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Intergenerational pathways linking maternal early life adversity to offspring birthweight

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ABSTRACT

Adverse birth outcomes can lead to problematic long-term outcomes for children, and are also known to transmit socioeconomic disadvantage across generations, thereby amplifying the importance of identifying their social determinants. However, the full set of factors causing adverse birth outcomes remains unknown. Drawing together theory describing intragenerational (life course) processes linking early life adversity to adult health, and intergenerational transmissions of inequality via birthweight, this study tests a chain of risk that originates within early adolescence, impacts young women's risky health behaviors in late adolescence/early adulthood and risky health behaviors during pregnancy, and ultimately decreases offspring's birthweight. We do so using structural equation models and prospective, population-level data on a racially and socioeconomically diverse cohort of young adults (National Longitudinal Study of Adolescent to Adult Health). Results (a) reveal four pathways that fully mediate the association between a young woman's family-of-origin socioeconomic status in adolescence and her offspring's birthweight, and (b) identify a trigger effect—a place in the chain of risk where prevention efforts could be targeted, thereby breaking the chain of risk leading to poor offspring health at birth for vulnerable individuals.

1. Introduction

Adverse birth outcomes, such as lower birthweight (measured as a continuous variable, in grams) or low birthweight (< 2500 g), are one mechanism through which socioeconomic disadvantage can be transmitted across generations (Aizer and Currie, 2014; Case and Paxson, 2006; Conley et al., 2003; Palloni, 2006). This classification holds based on the stark social patterning of adverse birth outcomes observed in the U.S. (meaning, poor mothers tend to exhibit higher risk of adverse birth outcomes), in combination with the generally increased risk of problematic longer-term outcomes (e.g., neurodevelopmental problems, learning disabilities, behavioral problems, lower educational attainment, poorer cardiovascular health) observed among children born with an adverse birth outcome (Behrman and Butler, 2007; Conley et al., 2003; Goldenberg and Culhane, 2007). It follows that identifying the etiology of adverse birth outcomes could not only create new opportunities to develop programs that effectively prevented such outcomes, but also potentially disrupt intergenerational transmissions of disadvantage via birthweight.

Despite the considerable attention placed on identifying the determinants of adverse birth outcomes however [for reviews, see (Kramer, 1987; Paneth, 1995; Goldenberg et al., 2008; Goldenberg and Culhane, 2007)], we still do not understand what causes adverse birth outcomes (Savitz and Murnane, 2010). Studies in this area have been hampered by a lack of appropriate data and/or methods available to test causal claims at the population-level (Kane and Margerison-Zilko, 2017). But, another key contributor is that this literature has historically focused on risk factors operative within the prenatal or immediate preconception period (Johnson et al., 2006; van Dyck, 2010), to the exclusion of earlier life course events and risk factors that could set into motion a chain of events that ultimately lead to poor health at birth among offspring (Richardson et al., 2012; Atrash et al., 2006). This stands in contrast to a wide body of theory, spanning multiple disciplines from the social sciences to public health, that emphasizes the prominence of the early life environment in shaping health outcomes that unfold within individuals over time (Center on the Developing Child at Harvard University, 2010, Hertzman and Boyce, 2010, Kuh and Shlomo, 2004, Halfon and Hochstein, 2002, Elder, 1977). Additionally, birth outcomes are a unique case in which both intra- and intergenerational mechanisms are at play (Kane, 2015; Palloni, 2006).

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Therefore, fully understanding the complex set of pathways that influence an offspring's birth outcomes requires drawing from theoretical perspectives spanning both intra- and intergenerational processes.

The current study identifies a set of theoretically-informed mechanisms that operate across women's early life and impact offspring birthweight, using data from a contemporary, population-based cohort. To do so, this study first draws together theory from sociology and epidemiology describing life course processes linking early life adversity to adult health. From this foundation, we formulate a set of hypotheses relating a mother's early life environment (in adolescence) to her risk and protective behaviors in late adolescence/early adulthood, and subsequently, her prenatal health behaviors. To describe the significant contributions these pathways may have on the life chances of the next generation (via offspring birthweight), we draw upon theory relating to intergenerational transmissions of inequality. We then test these hypotheses using a nationally representative, prospective dataset containing substantial racial and economic diversity that includes extensive information on individuals spanning multiple domains of health and wellbeing (biological, genetic, social, psychological, behavioral, environmental, and economic), measured at multiple stages in early life. We conclude with a discussion that integrates study findings related to intragenerational pathways with those related to intergenerational transmission of inequality, thereby providing a richer description of the sets of processes shaping adverse birth outcomes.

1.1. Background

Social conditions are a fundamental cause of disease (Link and Phelan, 1995). Early life adversity, in the form of material hardship or socioeconomic disadvantage, is one type of social condition that can adversely impact adult morbidity and mortality as an intragenerational process (Montez and Hayward 2014; Hayward and Gorman, 2004; Haas, 2008; Almond and Currie, 2011), and can also play a key role in the intergenerational transmission of inequality (Kuh and Shlomo, 2004; Elder, 1977, 1998; Elder et al., 2003; Palloni, 2006). These intra- and inter-generational literatures tend to parallel one another, but need not be mutually exclusive. Indeed, more rigorous study of the intersection of intra- and intergenerational pathways could reveal new insights into disrupting intergenerational transmissions of inequality (Kane, 2015; Palloni, 2006).

1.2. Theoretical framework

The prominence of early life social conditions for understanding patterns of adult health and wellbeing is perhaps most clearly articulated by life course theory. Within this framework, social conditions and exposures within each life stage are intricately linked across the life course; macro- and micro-level factors interact to shape the exposures, constraints, and opportunities individuals encounter as they move from one life stage to the next. Importantly, individuals enact human agency and personal control over situations they encounter, acting planfully (Elder et al., 2009; Elder, 1977, Elder, 1998).

These principles are also central tenets of life course epidemiology—a growing subfield of public health that builds on life course theory by bringing together social and biological origins of disease from across the life course (Ben-Shlomo and Kuh, 2002; Kuh et al., 2003). Situated within this framework, adverse exposures—whether environmental, socioeconomic, or behavioral—are thought to accumulate over time, ultimately degrading health by wearing down the body's ability to continually repair damage. Oftentimes, this accumulation involves multiple risk factors that independently influence a health outcome, but are also clustered, meaning they stem from a common source—such as family socioeconomic status; this is known as an accumulation model with risk clustering. A special case of this model is an additive chains of risk model, wherein one adverse exposure increases the risk of a subsequent adverse exposure and each exposure maintains both an indirect and direct effect on the health outcome. Another type of chain of risk model involves a trigger effect, whereby a chain of risk may stem from a trigger event, such as a health event, a move, the death of a loved one, experiencing abuse or trauma, etc., that increases one's risk of engaging in unhealthy behaviors and ultimately increases the risk of a poor health outcome (Kuh et al., 2003). Trigger events hold particular importance because these are places where chains of risk could be averted or broken for vulnerable individuals (Kuh and Shlomo, 2004).

Another aspect of life course epidemiology that relates to the present study is the notion that the timing of the exposure matters. Sensitive periods are stages in the life course in which the effect of a stressor on health is unique, such that, the same effect on health would not be observed if the stressor were encountered in a different stage of life. A prime example is adversity encountered in the sensitive period of childhood which has distinct and critical implications for adult health and wellbeing (Umberson et al., 2014; Ben-Shlomo and Kuh, 2002; Miller et al., 2011; Shonkoff et al., 2009; Hayward and Gorman, 2004; Haas, 2008).

Applied to the case of adverse birth outcomes, an accumulation model with risk clustering would suggest that adverse exposures across childhood and adulthood accumulate, each maintaining a direct effect on adverse birth outcomes while also sharing a common source, such as family socioeconomic status. The special case of this model, an additive chain of risk, links these adverse exposures through a chain, or pathway. Each adverse exposure may retain a direct effect on the birth outcome (an additive chain of risk model), or not (a trigger effect chain of risk model). The logic underlying these claims is that early life stressors induce physiologic dysregulation that ultimately impact adverse birth outcomes, either directly or indirectly through social and biological risk factors. Next we seek to identify adverse exposures that may comprise such a pathway.

1.3. Maternal early life adversity and offspring adverse birth outcomes

Using data from the 1958 birth cohort in Great Britain, Harville et al. (2010) demonstrated a link between early life adversity, indicated by material hardship and childhood neglect/abuse, and an increased risk of low birth weight and preterm birth; some of this association was mediated by prenatal smoking. Using more recent data from the U.S. [Waves I and III of The National Longitudinal Study of Adolescent to Adult Health (Add Health)] Gavin et al. (2012) found that childhood SES and childhood maltreatment lowered offspring birthweight; some of this association was mediated by adolescent substance abuse (measured as a latent variable indicated by adolescent smoking, heavy drinking, and drug use) and prenatal smoking. The U.S. study was limited however by (a) including a select group of young (largely teen) mothers, (b) using data (from Wave III) that systematically undercounted births, and (c) not examining adolescent smoking, drinking, and drug use as independent risk factors. The sample issue described in (a) is problematic because the salience of adolescent substance abuse for normative, later-timed births is unknown. As a result of the measurement issue described in (b) it is impossible to know if the study's results are generalizable to the U.S. population. Additionally, without being able to distinguish the unique effects of each form of substance abuse [implied by (c)], each of which may well imply a distinct prevention strategy, scholars have yet to clearly identify modifiable risk factors on the pathway between mother's early life environment and offspring's birth outcomes in the U.S.

Fig. 1 presents the measurement model guiding the present study. We begin by drawing a path linking a young woman's family-of-origin socioeconomic status (SES) in adolescence to her offspring's birthweight. Next we identify risk factors that potentially mediate this association. Based on past research (Harville et al., 2010; Gavin et al., 2012), we consider prenatal smoking to be a probable mediator. Given that the association between prenatal smoking and offspring birthweight is well-supported by evidence emerging from economics