abu-Saad and Fraser (2010) presents evidence, some based on randomized controlled trials, that energy, protein, essentially fatty acids, iron, and folate play crucial roles in influencing fetal growth and birth outcomes, which may affect health across the lifecourse (also see Barker, Chap. 1) and into the next generation (as briefly discussed later in this chapter). The authors suggested that because nutritional deficiencies are more often found in low-SES populations, prenatal nutrition may mediate the effects of SES on infant and child health disparities. The role of maternal nutrition during the gestational period appears to be a potentially fruitful area of inquiry for uncovering sources of SES disparities in health of both children and adults.

Prenatal Substance Use

Methodologically rigorous studies have confirmed that both cigarette smoking and illicit drug use during pregnancy reduce birth weight and increase the probability of low birth weight and that prenatal illicit drug use increases the probability of abnormal infant health conditions (see Reichman, Corman, et al., 2009). The hypothesized biological mechanisms depend on the specific substance. Heavy prenatal alcohol use is associated with fetal alcohol syndrome, which is clearly an adverse child health condition, but the theoretical and empirical links between alcohol and birth outcomes are relatively weak.

Reichman et al. (Reichman, Corman, et al., 2009) found that both prenatal smoking and prenatal illicit drug use are negatively associated with education, positively associated with Medicaid coverage for childbirth (proxy for poverty), not independently associated with living in a poor neighborhood (proxy for individual-level poverty), and negatively associated with being black (which, as discussed earlier, is strongly associated with low socioeconomic status in the USA). That is, the associations between these two health-compromising maternal prenatal behaviors and socioeconomic status are not clear cut. Thus, it does not seem likely that these behaviors play a large role in shaping SES disparities in child health.

Environmental Exposures During Pregnancy

Environmental contaminants could adversely affect maternal gestational health, fetal development, birth outcomes, and/or child health. The mechanisms, which are complex and depend on the specific substance, timing, and intensity of exposure, are comprehensively reviewed by Perera and Herbstman (2011). In a review of the empirical literature, Stillerman, Mattison, Giudice, and Woodruff (2008) found associations between outdoor air pollution and both reduced birth weight and preterm delivery, suggestive associations between both pesticides and polychlorinated biphenyls and decreased fetal growth and length of gestation, and links between

environmental tobacco smoke and reduced birth weight and preterm delivery. The authors also discussed linkages between gestational prenatal exposures and health conditions of offspring that may operate through pathways other than birth weight, gestational age, or birth defects. Currie (2011) discussed several recent studies that provide strong evidence of (adverse) causal effects of specific prenatal environmental exposures on birth weight and related infant health outcomes.

Some prenatal environmental exposures take place in the workplace. The research on prenatal occupation and infant/child health is large, and studies tend to focus on very specific occupations and outcomes. Recent research by Herdt-Losavio et al. (2010) investigated the associations between maternal occupation during the first trimester of pregnancy and 45 different birth defects. Among the many findings were that women working as janitors are at increased risk of giving birth to a child with a specific set of birth defects, those working as scientists are at increased risk of giving birth to a child with a different set of birth defects, and those working as teachers have a significantly *reduced* risk of giving birth to a child with yet a third set of defects. Thus, although environmental contaminants associated with place of residence are strongly related to SES, with more disadvantaged individuals facing greater health risks, the relationship between gestational occupational exposures is less patterned along SES lines and therefore not likely to explain disparities in child health. That is, the former appears to be a better candidate than the latter for explaining SES disparities in child health. Currie (2011) recently estimated that 6% of the gap in low birth weight between white college-educated mothers and black high school dropout mothers could be attributed to differences in just one type of prenatal environmental exposure—residential proximity to toxic emissions.

Stress During Pregnancy

Many studies have found associations between psychological stress (usually self-reported), particularly in the first trimester of pregnancy, and birth weight. Several recent studies have exploited "natural experiments," such as terrorist attacks, earthquakes, or armed conflict, in an attempt to isolate causal effects and have found similar results—namely, that exposure to a catastrophic event during the first trimester of pregnancy (but generally not later in the pregnancy) appears to increase the likelihood of delivering a low birth weight infant (e.g., see Mansour & Rees, 2012, for a general review of this literature plus original research specifically on the effects of armed conflict). Other studies have found linkages between stress during pregnancy, which in some studies is characterized by experiencing adverse life events such as death of a loved one or divorce up to 3 years before conception, and other conditions of offspring including neurologic disorders, psychiatric disorders, and infectious disease (see Nielsen, Hansen, Simonsen, & Hviid, 2011). The hypothesized biological mechanisms depend on the specific outcome. While this line of research is very interesting and important, it is not clear that low-SES women are more likely than high-SES women to report stress, to experience stress, or to be exposed to catastrophic events. As such, it does not seem likely that exposure to

stress during pregnancy is an important determinant of SES disparities in child health.

Epigenetic Processes

We briefly discussed the process by which exposures during childhood may alter children's gene expression and result in disease. Such epigenetic processes may actually begin during fetal development through maternal nutrition or other in utero exposures. The programming of gene expression during fetal development could predispose a child to develop specific diseases or affect his/her ability to recover from illnesses. This field is still in its infancy but its potential applicability to the study of health disparities is very promising.

Intergenerational Exposures

A growing literature, including Barker (Chap. 1), reveals evidence that child health does not begin at conception. That is, a child's health can be programmed by the exposures of his or her parents prior to the child's conception. One example we have already pointed out is that epigenetic imprinting may be transmitted intergenerationally to perpetuate behaviors, which in turn may affect health across generations. In this section, we discuss just two additional examples, paternal occupational exposures and maternal nutritional deprivation during childhood, recognizing that the potential role of intergenerational exposures in shaping child health is much more multifaceted and far-reaching than what is covered in this chapter.

Paternal Occupational Exposures

Hundreds of studies have explored associations between paternal occupations and birth outcomes. Most have considered specific types of birth defects and specific types of occupations or exposures. According to a fairly recent in-depth review, the following paternal occupational groups are most consistently associated with birth defects: janitors, painters, printers, and workers exposed to solvents; fire fighters and other workers exposed to heat; and agricultural workers who are generally exposed to pesticides (Chia & Shi, 2002). The presumed mechanisms by which paternal occupation might affect fetal development are not well understood. Possible mechanisms include mutagenic damage to paternal germ cells or sperm DNA, transmission of teratogenic agents through the seminal fluid and sperm, and household contamination by substances brought home by the father (Chia & Shi). At least the first two mechanisms would involve occupational exposures prior to conception. Although it appears that the at-risk occupations are related to low SES, to our knowledge that association has not been systematically investigated. For this reason

and because the mechanisms by which paternal occupational exposures may affect birth outcomes are not well understood, the potential of paternal occupational exposures in explaining SES disparities in child health is far from clear.

Maternal Nutrition During Childhood

Barker (Chap. 1) discusses mechanisms by which intrauterine undernutrition may affect children's growth and development. There is some evidence that intrauterine undernutrition leads to adverse child health in the next generation. For example, Lumey and Stein (1997) investigated the effects of maternal intrauterine undernutrition on birth weight of children of women born during the Dutch Hunger Winter, a war-induced famine. The authors found that the typical increase in offspring birth weights with increasing birth order was not seen for women who experienced the famine in the first trimester of pregnancy, even after controlling for numerous potentially confounding factors including maternal birth weight and birth weight of the oldest child. There were no abnormal patterns in birth weight of children born to women who experienced famine only in the second or third trimester of pregnancy. The authors ruled out many competing explanations and concluded that their findings are consistent with adverse intergenerational health effects of intrauterine undernutrition.

A mother's nutrition during childhood may also affect her child's birth weight and subsequent growth. For example, using data from a randomized controlled trial of child nutritional supplementation in Guatemala, Behrman et al. (2009) found that nutritional supplementation in girls is associated with substantial increases in birth weight, height, head circumference, and growth among their offspring at ages 0–12 years.

This line of research is intriguing and has the potential for advancing knowledge about the causes of SES disparities in child health, since, as discussed earlier, nutritional deficiencies are more often found in low-SES populations. However, as the intergenerational focus is relatively new, there is not as yet a great deal of relevant published work and the findings need to be replicated and further explored (e.g., by investigating other settings and elucidating pathways).

Feedback Loops from Child Health to SES

A substantial literature has investigated bidirectional effects between adult health and SES. However, until the past decade, relatively few studies had examined effects of poor child health on their families' socioeconomic circumstances—perhaps because most children do not earn money so their health could not have direct labor market effects. The small literature at the time indicated that child health problems can have adverse effects on family structure and employment; in particular, studies found that having a child in poor health (defined various ways) increased the

likelihood of divorce (Corman & Kaestner, 1992; Mauldon, 1992) and decreased maternal employment (e.g., Powers, 2003).

In a recent 7-year NICHD-funded project, Nancy Reichman and colleagues used the Fragile Families and Child Wellbeing survey data augmented with data collected from medical records to estimate the effects of poor child health (using measures and econometric techniques designed to capture health shocks) on parents' relationship status, employment, material hardship, health insurance status, childcare arrangements, housing conditions, and other family resources. They found that poor child health increases the risk of parental breakup (Reichman, Corman, & Noonan, 2004), decreases legal employment (Corman, Noonan, & Reichman, 2005; Noonan, Reichman, & Corman, 2005) but increases illegal work (Corman, Noonan, Reichman, & Schwartz-Soicher, 2011), and is associated with overcrowded housing (Curtis, Corman, Noonan, & Reichman, 2010). It also increases reliance on several forms of public assistance (Reichman, Corman, & Noonan, 2006). These results indicate that children's poor health exacerbates their families' socioeconomic disadvantages. This self-reinforcing vicious cycle no doubt compounds health disparities and may contribute to the widening SES gradient as children age. It also makes it difficult to precisely identify the magnitude of effect of any one risk factor for poor health.

Conclusion: Going Forward

Our glance at some primary data and review of the literature indicate that SES disparities in child health are substantial, begin at very young ages, persist, and appear to compound over the lifecourse. Children's environments and exposures may modify their health trajectories, but no single factor has been shown to explain SES gradients in child health to any significant extent. Few types of exposures specifically during childhood appear promising for explaining observed linkages between SES and child health, although neighborhood and city characteristics, environmental toxins, parent behaviors, and children's epigenetic responses to exposures deserve more scrutiny. That said, existing panel studies have not found evidence that maternal health-related parenting behaviors, such as breastfeeding, smoking in the home, and children's diets, play a substantial role in shaping SES disparities in child health (e.g., Dowd, 2007; Propper, Rigg, & Burgess, 2007). As such, it may be important to expand the array of behaviors considered.

An increasing number of studies point to the importance of in utero and intergenerational exposures, suggesting that health disparities emerge earlier than previously thought. Findings from the UK (Propper et al., 2007) and Australia (Khanam et al., 2009) that the widening SES gradient in child health with age can be accounted for by mothers' physical (self-reported) and mental health are consistent with this perspective. Maternal nutrition (in utero, prior to conception, and during the mother's own fetal development and childhood) is a promising area of research for understanding SES disparities in child health.

Prenatal and preconceptional exposures to toxins through occupations and residential environments (for both mothers and fathers) represent another. Finally, unintended childbearing as reflected in short inter-pregnancy intervals is worth investigating.

A simple but important take-home message from this chapter is that there is a long way to go in uncovering sources of SES disparities in child health. In this concluding section, we identify key areas that we believe should receive attention in order for significant advances to be made in understanding health disparities among both children and adults. These involve (a) a better conceptualization of the process leading to health disparities, (b) expanding our knowledge about determinants of health, and (c) better conceptualizing and measuring health. We elaborate on each of these below. In addition to these substantive issues, more and better data on child health disparities are sorely needed.

(a) We may be placing too much emphasis on individuals' contemporaneous or recent exposures and looking for direct or short-term effects. The processes at play may involve more complex and long-term mechanisms that accentuate inherited or early disparities rather than produce them. Existing theories provide an incomplete framework for understanding how disparities are compounded or aggravated over time and cannot adequately account for what appears to be a health gap at the starting gate. Based on the limited evidence to date, our best guess is that the life-cycle point of origin for disparities is situated in the experiences and environments of past generations and that initial health disadvantages are compounded by feedback loops in a vicious cycle. In Fig. 9.3, we illustrate this dynamic intergenerational process, which involves a complex interplay between SES and health over the lifecourse. Against this backdrop, exposures and experiences can compound existing disadvantages and perpetuate inequalities well into the future.

The model is stylized and not all potential directional arrows are shown. Health and SES are closely interconnected over the lifecourse and both transmit to future generations (illustrated with the feedback loop at the top of the diagram). The bold arrows represent the focal linkages of this chapter—those between family SES and infant/child health and from child health to family SES. Specifically, we considered the myriad exposures that may explain or modify associations between SES and child health. Linkages between birth outcomes and child health, between child health and adult health, and between adult health and adult SES have often been studied. The arrow from child health to adult SES, which could just as easily have been drawn from birth outcomes to adult SES because birth outcomes and child health are so strongly associated, has also received a good deal of research attention. We know that LBW crosses generations. Hundreds, if not thousands, of studies have investigated determinants of health at various stages of the lifecourse. All too often, studies focusing on specific linkages within the much broader process reveal weak associations, potentially missing more complex or longer term mechanisms. The model we have laid out, which guided our work for this chapter by leading us to investigate

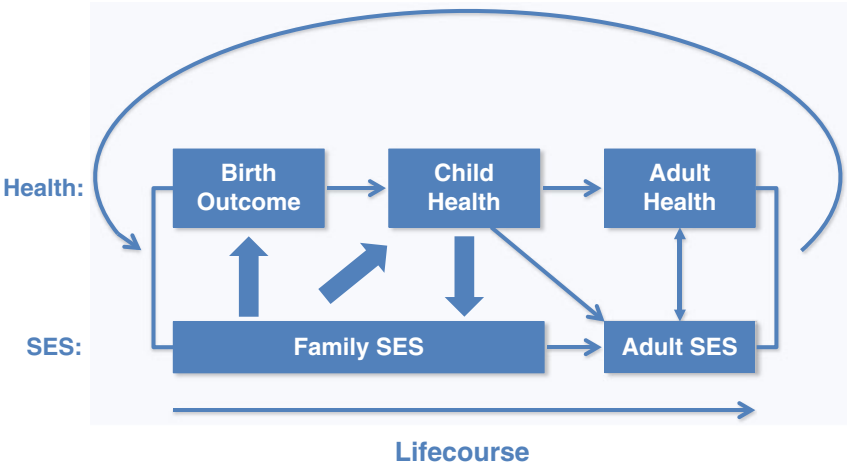

Fig. 9.3 Conceptual Model of Socioeconomic Disparities in Health

exposures occurring prior to birth and even prior to conception, is intended to focus more attention on the overall process within which small proximal effects may compound into large disadvantages over the lifecourse and across generations.

(b) To better understand health disparities, our knowledge about the determinants of health that are related to SES needs to be expanded. There may be important factors that are as yet unknown or that have not been studied. For example, we pointed out earlier that family exposures such as parental relationships and transitions have rarely, if ever, been explored in the context of explaining SES disparities in child health. More generally, the potential role of adverse life events or "turbulence," which may disproportionately affect the poor, may warrant attention given recent research pointing to the importance of income volatility, as distinct from income per se, as an important determinant of material hardship, emotional distress, and outcomes such as child well-being and obesity (see Hacker & Jacobs, 2008). Genetic factors that have not yet been identified may be important, though they are unlikely to explain a large portion of health disparities given rapid changes in health disparities that have followed advances in medical knowledge (see Phelan et al., 2004). That said, more research is needed on both genetic and epigenetic determinants of health among humans, with particular attention to gene–environment interactions and interactions by socioeconomic status.

(c) More work needs to be done on conceptualizing and measuring health, particularly for children. Should the focus be on functioning/limitations or on the absence/presence of disease? How broadly should health be conceived? Should it include behavioral, developmental, and cognitive disorders, which appear to be driving dramatic increases in child disability rates over time (Halfon,

Houtrow, Larson, & Newacheck, 2012)? Should health be characterized using measures of overall health status, which have reporting biases, lack specificity, and have been shown to be problematic when studying socioeconomic health disparities among adults (e.g., Dowd & Zajacova, 2010), or using more clearly defined measures of specific conditions, which may be relatively rare?

If using specific markers or conditions, should all be given equal weight? We implicitly do so when comparing prevalence or incidence of diseases by SES across different illnesses (as in Table 9.1). But perhaps the implication of a 50% higher age-specific mortality rate, for example, should be thought of differently than a 50% higher rate of asthma. This quandary has implications for measuring gradients over the lifecourse.

Measuring health specifically in children presents additional challenges. Because most children appear healthy, it is difficult to characterize health early in the lifecourse. The indicators we typically focus on (e.g., birth weight, asthma, BMI) are limited and fairly crude. Recent advances in the biological sciences have made it possible to measure physiological deterioration prior to the emergence of symptoms of ill health or detection of disease (e.g., telomere length, immune system response, allostatic load). For children, among whom disease is rare, developing these sciences might allow us to better capture variations in health. In parallel, expanding and validating new types of survey questions to capture more symptoms of ill health (e.g., on frequency and occurrence of infections and cold symptoms) might produce more granular distinctions of health status among seemingly healthy children.

The various measurement issues make it difficult to characterize SES disparities and trajectories in child health and to determine when in the lifecourse disparities emerge. For example, the widening of SES health disparities with age is most often observed when considering maternal reports of their children's health. This pattern may reflect exposures during childhood, but it could also potentially reflect measurement error in the dependent variable or effects of adverse health endowments at birth that may not be captured by birth weight or gestational age outcomes and that become apparent only when children get older. Given the importance of the observed widening of the SES gradient in health with age for understanding health disparities and trajectories, further validation and investigation of this pattern is necessary, including studies of how maternal reports of their children's health may vary by SES and the types of conditions that are reflected in maternal ratings of their children's health.

We end this chapter with bad news, good news, and an ethical issue. The bad news is that despite many years of rigorous science, we have been unable to adequately identify the factors that give rise to health disparities. The good news is that enough of a foundation has been laid for us to move forward and make significant advances. The last point involves our interpretation of trends in health disparities over time. Improvements in population health resulting from technological advances or public health campaigns can benefit everyone but actually increase health disparities. For example, advances in neonatal care technology over recent decades

dramatically increased the survival of LBW infants, resulting in sharply lower infant mortality rates among both blacks and whites. While the yearly percentage point decreases were about the same for the two groups, the percentage decreases were higher for whites than for blacks. The bottom line is that in the pursuit of reducing or eliminating health disparities, it is important to consider both absolute and relative changes in health over time and to be aware of the normative trade-offs those patterns may present.

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Chapter 10
Life Course Exposures and Socioeconomic Disparities in Child Health: Opportunities for Intervention

Marianne M. Hillemeier

Abstract Reichman and Teitler (Chap. 9) review the literature on socioeconomic disparities in child health. The life course-based conceptual framework they develop implies that there are multiple avenues for intervention, beginning with the mother's health prior to conception, which could ultimately be effective in decreasing child health disparities. In this chapter, results are presented from a randomized controlled trial of one such intervention, *Strong Healthy Women*, designed to promote health and reduce risks for adverse birth outcomes among women living in low-income communities. More generally, advances in health- and healthcare-related knowledge and technology can lead to significant improvements in child health and well-being. If disparities in child health are to be reduced, the barriers experienced by families of lower socioeconomic status in accessing these improvements must be identified and addressed.

Reichman and Teitler (Chap. 9) provide a thoughtful and cogent discussion of what is currently known about socioeconomic disparities in child health, including the potential role of social and physical exposures before birth and in childhood, as well as influences transmitted across generations. At times they also remind us, as economists such as Dr. Reichman often do, that some things believed to be true may not yet be settled fact and that competing explanations bear consideration. For example, Reichman and Teitler describe persuasive work by Case and her colleagues (2002) that identified a widening SES gradient in parent-reported child health status over childhood in the USA. They also describe findings consistent with this pattern from research in a Canadian child sample by Currie and Stabile (2003).

M.M. Hillemeier, Ph.D. (✉)
Department of Health Policy & Administration, The Pennsylvania State University, University Park, PA, USA
e-mail: mmh18@psu.edu

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These findings are consistent with an interpretation that many find highly plausible—that health disadvantages compound over childhood among poor children due in large part to repeated adverse physical and social exposures differentially experienced by this group. Reichman and Teitler are careful to point out, however, that error in the measurement of children's health status remains an important issue that must be kept in mind when interpreting the research to date. They also raise the interesting possibility that what looks like a compounding trajectory of health disadvantage could in fact be related to adverse health endowments present at birth that only become manifest as children get older. This insight is emblematic of the thoughtful approach taken by the authors to explain what the existing literature can tell us and what questions are not yet fully answered.

Seminar presenters in my home department of Health Policy and Administration at Penn State rarely get through the hour, or even their introductory remarks, without a question from the audience about endogeneity. Reichman and Teitler's work would be well received by this group in that while they focus on potential causal forces arising from the socioeconomic status of families that can influence child health, they also pay careful attention to mechanisms that are likely to work in the opposite direction. They describe a large program of research that Dr. Reichman and her colleagues have developed using data from the Fragile Families and Child Wellbeing Survey and medical records to estimate the effects of poor child health on aspects of family socioeconomic status. They find evidence that poor child health is associated with dissolution of parental relationships, less optimal employment status, and residential overcrowding (Corman, Noonan, & Reichman, 2005; Corman, Noonan, Reichman, & Schwartz-Soicher, 2011; Curtis, Corman, Noonan, & Reichman, 2010; Reichman, Corman, & Noonan, 2004). This complexity of directional effects makes it difficult to cleanly delineate the role of specific risk factors in determining child health outcomes. However, it is crucial to understand and take such reciprocal effects into account if we are to improve our understanding of the mechanisms driving child health disparities.

The discussion by Reichman and Teitler (Chap. 9) of the effects of prenatal care on children's health was also interesting, especially the description of a study conducted by Reichman and colleagues which found that early initiation of prenatal care is associated with less maternal smoking after an infant's birth and increased breastfeeding and use of well-child care in early life (Reichman, Corman, Noonan, & Schwartz-Soicher, 2010). These are positive findings. However, overall this chapter concurs with a growing consensus that adequate prenatal care has not been shown to effectively reduce the incidence of adverse infant outcomes (e.g., low birthweight, preterm birth) in rigorous studies that adequately control for selection bias regarding which women seek early and regular prenatal care (Moos, 2010). This is not to say that women should not be advised to obtain prenatal care, but rather that this intervention is likely to be "too little too late" to affect some birth outcomes that can seriously impact children's health. In response, the Centers for Disease Control and Prevention have repeatedly called for clinical- and community-based programs that can improve women's health before they become pregnant. This is seen as a potentially important strategy to improve birth outcomes, based on

an increasing body of evidence that women's preconceptional health is associated with birthweight and other aspects of infant health (Centers for Disease Control and Prevention, 2006).

The use of interventions designed to improve women's preconceptual health is also consistent with the conceptual framework presented by Reichman and Teitler (Chap. 9), which stresses a life course perspective. In this model, adult health is shown to have an important influence on subsequent birth outcomes. In thinking about how disparities in birth outcomes might be reduced, a logical next step is to consider interventions that could promote better health among women of lower socioeconomic status who are in their prime childbearing years but are not yet pregnant. Unfortunately, while there are numerous health promotion strategies available, very few evidence-based intervention programs have been developed which specifically focus on the unique health needs and vulnerabilities among the population of disadvantaged women of reproductive age, and even fewer have been systematically evaluated.

My colleagues and I have developed and tested one such program, the Strong Healthy Women Intervention, in the context of the Central Pennsylvania Women's Health Study (CePAWHS) (Hillemeier et al., 2008; Weisman et al., 2006, 2011). This intervention was designed for women prior to and between pregnancies living in low-income communities. It targets known risk factors for poor health and birth outcomes including elevated psychosocial stress, poor nutrition and inadequate physical activity, tobacco and alcohol use, toxic environmental exposures, infection risks, and prevention of unwanted pregnancy. Women participating in the intervention meet in small groups that promote interaction and social support as well as encourage active learning and behavior change.

In order to evaluate the effectiveness of this intervention, a randomized control trial was conducted as part of the Central Pennsylvania Women's Health Study. We recruited 692 women living in low-income communities. After a comprehensive baseline risk assessment, the women were randomly assigned to either the intervention or control group. Follow-up assessments were conducted after the 12-week intervention period, and again in 6 and 12 months. Birth records were obtained for infants born to women who became pregnant after the study began.

We were encouraged to find that at the conclusion of the intervention period, the participating women were significantly more likely to have changed their attitudes and behavior. Regarding behavior change, the intervention participants were significantly more likely to read food labels for nutritional values. They were also more likely to be taking a multivitamin with folic acid daily, which is important in preventing some birth defects, and to have increased their physical activity level. In addition, women in the intervention group were more likely to believe that they could continue to make positive changes in their health-related behavior (i.e., demonstrated self-efficacy) and to report their intention to make such changes (Hillemeier et al., 2008). Long-term follow-up confirmed that after 12 months, women in the intervention group continued to exhibit significantly higher rates of multivitamin use, and at that point, these women had also significantly reduced body weight and BMI compared to women in the control group (Weisman et al., 2011).

We were also interested in women who became pregnant after the intervention period and obtained birth records for 37 births which occurred in the follow-up period. Although this small number of births precluded analysis of differences in low birthweight or preterm birth, we did examine pregnancy weight gain, which if excessive can have important negative effects on maternal and infant outcomes. We found that weight gain among intervention participants was lower (Weisman et al., 2011), and more likely to be consistent with the Institute of Medicine guidelines (Institute of Medicine, 2009). In this small sample, the results were not statistically significant in multivariate analyses but were in the expected direction. This suggests that health promotion prior to pregnancy among low-income women can have important positive effects on aspects of their health status that are associated with later birth outcomes.

These findings support the optimistic "good news" conclusion by Reichman and Teitler (Chap. 9) that a sufficient foundation of knowledge exists to move forward in designing interventions aimed at reducing socioeconomic disparities in child health. An important implication of their conceptual framework is that there are multiple intervention points throughout the life course and that improvements in health at one stage could have positive effects that influence health outcomes at other life stages, as illustrated in Fig. 10.1. These interventions could include (but are not limited to) innovations in healthcare quality and access, which were not emphasized by Reichman and Teitler (Chap. 9) but are potentially effective avenues for the reduction of disparities in health (Beal, 2004; Chin, Alexander-Young, & Burnet, 2009). Of course, many would argue that the optimal range of intervention opportunities would also include policies that serve to increase economic and other material support available for low-income adults, families, and children (Phelan, Link, & Tehranifar, 2010), as indicated by dashed lines in Fig. 10.1.

The "bad news" conclusions by Reichman and Teitler (Chap. 9) relate to the fact that our understanding of the factors influencing child health disparities remains incomplete. As one example, the physiological effects of stressful events and exposures on biological processes such as central nervous system reactivity and immune functioning are becoming increasingly well documented (McEwen & Gianaros, 2010; McEwen & Tucker, 2011). Stress prior to and during pregnancy has been related to lower birthweight, although the processes involved are not yet well understood (Behrman & Stith Butler, 2007). Women and children with lower socioeconomic status would plausibly be expected to experience elevated levels of stress, although as Reichman notes these relationships have not been conclusively demonstrated. There is research currently underway, however, that will shed light on this issue. One example is the NICHD-funded Community Child Health Network (CCHN), which includes five race/ethnically and socioeconomically diverse populations of mothers and fathers and their infants located in various regions of the USA, as well as a data coordinating center located at Penn State. This study includes multiple longitudinal measures of psychosocial stress, with both self-reported chronic and acute stressors and biological stress markers. The analyses will provide new evidence about socioeconomic and race/ethnic disparities in perceived stress prior to and during pregnancy among mothers and fathers and how perceived stress relates to

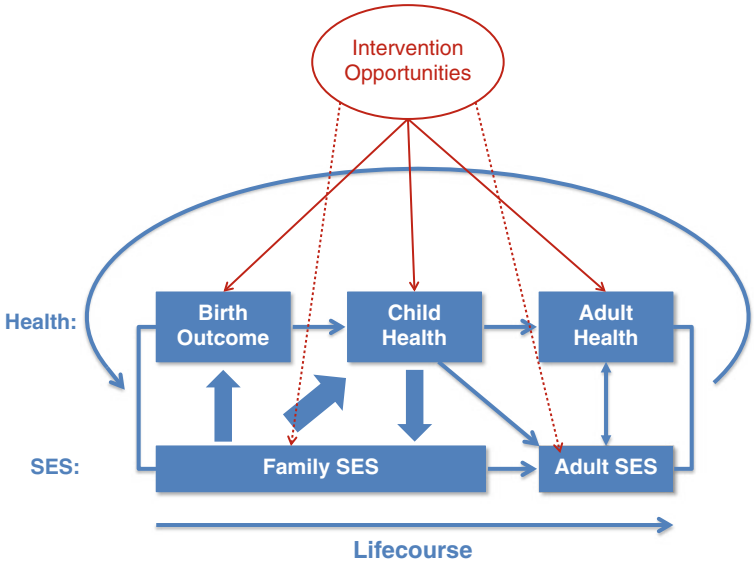

Fig. 10.1 Optimal range of health-related intervention opportunities

objective measures of cortisol and other stress-related biomarkers (see http://www.nichd.nih.gov/research/supported/cchn.cfm).

Among the other areas of incomplete knowledge mentioned by Reichman and Teitler (Chap. 9) are epigenetic processes, whereby social and environmental exposures can potentially alter the expression of a person's genes. An important point that is stressed is that a better understanding is needed of these processes as well as the interactions between these processes and socioeconomic status. This part of the discussion section also contains a brief but key caveat stating that genetic factors in themselves are "unlikely to explain a large portion of health disparities given rapid changes in health disparities that have followed advances in medical knowledge" (p. 19). This point warrants additional discussion and emphasis, particularly in view of the tendency among some in the medical community and elsewhere, to be drawn to simplistic genetic explanations for complex phenomena like health disparities.

I would also like to highlight the final point made in the conclusion, which is that improvements in population health can provide widespread benefits but at the same time increase disparities in health. The example given is neonatal care technology, which has dramatically increased the survival of both black and white low-birthweight infants but also resulted in increased racial disparity in mortality rates as the percentage decrease was more pronounced for white infants. Another example in the area of child health involves prevention strategies for Sudden Infant Death Syndrome or SIDS. After clinical evidence emerged in the early 1990s that placing babies on their backs was associated with lower risk of SIDS, a public education campaign was launched with a simple message stressing "Back to Sleep" for all

infants (Willinger, Hoffman, & Hartford, 1994). Pickett and colleagues (2005) used national linked infant birth and death certificates to examine SIDS mortality rates before the campaign in the years 1989 through 1991, and after the campaign in 1996 through 1998. They found that while rates of SIDS death were reduced in all social class groups, gaps in risk by maternal education actually widened. At both time points, the odds were lowest for women who were college graduates and generally increased with decreasing education. After the public education campaign, the SES gradient was steeper, with the exception of the very small and likely anomalous group of women who had no education at all at the high school level. Overall, more educated women and their infants generally experienced greater benefits from the informational campaign. Taken together, these examples suggest that advances in knowledge and technology can make significant improvements in children's health and life chances. However, it is crucial to specifically look for and address barriers experienced by families of lower socioeconomic status if disparities in child health are to be reduced.

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Chapter 11
Social Class and Child Health: Our Complexity Complex

Thomas A. Glass

Abstract Reichman and Teitler (Chap. 9) review the complex literature on social class and child health and find ample evidence of consistent associations, operating through multiple pathways that arise early in life and compound over the life course. They conclude, as do most such reviews, that the relationship between social class and child health is complex and that much is still unknown. This reflects what I describe as a *complexity complex* that impedes effective research and intervention. In this commentary, I make three main points: (1) we lack an adequate theory of social class gradients in children's health as well as a coherent understanding of what a good theory is and how it can be used, (2) the perception of inscrutable complexity is partly a function of the lack of fit between social class as an explanatory concept and currently accepted ideas about what constitutes a cause, and (3) the failure to explain the social class clustering of health behaviors and risk factors is partly a function of our inability to account for the role of culture.

Introduction: The Complexity Complex

I should clarify that I do not have extensive knowledge of the field of childhood health status and social class. As such, my remarks come from the perspective of someone who studies adulthood and late life. Also, while there are many terms that could be used to describe socioeconomic position, I will use the term social class for reasons that may not be clear but which provide clues as to my own theoretical orientation.

T.A. Glass, Ph.D. (✉)
Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, USA
e-mail: tglass@jhsph.edu

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Chapter 9 is a fine and well-written overview of a difficult and voluminous literature. There are many aspects of this account that are laudable. Reichman and Teitler cover a vast literature from preconception through the early life course. They consider a wide range of exposures from genes to societies. I am particularly interested in a point they made in reviewing the role of neighborhood factors. Reichman and Teitler speculate that we may be looking at the wrong level:

> The extent to which neighborhood social and compositional factors explain socioeconomic disparities in health is not known, but we are doubtful that they account for a substantial portion of the disparities given the moderate effect sizes reported in the empirical literature. The effects of city level compositional factors may be more important (p. 15).

As someone who has examined neighborhood factors and health, I am beginning to believe that they are right, but I have rarely seen this argument in print. I would go further and say we have not paid sufficient attention to larger spatial geographic differences in health at the county, state, and national levels. At those levels, policies may prove to be more fruitful explanations of social class gradients in child health than we are able to call forth from the study of the small "neighborhood" geographies more typically employed. There is also a notable bravery to this review, perhaps most clearly seen when Reichman and Teitler confess that the literature on early prenatal care shows that for birth outcomes, it may be "too little too late." Their careful consideration of reciprocal effects of child ill-health on family social class is also noteworthy.

In general, Reichman and Teitler (Chap. 9) conclude that despite the fact that we do not have the right kinds of data, and that we are not good at measuring social class or health in children, social class is strongly and consistently associated with child health. The authors further conclude that the relationship starts earlier than thought and that it appears to compound over the life course. The general theme of their review, and the one I will focus my remarks upon, is that we remain baffled as to how and why this is the case. The social class–child health relationship is, in their view, extremely complex. They suggest that "Very little is known about how exposures produce or mediate the effects of SES on health, particularly among children" (p. 11) and that "…no single factor has been shown to explain SES gradients in child health" (p. 28).

These are common conclusions in almost all reviews of social class and health (Adler & Newman, 2002; Berkman & Macintyre, 1997; Browning & Cagney, 2003; Chen, Matthews, & Boyce, 2002; Link & Phelan, 1995; Marmot, 1996; Syme & Berkman, 1976). Apparently, we have known since Hippocrates, the great Victorian sanitation reformers, and what we see everywhere in the developing world that impoverishment is bad for the health of children. Yet, we remain bafflingly baffled as to how and why. That is my point of departure and central concern. I will make three general points. All three points have to do with potential barriers to a deeper understanding of this mysterious complexity. The three main points are roughly that:

1. *We are quite confused and lost about theory*. We don't actually know what a theory is or how to use it.

2. Part of the problem is that *we have put ourselves in a box* with our definition of cause. Because social class does not fit the cause box, we appear to be unable to figure out what is going on.
3. *We are afraid of culture*. Partly because we want to view health behaviors as freely chosen and amendable to educational/informational manipulation, we do not have any useful account of why all the important health behaviors cluster by social class.

The main thesis of this commentary is that we as a field have developed a complexity complex that impedes research, clouds our methodological lens, and blocks effective intervention.

The Trouble with Theory

So what exactly makes the social class–child health association so bafflingly complex? Part of the answer is our need to identify the causes or mechanisms that underlie this association. That need is well represented by Reichman and Teitler's (Chap. 9) extensive list of potential "risk factors" or "exposures" that seem to "mediate" the association of interest. The list of such exposures is long indeed. There is no priority given to any particular set of factors, and no overall logic or narrative account of how the process unfolds. That would require a strong theory, which the authors confess they lack. The attempt to catalogue the relevant exposures is laudable, but may be a function of having chosen, or defaulted to a particular scale of vision. And of course we are left to conclude that the problem is very complex.

Consider a thought experiment. Suppose a person fell out of a plane and fell to earth. What is the cause of death in this case, and what are the mechanisms? At one level, this case would give rise to infinite complexity. We might examine the body carefully and decide that the skull was crushed, the chest cavity compressed, leg bones shattered, heart ruptured, etc. Determining the cause of death would at one level of examination be extremely complex. Microscopically, it would be apparent that every cell of the body had been impacted (so to speak). The consequences could be seen as far down as the quantum scale. On another level, the problem is very simple. The person fell out of a plane. How does this example help? I will get to that in a moment.

Reichman and Teitler (Chap. 9) discuss three "theories" to account for the relationship between SES and health. These include the "economic theory of health production" or the "Grossman theory" (1972), the "fundamental causes" theory put forth by Link and Phelan (Link & Phelan, 1995), and, on page 3, they list "psychosocial theory developed in the epidemiological literature…." These theories are listed and given brief descriptions in about one page; they are not discussed further in this chapter. It is unclear which of the theories the authors have chosen as their guide. Perhaps most importantly from my perspective, it is not clear exactly how the choice of a theory matters or is even necessary.

Many authors have argued persuasively that theory is indispensible for understanding the relationship between social class and health (Krieger, 2001, 2011; Link & Phelan, 1995; Macintyre, 1994; Macintyre, McKay, Der, & Hiscock, 2003). It is felt to be especially important given that social class is considered a distal or uphill factor in disease etiology, requiring a clear understanding of the risk factors or mechanisms through which social class position gets "under the skin." Almost everyone agrees that a theory is needed, but no one seems to be clear on what a good theory really looks like or how it should be used. As Reichman and Teitler (Chap. 5) have done, we often list possible theories but rarely do we choose one. I know little about the Grossman model, but I am less than convinced that Link and Phelan (1995) would claim that the fundamental causes idea is a fully realized theoretical account of social class effects. There is an astonishing absence of clearheaded discussion of just what constitutes theory in epidemiology and public health, or how we might go about developing or using one (for notable exceptions, see Campaner, 2010; Cassel, 1964; Cziko, 2000; Dunn, 2006; Frost, 1936; Hamlin, 2006; Krieger, 1994, 2011; Susser, 1991; Weed, 2001). It is possible that the complexity complex is an artifact of the failure to select a theory that has the carrying capacity to make sense of what is most important and why. The point of theory should be to actually use it to sort through the complexity and specify what are the important *drivers* of population health and what are the downstream *passengers* set in motion by particular social arrangements. In short, a theory could help us focus attention on who pushed the guy out of the plane, rather than getting caught up in the mess on the ground.

Of the three theories Reichman and Teitler (Chap. 9) mention, I am particularly troubled by the "social psychological theory developed in the epidemiological literature." Here the authors cite Marmot and Wilkinson's (2006) textbook The Social Determinants of Health (Marmot & Wilkinson). I do not find evidence of a "social psychological theory" in that volume, but that is not my primary concern. Reichman and Teitler argue that the central idea of the psychosocial theory is that stress is the explanation for socioeconomic gradients in health. This raises important questions about what stress is and how it works. They, following the literature on stress, suggest that it operates both directly (through neuroendocrine pathways) and indirectly (inducing health-compromising behaviors). The problem with this view (aside from the fact that the term stress is never defined or clearly operationalized) is that while a great deal is known about what goes on under the skin during the acute stress response (i.e., from studies of alcoholics and other subgroups, we know what happens physiologically with long-term dysregulation of the HPA axis), almost nothing is known about how social class position triggers stress. Some studies show that individual social class is associated with biomarkers of stress. However, almost no studies have actually connected exposure to particular environmental features associated with low socioeconomic position and any biomarker of stress (for a notable exception, see Do et al., 2011). Many hypothesize that being poor is associated with many stressors, but no one has worked out what it is that people of low SES are actually exposed to that evokes differential stress responses. Studies of allostatic load show that poor people look differently, but the actual environmental stimuli

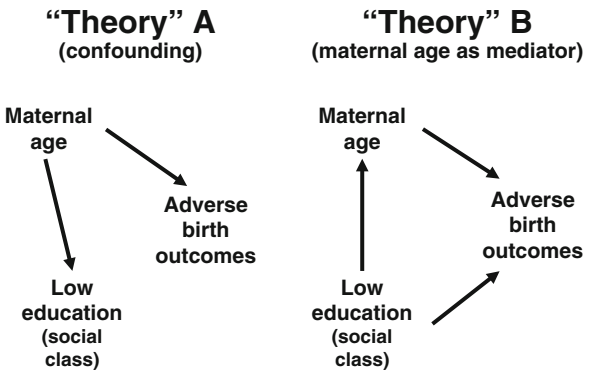

Fig. 11.1 Competing directed acyclic graphs for relations among maternal age, social class and adverse birth outcomes

that lead to AL remain a dark black box. I am reminded of John Cassel's famous fourth Wade Hampton Frost lecture (1976), credited with launching the so-called psychosocial research program. In it, he warned that "I think the simple-minded invocation of the word stress in such thinking has done as much to retard research in this area as did the concepts of miasmas at the time of the discovery of microorganisms" (Cassel, 1976, p. 108).

My own view is that the stress literature has been sloppy in not clearly identifying the psychosocial hazards to which those of low social class are exposed, making it difficult to connect stress research and studies of social class gradients. We have relied heavily on self-reported measures of subjective distress rather than direct measures of the biology. Attempts to get more biological by measuring allostatic load may be a step in the right direction, but most measures to date have confused the consequences of stress (adiposity, insulin insensitivity, hypertension) with actual stress. More to the point, stress may be a useful concept, but there is not yet a unified and coherent "psychosocial theory" to provide an explanation for social class gradients in child health.

In the absence of a clear theory, we frequently get tangled in methodological knots. Consider as an example the relationship between social class, age, and adverse health outcomes. Reichman and Teitler (Chap. 9), in reference to Fig. 9.1, argue:

> Women with less than 12 years of education are at the highest risk of adverse birth outcomes (not shown), but maternal age is so confounded with education for that group that comparisons of SES differences based on education are likely to be misleading. (p. 6)

This is a point often made. Age and maternal education are closely coupled. Consider the two directed acyclic graphs depicted in Fig. 11.1. If Reichman and Teitler (Chap. 9) mean that this is true confounding, then the association with education is false or spurious because maternal age is the real driver ("Theory" A, left). Or the association of maternal age is false and spurious because social class (here, maternal education) is the actual common cause ("Theory" B, right). It is not simply that the association is muddled. If confounding is at play, we should "control" for

education and assess the importance of maternal age. This would lead to interventions to get poor young women to delay childbearing. Or Theory B is correct, and we should *not* adjust for maternal education (to do so will induce bias). Here we are without a theory when we need one most. Which model is correct? Without a theory, we are inclined to adjust for maternal age and education as if they were competing risk factors. What if social class position determined the age at childbirth? What if there was something about social class that actually *caused* poor women to have children at much earlier ages? The appropriate interventions here would be different (reduce social class inequality through policy).

This raises a fundamental question: How would such a process work, and can social class actually be a "cause" of anything? This is where I turn next.

The Cause Box

As epidemiologists, we are interested in causes. The history of our field can be seen as one long discourse on disease causation (Hamlin, 1995, 2006; Hill, 1965; Krieger, 1994; Morabia, 2004; Susser & Stein, 2009). Epidemiologists are at ease in a world in which we can identify agents and the hosts that are invaded by them. We are on solid ground with cholera vibrio, asbestos, sugary beverages, drug A versus placebo, and even wearing a condom versus not. We have Koch's postulates, Hill's criteria, and Pearl's computer chips to help us sort out what the causes are. Epidemiologists have chosen to worship at the altar of specificity, strength of association, and the basic ontological principal that causes can be teased apart. We have worked out a sophisticated methodological machinery for detecting the causes of disease—one that requires us to imagine possible worlds—one in which some population is exposed to a candidate cause, and another identical—but counterfactual world in which the same population is unexposed (Hernan, 2004; Rubin, 1997).

Enter social class: Is it a cause? Is it a proxy for some set of causes? If social class is a cause, what kind of cause is it? Our complexity complex is, in part, a byproduct of uncertainty about whether social class qualifies as a causal risk factor. Social class does not fit the epidemiologist's way of thinking about causes. It is not like cholera, asbestos, sugary beverages, heroine, or drug A. Yet, we are faced with the undeniable fact that knowing someone's social class tells us a great deal about the probability that a person is "exposed" to all these things. We get around the direct question of whether social class is a cause by saying instead that social class is complex and then, as Reichman and Teitler do, proceed to list all the "exposures" (the traditional causal risk factors) that happen to be passengers on the social class train. Generally, there is no theory, no account of how or why these risk factors cluster, covary, and are chained together in ways that create geographically arranged pockets of what Rhodes calls "risk environments" (Rhodes, 2009; Rhodes & Simic, 2005) or what I call risk regimes (Schwartz, Bellinger, & Glass, 2011a, 2011b, 2011c). To risk sounding a bit simplistic, our definition of cause blocks us from declaring that social class itself is a cause of all the other causes. It is impossible to

imagine a counterfactual world in which the rich and the poor might be regarded as exchangeable on all other things except for their different social class positions. Social class starts at conception (or most likely before) and so, like genes, is not amendable to our discretization—our need to tease apart—and our insistence that anything that cannot be experimentally manipulated in isolation from other factors cannot be admitted to the club of causes.

What I would like to point out is that social class is so confusing and complex, in part, because we are constrained by a definition of cause that impedes good public health and forces us to look for determinants of disease that, like the keys at the base of the lamppost, are better suited to our mental picture of what a cause should be.

Consider the example of smoking. The 1964 US Surgeon General Report pointed out that smokers were at ten times the risk of death from lung cancer based on different studies of various population groups (U.S. Department of Health, Education and Welfare, 1964). At the time, there were no randomized trials; we lacked an understanding of the biology; we knew that there were many potential mechanisms through which tobacco smoke could damage the lungs; we did not yet have the vaunted Hill "criteria" (Phillips & Goodman, 2004) to tell us whether to declare smoking a true cause. (These were published later in response to confusion about whether smoking was a cause.) As evidence continued to mount implicating smoking as a cause of ill health, a shift in language and a new emphasis on developing a formal definition of disease causation emerged in the revised 2004 SGR (U.S. Department of Health, Education and Welfare, 2004).

What is meant by cause has been an ongoing controversy since then. In a very provocative and important paper, Lipton and Odegaard (2005) question whether the statement that smokers have ten times the risk of lung cancer (assuming we have adjusted for biases) is actually improved by adding the claim that smoking *causes* cancer. Do we learn anything new? Are we aided in some way as public health professionals? Do we have any additional interventions at our disposal? Odegaard and Lipton say no. By setting aside certain factors and calling them causes, we are engaging in what David Hume (1910/1748) would have called an act of metaphysical hubris. In the absence of strong, experimental evidence, declaring one factor a cause simply provides industry lawyers and epidemiologic provocateurs (e.g., famously Fisher, 1958) an easy target for criticism. Given that causation can rarely be proven, and certainly cannot be proven for variables like social class that operate through many pathways and that begin prior to conception, what good is such a restrictive definition? We know that bullets are the *cause* of injury and death from firearms, but that does not imply that distributing Kevlar vests is the ideal policy intervention. Returning to smoking, we still do not know the precise mechanisms through which smoking causes cancer. There are many overlapping pathways and mechanisms that cascade at the cellular level. The changes that occur in the lung or in the heart are infinitely complex. This does not preclude us from taking effective public health action. It is sufficient to know that smoking increases the risk of lung cancer tenfold.

Returning to the theme of this commentary, the evidence makes clear that children born into poverty and low social class have dramatically worse health through

a myriad of mechanisms and exposures. This alone may be sufficient evidence for public health action to improve the health of children. We remain perplexed by the mechanisms and pathways involved. Would knowledge of those mechanisms yield better interventions, or is our complexity complex a pointless source of obstruction? Being born poor may be like falling out of a plane. Social class will never be like cholera, asbestos, sugary beverages, heroine, or drug A. However, knowing someone's social class position tells one a great deal about the probability that all those things will be part of a unified risk regime of exposures that are not discrete, not separable, and are socially patterned because of the system that distributes rewards and risks that generates population health inequities. We could use a better theory for that.

The Problem with Culture

Part of the reason we find social class inequities so inscrutable is that we know that there are a suite of behaviors that have a lot to do with health, which vary systematically according to social class. In explaining the social class gradient in children's health, Reichman and Teitler (Chap. 9) say:

> Several health-related behaviors—regular bedtime, seat belt use, regular source of care, well child care, parents' body mass index (a proxy for diet), mother's well care, and smoking in the home—accounted for a small portion of the increasing income gradient in children's overall health status with age, but most of the increase remained unexplained (p. 9).

Explaining why it is that behaviors cluster by social class position has proven to be beyond our capacity. Vague theories are invoked suggesting that unhealthy behaviors happen to poor people because they are under stress, or because they lack resources, or live in places with broken windows, or they do not know any better. There is no consensus about why.

One reason we have difficulty explaining behavioral clustering is that public health, as a field, generally accepts the fundamental premise that behaviors are chosen by individuals. Hence, we have theories that focus on individual-level explanations like stages of change (Prochaska, Redding, & Evers, 1997), health beliefs (Anderson, 1968), or resiliency (Rutter, 1993). We invoke concepts like self-efficacy, coping styles, work–life balance, type A personality, attachment styles, and lifestyle. The frequent use of the term "style" is instructive. I (and others) argue that the concept of lifestyle is misleading and problematic (Bickenbach & Glass, 2009; Coreil, Levin, & Jaco, 1985; Krieger, 1994; Rose, 1985; Stokols, 1992). Style invokes the notion of unconstrained choice, based on idiosyncratic taste, whim, or fashion. We in public health prefer to think of health behaviors in this way because it undergirds our primary public health intervention tools (health education, tailored fact giving). The rise of lifestyle as the dominant umbrella concept has pushed out a primary concern with context and has pointed us in the direction of individual-level interventions that privatize risk and that are largely ineffective at a population level (Glass & McAtee, 2006; Rockhill, 2001; Rose, 2001).

The behaviors that underpin social class gradients, like smoking, eating bad food, risky sex, taking drugs, violence, and ignoring the sound advice of doctors and public health experts, may not be chosen at all, not a matter of personal style or taste, but may in some fundamental way be determined, forced, or cajoled. Because we think of health behaviors as chosen, we often do not consider the possibility that the forces that determine these behaviors are related to social class or may actually *be* social class.

What I would like to suggest (Glass, 2006) is that one reason we do not understand the way social class works is that we do not admit or allow for the role of culture. Culture is a highly problematic and inflammatory concept rarely taken seriously within public health (for noteworthy exceptions, see Dressler, 1985, 2001, 2006; Eckersley, 2006, 2007). Culture is difficult to measure, is not seen as a target of intervention, and may not even exist at the individual level. The mention of culture invokes the charge of blaming the victim, as was evident in the public outcry after the discourse on the "culture of poverty" in the 1960s (Morris, 1989; Ogien, 1978; Page, 2005). Culture is an emergent property of complex human systems; it is very difficult to measure, to change, or to study.

Tobacco advertisers are an exception in our society. They understand culture. Furthermore, they understand that behaviors are not freely, rationally, and individually chosen, but shaped, induced, and redirected. Advertisers also understand that culture *is* social class. Research on advertising of diverse products from cigarettes to alcohol to snack food shows that advertisers segment target audiences according to socioeconomic and race/ethnic groupings and customize messages to change purchasing behavior within each market niche (Adams, Tyrrell, Adamson, & White, 2012; Bansal, John, & Ling, 2005; Garman, Tavakoly, & Gilmore, 2010; Gentry et al., 2011; Kwate & Lee, 2007).

Conclusion

Like most such reviews, Reichman and Tietler (Chap. 9) conclude that social class matters, but that much mystery remains. In this commentary, I have explored the nature and contours of this complexity complex to better understand what has us so baffled and how the situation may impede needed public health action. The problem lies not in our lack of a solid research foundation. No further studies are needed to show that children who are born into poverty will have worse health than those born into wealth. Social class inequalities are generated in part by social, economic, and tax policies. In the USA, data clearly shows that income inequalities have increased substantially since the 1970s to levels not been seen since the days of the robber barons of the 1930s (Johnston, 2007). Evidence shows that lowered corporate income, estate and capital gains taxes, have weakened the redistributive power of federal tax policy since the 1970s contributing to higher inequality levels (Piketty & Saez, 2007). If policies can make inequality worse, they can just as certainly make it better. A detailed understanding of the precise mechanisms linking social class

and child health will not necessarily lead to a better policy solution to the problem of inequality. I argue that what will lead to effective policies is a better theory actually put to use, an alternate framework for thinking about disease causation, and serious and systematic attention to the role of culture.

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Part IV
Impact of Social Policies and Programs on Children's Health

Chapter 12
The Pro-Family Workplace: Social and Economic Policies and Practices and Their Impacts on Child and Family Health

Lisa F. Berkman and Emily M. O'Donnell

Abstract Social and economic policies designed to improve working conditions and employee well-being in adulthood have often resulted in the unintentional improvement of the health of children and their parents. Unfortunately, the USA is behind in implementing such policies and is losing ground in the health of its families compared to most other industrialized countries. We present historical patterns of infant mortality and women's life expectancy, both indicators of child and family health, over time and across the USA and other industrialized countries. Using a predominantly ecosocial framework, we review the channels or mechanisms that may link social or economic policy to a physiological change in children and/or their close family members. We continue to review a range of family and labor policies and evidence linking specific family and work policies to child and family health outcomes. We argue that, despite challenges, the identification of social and economic policies that impact the work/family interface and promote family health and well-being is critical and that the conditions which improve health for families will likely require modification in the public policy arena.

Introduction

Public policies have been among the major drivers of improvements in child and family health over the last centuries. Initially, public health policies regarding water sanitization, milk pasteurization, vaccination (Rosen, 1958), and basic hospital

L.F. Berkman, Ph.D. (✉)
Harvard Center for Population and Development Studies,
Cambridge, MA, USA
e-mail: lberkman@hsph@harvard.edu

E.M. O'Donnell, M.S.
Harvard School of Public Health,
Boston, MA, USA

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practices regarding infectious diseases (Jarvis, 1994; Semmelweis, 1861/1983) were responsible for major decreases in infant and maternal mortality. In the mid-twentieth century, medical practices reduced infant mortality substantially as the profession learned how to prevent neonatal deaths (Cutler & Meara, 1999; Paneth, 1995; Williams & Chen, 1982). There is a great deal that has been written about the ways in which these critical public health and medical policies and practices have impacted infant and child health and development. Our goal is substantially different; it is to focus on the social and economic policies that have been implemented over the last 40–50 years primarily aimed at improving working conditions, the work/family interface, workplace flexibility, and retirement policies so that men and women in the labor force would be able to take care of their own health and that of their families. Our hypothesis is that these *non-health care policies* designed to improve working conditions and employee well-being in adulthood (albeit with some attention to well-being for children and families) had fundamental and often unintentional consequences of being "pro-family," thus improving the health of children and their parents. To be clear, some labor policies, including child labor policies and antipoverty policies especially for single mothers, had quite explicit intentions in terms of improving children's health and well-being; however, our point is that these policies have not been adequately evaluated so that the health benefits can be understood and documented. Included in this category of social and economic policies and practices are those related to paid or unpaid vacation or leave, workplace flexibility, family policies more generally (childcare, parental leave), and economic incentives including the Earned Income Tax Credit (EITC) in the USA.

We begin with a brief description of the historical patterns of infant mortality and women's life expectancy over time and across industrialized countries. We focus initially on these outcomes as bellwethers for other health indicators. Infant mortality as an indicator of child well-being has the advantage of being available for many countries over long periods of time, though harmonization is not as tight as we would optimally like. Here, we also discuss cross-country comparisons in health and, briefly, raise the notion of relevant work and family policies that might account for some of the variations we see across time and place. Following the discussion of infant mortality and women's life expectancy, our discussion is built on a theoretical model drawing largely on ecosocial frameworks of disease causation. The goal is to understand at a theoretical level what the channels or mechanisms might be that could lead from a social or economic policy to a physiological change in children and/or their close family members. This model incorporates dimensions that interact and are capable of explaining spillovers across networks as well as crossovers in domains of well-being (meaning that policies improving worker productivity, for instance, might also impact health—for better or worse). In the next section of the chapter, we review and interpret the evidence linking specific family and work policies to child and family health outcomes. There are several outstanding studies that have been done in this area and a larger number of studies in which the links to health outcomes are not as well defined, though the labor or economic benefits of such policies are well known. Finally, we draw conclusions and make recommendations.

A major rationale for evaluating social and economic policies in terms of child and family health is that the conditions which improve health for families will likely

have to be modified in the public policy arena. Identification of policies that prove to be harmful to health is as important as evaluating those that are likely to have positive effects. Over and over again, we have learned that asking parents, for instance, to change behavior in the absence of a supportive social and economic context is not fruitful. Few people change without a supportive environment. We have also learned from a host of public health interventions that changing public policy, for instance, prices of tobacco or occupational and environmental exposures, has more substantial health impacts than asking individuals to stop smoking or to wear safety gear and avoid toxic exposures. We suspect the same is true with regard to family health. Finally, if social policies have spillover effects and thus improve the health of children and their families, they may be much more cost-effective than policy makers realize. In current times of economic cutbacks, such policies need to be evaluated fully for the range of outcomes that may result so that the true costs and benefits are understood.

By way of background, from 1950 to 1990, the lives of American women changed dramatically: Women flooded into the labor force but maintained high fertility. Divorce and single parenthood increased, while mobility away from extended family became common. In 1950, 59% of American women aged 25–45 lived with children under 14, 94% of those mothers were married, and only 18% were employed (Ruggles et al., 2004). In 1990, the percent of women aged 25–45 who lived with children was similar (55%), but 80% of those mothers were married, and 64% were employed. Most single mothers were employed (65%), working an average of 38 h per week. By 2000, 79% of mothers with children from 6–17 were in the labor force (Ruggles et al., 2004). Trends between 1950 and 2000 in Europe looked differently: Although female labor market participation grew quickly in many countries, fertility declined; single parenthood grew more slowly; and annual hours of work per employee declined (Eurostat Yearbook, 2002; Ruggles et al., 2004). By 1990, 22% of US children under age 14 lived in a household with only one adult, compared to only 6% in the EU-15 countries. Despite these social changes, US policies most relevant for working mothers changed little. The USA was an outlier among high-income countries for having weak labor laws and limited family protection policies (Gornick & Meyers, 2004). As we illustrate below, in 1960, the USA ranked 12th in infant mortality and 11th in women's life expectancy among the 34 OECD countries. By 1980, the rankings were 18 and 13 and, in 2008, 30 and 28, respectively (OECD, 2009b, 2009c). Most other OECD countries have now overtaken the USA in health and longevity for children and adults.

Infant Mortality in Industrialized Countries, 1960–2009, and Parallel Trends in Women's Life Expectancy

Comparisons over time and place in infant mortality can provide clues about country-level exposures, including work and family policies and practice. In addition, because child health and well-being is heavily dependent on parental (especially maternal) health, we also show parallel trends in life expectancy (LE) for

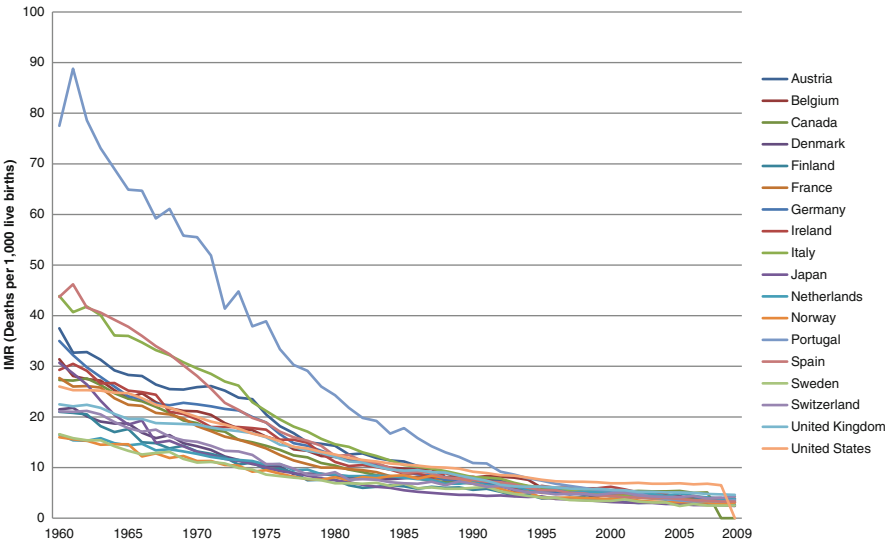

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Fig. 12.1 Infant mortality rates (1960–2009)

women. Health is an intergenerational and family experience and is dynamically interwoven with family-level exposures (see, for instance, Barker, Chap. 1). Infant mortality rates are widely accepted as a proxy for well-being. Aside from being widely available, the indicator is closely linked with socioeconomic status, access to health care, and the health status of women of childbearing age (CBO, 1992; MacDorman et al., 1994). Infant mortality has also been found to correlate strongly and significantly with other measures of overall population health, including disability-adjusted life expectancy. Thus, infant mortality data remains a strong tool for understanding health trends of children and adults alike (Reidpath & Allotey, 2003). Despite its utility, it is important to note that cross-country comparisons of infant mortality are not without methodological shortcomings. Rates are calculated based on the number of deaths of infants up to 1 year of age per 1,000 live births in a given time period. However, what constitutes a live birth is controversial. In the United States, for example, very premature births are often included in birth and mortality statistics, whereas in other industrialized countries with lower infant mortality rates, they may not (CBO, 1992).

The following figures show trends in infant mortality rates and female LE for a subset of 18 OECD countries over the last half century.

As shown in Fig. 12.1, beginning first with trends in infant mortality, in 1960 the infant mortality rate in the USA was 26 deaths per 1,000 births, compared to an average of 40.4 among all OECD countries. Countries such as Iceland, Norway, and Sweden had infant mortality rates ranging from 13 to 16.5, with the UK at 22.5 and Spain and Italy at roughly 43 deaths per 1,000 births. By 1970, infant mortality

in all OECD countries had improved but by varying degrees for different countries. Nordic countries again excelled in infant health with mortality rates between 11 and 13 deaths per 1,000 live births (though Iceland's rate did not change and, thus, its relative rank fell). Most impressive, infant mortality in Japan declined from 30.7 to 13.1 (rank 19 to 4) between 1960 and 1970. Mortality rates and ranking improved in countries like France, Germany, and Canada during this period, and while absolute rates declined, relative mortality in Spain, Italy, and Portugal remained fairly constant from 1960 to 1970. In other countries, such as the USA and UK, the relative rankings among OECD nations worsened while overall infant mortality rate improved (20 and 18.5 deaths per 1,000 live births and ranking 13 and 16 out of 34 countries, respectively).

In 1980, the infant mortality rates of Sweden, Finland, Norway, Denmark, the Netherlands, and Switzerland had decreased further (6.9, 7.6, 8.1, 8.4, and 9.1 deaths per 1,000 live births, respectively). Rates in Japan continued to improve immensely, securing the second lowest infant mortality rate among OECD countries (7.5 deaths per 1,000 live births). France and Canada also experienced relative and absolute progress (roughly 10 deaths per 1,000 live births). Perhaps most notably was Spain's mortality decline to 12.3 deaths per 1,000 live births (ranking 16 out of 34 OECD countries, compared to a rank of 24 ten years earlier). Yet again, mortality in the USA and UK improved (12.6 and 12.1 deaths per 1,000 live births, respectively), but their relative ranks fell. Mortality rates in Portugal remained strikingly high compared to other European nations at 24.3 deaths per 1,000 live births.

At the start of 1990, Japan ranked best in infant mortality among all OECD nations (4.6 deaths per 1,000 live births). With the exception of Denmark whose ranking fell to 12 in 1990 from 6 in 1980, rates in Nordic nations remained relatively low as well. Canada, Germany, and Italy experienced notable improvements in relative and absolute infant mortality (6.8, 7, and 8.1 deaths per 1,000 births, respectively), and Spain's ranking among OECD nations also continued to improve. The UK remained ranked at 15, while the relative mortality rate in the USA continued to worsen (7.9 and 9.2 deaths per 1,000 births in the UK and the USA, respectively).

A new millennium brought with it sustained improvements in infant mortality in industrialized countries. In 2000, Iceland, Japan, Sweden, Finland, and Norway continued to boast the lowest infant mortality rates in the industrialized world, though relatively poor rankings in neighboring Switzerland, Denmark, and the Netherlands persisted. Italy's overall infant mortality improved most drastically (4.3 deaths) resulting in a rank of 7 out of all OECD nations, followed by Germany, Spain, and France. Mortality in Canada, however, did not continue to improve with the same vigor as in the prior decade, and its ranking fell from 5 to 18, despite absolute infant mortality declining to 5.3 deaths per 1,000 births. Similarly, mortality in the USA and UK continued to exhibit far less impressive declines compared to their OECD counterparts, with Portugal's rank exceeding both in 2000.

Despite drastic variation in infant mortality rankings over time, almost three-fourths of OECD currently boasts infant mortality rates lower than 5 deaths per 1,000 births. In 2008, Luxemburg and Slovenia exhibited better mortality rates

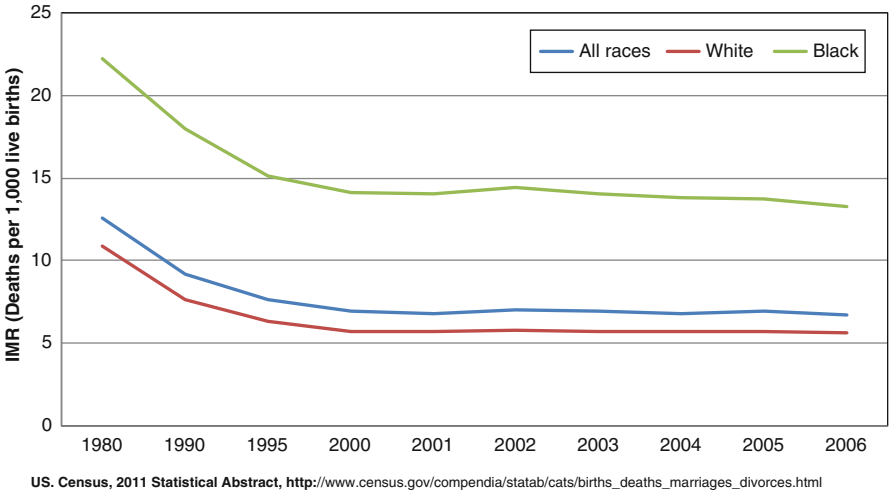

Fig. 12.2 US infant mortality rates by race (1980–2006)

(1.8 and 2.4 deaths per 1,000) than Japan, Iceland, Sweden, or Finland, despite having had rates as high as 31 and 35 deaths per 1,000 in 1960, respectively. Infant mortality in the USA has shown little additional improvement since 2000 and ranks even lower today (30 among 34 OECD countries with 4.7 deaths per 1,000 births). In addition to its lack of relative progress, drastic racial disparities within the USA are present as depicted in Fig. 12.2. Though data from all races indicate a gradual decline in infant mortality from the 1980s until just before 2000, when rates began to taper, non-Hispanic blacks in the USA consistently experienced mortality rates more than two times higher than their white counterparts.

Similar to the infant mortality rates, trends in female life expectancy indicate that the USA is losing ground, as shown in Fig. 12.3. In 1960, life expectancy for women at birth was estimated to be 73.1 years, and the USA ranked 11th behind Nordic countries (except Finland), Australia, the UK, France, and the Czech Republic where women were expected to live as long as 76 years. At this time, female life expectancy was as low as 53.7 and 66.7 years in Turkey and Portugal, respectively. Throughout the next decade, all OECD countries experienced increases in female life expectancy, and while women in nations like Norway, Iceland, and Sweden lived the longest, Finnish and Spanish women experienced the biggest strides in longevity with life expectancies rising to roughly 75 years, increasing their rank among OECD nations to 10 and 11 (up from 14 and 15 in 1960), respectively. Women in Japan and the USA were expected to live until 74.7 years of age, up from 70.2 to 73.1 ten years before, respectively. While this improvement in longevity represented a substantial increase in ranking for Japan, the USA had actually experienced a relative decline.

In 1980, America's rank fell to 13 (77.4 years). Iceland, Norway, the Netherlands, Switzerland, and Sweden all remained leaders in female longevity, and Canada joined their ranks when it was first surveyed this year (78.9), ranking 5 among all

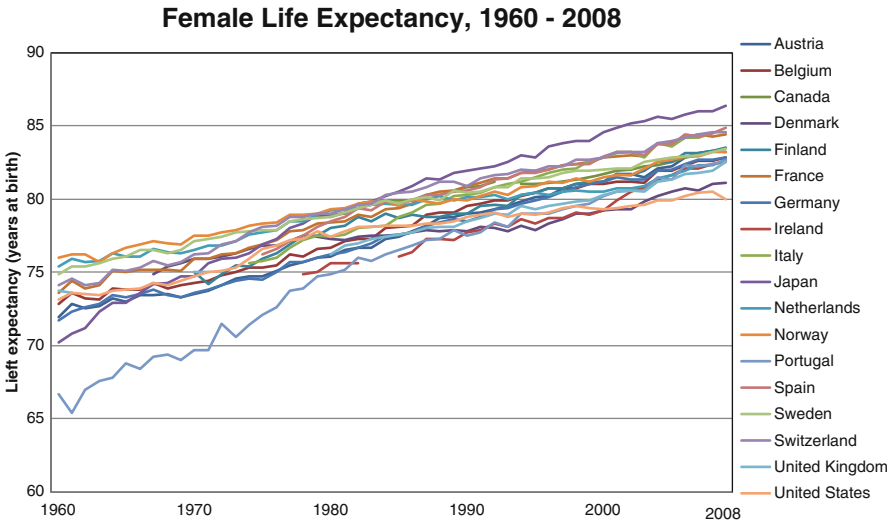

Fig. 12.3 Female life expectancy (1960–2008)

OECD nations. Female life expectancy in Japan also continued to climb to 78.8 years, ranking it 7. Similarly, Australian women began to live to an average of 78.1 years, ranking it 10 (up from 16 in 1970). Relative longevity in the UK and Ireland saw declines (ranked 17 and 23 in 1980, respectively), despite absolute female life expectancy in these countries increasing to 76.2 years and 75.6 years, respectively. A decade later, Japan was ranked number 1 in female life expectancy among OECD nations (81.9 years), followed by Switzerland, France, and Canada at roughly 81 years. Female longevity in many Nordic countries including Iceland, Sweden, the Netherlands, Norway, Finland, and Denmark experienced relative declines, and Italy's rank increased to 8. Yet again, life expectancy increased in the USA but its position among OECD nations continued to plummet.

The year 2000 continued to bring improvements in female longevity in all OECD countries. Japan continued to rank highest at 85.6 years, followed closely by Spain, France, and Italy (roughly 83 years). These rankings marked a substantial relative increase for women in Italy, compared to 10 years before. Women's longevity in Germany, Austria, and Portugal also made impressive strides this year. Accordingly, the relative position of Sweden, Iceland, and Norway declined. Similarly, the USA was ranked 24 with an estimated female life expectancy of 79.3. Eight years later, female life expectancy was ranked 28 in the USA, tied at 80 years along with Poland, Estonia, and the Czech Republic. In this short time, the following countries impressively ascended the ranks: Ireland (ranked 26 in 2000 to 20 in 2008), Israel (ranked 16 in 2000 and 8 in 2008), and Korea (23 in 2000 to 6 in 2008), although Japan, Spain, and Switzerland continue to have the highest expected female longevity among OECD nations.

Thus, in looking at both trends in infant mortality as well trends in women's life expectancy, we are struck by the parallel patterns. Particularly, with regard to the USA, we are struck by the low international standing as we enter the twenty-first century and with the fact that the USA has lost ground during this time for both women and children. We suspect that social and economic policies that frame the ways in which women can participate in the labor force and continue to participate fully in family life are taking a toll on women and their children.

An Ecosocial Framework of Work/Family Demands, Control, and Formal and Informal Support

A variety of theories exist to explain the impact of social policies on population health and, in this case, the health and well-being of children and families. Social science theories of relevance to parents, such as role enhancement theory, posit that participation in multiple roles, such as home and work, may lead to energy expansion, which in turn generates opportunities and resources such as income and self-esteem to promote health. The role enhancement literature has indicated that job–role satisfaction, also referred to as work–family enrichment or positive spillover (Greenhaus & Powell, 2006), contributes positively to individual and partner mental health as well as the psychological well-being of children of working parents (Barnett, 2004). Taking this theoretical approach one step further, it is reasonable to suggest that family-friendly policies, which contribute to the efficient and satisfying participation in roles at home and work, have the potential to positively impact the well-being of children.

Frameworks related to social networks and relationships may also be pertinent to understanding the link between social policies and child and family health. Attachment theory, first introduced by John Bowlby, suggests that formation of close emotional bonds, particularly among mothers and infants, is crucial to child and adult development as well as creation of future social relationships (Bowlby, 1969). In this model, the presence of social policies that provide parental leave, for example, are presumed to have a direct effect on child well-being by allowing parents to spend time with their offspring at key developmental junctures. Social network theorists are also concerned with the psychosocial environment, particularly those ties that "cut across traditional kinship, residential, and class groups to explain behaviors they observed such as access to jobs, political activity or marital roles" (Berkman, Glass, Brissette, & Seeman, 2000). These models propose that network structures determine individual behaviors and attitudes through the resources they make available as well as constrain. Here, traditional theorists emphasize the role of social institutions in guiding resources, and later work in this area suggests that networks operate through four primary pathways, including the provision of social support, social influence, social engagement and attachment, as well as access to resources and material goods (Berkman et al.). Thus, according to this perspective, the presence of family-oriented policies, whether they be in the form of child

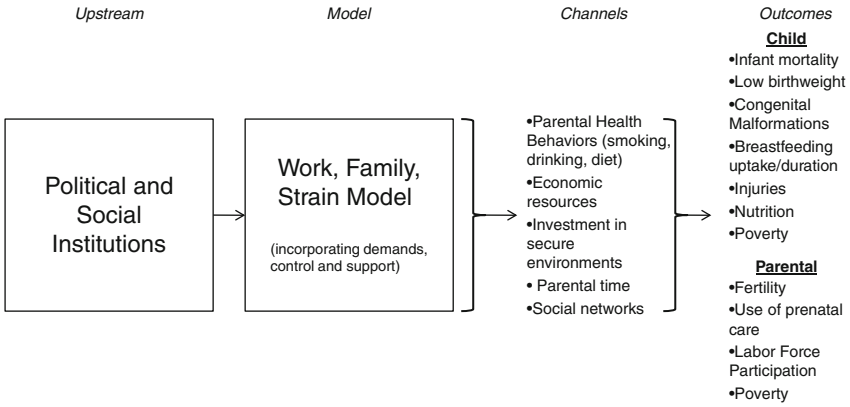

Fig. 12.4 Work, family, strain model

allowance, parental leave, childcare leave, or maternity grants, is viewed as capable of shaping health decision-making processes that impact parental and child health.

From an economic point of view, child health can be framed as a function of baseline health status, including child genetics, medical care and technology, parental time investments, income, and stochastic shock. Economists also concede that children possess a certain level of health capital at birth, as determined by attitudes surrounding prenatal care, for example, but that health capital can be improved through inputs like parental time and household goods such as immunizations and diet. Predominantly, however, this school of thought posits that parental time investments or parent's time away from work are the predominant mechanisms by which family-oriented policies affect child health and that pathways such as child genetics and medical care would not be influenced by policy-level interventions (Ruhm, 2000; Tanaka, 2005).

Despite the many theories that seek to explain how the confluence of conditions facing American children and women may impact morbidity and mortality and overall LE, the model employed here builds on a general framework, an ecosocial model of disease (Krieger, 1994). We refine this framework more explicitly to identify the dynamics between labor and family policies and demographic changes and child and family health. The ecosocial model of disease causation proposes that epidemiologic frameworks are strengthened by linking societal and biophysical determinants of disease over the life course and over historical periods. Our work–family strain model (Fig. 12.4) incorporates aspects of family context into the well-established job strain model, which relates job demands, job control, and social support to a broad range of outcomes for parents, but especially for mothers. We hypothesize that these impacts spill over to children. American women (and to a certain extent men as well) encounter demands from full-time work and high family needs, coupled with low formal support (social protection policies) and often limited informal family support. This combination is exacerbated for low-wage and

low-education workers who have little job control and often live near the poverty level. The conflicting demands associated with single parenthood and work–family tensions strongly affect cardiovascular risks such as smoking and BMI and likely also affect CVD via direct physiological consequences of chronic stress. We hypothesize that the interaction of high demands, low control, and low support leads to sustained stress, damaging health behaviors, and cumulative cardiovascular damage. Women are especially affected, but adult men increasingly suffer from many of the same work–family demands. These health effects among mothers may spill over directly to infants during in utero experiences as well as play out over their childhood via related behavioral and environmental interactions (e.g., an employed woman's ability to breastfeed). We also hypothesize that regional variation in the *distribution* or the *toxicity* (effect of exposure on disease) of work–family strain influences variations among countries and within the USA in child and family health. Variations in work–family strain may arise from differences in family demands, workplace conditions, family protective policies, or informal family supports. Of central interest in this chapter is a focus on formal or institutional supports based on family policies. Figure 12.4 depicts this model. Here, one can see the three dimensions that create work–family strain. On the left-hand side is the control dimension. On the bottom is work–family demand and along the third dimension is support. Relevant to our work here is the view that social and economic policies serve as a form of institutional support for families. We also hypothesize that families with low control and high demand will be most vulnerable and, in fact, most in need of formal and institutional support in the form of public policies.

The Evidence Linking Family and Work Policies to Child Health

Overview of Family Policies

Gornick (Gornick, 2004; Gornick & Meyers, 2004), Ferrarini (Ferrarini & Norstrom, 2010; Ferrarini & Sjoberg, 2010), Heymann (Earle & Heymann, 2007; Heymann, Earle, & Hayes, 2007), Ruhm (Ruhm, 2000), Winegarden (Winegarden & Bracy, 1995), and others have identified the range of family or work–family policies that are linked to child well-being. In so doing, they often point out that American policy makers and researchers have long noted the strength of European policy supports for working parents (Gornick, 2004; Gornick & Meyers, 2004) so that many of the common models or typologies of family policies have grown from Northern and Western Europe. There are far more types and ranges of policies regarding family well-being or work–family interface than there are rigorous evaluations of such policies. Theoretically, we are interested in the full range of policies though our review here reflects the narrower range. As Ferrarini notes, family policy institutions have a potential to affect infant mortality in different ways:

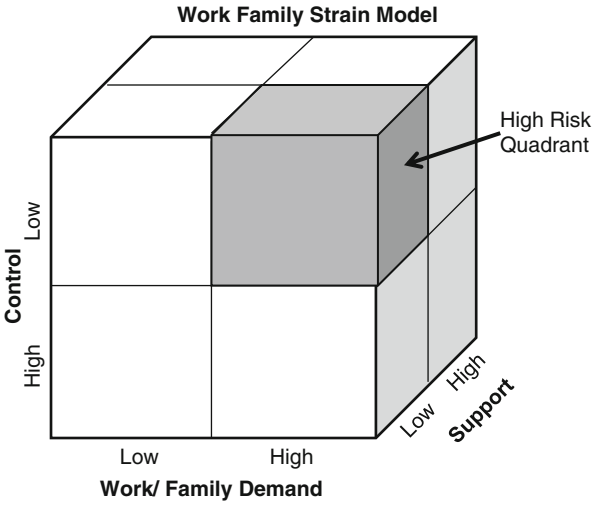

Fig. 12.5 Theoretical causal model

They can structure the time that parents can spend with their children or the income of the household either through direct contributions or indirectly through labor market behavior (Ferrarini & Norstrom, 2010). These midrange outcomes may influence child health via another set of pathways or channels including direct physiological pathways related to stress, consumption and patterns of health behaviors, and economic resources resulting in physically, emotionally, and cognitively healthier environments. Ultimately, these exposures may influence child health along a spectrum of life course exposures from basic decisions about fertility to in utero exposures and exposures during infancy and early childhood. Figure 12.5 shows this theoretical causal model.

Several models have emerged to classify types of family policies. Ferrarini and Norstrom (2010), for instance, have ways of classifying models as they provide traditional family support and/or support to dual earner families. The traditional family policy model present in many western European countries, for example, has approaches that reinforce traditional family support and highly gendered division of labor in families (e.g., flat-rate childcare leave benefits, tax deductions for the less economically active spouse). In contrast, in some Northern European countries, a newer model has emerged which encourages dual earner families to remain in the labor force (e.g., earning related parental leave, publicly subsidized services for the youngest children and the elderly) and supports gender equality. A third type is more market oriented, such as that present in the USA which falls into a minority category of countries that does not offer paid leave following childbearing, support for breastfeeding, or paid annual leave (Heymann et al., 2007). Finally, in considering the impact of these policies, one would optimally incorporate generosity of benefits into the framework as well as breadth of distribution of benefits. In this chapter, we focus essentially on those policies which are related to work or offer financial incentives to remain in the labor force while caring for family members.

We have included a few references to pension reforms which have the surprising intergenerational impact of spillover to impact the health of young children. Because the literature is sparse in this area, we have a review which points us in the direction for future evaluations of a much broader set of "family" policies.

Family Policy and Infant Mortality

Research indicates that family-oriented policies may reduce infant mortality. Almost 20 years ago, Wennemo investigated the link between public policy provisions (including family benefits) and the quality of unemployment insurance on infant mortality rates in 18 industrialized countries, including the USA, from 1950 to 1985 (Wennemo, 1993). Wennemo also demonstrated that, although economic development does impact infant mortality, it does not sufficiently explain variability across countries and that gaps may be addressed by assessing sociopolitical factors. Findings suggested that one percentage point increase in levels of family benefits may be accompanied by a reduction in infant mortality of about 0.65 per 1,000 births. Additionally, an increase in unemployment rate was associated with lowered infant mortality but much more modestly.

More recent research has continued to reveal the importance of social policies on infant mortality. In 2010, Ferrarini and colleagues examined the impact of higher family policy generosity, defined as the annual replacement of family policy benefits after taxation, on infant mortality (Ferrarini & Norstrom, 2010). These benefits, inclusive of earnings-related parental insurance, childcare leave benefits, child allowances, maternity grants, and tax deductions, were assessed in eighteen OECD countries, including the USA, from 1970 to 2000. Unlike Wennemo, Ferrarini concluded that increases in GDP, operationalized as purchasing power parity in US dollars, in postwar industrial societies may have weak or even unfavorable effects on infant mortality. Similar to Wennemo, Ferrarini suggests that family policies may be driving declines in infant mortality through mechanisms such as time to facilitate breastfeeding and other healthy newborn practices as well as availability of disposable income. Family leave policies which provide income support during leave and enable women to remain in the workforce are related to availability of disposable income.

Two years prior, Lundberg and colleagues studied the same OECD countries during the same time frame to determine the impact of family policy generosity using a measure of annual wage replacement on infant mortality (Lundberg et al., 2008). Lundberg's work, however, distinguishes dual-earner support and general family support in the analysis. The dual-earner model, embraced largely by Nordic nations, allows both mothers and fathers to combine paid employment with childcare through earnings-related parental leave benefits, universal child benefits, and childcare support. A general family support policy, on the other hand, is described as "highly

gendered" and includes flat-rate benefits for leave and childcare as well as subsidies for dependent spouses. Interestingly, Lundberg's results indicate that an increase by one percentage point in dual-earner support lowers infant mortality by 0.038 deaths per 1,000 births but that general family support was not related to infant mortality. The authors also acknowledge potential issues in extending the Nordic, dual-earner model to other countries.

Scholars have also explored the specific impact of parental leave on child health outcomes, including infant mortality. In 17 OECD countries (1959–1989), Winegarden and Bracy (1995) found that an additional week of maternity leave was associated with a modest decline in infant deaths (0.5 deaths per 1,000 live births). Pathways such as encouraged or prolonged breastfeeding and income effects are cited; however, other researchers have critiqued the study's methodological rigor (Ruhm, 2000; Tanaka, 2005). In 2000, Ruhm similarly investigated the link between paid leave (rights to job absences where the level of income support depends on prior employment) and child health in Europe. Using data from sixteen countries over the course of more than two decades (1969–1994) and controlling for time and country effects and relevant confounders, results suggest a negative relationship between leave durations and infant mortality, particularly postneonatal mortality. Specifically, a 10-week extension in paid parental leave may reduce infant deaths by 2.5–3.4%, which translated to roughly 13 fewer deaths per 1,000 live births, or upwards of 4.5% for postneonatal mortality. A limited investigation into unpaid leave found null results (Ruhm, 2000).

Building on Ruhm's work, Tanaka (2005) conducted an analysis of the impact of job-protected paid leave and other forms of parental leave on infant mortality and other child outcomes, including low birth weight and child immunization coverage in the same European countries, plus the USA and Japan, between 1969 and 2000. Here, a 10-week extension in paid leave resulted in a decrease in infant mortality of 2.3–2.5%, or roughly 10 per 1,000 live births. Consistent with Ruhm's findings, the strongest impact of paid parental leave was on postneonatal mortality. These results persisted despite controlling for other social policies such as public expenditures on parental leave and family services. Unpaid leave was not found to predict declines in infant mortality, and parental leave generally did not have a significant effect on the other child health outcomes assessed.

Other policies not specifically intended to benefit children and families appear to have spillover effects on infant mortality. In the USA, for example, it has been argued that federally mandated racial desegregation in the 1960s contributed to drastic reductions in infant mortality rates among African Americans. While racial gaps in infant mortality declined throughout the entire country during the years immediately following desegregation (1965–1971), blacks in the Mississippi Delta experienced the biggest relative gains. Almond and colleagues largely attribute this trend to dramatic improvements in access to hospitals in the Southern USA. In fact, racial integration of public health care facilities corresponds directly in terms of time and location to substantial declines in black infant mortality, which was cut in half in this 6-year period (Almond, Chay, & Greenstone, 2006).

Family Policy and Low Birth Weight

Birth weight also serves as an indicator of child health and prenatal exposures and has been shown to predict other child and adult outcomes, including infant mortality and educational level and income (McCormick, 1985; Strully, Rehkopf, & Xuan, 2010). A review by Spencer indicates the study of social policies and low birth weight may not be as pervasive as infant mortality, but that compelling evidence exists nonetheless (Spencer, 2004). Income inequality, in particular, may have significant effects on a baby's birth weight. Using national data for the USA, Kaplan and colleagues found that the percentage of total household income received by the poorest half of the population was significantly correlated with the proportion of live births weighing <2,500 g ($r=0.67$; $p<0.001$) (Kaplan, Pamuk, Lynch, Cohen, & Balfour, 1996). Two studies assessing inequities in Europe and North America identified similar trends. Lynch and colleagues concluded that inequality based on household disposable income and measured by the Gini coefficient was associated with a greater proportion of low birth weight infants ($r=0.79$, $p=0.001$) (Lynch et al., 2001). Interestingly, this relationship was reduced when the USA was excluded from the analysis. Building on the work by Lynch and colleagues with the inclusion of Israel, Muntaner et al. (2002) arrived at identical results and, while a bit qualitative, claimed that the best infant outcomes were found in countries with the strongest "welfare state" and greatest family and child supports.

Stronger empirical evidence of the impact of welfare programs on low birth weight also exists. A compelling study by Strully et al. (2010) tested the effects of prenatal poverty on low birth weight through a natural experiment of changes to Earned Income Tax Credits in the USA. The EITC is a refundable tax credit for low-income, single mothers. Similar to other welfare programs in the USA, such as Temporary Assistance for Needy Families (TANF), the policy requires women to work and is believed to improve standards of living through both increased engagement in the labor force and wages. However, unlike TANF, EITC is administered through the IRS independent of other social programs. Strully and colleagues tested a number of hypothesis including the effect of EITC on low birth weight, the potentially perverse effects of an income credit on maternal smoking, the extent to which EITC has indirect effects on family income (through unemployment incentives and earnings), and whether the effects of the credit vary by maternal characteristics such as age and education.

The sample was limited to US unmarried mothers with low education (high school degree or less) who previously experienced at least one live singleton birth in their state of residence. After controlling for state effects, including states' economic circumstances and social policies, as well as time fixed effects, Strully et al. (2010) found that the EITC increased birth weights by roughly 16 g and that income credits were associated with higher earnings and employment. No evidence of moderation by maternal education was present. However, the EITC may be most beneficial for children born to women aged 19–34. Small effects were found among young women, possibly due to their relatively modest labor participation. Negative

effects were detectable in women over 35, perhaps due to pregnancy complications that occur more frequently among women of advanced maternal age. To assess whether the type of welfare program affects low birth weight, a similar analysis of TANF was conducted and produced mixed findings. This suggests that the nature of welfare is relevant to improving child health. Interestingly, EITC was associated with reduced odds of maternal smoking by about 5%, but TANF was associated with increased smoking odds.

Strully et al.'s (2010) work provides compelling evidence of the impact of income credit policies on child health. The findings are further validated through a series of sensitivity analyses. In these checks, the authors focused on three female subgroups expected to be less affected y the EITC (married women, those with over 16 years of education, and women giving birth to their first child), relative to the study sample. Strully and colleagues also assessed descriptive birth statistics for states immediately preceding the enactment of the EITC and argued that inappropriately strong EITC–birth weight associations among subgroups or changes in maternal descriptive statistics (age, education, etc.) would raise concerns about assumptions surrounding the natural experimental design. Results, however, indicated methodological soundness. Despite these compelling results, it is important to note that Ruhm (2000) did not find a significant effect of parental leave on low birth weight, in the study described above.

Family Policy and Breastfeeding

In the industrialized world, breastfeeding has been associated with reduced accounts of diseases and conditions as varied as acute lower respiratory infections, lymphoma, childhood-onset insulin-dependent diabetes, eczema, asthma, and Crohn's disease (Lawrence, 1997). Ever-breastfed children also appear to experience lower postneonatal mortality, and their risk of death declines with breastfeeding duration (Chen & Rogan, 2004). The WHO recommends that mothers breastfeed their children for 6 months exclusively and another year and a half in conjunction with other foods (WHO, 2011). Yet, ever-breastfeeding rates vary in OECD nations, ranging from below 70% in Ireland and France to just above 75% in the USA, and 100% in Denmark, Sweden, and Norway (OECD, 2009a). Of 173 countries studied by Heymann and colleagues, 107 countries protect a woman's right to breastfeed and 73 offer paid breaks for feeding. The USA falls into neither of these categories (Heymann et al., 2007).

Parental leave has been shown to benefit mothers' ability to initiate and continue breastfeeding. In the USA, Roe and colleagues concluded that duration of leave from work and work intensity significantly affects the duration of breastfeeding. They also tested whether the opposite was true—that breastfeeding had effects on work return and intensity—and reported null results (Roe, Whittington, Fein, & Teisl, 1999). Also, among American mothers employed before giving birth, Berger and colleagues found that maternal return to work within 12 weeks of birth was

shown to reduce breastfeeding and number of medical checkups before age one, improved immunization uptake in the first 18 months, and was associated with an increase in child externalizing behaviors including aggressiveness, impulsivity, and defiance, as reported by the mother at ages three and four. These results were stronger for full-time working mothers (Berger, Hill, & Waldfogel, 2005), which is echoed by other research citing part-time work, lack of long mother–infant separations, employer-sponsored childcare, supportive work environments and facilities, and the flexibility to work at home as facilitators to breastfeeding (Jacknowitz, 2008; Johnston & Esposito, 2007). Despite this evidence, only 21 states in the USA have passed legislation to protect a woman's right to breastfeed. Texas is the only state that has passed a law declaring that mothers can breastfeed in any place they are authorized to be and created a designation for mother-friendly businesses that address flexible work schedules; access to clean, safe water; and access to private areas that allow mothers to pump (Mills, 2009). Thus, despite the clear child benefits of breastfeeding, few formal policies exist in the USA, and the support of lactation programs and facilities for working mothers is overwhelmingly left to the discretion of the employer.

Family Policy and Other Child Health Outcomes

An emerging area of research focuses on parental employment and developmental outcomes. Though much work remains, this literature suggests that children's development may benefit if mothers had more flexibility to stay home for longer periods of time. Data from the National Institute of Child Health and Human Development Study of Early Child Care found that maternal employment at 9 months ($p<0.01$) and 6 months ($p<0.10$) of age was associated with Bracken School Readiness scores at 3 years of age, controlling for quality of childcare, home environment, and maternal sensitivity. These results were more pronounced for mothers working more than 30 h each week. Employment at the first, third, or twelfth months of a child life did not have significant effects on school readiness nor was there evidence that maternal employment at any age affected child memory, learning, problem solving, or early communication (Brooks-Gunn, Han, & Waldfogel, 2002). Ruhm (2004) also assessed the effect of 20 extra maternal employment hours weekly on child cognition and found modestly lower scores for assessments in picture vocabulary scores, reading, and mathematics for young children.

Other studies highlight the role of family policy and childhood injuries and childhood poverty. In a study of six transition countries (Estonia, Poland, the Slovak Republic, Slovenia, the Czech Republic, and Hungary), family policies consisted of child allowances, earnings-related parental leave, flat-rate childcare leave, and lump-sum maternity grants. In the Czech Republic, a marriage subsidy for households with one working and another less economically active spouse was also present. Graphical depictions of policy generosity, measured as the level of benefits after taxes, indicate a negative relationship with both childhood injuries and poverty, though only a correlation for poverty was provided ($r=-0.70$ and significant at the

0.01 level). A descriptive study conducted by Immervoll and colleagues reinforces the beneficial role of European child benefits, defined as cash transfers for parents or other caregivers on behalf of dependents, on childhood poverty. This work also included a simulation of the effects of removing these benefits and found that, using 1994 data, the child poverty rate would rise from 3.1% to 7.5% in Denmark, for example (Immervoll, Sutherland, & deVos, 2001). These findings are particularly relevant given that childhood poverty appears to increase morbidity and decrease mortality over the lifespan (Evans & Kim, 2007; Galobardes, Lynch, & Smith, 2004, 2008), even after controlling for social status (Kuh, Hardy, Langenberg, Richards, & Wadsworth, 2002).

Finally, social policies may also have positive effects on child anthropometrics. In South Africa, a public pension system administered predominantly to blacks in an effort to create equity in the post-Apartheid era has been shown to have positive effects on child health and nutrition. In the mid-1990s, roughly 80% of all black South Africans received the government pension, the maximum amount of which totaled <3 dollars per day, and roughly one-third of black children under five lived with an elderly pension recipient. Height and, in particular, height-given age are believed to reflect accumulated investments in nutrition and health care throughout a child's life, and these anthropometric measures were captured among a randomly selected cohort of South African households with children 5 years or younger. Nonparametric analyses indicated that the government-sponsored pension program improved the health and nutrition of children. Over 2 years, female children in pension households gained 2 cm in height more than girls living in households not eligible for the pension (effects for boys were not significant). Of additional interest is the finding that these anthropometrical differences were present only in households in which women received the pension. While not intended as a family policy per se, this study offers evidence that programs to address income inequality have the potential to drastically improve child health outcomes, particularly in reference to and when administered by females (Duflo, 2000).

Family Policy and Fertility

Evidence also suggests that family-oriented policies may increase fertility. France, the USA, and Nordic countries have already begun to experience a modest reversal of what was previously a decline in fertility rates (Bonoli, 2008). According to Rindfuss, the correlation between female labor force participation and fertility was negative in the 1960s and 1970s but, since the 1990s, has been positive and moderately strong, suggesting that contemporary policies that promote women working outside the home may be associated with increased fertility (Rindfuss, Guzzo, & Philip, 2003). Studies across OECD countries confirm this notion. In this context, Winegarden and Bracy (1995) assessed the impact of family leave, specifically one additional week of maternity leave, and estimated that the policy had the ability to increase general fertility rates from 1.1 to 1.4 births. Other research points to the role of female unemployment and social policies, particularly those policies on childcare

and maternity payments, in determining fertility (Bonoli, 2008; Castles, 2003; Drago, Sawyer, Shreffler, Warren, & Wooden, 2011). It is important, however, to note that Castles (2003) found no association between fertility and parental leave or expenditures on family benefits, and a recent review of social policies and fertility found mixed evidence across the empirical literature (Gauthier, 2007).

Family Policy and Women's Labor Force Participation

Links between family policy and female labor force participation are somewhat sparse. As Winegarden and Bracy (1995) point out, a challenge in this area of research is varying employment classifications for parents on leave. That is, whether a woman on maternity leave is considered employed or not differs across countries. Nevertheless, the authors estimated that a marginal effect of an added week of leave results in a slight increase (0.60–0.75% points) in the labor participation rate for women aged 20–34 (Winegarden & Bracy). Similarly, Sundström (1993) attributes family policies in Sweden, the first country to offer parents paid leave following childbirth, to women's increasing labor force participation, though the author did not examine correlations between the two factors or address causality.

Conclusions

Over the last decade, there has been a growing interest in exploring the unanticipated health impacts of social and economic policies designed mainly to improve well-being, reduce discrimination, maintain family stability, and improve mobility for disadvantaged populations. In this chapter we have explored the ways in which policies, especially those social and economic policies which relate to the work and family interface, may impact the health and well-being of children. We have relied heavily on studies of such policies which have strong experimental elements, thereby avoiding some of the downfalls of observational studies where selection undermines our ability to make causal inferences. However, these evaluations have not been without significant problems. In conclusion, we outline a few reasons to continue work in this area and outline some of the challenges that exist in the interpretability of results.

Rationale for Evaluating Child Health Impacts of Social and Economic "Family" Policies

1. A major rationale to evaluating social and economic policies in terms of child health is that it is likely that conditions which improve child health will have to be modified in the public policy arena. Action to change policies that prove to be

harmful to health are as important as implementing those that are likely to have positive effects. In an era of cutbacks in traditionally liberal and social democratic countries, it may be of critical importance to identify the health impacts of such cutbacks.

2. Policy makers need research that says more than poor children are worse off—or single parenthood may be hard for both parents and children. Policy makers need potentially effective solutions—or at least the suggestion for a promising solution—to make policy. Epidemiologists, psychologists, and other social scientists have been helpful in monitoring and identifying health inequalities but could be even more effective if they evaluated potential solutions. Policy makers want reliable information on what kinds of policy changes actually lead to improvements in child health and well-being. If not, they cling to what they understand about the narrow benefits of specific medical care policies.
3. Policies often have spillover effects making them much more cost-effective than policy makers realize. These spillover effects mean that policies designed to relieve poverty may have health impacts or they may actually cross over to improve not only the health of direct beneficiaries but other family members. We are currently engaged in a study to see if increasing work place flexibility will improve health of employees *and* their families (WFHN, 2005). If such spillover effects are common, it means we are regularly underestimating the cost–benefit of such policies. Furthermore, many of the spillover effects are intergenerational in impact. Positive intergenerational impacts may help to reduce resistance to policies that are seen as favoring one group (e.g., older workers, families, recent immigrants).

Challenges that Exist in the Interpretability of Results

1. Observing changes in child health in relation to the implementation of social and economic policies is not easy. In some cases, the period of exposure may not coincide with the most sensitive etiologic period for a large number of people. Some health benefits may take years, if not decades to appear. We often lack health indicators and biomarkers of risk that would be the most sensitive indicators of impact.
2. When one policy is implemented, others often coexist. Attributing effects to one single policy may be harder than we think. While policy implementations substantially reduce selection effects, they do not solve the problem of identifying the precise causal exposure. For example, the EITC may be implemented in US states at the same time states are increasing Medicare benefits or implementing other antipoverty programs. Strully et al.'s (2010) analysis of the EITC impacts on low birth weight is an outstanding example of considering these potential joint or interactive impacts. In Europe, family policies often simultaneously changed working conditions, day care, and financial incentives. Approaches which hold place constant (some econometric approaches or time varying

analyses) help to overcome some of this concern, but clearly more thought needs to go into understanding the confluence of policies which happen closely in time and place.

3. Multiple channels are likely to mediate policy impacts on child health. These channels may be behavioral, social network related, economic, or stress reducing. Because in most cases researchers have not anticipated policy analyses, it is often difficult to gain traction on identification of the most important channels linking a broad policy to health outcomes. In observational studies designed explicitly to test a series of hypotheses, we often have more information on potential mediating mechanisms. Understanding these channels or mechanisms would enable us to further refine policies or develop new strategies to improve health. For instance, in some cases, financial resources may be the central element of family policies in terms of their impacts on child health. In other cases, financial resources may have little direct impact and time factors enabling parents to spend time with children may be the critical elements.

Identifying social and economic policies that impact the work/family interface and turn out to be truly "pro-family" and promoting of family health and well-being is critical. The USA is behind in implementing such policies and is losing ground compared to most other industrialized countries in the health of its children and their families.

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Chapter 13
Work–Family Strain and Health Care For Children

Paul J. Chung, Katherine D. Vestal, and Mark A. Schuster

Abstract Berkman and O'Donnell's (Chap. 12) model of work–family strain provides a useful backdrop for an examination of family leave policies and parents of chronically ill children. About 15% of children in the USA are children with special health-care needs (CSHCN) (Bethell et al. Maternal and Child Health Journal 12:1–14, 2008), and their parents are at particularly high risk of employment instability and financial problems (Kuhlthau et al. Maternal and Child Health Journal 9:207–218, 2005; Looman et al. The Journal of Pediatric Health Care 23:117–125, 2009). Even the health-care needs of healthy children are substantial, and both the demands placed on parents of CSHCN and the destabilizing loss of control that their child's illness generates create often unsustainable work–family strain. Until recently, however, US government work–family policies and programs

P.J. Chung, M.D., M.S. (✉)
Department of Pediatrics, David Geffen School of Medicine and Mattel Children's Hospital, University of California, Los Angeles, CA, USA
e-mail: paulchung@mednet.ucla.edu

Department of Health Policy and Management, UCLA Fielding School of Public Health, Los Angeles, CA, USA

RAND Health, The RAND Corporation, Santa Monica, CA, USA

K.D. Vestal, M.P.H.
Division of General Pediatrics, Department of Medicine, Boston Children's Hospital, Boston, MA, USA

M.A. Schuster, M.D., Ph.D.
Department of Pediatrics, Harvard Medical School, Boston, MA, USA

Division of General Pediatrics, Department of Medicine, Boston Children's Hospital, Boston, MA, USA

RAND Health, The RAND Corporation, Santa Monica, CA, USA

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were generally not designed to address the needs of employees with ill family members. In 1993, the federal Family and Medical Leave Act (FMLA) became the first federal law to do so. Because of FMLA's limited reach, in 2004, California enacted the Paid Family Leave Insurance program, which has since been followed by a few similar programs in other states and by work on additional federal legislation. If strengthened, such programs have the potential to provide critical support for employed parents of CSHCN, as well as other employees with ill family members.

Introduction

Berkman and O'Donnell (Chap. 12) provide an illuminating evaluation of the maternal and child health effects of various family-related social policies in industrialized countries that are designed to help individuals enter or remain in the labor force while caring for their children or other family members. Policies examined by the authors include public policies such as parental leave, child allowances, maternity grants, flat-rate childcare leave, and tax deductions, with infant mortality serving as the primary indicator of child well-being. As a framework for their analysis, Berkman and O'Donnell employ a model of work–family strain to explain how social policies may affect the health and well-being of children and families. In this chapter, we use this work–family strain model to address an additional area in which federal, state, and employer policies intersect with family and child health: The ability of employed parents to take leave from work—either paid or unpaid—to care for their child's health needs.

Parent Roles in Child Health Care

A distinct feature of health care for children is that parents are expected not only to provide various direct health-care services themselves but also to perform nearly all of the support roles that make direct services by health-care professionals possible (Schuster, Chung, & Vestal, 2011). Even the healthiest children have substantial health-care needs (American Academy of Pediatrics [AAP] & Bright Futures, 2008; Owens et al., 2008). All children are expected to receive frequent routine preventive care that addresses not only the screening and prevention of disease but also the promotion of healthy development. Current preventive care recommendations specify a minimum of 7 visits in a child's first year, 6 more in the next 3 years, and a total of 26 before the age of 18, a frequency far greater than recommended for most adults (AAP & Bright Futures). Virtually all children also need acute care (at home, in outpatient settings, or in hospitals), often multiple times a year, for illnesses ranging from minor to serious. Moreover, a large and growing subset of children is chronically ill, with ongoing preventive, acute, and chronic health-care needs that may be dramatically greater than those of healthy children (Owens et al., 2008).

Whether the care is preventive, acute, or chronic, parents are expected to be present, and many of the processes of care have been arranged based on an assumption of parental presence (Schuster et al., 2011). For instance, during outpatient visits, parents

are responsible for scheduling the visit and arranging transportation; filling out paperwork, displaying proof of insurance, and handling co-pays; entertaining and supervising children in waiting and patient rooms; providing relevant historical information to clinicians; comforting the child during exams or procedures; developing appropriate care plans with clinicians; and arranging follow-up appointments, filling prescriptions, and following through on lab requests. When children are hospitalized, parents need to be available to communicate with inpatient clinicians, which may require spending an entire day waiting in a child's room. Parents must also participate in important health-care planning on behalf of their children and may need to share information and coordinate care among multiple clinicians. In addition, they act as a supplementary line of supervision and safety to prevent medical errors or other accidents, and they provide emotional support, comfort, and assistance to their children. Typical clinician offices, clinics, emergency wards, and inpatient facilities are generally unprepared to act as surrogates for all or even most of these functions and are highly dependent on this parental "shadow system" of care for even basic activities (Schuster et al.).

When children are at home throughout an illness, after hospitalization, or while receiving long-term care, parents' responsibilities related to communication, coordination, supervision, and provision of emotional support are no less pressing. They also have the responsibility of providing most or all of the actual health-care services the child needs while at home, which can be substantial and intensive. This is especially true for parents of children with serious complex illnesses. These parents now provide not only medications but also oxygen, respiratory treatments, intravenous nutrition, physical and occupational therapy, and developmental and behavioral interventions.

About 15% of children are considered children with special health-care needs (CSHCN)—children "who have or are at increased risk for a chronic physical, developmental, behavioral or emotional condition and who also require health and related services of a type or amount beyond that required by children generally" (Bethell et al., 2002; Bethell, Read, Blumberg, & Newacheck, 2008; McPherson et al., 1998; Newacheck & Kim, 2005; Stein & Silver, 2002; Stein, Westbrook, & Bauman, 1997). For instance, children with conditions such as ADHD, asthma, autism, cancer, cerebral palsy, cystic fibrosis, depression, diabetes, and sickle cell anemia generally fall into this category. These children require ongoing care, including frequent monitoring, interventions aimed at preventing or managing complications of the illness, and often high-intensity acute care for severe episodes of illness. They account for about three times as many medical encounters, hospitalizations, and school absences as other children (Neff et al., 2002; Newacheck et al., 1998; Newacheck & Kim). We will focus the remainder of our discussion on this population.

Children with Special Health-Care Needs: More Family Demand, Less Family Control

In recent decades, increases in children's preventive and chronic care needs, combined with increases in workforce participation among mothers, have made the need for parents to miss work on behalf of their child's health a ubiquitous part of workplace

life (Coontz, 2005). For the most part, though, workplaces have not developed effective strategies to adapt to these changing demands, creating the potential for work–family strain among employed parents of all children but most particularly parents of CSHCN.

In the work–family strain model described in detail by Berkman and O'Donnell (Chap. 12), parents experience varying levels of work–family demand, control, and support that together produce a range of outcomes not only for parents but also for their families. Families with parents who experience low control, low support, and high demands in both work and family domains would be considered most vulnerable; by contrast, families with parents who have high control and high support would be considered more resilient and less likely to suffer negative outcomes as a result of high work and/or family demands.

There is compelling evidence of the negative consequences created by the heightened family demands experienced by parents of CSHCN. These intense demands are often accompanied by parental loss of family control, as the demands must generally be met while accommodating the rules and schedules of someone other than the parents—e.g., the child (through the unpredictability of illness), the provider, the school. The additional time and effort that parents of CSHCN must devote to seeking and managing treatment, attending medical or therapy appointments, and working with day-care providers and schools to find accommodations for their child's complex and challenging needs can create a loss of control that contributes to financial problems, marital discord, sibling issues, and problems at work (Goodman, 2010). Across a variety of domains, parental caregivers of children with activity limitations are at a particular disadvantage compared with other parents. They report poorer quality of life, have slightly higher use of sick visits for their own medical issues, and have less favorable employment and financial outcomes (Kuhlthau, Kahn, Hill, Gnanasekaran, & Ettner, 2010).

In the context of high family demand and low family control, many families with chronically ill children face an enormous financial burden (Looman et al., 2009). About 40% of these families, or about four million families nationwide, report experiencing financial problems related to their child's condition (Kuhlthau, Hill, Yucel, & Perrin, 2005). In a 2005–2006 national survey of chronically ill children, 24% of parents reported work loss as a result of their child's health-care needs. Moreover, parents reported that having access to a coordinated care system (e.g., a medical home) was associated with a 50% reduction in the odds of work loss (Okumura, Van Cleave, Gnanasekaran, & Houtrow, 2009), presumably because the care system could absorb or reduce some of their illness-related demands and help restore family control.

As illness severity mounts, so does the risk of work instability. A large, nationally representative survey found that children's limitations were associated with parents' job changes and income loss. Specifically, functional limitations in mobility and self-care were associated with intensive home-care requirements, leading parents to make various job changes to accommodate these needs. Severe limitations in the child's learning ability, meanwhile, greatly increased both job changes and income loss (Rogers & Hogan, 2003).

Mothers' careers may be especially affected by caring for CSHCN. A study of families with autistic children found that in two-parent households, a large majority

of parents said the mother's work outside the home was the most affected by their child's autism. Three out of five mothers reported not taking a job because of their child's autism. Of those mothers who were employed, caring for their child had forced more than half to work fewer hours, one-quarter to take a leave of absence, and nearly as many to turn down a promotion (Baker et al., 2010). Another study found that mothers of chronically ill children requiring use of technical devices were much more likely to quit their jobs to care for their child, with single caretakers being 15 times more likely than mothers in two-parent families to quit employment (Thyen, Kuhlthau, & Perrin, 1999).

Work–Family Support: Family Leave Policies

As Berkman and O'Donnell (Chap. 12) note, social and economic policies can serve as a form of institutional support for families. This support can counterbalance the increased demand and diminished control experienced by parents of ill children. Employed parents in the USA tend to rely on a haphazard mix of support that includes federal, state, and local leave laws and programs but is determined mainly by working conditions established by their employer, including flexibility in duties, locations, and schedules, as well as other employer-provided benefits. In the USA, where the availability of employer-provided paid sick leave is not universal (unlike in many other countries), parents who have paid sick days are more than five times as likely to be able to care for their sick children themselves as parents who do not (Heymann, Toomey, & Furstenberg, 1999; World Adult Labour, 2011). According to the 2010 National Paid Sick Days Study, 64% of all workers report that they are eligible for paid sick days from their employer (including those receiving "paid time off," also known as PTO, which combines time off for sick leave, vacation, and other reasons) (Smith & Kim, 2010). However, substantially fewer workers (47%) receive paid sick days that can also be used to care for ill family members. Without flexible scheduling or paid leave to care for children's health needs, employed parents may opt to forgo important disease prevention or treatment activities or even expose other children to infectious diseases. Studies in Haiti, Indonesia, and the USA have found that parents report that work schedule conflicts are a significant barrier to getting their children immunized (World Adult Labour). Similarly, among US workers with paid sick days, 14% have sent a sick child to school or day care; among those without paid sick days, 24% have done so (Smith & Kim, 2010).

Federal Policies

The federal government, through the Family and Medical Leave Act (FMLA), provides some support for employed parents who need to take time off of work to care for their child's health needs. FMLA provides up to 12 weeks a year of unpaid leave

to certain workers to care for themselves or ill family members (Family and Medical Leave Act, 1993). FMLA provides job protection (i.e., protection from being fired) and also requires the employer to maintain the employee's group health benefits during the leave. Signed into law in 1993, FMLA was the first major federal leave legislation to specifically address the competing demands of work and family illness. About half (47%) of workers are eligible for FMLA leave (Han & Waldfogel, 2003). Eligibility depends on the size of the employer (at least 50 employees), the duration of current employment (at least 12 months with the same qualifying employer), and the number of hours worked there (at least 1,250 h in the past 12 months). Thus, eligibility is essentially restricted to long-term employees working more than half-time for public agencies and large private employers. In addition, many eligible employees cannot afford to take unpaid leave. Of the 3.5 million employees who needed leave but did not take it in 2000, 78% cited inability to afford leave as a reason. Of these, 88% said they would have taken leave if they had received either some pay or (if already receiving partial pay through their employer) additional pay (Cantor et al., 2001).

Two pieces of proposed federal legislation, the Healthy Families Act and the Family Leave Insurance Act (both currently stalled in Congress), would partially address concerns about employees who lack access to paid leave that can be used to care for themselves or family members. The Healthy Families Act would create a new national standard guaranteeing employees the ability to earn up to seven paid sick days a year that they could use for the health needs of themselves or family members. It would also apply more broadly than FMLA, with lower requirements for employer size (at least 15 employees), employment duration (60 days), and hours worked (1 h earned for every 30 h worked). Costs would primarily fall upon employers, who would be responsible for paying employees' wages when they use their sick leave. The Family Leave Insurance Act would create an insurance program that would revise FMLA, funded through employer and employee payroll tax contributions, to provide up to 12 weeks of paid benefits. Employees would receive a percentage of their daily earnings (from 100% for lower-income workers down to 40% for higher income workers) and be subject to a waiting period of five workdays before receiving benefits. The Family Leave Insurance Act would also have somewhat broader eligibility than FMLA: It would apply to employers with 20 or more employees and to employees who have worked at least 625 h for the same employer in the past 6 months.

There is strong public support for federal government-mandated paid sick days. According to the 2010 National Paid Sick Days Study, across all sociodemographic and political groups, most Americans believe that paid sick leave to care for themselves or for immediate family members should be a government-guaranteed right for workers (Smith & Kim, 2010). Sixty-nine percent of respondents said that paid sick days were "very important" for workers, and 75% favored a law that would guarantee paid sick days for all workers.

State and Local Policies

Several states have moved to extend FMLA's approach by instituting state-level paid leave programs. In 2004, California's Paid Family Leave Insurance (PFLI) program became the first such state program and contains innovative features that have set an example for other state and federal programs and proposals. First, it is entirely employee-funded, using a small mandatory payroll tax to create an insurance pool with broad eligibility (i.e., anyone who contributes to the pool) that partially funds up to 6 weeks of leave for an immediate family member's illness or the birth or adoption of a child (California Paid Leave Law, 2002). PFLI covers most part- and full-time employees at about 55% of their salary up to a maximum in 2012 of $987 a week (Paid Family Leave Benefits, 2011). It does not, however, include job protection, and employers are not required to maintain the employees' employer-sponsored health benefits during leave, an especially important consideration for parents of CSHCN. Benefits apply after employees miss 1 week of work for a given illness (continuously or cumulatively). A statement signed by a physician or other clinician documenting the illness is required.

Awareness and use of PFLI have been generally low among parents of CSHCN and the general population. About 18 months after the program was implemented, only 18% of parents of CSHCN knew about it, and only 5% had used it (Schuster et al., 2008). In the general California population, 28% were aware of the program in 2007 (Milkman, 2008). These low rates of PFLI awareness and uptake are probably due to a combination of factors. First, PFLI was not widely publicized and requires only that employers provide information about the program to new employees and employees who inquire about pay during leave for a covered purpose. In addition, the lack of full pay during leave prevents use by some parents (Schuster et al.); requiring the accrual of seven missed days of work for an illness before the benefit can start reduces its usefulness for limited absences; and the lack of a job protection provision raises the risk of job loss for parents not covered by FMLA, especially for parents who have extended absences to tend to their chronically ill children.

New Jersey implemented a similar paid leave law in 2009 (NJ Paid Leave Law, 2008). Washington State passed more limited family leave legislation (covering leave only for parents with a newborn or newly adopted child) in 2007 (Washington Paid Leave Law, 2007) but has yet to implement its program (Washington Family Leave Coalition, 2011). In addition, a number of states, including California, Connecticut, Hawaii, Washington, and Wisconsin, have flexible sick-leave laws that entitle all workers who have access to sick leave to use some of their sick days to care for a sick child (National Partnership for Women & Families, 2012). A few cities, including San Francisco, the District of Columbia, and Milwaukee, have also passed sick day ordinances that guarantee paid sick days for all or most workers (National Partnership for Women & Families, 2011; Washington, DC Accrued Sick and Safe Leave Act, 2008).

International Comparisons

Despite legislative activity at the federal, state, and local levels, the USA remains one of only a few industrialized countries that do not have national laws providing paid leave for children's health needs. At least 43 countries, including Australia, Canada, France, Japan, Nicaragua, and South Africa, specifically guarantee parents paid leave when their child is ill, and more than half of the 43 provide full wages (World Adult Labour, 2011; Heymann, Penrose, & Earle, 2006). Although guaranteed length of leave varies, an analysis of 37 countries that offer paid leave for children's health needs found that more than a third guarantee 11 or more workdays (i.e., more than 2 weeks) of paid leave (Heymann et al.). Types of paid leave arrangements also vary. For instance, Norway typically provides 10 days annually, but 15 if the employee has more than one child and 20 if the employee has a chronically ill child. In addition, at least 34 countries guarantee discretionary leave (17 with pay), which can be used for ill children (Heymann et al.).

Conclusion

Family leave policies can help parents by giving them the support they need due to the increased demands and decreased control they experience when their children are ill. The ultimate goal of such policies should be to allow parents the ability to remain fully engaged in the workforce while still caring for their children, regardless of their children's health or illness. For parents of CSHCN, who experience disproportionate levels of work loss due to their child's condition and consequent financial difficulties, support of this nature can have substantial effects on their own well-being and that of their children. Our own research has found that parents of CSHCN who received full pay during leave from work to care for their ill child were more likely than parents who received no pay to report positive effects on child physical and emotional health and parent emotional health and were less likely to report financial problems (Schuster et al., 2009). Additional research investigating the child health effects of social and economic policies related to family leave could point the way to more effective policy solutions.

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Chapter 14
Harder Than Rocket Science? The Science of Designing and Implementing Strong Family-Friendly Policies

Patricia O'Campo

Abstract Berkman and O'Donnell (Chap. 12) effectively argue that improvements in infant and child health will only come about with supportive family national policies. I pick up on two themes from the paper that identify potential gaps in the current literature. First, drawing from Berkman and O'Donnell's conceptual framework, I review the "state of the evidence" for key macro-social drivers of pro-family policies. Despite widespread recognition that macro-social are critical drivers of population well-being, a very small proportion of our research incorporates consideration of these contextual factors due, in part, to an overreliance on studies at the individual level in public health. Second, I build on their call for more evaluation of pro-family policies. In particular, I examine whether the tools we have to undertake such evaluations are adequately developed. We must take advantage of and help to refine emerging evaluation methods that accommodate complex multilevel policies and programs if we are to identify and implement effective family-friendly policies.

Introduction

Berkman and O'Donnell (Chap. 12) argue that improvements in infant and child health will only come about with supportive family national policies. I focus on two themes from their chapter. The first issue I discuss builds on the conceptual framework that the authors present (see Fig. 14.1). In particular, I examine the "state of the evidence" for key drivers of well-being depicted in that framework. My second

P. O'Campo, Ph.D. (✉)
Center for Research on Inner City Health, University of Toronto,
Toronto, ON, Canada
e-mail: pat.ocampo@utoronto.ca

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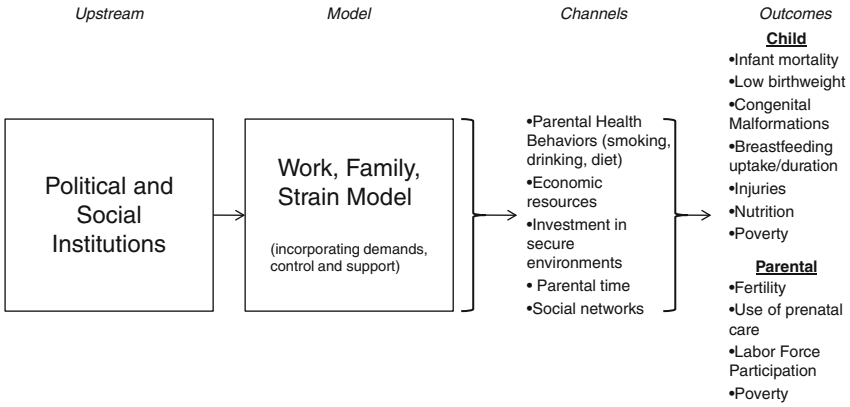

Fig. 14.1 Theoretical causal model (Fig. 12.5 in Berkman and O'Donnell, Chap. 12)

discussion point is on the theme of evaluation of complex family-friendly policies and programs that Berkman and O'Donnell argue we should be placing greater emphasis upon. Specifically, I discuss whether the tools we have to undertake such evaluations are adequately developed.

Eco-social Conceptual Framework

Berkman and O'Donnell (Chap. 12) present a conceptual framework that represents the influence of social policies on the well-being of families, parents, and children which is replicated in Fig. 14.1. The authors note:

> Theoretically, we are interested in the full range of policies though our review here reflects the narrower range. As Ferrarini notes, family policy institutions have a potential to affect infant mortality in different ways: They can structure the time that parents can spend with their children or the income of the household either through direct contributions or indirectly through labor market behavior (Ferrarini & Sjoberg, 2010). These mid-range outcomes may influence child health via another set of pathways or channels including direct physiological pathways related to stress, consumption and patterns of health behaviors, and economic resources resulting in physically, emotionally and cognitively healthier environments. Ultimately, these exposures may influence child health along a spectrum of life course exposures from basic decisions about fertility to in utero exposures and exposures during infancy and early childhood (p. 14).

The framework presents an opportunity to note a gap in the existing literature on social policies that improve family and child health. While the authors are appropriately interested in the full range of proximal and distal influences and state and national policies that can ultimately impact family and child well-being, too few studies in the fields of public health, social epidemiology, and social policy focus on the "upstream" factors noted in the Fig. 14.1.

The majority of research on infant, child, parent, and family well-being tends to focus on what is described in the Berkman and O'Donnell framework as "outcomes" and "channels" (O'Campo & Dunn, 2012a) (see Fig. 14.1). Admittedly, many of the studies on infant and child well-being focus on individual factors by design, for example, studies of parent–child attachment, breastfeeding, or infant development. Yet, there are two major limitations to the current literature on family, child, and infant well-being.

First, when such studies fail to explicitly consider the context in which individual level situations and behaviors occur, we miss an opportunity to fully understand how individuals are influenced by their surroundings and how social policies and programs can shape individual risk or resilience. The conclusions we draw about the importance of individual and social contexts may differ widely between studies focusing just on individual factors and those that examine cross level interactions, for example, between residential neighborhood context and child and infant outcomes (Caughy, O'Campo, & Muntaner, 2003; O'Campo, 2003; O'Campo, Caughy, & Nettles, 2010; O'Campo, Xue, Wang, & Caughy, 1997). Second, and perhaps the real gap in this field of inquiry about social policies and family well-being, are that there are too few studies that include a focus on macro-social policies (Muntaner et al., 2011; O'Campo & Dunn, 2012a; Putnam & Galea, 2008). Just as individuals are influenced by the contexts in which they live, work, attend school, and receive services to name a few examples, the existence and nature of the social and economic policies that Berkman and O'Donnell (Chap. 12) refer to—workplace flexibility, family policies such as child care or parental leave, paid or unpaid vacation or leave, and economic incentives such as the earned income tax credit (EITC)—are influenced by societal macro-social factors and processes (Muntaner et al., 2011; Putnam & Galea, 2008). Thus, whether a city, state, or country has family-friendly and workplace supportive policies and the nature of those policies are determined by macro-social factors such as the political party in power, welfare state, macroeconomic policies, and labor market to name a few (Beckfield & Krieger, 2009; Muntaner et al., 2010, 2011; Navarro et al., 2006; Navarro & Shi, 2001). Yet, a recent search of the health literature for the period 2008–2010 on interventions to reduce health inequities indicated that more than 97% of scholarly articles focused on downstream, usually individual-based, determinants of health, while <3% addressed structural-level policies and programs. This fact was recently recognized by the NIH when they noted that applications with a focus on "macro-level or structural features of the social environment that affect health are needed, as research has tended to focus on proximal dimensions of the social context" (National Institues of Health, 2010).

One related gap should be noted when focusing on the study of macro-social influences on families. In public health and social epidemiology, the unit of analysis in the majority of studies is the individual. Yet, studies of individuals will shed little light on whether and how macro-social factors influence and shape family-friendly policies. Policy makers recognize the importance of evidence about the full range of social determinants of health as they have noted that "much of the available evidence on health inequalities was from far down the causal chain, and was often concerned with health behaviors and clinical issues, rather than broader social

determinants of health" (Petticrew, Whitehead, Macintyre, Graham, & Egan, 2004, p. 813). Yet, the dominant explanatory model used in epidemiologic and social epidemiologic inquiry continues to be the biomedical or "disease-specific model," which seeks to identify mostly individual-based risk markers and risk factors for specified health conditions. Thus, the study of macro-social policies and programs necessitates the expansion of the study designs used to understand and document contextual and macro-level influences on family and individual well-being.

Evaluation of Family-Friendly Policies

Studies of social policies must go beyond demonstrating associations between macro-social determinants, family-friendly policies, and health outcomes of individuals. As Berkman and O'Donnell (Chap. 12) note, it is important to have these policies evaluated to capture their full range of impact on well-being. Moreover, if we are to scale up such programs, it would be important to have process evaluation data in addition to outcome evaluation information. Yet, in fields like epidemiology where there is strong research that demonstrates that social factors and policies may positively or negatively impact population well-being or health disparities, there tend to be far fewer evaluations of such programs and policies. One epidemiologist recently noted that while the field of epidemiology faces "a feast of descriptive studies of socio-economic causes of ill health, we still face a famine of evaluative intervention studies" (Bonneux, 2007, p. 483).

To draw from the conceptual framework provided by Berkman and O'Donnell (Chap. 12), it is clear that the pathways from macro-social factors to family-friendly policies to a broad range of family and child outcomes is multilevel and complex. Too often in epidemiology and public health, where evidence syntheses to measure program effectiveness have become the norm (e.g., Cochrane Reviews, CDC's Guide to Community Preventive Services), the study designs we rely on for such evaluations are not ideally suited to complex multilevel programs and interventions (Given, 2008). Moreover, policy makers have asked explicitly for more evaluations that unveil and test program theory and not just whether program components work (Petticrew et al., 2004). Thus, evaluations that focus upon, uncover, and test the effectiveness of the critically effective ingredients of programs, in particular for complex multilevel interventions, are much needed (Dunn, van der Meulen, Muntaner, & O'Campo, 2012).

As mentioned earlier, study designs that are appropriate for complex interventions with impacts at multiple levels are not often used in epidemiology. Moreover, epidemiologic evaluation tends to focus upon program effectiveness and less frequently on understanding and strengthening the theory of programs. While theories of change, especially in social epidemiology, have been an area that requires further development (Krieger, 2001; O'Campo & Dunn, 2012a), it is also true that the philosophical underpinning of epidemiology favors reducing causality and effectiveness to quantitative demonstrations of impact (O'Campo & Dunn, 2012a). This issue is slowly being explored and elaborated upon within our discipline (O'Campo & Dunn, 2012b).

One promising alternative that focuses upon theories of change and also acknowledges and accommodates evaluation of complex programs is the realist reviews (Kirst & O'Campo, 2012; O'Campo et al., 2009; O'Campo, Kirst, Tsamis, Chambers, & Ahmad, 2011). Realist reviews, derived from scientific realism, have at their core a concern for the development and refinement of theories to explain the world. Realist approaches are particularly well suited for complexity as context, contributing factors, and determinants at all levels are embraced rather than controlled for by realists. In one recent example, the focus on why programs work and in which settings such programs worked well served to advance our understanding of program effectiveness over evidence syntheses undertaken using traditional approaches to systematic review (O'Campo, Kirst, Tsamis, Chambers, & Ahmad, 2011). In part, this is due to our overreliance on quantitative evaluation studies. Rather, to support the successful design and implementation of such policies, the evidence generated by researchers must measure and document the complex pathways of influence of how such policies support families and children. This can be accomplished by conducting more studies that examine the causal pathways or by undertaking evaluations of existing policies.

Health in All Policies

One example of research being undertaken to study causal pathways of macro-social factors on health inequities is from the policy area of Health in All Policies (Shankardass, Solar, Murphy, Greaves, & O'Campo, 2012). Health in All Policies (HiAP) is a whole-of-government strategy that "seeks to improve health… through structures, mechanisms and actions planned and managed mainly by sectors other than health" (Ståhl, Wismar, Ollila, Lahtinen, & Leppo, 2006, p. xviii). HiAP has been implemented for decades all over the world, yet little evaluation work has been undertaken to determine just how this whole-of-government strategy brings about improvements in population well-being. The research that we are undertaking examines the theory behind HiAP and how the macro-social context facilitates the implementation of HiAP in different countries. In particular, this approach to evaluating evidence is explanatory (i.e., how "x" works) rather than simply judgmental (how well did "x" work) because it combines both theoretical thinking and empirical evidence about program workings and context. It attempts to explain what works for whom, in what circumstances, in what respects, and how (Pawson, 2006; Pawson, Greenhalgh, Harvey, & Walshe, 2004).

This recent research on how HiAP works began as a partnership with the Ontario Ministry of Health and Long-Term Care (MOH) as the MOH was seeking guidance on implementing a Health in All Policies approach to reduction of health inequities with the recognition that the real drivers of health inequities are macro-social intersectoral policies and practices. The MOH was seeking to learn lessons from other jurisdictions around the world about the critical ingredients for successful initiation and implementation of HiAPs globally. The MOH specifically asked for a realist review of the existing evidence knowing that a realist synthesis would reveal

what works, for whom, and why. The realist systematic review was to synthesize evidence that has been accumulating over decades since the first HiAP appeared over 20 years ago. While I can only go into a few details here in this chapter about the findings from that research to date, extensive reports and publications are available and can be consulted elsewhere (Shankardass et al., 2011, October; Shankardass et al., 2012; Shankardass et al., 2010a, 2010b, 2010c, December; Solar & Shankardass, 2011, February).

This research began with a theory of change as shown in Fig. 14.2. This theory of change covered several stages of HiAP from initiation of the policy, implementation of the policy, design of specific intervention and policy components, and ongoing implementation of the policy and was based upon the available literature on the topic to date (Solar & Irwin, 2007; Solar, Valentinen, Albrech, & Rice, 2009; Solar & Shankardass, February 2011). The framework in Fig. 14.2 presents the overall components, but each phase (e.g., initiation, or financing and sustainability) had specific theories of change associated with it. Figure 14.3 presents some of the components comprising the mechanisms of influence for financing and sustaining HiAP during implementation. Thus, we sought to find evidence to support or contradict this initial theory of change for HiAP. We have carried out this work with two grants awarded by the Canadian Institutes for Health Research (O'Campo et al., 2009; O'Campo, Shankardass, Murphy, Solar, & Bayoumi, 2011, October) and have relied on realist approaches—literature reviews and explanatory case studies—for our methodology (Shankardass et al., 2010c, December; Yin, 2003, 2009). The final products will facilitate a greater understanding of what strategies promote successful initiation and implementation of HiAP in given contexts and why those strategies work. Such information can be used to tailor the critical ingredients of programs and policies to other settings, in this case, Ontario.

While realist syntheses, realist evaluation, and explanatory case studies are but three new tools that can be used to evaluate complex multilevel interventions, there are other strategies that might be appropriate to consider in future work. Such strategies include, but are not limited to, systems analyses (Leischow et al., 2008) and developmental evaluation (Fagen et al., 2011). The point is that we will need to adopt and refine newer methods to accommodate evaluation of complex multilevel programs and policies if we are to fully evaluate these pro-family policies as suggested by Berkman and O'Donnell (Chap. 12). Our existing methods may not enable us to fully grasp the ways in which context shapes program or policy success. The good news is that these tools are emerging and are being put to good use in gaining a greater understanding of complex programs.

Conclusions

In this chapter, I sought to highlight two issues of relevance to Berkman and O'Donnell's (Chap. 12) discussion of pro-family policies. In particular, two gaps in the current research approaches have been highlighted. First, if we are to design

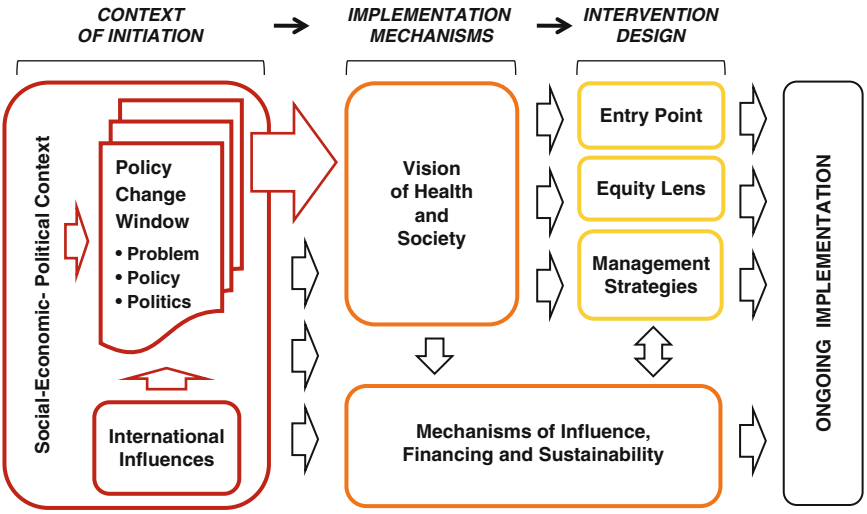

Fig. 14.2 Explanatory model of the process of implementing intersectoral action for health equity at the level of countries

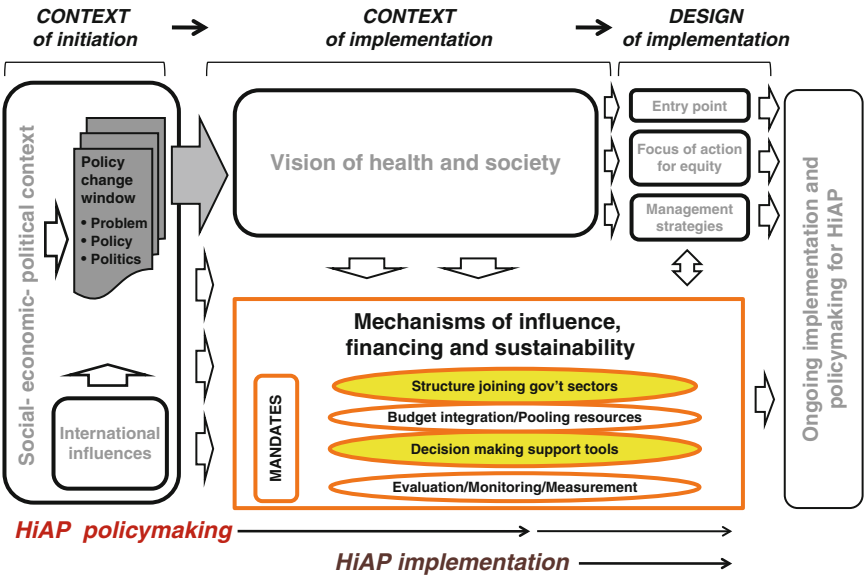

Fig. 14.3 Specific strategies undertaken to finance and sustain health in all policies

pro-family policies and programs to address infant mortality and child health, we need to focus on a full range of determinants of well-being with a particular focus on the "causes of the causes" (i.e., macro-level drivers of population well-being). Second, our research tools, in particular our evaluation tools, need to be expanded

to accommodate complexity and to generate information about the theory or critical ingredients that lead to successful programs. Merely understanding whether a program works will not facilitate the design and implementation of strong and effective pro-family policies, even if a comprehensive set of outcomes is evaluated. The good news is that we have already seen progress in the research arena for both challenges as described in this chapter. Our task ahead is to further refine these emerging approaches.

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Chapter 15
Emerging Evidence from Research That Can Improve Social Policies and Programs to Impact Child Health

M. Rebecca Kilburn

Abstract In this chapter I outline three emerging ideas from research that have implications for policies and programs that aim to promote child health. These ideas have relevance for a range of investments in child health, including the work–family policies and practices discussed by Berkman and O'Donnell (Chap. 12). The first is a growing body of evidence that psychological health, in addition to physical health, in childhood is associated with long-term outcomes and that the relationship between childhood psychological well-being and adult outcomes is sizeable. The second emerging research finding discussed in this chapter is mounting evidence related to the implementation and effectiveness of policies that aim to promote child health by affecting parents' human capital and family processes. The third issue that this chapter highlights is that research evidence can be put to better use by helping decision makers build a portfolio of effective approaches rather than using research to make one "best" choice off the menu of options.

Introduction

In this chapter I provide an overview of emerging evidence from social science research that can improve policies and programs aimed at promoting child health in the family context. These research findings have relevance for the types of work–family policies and programs that are discussed by Berkman and O'Donnell (Chap. 12).

M.R. Kilburn, Ph.D. (✉)
RAND Child Policy, RAND Corporation,
Santa Monica, CA, USA
e-mail: kilburn@rand.org

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In developing policies and programs to improve child health, decision makers in the public and private sectors often seek information from research on the following types of questions:

- Which child outcomes are most important to address?
- What types of policies and programs improve child health?
- Given limited resources, how should investments in policies and programs to improve child health be prioritized?

This chapter summarizes research findings that inform each of these questions and that could be considered to be "emerging evidence." The term "emerging evidence" encompasses findings that have been established relatively recently—within the last decade or even more recently—and that are backed by a body of research rather than just one study or research on one intervention. The following sections address each of these questions in turn, with the last section relating the main points to Berkman and O'Donnell (Chap. 12).

New Evidence Regarding Child Outcomes That Explain Variance in Adult Well-Being

A long line of research has documented the importance of childhood health from a public health perspective, generally emphasizing measures of physical health in childhood. Children's physical health has been shown to be associated with health and well-being throughout adulthood. Birth weight and children's anthropometric measurements (such as height for weight) have been the two classic measures of physical health for infants and older children, respectively (Case & Paxson, 2010, Center on the Developing Child at Harvard University, 2010). Reflecting the orientation of the bulk of research evidence and data collection related to the long-term relevance of child health outcomes, public health objectives and status indicators have also generally focused on physical health in childhood. (For examples, see Healthy People national health goals at http://www.cdc.gov/nchs/healthy_people.htm, and KIDS COUNT Data Books and essays at http://datacenter.kidscount.org.) Similarly, the nation's policies and programs that aim to improve child health have typically focused on promoting the use of health inputs that have been shown to boost measures of physical health. These include strategies such as encouraging the use of prenatal care, increasing rates of childhood immunizations, raising rates of children's health insurance coverage, and encouraging children's good nutritional intake (e.g., American Academy of Pediatrics Policy Collections at http://aappolicy.aappublications.org/misc/About.dtl).

Several new lines of research from different disciplines provide compelling evidence that policies and programs to improve child health should target behavioral health outcomes in addition to physical health outcomes. Specifically, these bodies of research all demonstrate that key measures of adult well-being are related to

children's exposure to adverse events, children's psychological problems, and children's early cognitive or behavioral measures. In one of these lines of research, Smith and Smith (2010) document that among adults in the USA, having had psychological problems in childhood is associated with lower education, lower income, fewer work hours, a lower probability of marriage, and the likelihood of experiencing psychological problems in adulthood. The authors' measures of childhood psychological problems include experiencing depression, substance abuse, or other psychological problems before age 17. They estimate that the economic costs of childhood psychological problems total $300,000 in lost lifetime income per individual. To put these findings in perspective relative to the relationship between measures of childhood physical health and adult outcomes, the estimates from Smith and Smith indicate that having a childhood psychological problem is associated with a reduction in completed years of education equivalent to a decrease of about six inches in height based on the estimates of Case and Paxson (2010). In other words, the size of the relationship between having a childhood psychological problem and adult outcomes is relatively large.

A second line of research that has been receiving growing attention from the public health community also points toward the important role of childhood experiences and mental health in explaining adult outcomes. Evidence is mounting that traumatic childhood experiences in particular are related not only to worse outcomes in childhood but also are predictive of worse health behaviors and physical health outcomes in adulthood (Felitti et al., 1998; Flaherty et al., 2006; Ford et al., 2011). A body of literature has shown that a set of "adverse childhood experiences" (ACEs) are strongly associated with adult health outcomes including morbidity, negative health behaviors, and mortality in adulthood (Dong, Anda, Dube, Felitti, & Giles, 2003; Dong et al., 2004; Felitti et al.). These experiences include abuse, neglect, and household dysfunction, such as having lived with someone with a substance abuse problem or having an incarcerated parent. In the ACE study, which included a relatively well-educated population enrolled in an HMO, one in four adults had experienced an ACE, and half had experienced between 1 and 3 ACEs (Anda, 2007). Similarly, children who are exposed to neighborhood violence and other forms of trauma exhibit high levels of PTSD and depression as a result (Berton & Stabb, 1996; Cuffe et al., 1998). The incidence of PTSD in older adolescents in the USA may be as high as 9–10% (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Children's exposure to violence is associated with higher levels of depression and problem behaviors, worse school outcomes, and attendance problems (Dyregrov & Yule, 2006). Research has also shown that it is possible to reduce children's symptoms of PTSD through interventions (Stein et al., 2003).

A third line of research that emphasizes the importance of psychological and mental health outcomes in early childhood comes from economists' work on "human capital." The term human capital refers to the productive capacities (e.g., knowledge, health, experience, and skills) embodied in people. While human capital theory was originally developed by Becker (1975), James Heckman has more recently applied and elaborated on this theory in the context of early childhood (Heckman, 2000, 2007). Heckman and colleagues have examined which types

of human capital from the early years are most predictive of success in adulthood, and they increasingly emphasize the critical role of "noncognitive" skills that children attain in childhood. The authors distinguish "noncognitive" skills, such as motivation, sociability, self-esteem, and mental health, from "cognitive" skills, such as achievement test scores. Their research indicates that noncognitive skills explain a large amount of variance in adult outcomes that they are malleable, and that intervening to improve them early in people's lives is likely to be more efficient than mitigating disadvantage at older ages (Heckman, 2007).

In sum, while policies and practices to promote child health have traditionally focused on improving physical health, evidence from different lines of research is converging on a similar theme: Policies and practices should include improving children's mental and psychological outcomes as a key strategy for promoting long-term well-being.

Mounting Evidence Points to New Ways of Thinking About Interventions to Promote Child Health

The importance of family characteristics in explaining child outcomes including health is one of the incontrovertible findings from all disciplines spanning the history of empirical research (see review in Haveman & Wolfe, 1995). Despite the primacy of the family in the "production" of child well-being, policies and programs to promote children's outcomes have generally considered the family to be outside the purview (see model in Kilburn & Karoly, 2008). Instead, the mainstay of US policies related to children has focused on institutions and sectors other than the family, such as education, health care, and work policies. The principal way that policies have related to the family is by affecting access to the inputs that parents use in the production of child health and well-being, through approaches like income supports, subsidizing children's health insurance and enriched child care, or environmental regulations.

Particularly in the last 5 years, policies and programs at all levels of government, as well as the private sector, have increasingly incorporated strategies that more directly get inside the "black box" of family processes as a way to promote child well-being. These strategies typically focus on improving human capital specific to parenting so that parents can be more effective in promoting the positive development of their children. This may take the form of raising parent's knowledge about child development and effective child-rearing or by otherwise enhancing their parenting skills. The dramatic expansion of publicly provided home visiting services across the country is an example of this approach (Pew Charitable Trusts, 2011a, 2011b). These strategies may also take the form of improving parent's "general" human capital, through such means as raising their overall educational attainment, treating parents' mental illnesses and substance-dependency problems, or promoting women's preconception health and general health as a way of improving birth outcomes (Oreopoulos, Page, & Stevens, 2006; Overpeck, Brenner, Trumble,

Trifiletti, & Berendes, 1998; Currie & Morretti, 2003; Behrman & Butler, 2007; Johnson et al., 2006). While not every policy or program that endeavors to build parents' specific or general human capital is successful, a growing body of research documents many options for building a better parenting "workforce" as a promising way to improve child outcomes ranging from birth weight to maltreatment to behavioral problems. (See http://www.promisingpractices.net Issue Briefs on birth weight, child abuse and neglect, high school graduation, and *Programs that Work* section). This approach is also consistent with a pervasive shift in health and child policy from a treatment orientation to a prevention and health promotion orientation (Kilburn & Karoly, 2008).

To summarize, there has been a disconnect between research findings of the centrality of parents' human capital and family processes in the production of child health and US policies and programs that strive to promote child well-being—the latter generally including few strategies that directly targeted parents' human capital or family processes. A groundswell of policies and programs are currently emerging concomitant with a body of research suggesting that this class of strategies is an important lever for decision makers to consider.

Growing Body of Cost–Benefit Analyses Can Be Put to Better Use[1]

In the current environment of tight budgets and an increasing emphasis on accountability, the demand for and supply of cost–benefit analyses related to children's programs have proliferated (Kilburn & Karoly, 2008). The third main point of this chapter is that decision makers may be using cost–benefit analysis and related research to answer the wrong question. The assumption is often that results from cost–benefit analyses will tell decision makers, "Which investment will give us the most bang for the buck?" Economists would argue that rather than using the results of cost–benefit and related analyses to select the one best investment, policymakers could obtain the greatest societal benefits at the same costs by using the results to assemble a portfolio of investments in child health.

A portfolio approach to investing in child health would select a range of interventions in a way that equalized the marginal contribution to child health across the different interventions. The utility of the portfolio approach in this context relies on a few assumptions about the "production" or development of child health:

- Children have varied health needs that would respond to different investments.
- Some families will benefit more from specific investments than other families (sometimes referred to as "targeting").

[1]This discussion draws heavily on Kilburn and Karoly (2008).

- The highest risk children are likely to benefit more from interventions than lower risk children, and as lower risk children are successfully served, the marginal benefit realized will begin to decline.
- It is feasible to assign dollar values to the costs and benefits gained from different interventions.

The basic idea is that if intervention A is providing $22 in health benefits for each dollar spent while intervention B is providing $12 in health benefits for each dollar spent, one would want to reallocate some of the investment portfolio away from B and toward A. As you provide more of intervention A successively to lower risk children, the marginal benefit from this intervention would begin to decline, and as you provided less of intervention B successively to higher risk children, the marginal benefit of intervention B would begin to rise. You would cease reallocating investments when the marginal benefit of the two interventions was the same—somewhere between $12 and $22 in this example (see a more extensive discussion in Kilburn & Karoly, 2008).

While using cost–benefit and related analyses to improve decision making related to child health is intuitively appealing and relies on some classic ideas from economics, it is not straightforward to put it into practice. This is because comparable cost–benefit calculations are not regularly available for all child health investments that might be under consideration (Karoly, 2010), and there is little documentation regarding the path of marginal benefit changes one would obtain when increasing or decreasing the amount of an intervention. Until the state of the art advances in the area of cost–benefit analysis, decision makers can at least use these concepts to steer away from choosing one best investment and toward assembling an informed set of child health investments. This shift in the way research is used in decision making has the capacity to yield better child health results with a given amount of funding.

Summary and Implications for Berkman and O'Donnell (Chap. 12)

I have outlined three emerging ideas from research that have implications for policies and programs intended to promote child health. The ideas have relevance for a range of investments in child health and have been discussed in very general terms. Here, I briefly highlight some ways that these ideas are related to the work–family policies and practices discussed by Berkman and O'Donnell (Chap. 12).

The first emerging research finding discussed above relates to the growing evidence that psychological health, in addition to physical health, in childhood is associated with long-term outcomes and that the relationship between childhood psychological well-being and adult outcomes is sizeable. The work–family strain model that Berkman and O'Donnell present emphasizes concepts like parental stress, competing time demands, and locus of control. Given the concepts that are at the heart of their model, it is likely that work–family policies are particularly well

suited for promoting children's psychological and mental health and reducing children's exposure to trauma.

The second emerging research finding discussed in this chapter is growing evidence related to the implementation and effectiveness of policies that aim to promote child health by affecting parents' human capital and family processes. In describing "*non-health-care policies* designed to improve working conditions and employee well-being in adulthood" that also improve the health of children, Berkman and O'Donnell (Chap. 12) clearly focus on the family as the predominant contributor to child well-being. They discuss work–family policies that increase the amount of time families can spend with children and the economic resources that families have to contribute to child-rearing. Berkman and O'Donnell could expand the scope of policies they discuss to include those that encourage employees to invest in themselves (e.g., PELL grants, G.I. Bill) and those that encourage employers to invest in their workers. The discussion might include investments that increase employees' general education level or workplace education programs specific to family issues (see Schuster et al., 2008 for an example of a program that improves parent–adolescent communication). This class of policies may also include efforts to promote employees' physical or mental health, such as employee assistance programs or providing immunizations at work, which could make them better caregivers for their children. While these human capital investments may be motivated by workplace outcomes (such as greater productivity), they are likely to have a spillover effect for child health.

The final issue that this chapter highlights is that research evidence can be put to better use by helping decision makers build a portfolio of effective approaches rather than using research to make one best choice off the menu of options. The work–family strain model that Berkman and O'Donnell (Chap. 12) present certainly emphasizes many domains where there are opportunities for intervention, and the authors provide a number of examples of policies that enhance work–family balance. The discussion by Berkman and O'Donnell is a good starting point for helping decision makers understand the full range of benefits that might be derived from the class of work–family policies and programs. A next step in helping decision makers apply research evidence on work–family policies and programs would be to develop more information about the relative effectiveness and costs of the options available in assembling a portfolio of programs and policies.

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Part V
Conclusions

Chapter 16
Integrating Perspectives on Child Health

Claudia Nau and Jessica Heckert

Abstract Penn State's 19th Annual Symposium on Family Issues incorporated diverse disciplinary perspectives on families and child health to advance understanding of how family processes contribute to health and health inequalities during childhood. This chapter critically examines the symposium's contribution to understanding family influences on child health via social structural, psychosocial, and physiological pathways and their interactions. Advantages and disadvantages of potential health improvement strategies ranging from broadscale policies to interventions targeting at-risk groups are discussed. Incorporating multiple strategies via a portfolio approach is one means of addressing a wide range of long- and short-term goals relevant to multiple health domains. Directions for future research are considered. Priorities should include more precise conceptualization and measurement of child health and risk factors in large-scale studies along with development and testing of middle-range theories to unravel the complex processes connecting families and child health outcomes. Focusing on these priorities and fostering interdisciplinary collaborations are critical steps for generating the evidence that can be used by policy creators and other key decision makers.

C. Nau, Ph.D. (✉)
Johns Hopkins Bloomberg School of Public Health, Johns Hopkins Global Center on Childhood Obesity, Baltimore, MD, USA
e-mail: cnau@jhsph.edu

J. Heckert, M.S.
Department of Human Development and Family Studies,
The Pennsylvania State University, University Park, PA, USA

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Why Study Child Health in the Context of Families?

Health inequalities, the avoidable differences in health between population groups (Braveman, 2006), begin early in life. The incidence of low birth weight and infant mortality, for example, varies strongly by race and maternal education (Blumenshine, Egerter, Barclay, Cubbin, & Braveman, 2010; Kleinman & Kessel, 1987; Parker, Schoendorf, & Kiely, 1994). Furthermore, differences in health appear to increase during childhood (Reichman and Teitler, Chap. 9). Health inequality is, of course, not limited to child health outcomes. It concerns most, if not all, major health conditions and has been observed across the entire life course (Marmot et al., 1991; Willson, Shuey, & Elder, 2007). Studying child health, however, is of particular importance for ethical, epidemiological, and economic reasons. Childhood is the most critical phase for physical and psychological development. Trajectories for future outcomes ranging from educational achievement to longevity begin to be established during this time (Haas, 2006; Hayward & Gorman, 2004; Palloni, 2006). Child health, thus, is an important policy target, not only due to concerns about children's immediate well-being but also to reduce the impact of poor child health on adult health, well-being, and achievement. Moreover, child health has important economic impacts. If an individual's health begins to fail in childhood and continues to do so, this burdens the health-care system disproportionally and increases the net economic burden for society because this individual's economic productivity is reduced from an early age.

Child health cannot be studied without studying the family. The family confers a child's social status and is a primary context in which behaviors and norms are formed and child development takes place. What are, however, the structural, developmental, psychosocial, and biological mechanisms that operate around and within families to affect child health? Which interventions and policies are effective in improving child health and family well-being? Penn State's 19th Annual Symposium on Family Issues and this resulting volume have brought together evidence from the latest research on families and child health as well as examples of interventions and policies that target the family–child health relationship. While the symposium showed that each discipline has a wealth of knowledge to contribute, it also raised numerous unanswered questions, including issues about measurement and conceptualization. The symposium itself is an example of the multidisciplinary efforts needed to harness this body of knowledge, better integrate theory and measurement, and develop a comprehensive approach to the issue of families and child health. Important issues for research include identifying the structural, psychosocial, and biological mechanisms that operate around and within families to affect child health as well as determining which family-oriented interventions and policies are effective in improving child health. This volume brings together evidence from the latest research on families and child health along with examples of interventions and policies that target the family–child health relationship. Although the volume demonstrates that there is a wealth of knowledge from multiple disciplines that addresses these issues, contributors have also raised numerous unanswered questions.

The volume itself is an example of the kind of multidisciplinary efforts that are needed to generate a body of knowledge, integrate theories and tie theory to measurement, and translate research into policies and practices directed at improving and supporting families' roles in child health. This chapter provides a critical review of the information presented and questions raised by the authors who have contributed to this volume. We begin by presenting the conceptual frameworks that have been used to explain differences in child health. We then discuss the types of influences that link families and child health and review the policy solutions brought forward to address them. Finally, we conclude by discussing the challenges that will face future multidisciplinary efforts to understand and address family–child health linkages.

A Puzzle of Theories on the Links Between Social Status and Child Health

There is ample evidence that children who grow up in poor families have poorer health than those growing up under more affluent circumstances (see, e.g., Aber, Bennett, Conley, & Li, 1997; Evans, 2006; Wagstaff, Bustreo, Bryce, & Claeson, 2004). What are, however, the mechanisms through which family socioeconomic status influences health? Reichman and Teitler (Chap. 9) review the three most commonly invoked theories that address the pathways through which social status "gets under the skin."

The fundamental cause theory is most commonly found in the sociological literature on health inequality (Link & Phelan, 1995). It is based on the observation that, compared to individuals with lower socioeconomic status (SES), individuals with higher SES have access to more and better social, human, and financial capital that allows them to avoid disease risk and to procure better treatment in the event of disease. Accordingly, children from families with a higher socioeconomic standing are assumed to be better protected from disease and more likely to recover in the event of disease. This pattern is found regardless of the specific disease or the specific proximate pathways examined.

A social psychological framework, in contrast, puts emphasis on individuals' reactions to living in a lower SES environment, including their interrelated behavioral, psychological, and physiological reactions. This framework suggests that chronic stress from lack of control over one's life debilitates individuals, in part through activation of neuroendocrine pathways and concomitant increases in the risk of unhealthy lifestyles, such as lack of exercise, a low-nutrient high-fat diet, or smoking (Wilkinson & Marmot, 2003). From this perspective, health inequalities should be smaller at birth and grow over the life course as the cumulative effects of stressors begin to take their toll.

Reichman and Teitler (Chap. 9) also discuss the economic approach to the social production of disease (Grossman, 1972). This approach suggests that children are born with varying levels of health stock, which then grow or shrink, depending on their parents' and their own health investments. Such health investments include pre- and postnatal medical care and knowledge and support for healthy lifestyles.

To date there is no overarching framework that combines these three principal theories. Each proposes a different set of causal pathways due to their disciplinary origins. Nevertheless, they are not exclusive of each other. In particular, Grossman's economic theory potentially allows for the integration of the different pathways suggested by the psychosocial framework and the fundamental cause theory by providing a general accounting scheme of health stocks, inputs, and outputs. If health stocks were broadly construed, they could take any form and accommodate variables ranging from economic resources to social stressors.

A comprehensive theoretical framework, though, might not be the solution to the conceptual dilemma faced by researchers interested in child health. Each of these theories tends to produce a long list of potential risk factors, but none serves to prioritize them (Glass, Chap. 11). Indeed, results from the large body of empirical research suggest that there are no major causal pathways; on the contrary, differences in child health are the result of the cumulative effects of a multitude of risk factors and causal pathways (Glass, Chap. 11; Reichman & Teitler, Chap. 9). From this perspective, the main challenge to establishing a useful theoretical framework to study child health is not creating a comprehensive theory of causes and pathways. Such a causal super-framework would be impractical, and its use would be limited by the lack of complete data and the inability to model such complex processes. An alternative could be to avoid theorizing on the web of causal pathways between social status and ill-health, refocusing instead on the "system of distribution of rewards and risks that generates population health patterns" (Glass, Chap. 11).

Child Health and Families: Family Context as Producer and Product of Child Health

Biological Processes In Utero

The developmental or fetal origins theory (Barker, Chap. 1) adds an important piece to the puzzle of causes of childhood health inequality. The theory emphasizes the prenatal and multigenerational influences on human physiological development. Barker states that the first months after conception constitute a critical period during which the most significant part of human physiological development takes place. If the fetus lacks sufficient nutrients to support a normal growth trajectory, a trade-off is made with the development of lower-priority organs or body systems such as kidneys and muscle mass in favor of more vital organs such as the brain. Malnutrition in utero also leads to alterations in gene expression, termed fetal programming. In utero experiences can thus increase individual frailty and render a person more susceptible to developing chronic diseases such as diabetes, heart disease, and certain cancers later in life. Barker states that intrauterine conditions are the result of the mother's lifetime health experiences and, indirectly, those of the grandmother, given the importance of the mother's own prenatal development. Fetal development, therefore, is assumed to link a child's health to maternal health across several generations.

Kuzawa (Chap. 2) recasts the fetal origins theory from the perspective of biological anthropology. He suggests that in utero conditions not only impede normal fetal development but also lead the fetus to adjust physical set points to increase its chances of survival in a nutrition-scarce environment after birth. He notes that fetal health is surprisingly robust to both public health interventions that promote nutrition and to severe nutritional deprivation during pregnancy. Kuzawa argues that the fetus does not use the immediate uterine environment to extrapolate on the post-pregnancy environment but instead uses a more robust measure of the extra-uterine environment, namely, the mother's body condition. Both Barker's approach and Kuzawa's extension suggest that the time scale of child health reaches back beyond childhood and that consequently effective interventions have to as well.

Despite its theoretical cohesiveness and empirical evidence for the importance of in utero experiences in shaping health inequality, there are several shortcomings of the fetal origins theory. Most importantly, the theory focuses on the physiological consequences of deprivation and treats the socioeconomic context that triggers these processes as an exogenous factor (Haas, Chap. 3). Also, the theory assumes that risk factors that act beyond the fetal and infant stage are only moderators of the fetal experience and not health risks in themselves. This assumption elevates in utero experiences to a necessary cause for health inequality and implies that individuals who are born with good health will not show significant deterioration in their health as a consequence of social inequalities they may experience in later life.

Importantly, the building blocks of the developmental theory, such as critical periods, epigenetic influences, and developmental trade-offs, pertain to other periods of the life course as well and allow for the integration of the concept of fetal development and theories about socioeconomic determinants of health. Critical and sensitive developmental periods, that is, developmental plasticity or times when individuals are most susceptible to the effects of environmental exposures, extend into early childhood and adolescence, when important mental, emotional, and physical developments take place (Haas, Chap. 3). Fox (Chap. 4) provides evidence of the operation of critical periods in the study of children in Bucharest orphanages, some of whom were placed in foster care. These children had experienced severe psychosocial deprivation in infancy and early childhood. Fox and colleagues' results show that timely interventions have some effect on selected outcomes but that there can be long-term and seemingly irreversible damage to mental and physiological health due to early childhood deprivation. If critical periods extend beyond the womb, a stronger focus on family processes and the broader social context is needed to understand the link between families and child health.

Family Processes

Building on biosocial pathways that shape child health, we now examine how child health emerges at the interface between families and the larger social environment. In addition to configuring a child's social status, families serve as a filter of the

social environment, and family members, particularly parents, serve as orchestrators, models, and teachers of health practices within this environment (Bronfenbrenner, 1989; Drotar, Chap. 7; Parke, 2004). We highlight how family processes shape child health with examples from family relationships, the work–family interface, and the health-care system. We begin with a discussion of the connections between family relationships and child mental health.

The parent–child relationship is one forum within which poorly resourced and problematic family and community environments can generate maladaptive mental health outcomes. Davies, Sturge-Apple, and Martin (Chap. 5) introduce an innovative revision of Emotional Security Theory (EST-R), which takes a closer look at pathways in the well-established but little-understood connection between inharmonious family environments and detrimental mental health outcomes for children. The theory emphasizes how conflict between parents spills into other family relationships to produce emotional insecurity in early childhood. In turn, emotionally insecure children are more likely to experience other negative psychological, social, and behavioral health outcomes in childhood and beyond through a process termed developmental cascading (Masten & Cicchetti, 2010). For example, emotionally insecure children may more commonly experience behavioral problems later in childhood or depressive symptoms as adults.

Though Davies et al.'s (Chap. 5) theory focuses on family processes, it can be expanded to allow for the potential that social factors external to the family can lead to marital discord and therefore contribute to negative child mental health outcomes. Additionally, because high conflict marriages may be more common for adults who were emotionally insecure as children or those who experienced health problems, these processes may explain how poor mental health outcomes are transferred from one generation to the next.

A further connection between family processes and child health comes via the work–family interface. The process of spillover from parents' workplace environments into various domains of family life is well established (Perry-Jenkins, Repetti, & Crouter, 2000). Berkman and O'Donnell (Chap. 12) emphasize how a demanding work environment accompanied by low support and low control creates a triple jeopardy of risk for working-class families. High-stress work environments influence family environments and can have their first detrimental impacts during intrauterine development; later they can restrict parents' care giving and monitoring capacities. Parents also serve as the intermediary between children and the health-care system, and evidence of work–family conflict is particularly apparent among families of chronically ill children (Chung, Vestal, & Schuster, Chap 13). These parents must negotiate competing work and family demands including their ill child's frequent medical appointments and treatments. This stress is particularly pronounced among parents who have a limited ability to negotiate flexible schedules that accommodate their children's health-care needs.

Beyond simply providing health inputs, parents also orchestrate children's health care and behavior within the home and health-care system. This role includes negotiating with the health-care system on behalf of their children and managing their children's health behaviors (Chung et al., Chap 13; Drotar, Chap. 7). The role of

orchestrator becomes increasingly complex for parents of adolescents, when youth gain autonomy with respect to their health behaviors but continue to rely on their parents to manage their contacts with the health-care system (Harris, 2010). Parents who are highly stressed because of their work or other family demands may be less effective than other parents in this role (Drotar, Chap. 7). Furthermore, many aspects of health behavior and how adolescents disclose these behaviors are set in place early in the parent–child relationship (Morrongiello, Chap. 6). This suggests the importance of promoting positive parent–child dynamics around health and health behavior early in children's lives.

The Role of Structural Risk Factors in Child Health

Family processes are embedded within the broader social structure (Haas, Chap. 3). Socioeconomic and demographic characteristics such as parental income, education, occupation, and racial and ethnic origin influence exposure to stressors (Wilkinson & Marmot, 2003) and the resources parents have available to protect their health and that of their children (Grossman, 1972; Link & Phelan, 1995). Reichman and Teitler (Chap. 9) evaluate numerous structural exposures ranging from neighborhood conditions to environmental exposures during pregnancy.

Data sets such as the Los Angeles Family and Neighborhood Survey (Peterson et al., 2004), the Project on Human Development in Chicago Neighborhoods (Earls, Visher, & Justice, 1997; Tonry, Ohlin, & Farrington, 1991), or national data sets such as the Child Supplement of the Panel Study of Income Dynamics (Hofferth, Davis-Kean, Davis, & Finkelstein, 1997), the National Longitudinal Study of Adolescent Health (Bearman, Jones, & Udry, 1997), and the National Health and Nutrition Examination Survey (CDC & NCHS, 2011) have provided researchers with the opportunity to study the effects of structural factors on child and adolescent health. Findings from studies conducted on these and other data sets reveal that risk factors abound, but when considered individually, each explains only a small piece of child health disparities. Neighborhood physical and social conditions, for example, matter for some health and developmental outcomes, but in general, the effects are small and often statistically nonsignificant (Evans, 2006; Sastry & Pebley, 2003). Research on environmental toxins such as lead (Muennig, 2009) or ambient air pollution (McConnell et al., 2003; Peters et al., 1999) provides conclusive results that these exposures pose threats to particular child health outcomes such as asthma or child development (McConnell et al.; Stein, Schettler, Wallinga, & Valenti, 2002). Parents' health-related behaviors including breastfeeding and smoking, however, have been found to affect child health but contribute little to the SES gap (Reichman & Teitler, Chap. 9). Reichman and Teitler do not discuss the role of schools, which nevertheless should not go unnoted in this volume since school quality is closely linked to family socioeconomic status. Children of poor families are likely to attend schools with fewer resources (Anderson, Hollinger, & Conaty, 1992) where factors such as limited curricular and extracurricular opportunities, unhealthy food options,

and violence contribute to child health inequalities and the perpetuation of inequalities in educational attainment (Anderson et al.; Delva, O'Malley, & Johnston, 2007; Leviton, 2008; Mrug, Loosier, & Windle, 2008).

After reviewing a broad range of contextual risk factors, Reichman and Teitler (Chap. 9) identify a set of preconceptual and in utero exposures that they consider to be the most promising directions for studies aimed at understanding the origins of health inequalities. Such risk factors include mothers' prepregnancy health status, in utero environmental exposures, and nutrition and paternal exposures, including occupational hazards, for example. By focusing on socially stratified in utero exposures and by expanding the set of exposures from those that are maternally mediated to a broader set of risk factors, Reichman and Teitler embed the developmental or fetal origin perspective within the theory of social determinants of disease. In addition, they consider not only the direct influence of exposures to health risks across the life course but also the recursive effects of child health on social status and family well-being. Their conceptual framework presents a complex picture and poses an important question: In a scenario where differences in health outcomes are explained by a multitude of interconnected and recursive causal pathways, what interventions and policies will be effective in reducing childhood health disparities?

Intervention Points

Following a discussion of the factors that shape child health, we now turn to potential pathways for improving child health. The authors in this volume discuss a wide variety of intervention points that traverse the spectrum from intensive approaches that target high-risk populations to national policies that can be applied broadly. Their findings also suggest interventions at a variety of time points: during the child's lifetime, the prenatal period, and even reaching back to when the child's own grandmother was in utero. This volume develops a more complex understanding of the timeline along which different child health inputs occur, and these insights should help shape attempts to improve child health.

In conceptualizing how to improve child health, there is often a division between microlevel and macrolevel approaches. Microlevel approaches are typically more in-depth and targeted towards a specific risk group, often because it is not feasible to apply intensive interventions broadly. Specific examples of microlevel approaches discussed in this volume include Strong Healthy Women (Hillemeier, Chap. 10) and the Strengthening Families Program (Coatsworth, Chap. 8). Advantages of such programs are that they can target known vulnerable groups and that often they produce measurable improvements, at least from a short-term perspective. However, these programs may be available only to a select portion of potential beneficiaries (McGowan, Nix, Murphy, & Bierman, 2010). Many barriers to successful implementation remain, especially among high-risk groups that would likely garner the greatest benefits. Logistical barriers, such as lack of transportation and less-flexible

work schedules, as well as unwillingness to participate exist (Spoth, Redmond, Hockaday, & Shin, 1996). Our improved knowledge of the timeline of child health calls into question the extent to which microlevel approaches can improve outcomes. As both Barker (Chap. 1) and Fox (Chap. 4) describe, many poor health trajectories, both physiological and mental, are set in place by prior adverse experiences. Hence, even intervening during the months immediately prior to pregnancy may be too late, due to the mother's prior adverse exposures.

In contrast to microlevel approaches, national- and state-level policies are large-scale approaches that can potentially reach a broader segment of the population to offer specific health services or reduce poverty and inequality. These strategies aim to address the root of the problem rather than split resources among multiple pathways, each of which explains a small part of the difference in health status. National- and state-level policies may produce rewards slowly but will potentially reach a larger segment of the population with benefits that persist across generations. But again, there may be barriers to uptake, and potential beneficiaries often fail to access services and programs (Chung et al., Chap. 13).

Though different approaches may compete for funding, and there are trade-offs such that investment in one program automatically dictates that there are fewer resources to invest in another, one strategy is to formulate a combination of interventions that are successful across multiple domains and time frames. The success of programs is commonly judged using a cost–benefit analysis, which calculates the money saved by investing in a program. For example, the cost of the WIC, a federal program that provides nutritional and health support to low-income women, infants, and children, can be compared to how much is gained from the program using a variety of time forecasts. These include short-term benefits, such as fewer emergency room visits in infancy, and longer-term benefits, such as school achievement (Reynolds, Temple, Robertson, & Mann, 2002). As one means of combining multiple approaches, Kilburn (Chap. 15) describes a portfolio approach, which extends the cost–benefit analysis and quantifies how to strategically select multiple policies and programs for optimal child health investments. A comprehensive portfolio of child health investments could incorporate both universal investments that reduce long-term inequality and more targeted interventions for high-risk families. It can also invest across multiple outcomes (e.g., mental health, chronic disease) and target a variety of time scales, such that short-term and apparent child health needs are addressed alongside long-term investments with the potential to reduce persistent intergenerational health inequalities. The challenge then remains identifying the critical components that will optimize returns.

Looking Beyond Health-Care Policies to Improve Child Health

Though the current US political agenda is immersed in health-care policy reform, non-health-care policies are another potential approach for improving child health. Berkman and O'Donnell (Chap. 12) describe how many non-health-care policies,

particularly pro-family workplace policies, have the added benefit of improving child health. These approaches are often palatable to employers who can improve their bottom line by improving employee health and by being viewed by consumers as innovative. Improving child health via workplace policies may also be more politically feasible than improving it via health-care policies, which are often blockaded by partisan politics.

Specific examples of pro-family policies are flexible work schedules and family leave. Historically, work–family policies that provide time off to parents to care for their children have been more generously available to white-collar and full-time employees, whereas such policies are not often extended to working-class and part-time employees (Berkman & O'Donnell, Chap. 12). Recently, several state-level policies have attempted to distribute these benefits more universally (see Chung et al., Chap. 13). These reforms allow more parents the flexibility to attend to their children's needs, particularly during emergencies. However, their uptake remains a problem, and many workers fail to use the available benefits. Both national- and state-level policies that improve the work–family interface have the potential to make dramatic improvements in child health.

Intersection of Science and Policy

Finding ways to improve child health must not only include cooperation between academics and critical stakeholders; it must also incorporate child health research from across academic disciplines with disparate approaches. Historically, economists have maintained the closest ties to policy makers, whereas other disciplines have struggled to earn policy makers' attention. This may be because economists focus on the specific dollar benefits of policy actions. The insights that come from joining forces across disciplines have improved research on policies that promote child health, and scholars should continue these collaborative efforts.

Child health researchers must also learn how to meet policy makers on their terms. When infiltrated with a broad array of issues, policy makers want to know what outcomes should be prioritized and what specific steps will achieve particular goals (Kilburn, Chap. 15). Policy makers also want clearly delineated evidence based on theory (O'Campo, Chap. 14). It is important to lay out the specific pathways that show how intervention and change in one domain will achieve positive results in another domain. A challenge to researches is to provide this evidence.

Conclusions

This volume is based on presentations at Penn State's 19th Annual Symposium on Family Issues, held on October 4–5, 2011. Speakers brought together a wealth of evidence showing how family contexts matter for child health. They also amassed

important information on the potential of various policies and interventions for addressing family–child health linkages. A picture of complex causal pathways and possibilities for intervention emerged, but much remains unknown and many conceptual and methodological questions need to be addressed. Intrauterine exposures and epigenetic processes have been considered promising areas for future research. It remains to be seen, however, if those areas actually hold promise. Relatively little is known about intrauterine exposures and epigenetic processes or whether they indeed constitute pathways that explain a significant part of childhood health inequalities. As Haas (Chap. 3) notes, more needs to be known about the specific types of exposures that affect fetal development and how those exposures cause health vulnerabilities later in life. The concept of critical periods has emerged as crucial for understanding and addressing child health inequalities. Further research should address how the timing and duration of exposure to both health risk factors and interventions influence health (Haas, Chap. 3).

Reichman and Teitler (Chap. 9) also call for better conceptualization and measurement of child health. Most population studies focus on a particular health outcome such as asthma, obesity, or certain behavioral problems such as internalizing and externalizing symptoms (e.g., Halfon & Newacheck, 1993; McLeod & Shanahan, 1993; Whitaker & Orzol, 2006). Reichman and Teitler discuss the potential for a more comprehensive approach via a summary measure of "child health." The main problem with a summary measure that includes various physical pathologies and mental health outcomes is to decide on the weight that is accorded to each condition. Realistically, the chances of constructing a synthetic measure that is accepted by researchers and policy makers appear to be slim. A possible alternative could be translating ill-health and its short- and long-term consequences into dollar amounts of health-care costs, parental days off work to care for their children, and individual and societal costs of suboptimal educational and professional attainment (Muennig, 2009).

In addition to defining the outcomes, we also need to consider how to conceptualize and measure the "causes" of disease. Glass (Chap. 11) cautions researchers against entrapping themselves in a stringent definition of cause that forces them to consider the multitude of downstream factors that translate social inequality into ill-health. Instead, he suggests considering the role of social class per se in generating health inequalities. This would allow researchers' attention to be refocused away from the infinite complexity of pathways to broader mechanisms and to large-scale interventions addressing the roots of poverty. In addition, Glass suggests including a largely omitted factor into consideration: structurally driven class culture. Social structure conditions attitudes, expectations, decision making, and behaviors, generating a class culture that could serve as an explanation for a host of health behaviors and lifestyles.

Though some authors in this volume emphasize moving out and addressing inequality as the root cause of poor child health, others highlight the need for future research that improves explanations of the links between low health status and its proximal causes. Discussants of Davies et al.'s (Chap. 5) new conceptualization of Emotional Security Theory (EST-R) and Berkman and O'Donnell's (Chap. 12)

work–family strain praise the utility of such middle-range theories that begin to unravel the complex processes connecting family dynamics and child health. They suggest that more middle-range theories should be developed to explain the specific processes that connect other well-established links between broadly defined themes (Coatsworth, Chap. 8; Drotar, Chap. 7; O'Campo, Chap. 14). New research should also highlight factors that moderate these pathways (Morrongiello, Chap. 6). Additionally, Reichman and Teitler (Chap. 9) call attention to understudied factors, such as parental relationships, adverse life events, or genetic and epigenetic factors.

The plethora of potential research directions brings up several important questions: What does research on childhood health inequality need most at present? Do we need to uncover more causal pathways? Is it likely that there is a set of pathways that will explain a significant portion of the variability in child health outcomes? In what direction should policies and interventions go? Are small-scale intensive interventions, large-scale policies, or a portfolio approach to be favored? Resources are limited, and trade-offs have to be made. Postponing decisions on priorities risks inefficient allocation of time, money, and research and intervention capacities. Scholars must consider trade-offs when pursuing future research paths and data collection efforts; in-depth data that examine multiple health indicators across key time points inevitably need to be balanced with the breadth of large, nationally representative data sources.

Penn State's 19th Annual Symposium on Family Issues, and now this volume, offered researchers of family and child health the rare opportunity to bring together a wealth of knowledge from multiple disciplines. While the scope of research and knowledge is impressive, it is also clear that disciplinary strengths need to be combined to elaborate coherent and efficient solutions that are feasible from a policy maker's perspective. It is therefore crucial to formulate common goals and develop theoretical premises that guide sampling, measurement, intervention, and policy and allow child health researchers and advocates to determine above all *what* questions need to be answered next and *how* program and policy recommendations can be made.

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