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

Steven A. Haas

Department of Sociology and Crime, Law & Justice, Penn State University, University Park, PA, USA, sah49@psu.edu

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 under-developed. 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 & tuberculosis) were replaced by degenerative and chronic diseases (i.e. cancer, cardiovascular disease, & 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.

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 life style, 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 1000 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 and Ben-Shlomo, 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 under-developed. 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 inter-uterine 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 (Burtin, 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 pre-natal 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, pre-conception 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 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 & Hansen, 2004). Gluckman and Hansen (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 & Hansen, 2005). The key argument is that nutritional scarcity during these periods of gestation requires the fetus to make tradeoffs 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, Fewtrell, & 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 under developed. 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 1000 days post conception. 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 post-natal 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 pre-natal and neo-natal life at the expense of the rest of the life course. It is often argued that what is most important is the first 1000 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-Shlomo, 1997). Part of the problem is that research in the DOD tradition has tended to conceptualize the post-natal 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 1000 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 US 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 less than 2500 grams (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 (Kimbrow, 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 1000 days of life can explain this shift. What differences over their pre-conceptional life course leads Mexican-origin and African American mothers to have such different birth outcomes, yet very quickly leads their children towards 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 1000 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 & 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; Curie &

Moretti, 2005); early life health plays an important role in both subsequent health (Haas, 2007) and socioeconomic attainment (Case, Fertig, & Paxon, 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 can not 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 and Ben-Shlomo, 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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