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Utilizing life history theory to assess the effect of the accelerated maternal reproductive index on infant and child health outcomes

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Utilizing life history theory to assess the effect of the accelerated maternal reproductive index on infant and child health outcomes

by

Meghan T. Gillette

A dissertation submitted to the graduate faculty
in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

Major
Human Development and Family Studies

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Iowa State University
Ames, Iowa
2014

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CHAPTER 1. INTRODUCTION

My primary research interest, and thus my dissertation, centers on a key life event: menarche, or first menstrual period. Scholars have spent the last few decades documenting the declining age of menarche around the world (see Posner, 2006 for a review). Assessing the correlates and consequences of reaching menarche at a young age has contributed to understanding how important menarcheal timing is for human health. This research has also significantly developed our understanding of how pubertal development unfolds in humans as well as the interplay between genetics and the environment in terms of human growth and development. Thus, a large body of knowledge regarding individual predictors and consequences exists, providing an excellent foundation for broader exploration of the larger implications of a decline in menarcheal age.

This dissertation expands on this foundation to explore a long-term effect of a decline in menarcheal age. Specifically, I am interested in expanding knowledge regarding the long-term influence of menarcheal timing and subsequent reproductive development by examining next-generation health effects of the timing of maternal reproductive development, including the timing of menarche, sexual debut, and first childbirth, from an evolutionary perspective. While some researchers have begun to examine next-generation health effects of individual facets of reproductive development (Kirchengast & Hartmann, 2000; Basso, Pennell, Chen, & Longnecker, 2010; Jolly et al., 2000; Lenders et al. 2000; Jokela et al., 2007; Coall & Chisholm, 2003), very few have framed this research within an evolutionary perspective; yet, evolutionary theories provide a unique lens through which to examine modern human health. Indeed,
individual predictors and consequences of the timing of reproductive development have been explored, but in order to understand broad, long-term, intergenerational consequences, further research in this area is needed.

The Evolutionary Perspective.

Evolutionary theories that are used to examine human health and development fill a gap left by other psychological theories of development. Buss (1995) noted that human behavior is made possible by underlying psychological mechanisms, and that other psychological theories of development imply the existence of these mechanisms. For example, Bowlby suggested that human development is influenced by attachment, especially to caregivers (Bowlby, 1969). Bronfenbrenner posited the bioecological framework, which suggests that individuals are influenced by both genetics and the environment, including the physical and social environments (White & Klein, 2008). While these theories have proven extremely useful in understanding human relationships and behavior by answering the question, “How?” (i.e. how does the parent-child bond affect the child’s development?), they do not address the question of, “Why?” Why does the parent-child bond affect the child’s development? Why does the family environment during childhood affect psychological, emotional, and social development in adulthood? Ultimately, why are humans so sensitive to physical and social surroundings? Inarguably, an understanding of “how” is vital, though it is only the evolutionary perspective that can examine both questions of, “Why?” and, “How?” (Belsky, 2010). For example, how does maternal reproductive development influence offspring health, and why does maternal reproductive development influence offspring health?
These questions can be addressed using an evolutionary perspective, which considers the way in which humans evolved and the underlying mechanism, or force, behind human development and behavior. Specifically, the evolutionary perspective posits that the ultimate force behind human development and behavior is the need to maximize reproductive fitness by producing viable offspring and thus passing on genetic material. Because humans are born immature, have large, complex brains, require significant parental investment, and take a long time to reach reproductive maturity, we have evolved to be especially sensitive to our environment in terms of the availability or non-availability of resources (Draper & Harpending, 1982). Specifically, the hypothalamic-pituitary-adrenal (HPA) and hypothalamic-pituitary-gonadal (HPG) axes in the brain execute two different but related functions: the HPA axis acts as an internal barometer that informs the brain of the conditions of the external environment, while the HPG axis, which is connected to the HPA axis, controls the hormones that are released for pubertal maturation. It is through these evolved mechanisms that the physical and social environment influences human reproductive development, and sets off a life history strategy intended to maximize reproductive fitness in the context of the environment. Thus, the evolutionary perspective can be used to examine environmental influences on an individual’s reproductive health and development, and the long-term consequences of the timing and tempo of reproductive development.

In addition, the evolutionary perspective is the only perspective that allows for an understanding of reproductive health and behavior in terms of tradeoffs between quality and quantity of offspring. For example, evolutionary theories can be used to explore the long-term, intergenerational effects of maternal reproductive development on the number
of children the mother is able to have and the health of her children. By assessing public health issues from an evolutionary perspective, researchers may further our understanding of the long-term effects of cultural and environmental influences on human health and development by elucidating the ways in which the mechanisms we evolved in an ancestral environment serve (or disserve) us in today’s environment.

**Life History Theory.** In an effort to contribute to knowledge regarding the importance of the timing of reproductive development on individual next-generation health, this research utilized life history theory, an evolutionary perspective, to explore why and how maternal reproductive development influences offspring quantity (number of offspring) and quality (infant and child health). This theory is used to explain the ways in which an organism allocates finite metabolic energy for growth, maintenance, or reproduction in order to maximize reproductive fitness (Ellis, 2004). In an ideal environment, humans will allocate a great amount of energy to growth during the first decade of life (childhood) and maintenance during the second decade of life (adolescence). In the second decade of life, humans must allocate energy to growing reproductive organs and, especially for females, provisioning offspring. But, life history theory posits that, in adverse conditions, reproductive maturation will be accelerated (the transition from growth to maintenance to reproduction will be hastened) so as to ensure the propagation of the genetic line in case the parent does not survive (Ellis, 2004). Earlier reproductive development can be achieved by accelerating the reproductive index, and thereby lengthening the reproductive lifespan, thus allowing the mother to have her first child at a younger age. However, earlier reproductive development may come at a cost, in terms of the tradeoff between the quantity and quality of offspring (in terms of
infant and child health and well-being). Thus, life history theory was utilized in this dissertation in order to test hypotheses regarding the impact of an accelerated reproductive index on next-generation health.

**Dissertation Organization**

Following the manuscript dissertation format, this dissertation includes two studies that utilize restricted-use data from the *National Longitudinal Study of Adolescent Health* (*Add Health*) to examine the relationships between an accelerated maternal reproductive developmental index (AMRI; early menarche and/or early sexual debut and/or early first childbirth) and the quantity and quality of offspring. Specifically, in Chapter 2, the first study, entitled, “The influence of the accelerated maternal reproductive index on the quantity and quality of offspring” explores whether or not an AMRI influences the number of children a woman has and the birth outcomes (low birth weight or LBW and premature birth) of her first child in light of many familial, socioeconomic, and individual factors. Specifically, I hypothesized that an AMRI would significantly predict a higher number of offspring, as well as LBW and preterm birth. This study contributes to the literature by examining the effect of an AMRI on the quantity and quality of offspring; previous studies have only examined individual facets of the AMRI, not the cumulative reproductive index, on offspring outcomes. Thus, results from this study will be beneficial for future research that examines trade-offs between quantity and quality of offspring. In addition, this study contributes to the literature by controlling for a plethora of familial, socioeconomic, and individual factors that are also known to influence quantity and quality of offspring.

In Chapter 3, the second study, entitled, “The influence of an accelerated
maternal reproductive index and infant birth outcomes on subsequent child health outcomes” builds on the first paper. While the first paper explored health in infancy, the second paper explored whether or not an AMRI impacts offspring’s health into childhood in light of familial, socioeconomic, individual, and infant factors. Specifically, I hypothesized that an AMRI would predict more incidences of health conditions for offspring in childhood even after controlling for many familial, socioeconomic, and individual maternal factors, as well as LBW, preterm birth, and offspring’s age. This study contributes to existing knowledge about the impact of maternal reproductive development on next-generation health by examining the long-term effects of an AMRI on the health and well-being of offspring into childhood in terms of the number of health conditions a child had.

Analyses for both of these studies focused on the health outcomes (LBW, preterm birth, and health conditions) of the mother’s first child. The first child was selected for these studies because, from an evolutionary perspective, the first child would have likely received the most parental resources (whether socioeconomic, emotional, or somatic), at least at the beginning of his/her life, and thus would theoretically have access to more parental resources and potentially enjoy better health. The resources an only child receives are not shared with any other sibling; when a second or subsequent sibling enters the family, these resources are reallocated (and, in many ways, decreased; for a review, see Schlomer, Del Giudice, & Ellis, 2011). In addition, the effectiveness or viability of the mother’s reproductive developmental index would be revealed in her first offspring’s health and development, since any subsequent offspring would have developed in utero with a mother who was already allocating resources to her first child. Thus, the analyses...
focused on the first child only. Lastly, Chapter 4 comprises a broad, overall conclusion, limitations, and implications of this research.
CHAPTER 2. THE INFLUENCE OF THE ACCELERATED MATERNAL REPRODUCTIVE INDEX ON THE QUANTITY AND QUALITY OF OFFSPRING

Modified from a paper to be published in *Evolution and Human Behavior*

Meghan T. Gillette\(^1,2\) and Brenda J. Lohman\(^3\)

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Abstract

Evolutionary theories are useful for examining public health issues as well as elucidating the ways in which the health and development of one generation impacts the next. Indeed, previous studies have found relationships between individual facets of maternal reproductive development (e.g. age at menarche, sexual debut, and first childbirth) and two primary components of the human reproductive strategy: the quantity and quality of her offspring. In this study, life history theory was used to, for the first time, examine the effect of a cumulative accelerated maternal reproductive developmental index (consisting of early menarche, early sexual debut, early first childbirth; AMRI) on the quantity and quality (in terms of preterm birth and low birth weight; LBW) of offspring in light of a myriad of control variables. Using data from the *National Longitudinal Study of Adolescent Health* (Add Health), results showed that an AMRI significantly predicted a

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higher number (quantity) of offspring, but did not significantly predict the quality of offspring. This may have been due to the plethora of life factors (familial, individual, and socioeconomic) included in the model, since previous studies did not control for all of these influences. Most notably, father absence during the mother’s childhood significantly impacted quality of offspring, a finding that requires further exploration. In conclusion, this study provides a starting point for future investigations regarding cumulative maternal reproductive trajectories and the extent to which they can be used to examine trade-offs between the quality and quantity of offspring among humans.
1. Introduction

Many scholars have utilized evolutionary theories to examine public health issues, as evolutionary perspectives can be used to assess not only the proximate ("how") but also the ultimate ("why") causes of a particular issue (Belsky, 2010). Evolutionary theories are especially helpful in elucidating the ways in which the health and development of one generation impacts the next. An understanding of the intergenerational transmission of health and developmental patterns is vital for addressing current health issues, including high rates of low birth weight (LBW; less than 5.5 pounds; World Health Organization Statistical Information System, 2012) and preterm birth (birth before 37 weeks; Centers for Disease Control, 2012). Indeed, LBWs and preterm births are on the rise in the U.S. (Donahue et al., 2010; Martin et al., 2010; Martin et al., 2002; Annie E. Casey Foundation, 2009) and tend to occur from one generation to the next (Annie E. Casey Foundation, 2009). Since the consequences of LBW and preterm birth are both extensive and expensive (e.g., preterm births cost the U.S. healthcare system more than $26 billion dollars per year; Centers for Disease Control, 2011; LBW babies are more likely to experience disabilities, chronic disease, and/or morbidity; Goldenberg & Culhane, 2007), exploring both why more LBW and preterm babies are being born and how this cycle is perpetuated is essential. Understanding the pathways through which the propensity for preterm birth and/or LBW operates may inform policy makers, public health officials, and interventionists, and may contribute to a reversal in these unhealthy trends.
1.1 Life history theory

In this study, life history theory (a type of evolutionary theory; Stearns, 1992) was used to explore the intergenerational transmission of health. Briefly, this theory is used to explain the ways in which an organism allocates finite metabolic energy for growth, maintenance, or reproduction in order to maximize reproductive fitness, or their ability to successfully pass on genetic material to reproductively viable offspring (Ellis, 2004). Specifically, humans have evolved flexible developmental patterns (i.e. variable timing of menarche) so as to maximize the ability to reproduce offspring and successfully continue propagation of the genetic line. Furthermore, humans have evolved a reproductive strategy that favors quality over quantity (Gluckman et al., 2011; Gluckman & Hanson, 2006); unlike other animals, humans tend to have fewer offspring (lower quantity) who require significant parental investment (higher quality). But, theoretically, a trade-off must occur between current and future reproduction and the quantity and quality of offspring. That is, maximizing quality of offspring (at the expense of a high quantity of offspring) may ensure high-quality offspring over generations and thus a higher likelihood of propagation of the genetic line, but only if the offspring survive. Yet, maximizing the quantity of offspring may ensure the probability that at least some will survive and reproduce, even if the parent is not able to invest heavily in each one, potentially resulting in lower quality (Chisholm et al., 2005).

In the context of this study, life history theory would suggest that girls who reach menarche (first menstrual period) early would be more likely to engage in an accelerated reproductive pattern (including early age at sexual debut and/or early age at first childbirth) that favors early reproduction rather than late reproduction and quantity over
quality of offspring. Engaging in this reproductive pattern is not necessarily a conscious decision; life history theory suggests that favoring quantity over quality of offspring may be an adaptive strategy left over from living in a stressful ancestral environment. Thus, this study assessed the impact of an accelerated maternal reproductive index (AMRI) on both quantity (the number of offspring) and quality of offspring (LBW and preterm birth of the first born).

1.2 Accelerated maternal reproductive index

Since this study examined the influence of an AMRI on quantity and quality of offspring, an operational definition of an AMRI is needed. Based on previous literature, the AMRI was comprised of early menarche (before age 12; Ge, et al., 2007; Watson et al., 2007; Ge et al., 2001; Cavanagh, 2004; Foster et al., 2008; Manlove et al., 2007; Zabin et al., 2005; Romans et al., 2003; Wise et al., 2009), and/or early sexual debut (first sexual intercourse before age 16; Ellis et al. 2003; Fergusson & Woodward, 2000; Paul et al., 2000; Spriggs & Halpern, 2008; Neberich et al., 2010), and/or early age at first childbirth (before age 21; Boden et al., 2008; Dupont & Page, 1986; Chisholm et al., 2005). Indeed, menarcheal age, sexual debut age, and age at first childbirth are often correlated; girls who reach menarche early are more likely to experience early sexual debut (Neberich et al., 2010; Downing & Bellis, 2009; Edgardh, 2000; Chodick et al., 2005; Watson et al., 2007; Udry, 1979). Similarly, girls who reach menarche early and/or sexual debut early are more likely to have their first child at an earlier age (Chisholm et al., 2005; Quinlan, 2003; Udry, 1979).

Women who reach all or some of these milestones earlier than the average woman are said to have an accelerated reproductive index (Belsky et al., 2011; Quinlan, 2003).
Quinlan (2003) regarded these three constructs as comprising a woman’s reproductive index, but only evaluated them separately, not cumulatively. While scholars have explored these individual reproductive milestones on quantity and quality of offspring among humans (see Kirchengast & Hartmann, 2000; Basso et al., 2010; Jolly et al., 2000; Lenders et al. 2000; Jokela et al., 2007; Coall & Chisholm, 2003), none have, to our knowledge, examined the effect of a cumulative AMRI on quantity and quality of offspring.

1.3 Antecedents of the AMRI and quality and quantity of offspring

In addition to utilizing a cumulative maternal reproductive index variable for the first time, this study is also unique in that it incorporated a plethora of familial, individual, and socioeconomic life factors that may influence the maternal reproductive index and quantity and quality of offspring. For example, LBW girls are more likely to experience compensatory catch-up growth in childhood, which leads to early menarche (Ibanez et al., 2006; Ong & Dunger, 2004; Sloboda et al., 2007). In addition, LBW mothers are four times more likely to have a LBW baby (Annie E. Casey Foundation, 2009). Father absence during childhood predicts early menarche (for an explanation, see: Draper & Harpending, 1982; Belsky et al., 1991; Ellis, McFayden-Ketchum et al., 1999; Hoier, 2003; Maestripieri et al., 2004; Matchock & Susman, 2006; Bogaert, 2005; Quinlan, 2003; Ellis et al., 1999). Parent’s income and education level have been shown to influence menarche, sexual debut, and age at first pregnancy wherein those with a lower socioeconomic status are more likely to experience these reproductive events at an earlier age (Quinlan, 2003; Blum et al., 2003). In the current study, access to health insurance in childhood was also included as an indicator of socioeconomic status. Finally,
girls who are overweight and/or obese are more likely to start puberty earlier (Rosenfield et al., 2009; Adair & Gordon-Larsen, 2001; Anderson & Must, 2005; Currie et al., 2012). Jokela et al. (2007) found that women who were underweight or obese as adolescents had fewer children in adulthood.

Individual, family, and socioeconomic life factors during adolescence and adulthood also impact reproductive development and the health of offspring. Girls who experience early sexual debut are less likely to initiate postsecondary education (Spriggs & Halpern, 2008; Parkes et al., 2010); in addition, an inverse relationship between secondary education and the number of children exists (Cohen et al., 2011; Castro, 1995). In terms of weight status, scholars have found that underweight women are more likely to have a LBW infant and preterm birth (Goldenberg et al., 2008; Hendler et al., 2005; Doherty et al., 2006). Overweight and obese mothers are more likely to give birth to a high birth weight infant and to have a preterm birth (Baeten et al., 2001). Access to health insurance in adulthood as well as if the mother received prenatal care were included in the current study, despite the debate about the effectiveness of prenatal care in improving health outcomes (see Lantos & Lauderdale, 2011; Fiscella, 1995; Walford et al., 2011; MacDorman & Singh, 1998). Finally, ethnicity was also considered, as ethnic disparities in reproductive development and offspring health exist (Wu et al., 2002; Anderson & Must, 2005; Cavanagh, 2004; McDowell et al., 2007; Cavazos-Rehg et al., 2009; Blum et al., 2000; Schieve & Handler, 1996; Annie E. Casey Foundation, 2009). These individual, family, and socioeconomic factors have been included, in some combination, in previous studies, but not collectively in one analysis, as in this study.
1.4 Hypotheses

The aim of this study was to fill the current gap in the literature by being the first, to our knowledge, to explore the impact of a cumulative AMRI on two primary components of the human reproductive strategy, quantity and quality of offspring (LBW and preterm birth), in light of a myriad of confounding individual, family, and socioeconomic factors (see Figure 1). The findings will set the stage for future research on the intergenerational transmission of health, the pathways by which preterm birth and LBW occur, and the trade-offs between quantity and quality of offspring. Therefore, guided by life history theory and based off of previous studies, three hypotheses were formulated:

1. Quantity: An AMRI would significantly predict a higher number of offspring;

2. Quality:
   a. An AMRI would significantly predict LBW of the offspring;
   b. An AMRI would significantly predict preterm birth of the offspring.

2. Methods

2.1 Sample

Longitudinal data is useful for studying the intergenerational transmission of developmental patterns and disease. Thus, the National Longitudinal Study of Adolescent Health (Add Health) was utilized, as it contains data following the participant from adolescence to adulthood, as well as outcomes of his/her offspring. Add Health is a nationally representative panel study of adolescents initially drawn from a random cluster sample of public, private, and parochial high schools (and their feeder schools) in the United States. The first wave of the study (1994-1995) gathered information from
students in grades 7-12 as well as information from a portion of the adolescent’s caregivers. A second wave followed one year later. A third wave of the study took place six years later (2001-2002) when most respondents were ages 18-26, and a fourth wave of the study was completed in 2008, when the respondents were between the ages of 24 and 34 (Harris et al., 2009). Only data from those females who participated in, at the very minimum, Waves I and IV, whose caregiver completed the parent in-home questionnaire in Wave I, and who reported at least one live birth were used, yielding a sample size of 2,797. These participants, referred to for the duration of the paper as “targets” answered questions about their offspring. A description of the sample is provided in Table 1.

2.2 Measures

2.2.1. Dependent variables

*Number of offspring (target).* In Wave IV, the target reported how many live births she had ever had. To reduce skewness, this variable was top-coded to represent one to four or more births.

*Preterm birth (offspring).* In Wave IV, participants were asked if their first baby was born before, on, or after their due date. For those who reported their baby was born before the due date, the number of weeks early was recorded. The variable was coded as 1 = birth before 37 weeks and 0 = birth 37 weeks or later (Centers for Disease Control, 2012).

*Low infant birth weight (offspring).* In Wave IV, participants were asked, “Did [baby] weigh less than 5.5 pounds at birth?” They responded 1 = yes or 0 = no.
2.2.2. Independent variable.

**Accelerated maternal reproductive index (target).** Three variables, early menarche, early sexual debut, and early age at first childbirth, were dichotomized where 1 = early and 0 = not early. These were then summed to create a composite variable wherein a higher value represented a more accelerated reproductive index. A description of each dichotomous variable follows.

*Early menarcheal age.* Participants were asked in Waves I and II, “How old were you when you had your very first menstrual period?” Menarcheal age at Wave II was used, though age at Wave I was used if the information was not given in Wave II. This variable was dichotomized where 1 = reached menarche 11 years or younger and 0 = reached menarche at age 12 or later, consistent with previous methods (Ge et al., 2007; Watson et al., 2007; Ge et al., 2001; Cavanagh, 2004; Foster et al., 2008; Manlove et al., 2007).

*Early sexual debut age.* Participants were asked in Waves I and II to provide the year they first engaged in sexual intercourse; in Wave III, participants were asked to report their age at first sexual intercourse. Ages at sexual debut in Waves I and II were calculated by subtracting the participant’s birth year from the reported year of sexual debut. Following Brückner and Bearman (2005), age of sexual debut was taken from Wave I if reported; from Wave II if no age at Wave I was reported; and Wave III if no age at Wave I or II was reported. This variable was dichotomized where 1 = reached sexual debut age 15 years or younger; and 0 = reached sexual debut age 16 years or older, as is consistent with previous studies (Neberich et al., 2010; Downing & Bellis, 2009; Edgardh 2000; Chodick et al., 2005; Watson et al., 2007).
Early age first childbirth (target). In Wave IV, the age of the first child reported was subtracted from the mother’s age, yielding the age at which the mother had her first live birth. This variable was dichotomized where 1 = had first child early (before age 21), or 0 = had first child at 21 years or after (Boden et al., 2008; Dupont & Page, 1986; Chisholm et al., 2005).

2.2.3. Individual, familial, and socioeconomic covariates

Low maternal birth weight. In Wave I, a parent questionnaire was completed by a caregiver of the adolescent, wherein the caregiver was asked to report the birth weight of the adolescent in pounds and ounces. The variable was dichotomized where 1 = birth weight below 5.5 pounds and 0 = birth weight of 5.5 pounds or more (Centers for Disease Control, 2013).

Father absence during target childhood. In Wave I, the caregiver reported whether or not the adolescent’s father was living in the home or not. This variable was dichotomized where 1 = father was absent at any point during childhood (including those who never lived with the adolescent) and 0 = father was not absent during childhood.

Target adolescent weight status. Body mass index (BMI) in Wave I was calculated using the adolescent's self-reported measurements of height and weight (kg/m²). Up to age 20, BMI is age and gender-specific; thus, BMI percentages were used to categorize the adolescents into four weight categories based on the Centers for Disease Control (CDC) classification system: underweight (BMI <5th percentile); healthy weight (BMI 5th to 84th percentiles; overweight (BMI 85th to 94th percentile); and obese (BMI ≥ 95th percentile) (Centers for Disease Control, 2011). Dummy variables were created for
each of the categories where 1 = part of that weight status and 0 = not part of that weight status; participants who were “healthy” comprised the referent group.

*Parent income during target adolescence.* In Wave I, the caregiver reported the total household income (in 1995 dollars). Following Blum et al.’s (2000) measure using Add Health data, income was rounded to the nearest thousand dollars. For those participants whose parents did not report income, the median value of the participant’s subgroup was imputed (based on sex, race/ethnicity, marital status, welfare status, and education).

*Parent education level during target adolescence.* In Wave I, the caregiver reported his/her highest level of education attained. This was transformed into four categories: less than high school diploma; high school diploma or GED; some postsecondary education; bachelor's degree or higher (Spriggs & Halpern, 2008). Dummy variables were created for each of the categories where 1 = attained that education level and 0 = did not attain that education level; those who reported having a bachelor’s degree or higher comprised the referent group.

*Target access to health insurance in adolescence.* In Wave I, caregivers were asked, "What type of health insurance does [child] have?" Participants could specify what type of insurance they had, or could indicate they did not have any. This variable was dichotomized where 1 = had health insurance of some kind; or 0 = did not have health insurance.

*Target age.* Age of the participant at Wave IV was included. Ages ranged from 24 to 32 years.
**Target ethnicity.** Race/ethnicity information was reported at Wave I. Participants were categorized as either Hispanic/Latino, White, Black or African-American, American Indian or Native American, and Asian, or Other (Add Health, n.d.), where 1 = part of that ethnic category and 0 = not part of that ethnic category. Participants who identified as White comprised the referent group.

**Target adult education level.** In Wave IV, the participants were asked to report their highest education level they had achieved to date. This variable was categorized into four categories: less than high school diploma; high school diploma or GED; some postsecondary education; Bachelor's degree or higher (Spriggs & Halpern, 2008). Dummy variables were created for each of the categories where 1 = attained that education level and 0 = did not attain that education level; those who reported having a bachelor’s degree or higher comprised the referent group.

**Target access to health insurance as an adult.** Participants were asked to report their current health insurance situation in Wave IV. They reported the type of health insurance they had (e.g., private insurance, on parent’s insurance, Medicaid) or reported that they did not have any health insurance. This variable was dichotomized, where 1 = had health insurance of some kind; or 0 = did not have health insurance.

**Target adult weight status.** Add Health field interviewers took weight and height measurements of the target in Wave IV, from which the Add Health team created a BMI variable. Participants were categorized into the following four CDC BMI categories: underweight (<18.5); normal (18.5-24.9); overweight (25-29.9) and obese (30+) (Centers for Disease Control, 2011). Dummy variables were created for each of the categories
where 1 = part of that weight status; or 0 = not part of that weight status; participants who were “healthy” comprised the referent group.

Visited a midwife/doctor for prenatal care. Female participants were asked in Wave IV if they ever visited a doctor, nurse-midwife, or any other type of health care provider for prenatal check-ups. Participants answered 1 = yes or 0 = no.

2.3 Analytic Plan

Since the purpose of this study was to explore the influence of maternal reproductive development on the number of offspring and infant birth outcomes, a multiple linear regression and logistic regressions were completed in SPSS 22.0. Iowa State University’s Institutional Review Board deemed this study exempt from approval for research, since it is deidentified secondary data.

3. Results

Results of the linear and logistic regressions are presented in Tables 2 - 4. Significant findings for each of the regressions are discussed in turn.

3.1 Hypothesis 1: Quantity: An AMRI predicts a higher number of offspring.

As hypothesized, an AMRI significantly predicted a higher number of offspring (more children; \( \beta = .18, p < .001 \)). The regression also revealed significant individual and socioeconomic factors. First, African Americans had more children compared to Whites (\( \beta = .04, p < .05 \)). Second, the older the mother was when surveyed in adulthood, the more children she was likely to have (\( \beta = .10, p < .001 \)). Third, those who were less educated, such as mothers with less than a high school degree (\( \beta = .22, p < .001 \)), a high school degree only (\( \beta = .18, p < .001 \)), and some college credits completed (\( \beta = .12, p < .001 \)), had more children compared to those women with a college degree. Finally, those mothers who
received prenatal care during pregnancy had more children than those who did not receive prenatal care ($\beta=0.09, p<0.001$).

3.2 Hypothesis 2a: Quality: An AMRI predicts LBW of the offspring.

An AMRI did not significantly predict the offspring’s LBW; in other words, experiencing reproductive milestones at an early age such as menarche, sexual debut, and childbirth did not lead to having a LBW baby, contrary to previous studies. Four individual, family and socioeconomic factors were significant, however. First, a one standard deviation (approximately $26,000) increase in parental income the mother experienced when she was an adolescent decreased the chances of having a LBW baby by 1% ($p<.05$). Second, if the mother was a LBW baby herself, her baby was 39% less likely to have a LBW ($p<.05$; a possible explanation for this counterintuitive finding is presented in the Discussion section). Third, if the mother, as an adolescent, had experienced father absence at any time, the odds of her baby having a LBW increased by 39% ($p<.05$). Finally, if the mother was overweight in adulthood, her baby was 37% less likely to have a LBW ($p<.05$).

3.3 Hypothesis 2b: Quality: An AMRI predicts preterm birth of the offspring.

Similar to results for Hypothesis 2a, an AMRI did not predict having a preterm baby. Three individual and family factors reached significance. First, if the mother as an adolescent experienced father absence at any time, she was 41% more likely to have a preterm birth ($p<.05$). Second, if the mother identified as Asian, she was 71% less likely to have a preterm birth compared to mothers who identified as White ($p<.05$). Third, if the mother was underweight in adulthood, she was 122% more likely to have a preterm birth compared to mothers who had a healthy weight in adulthood ($p<.05$).
4. Discussion

This study expanded current knowledge regarding maternal reproductive development and its impact on the quantity and quality of offspring. Based on life history theory and previous studies, we explored whether an AMRI would significantly predict both quantity and quality of offspring. Another unique facet of this study was the inclusion of a plethora of individual, family and socioeconomic factors. With such a rich dataset like Add Health, we aimed to include a large number of individual, family and socioeconomic confounding factors that may not have been included in previous studies.

Results of this study found that an AMRI was related to the quantity of offspring but not the quality. Specifically, an AMRI significantly predicted the number of offspring a woman had, but did not predict LBW or preterm birth of her first born. That is, when early menarche, early sexual debut, and early age at first childbirth were combined into one variable to represent an AMRI, it only predicted the quantity of the offspring, not necessarily the quality of the offspring. The lack of significant findings regarding quality is unexpected given previous work, where others found a relationship between mother’s age at menarche and offspring quality in terms of offspring size (Basso et al. 2010; Kirchengast & Hartmann, 2000) as well as the mother’s age at childbirth and preterm birth and low birth weight (Jolly et al., 2000; Fraser et al., 1995; Chen et al., 2007). However, previous work did not examine maternal reproductive development cumulatively, but rather only individual facets of reproductive development (i.e. age at menarche, age at childbirth).

Our findings regarding quantity partially support life history theory, which would suggest that a faster reproductive developmental index would represent a quantity over
quality reproductive strategy. Indeed, if a mother experienced an accelerated reproductive index she was significantly more likely to have more children. However, we do not know if there was a trade-off between quantity and quality of offspring. That is, we do not know if those who have many children also have children with more health problems, as life history theory may suggest. Furthermore, we do not know the extent to which parents, or at least mothers, make conscious decisions regarding current and future reproductive trade-offs in terms of quantity and quality of life for their offspring. Future work that is qualitative in nature could help explore the ways in which parents make decisions about whether or not to continue to reproduce.

Our hypothesis regarding quality of offspring was not supported; an AMRI did not predict quality of the offspring as measured by LBW or preterm birth. We postulate that this may be because of the low number of offspring who had LBW (8%) and preterm birth (9%). The result may also be attributed to the many individual, family and socioeconomic factors that were included in the analyses. Previous studies had not included as many control variables in one model, and this may have washed out any effects of maternal reproductive development on offspring quality, suggesting that perhaps it is not necessarily the mother’s reproductive development but other characteristics in childhood and adulthood that predict LBW and preterm birth. Notably, the control variables that were significant, such as the mother’s LBW, her environment in adolescence (father absence, parent’s income), and weight status as an adult, represent a range of influences, including familial, socioeconomic, and physiological, on her offspring’s health. In addition, these influences represent a range of stages in the mother’s life history: characteristics at birth, in adolescence, and in adulthood all play a
role in predicting the quality of her offspring. Together, these findings support the notion that the intergenerational transmission of health is multifaceted and complex, but reveals many points of intervention for improving health and well-being.

One unpredicted finding was that those mothers who had a LBW themselves were 39% less likely to have a low birth weight baby. This may be due to the compensatory catch-up growth that many LBW babies experience (Ibanez et al., 2006; Barker & Osmond, 1986). Briefly, LBW babies undergo fetal programming that wires their bodies to conserve metabolic resources in the womb (resulting in a small body size) but to over compensate in childhood and beyond when exposed to adequate calories, in case those resources become unavailable later in life. Thus, LBW babies tend to become obese in adulthood (Hales et al., 1991). Indeed, of those targets who had a LBW themselves (approximately 8% of the sample), 34% were obese and 26% were overweight in adulthood. Only 13% of LBW targets had a LBW baby. Compensatory catch-up growth may possibly provide some sort of buffering effect against having a LBW offspring. However, further research is needed to confirm this finding.

A second important finding was the effect of father absence on the quality of offspring; if the father was absent during the mother’s childhood, she was more likely to have a LBW or preterm baby. The impact of father absence on menarcheal timing was discussed in the introduction. Briefly, evolutionary theories (such as psychosocial acceleration theory) suggest that father absence from the home signals to the child that the future is unstable, that caregivers cannot be trusted, and that quality mates are unavailable, thus prompting an accelerated maturation in response to these stressful cues (Draper & Harpending, 1982; Belsky et al., 1991; Ellis, 2004). However, the significant
impact of father absence on the third generation’s health is unprecedented. We are not able to affirm a specific reason as to why father absence may impact the quality of his infant grandchildren. Perhaps the same mechanism(s) (e.g. epigenetics, fetal programming) that elicit(s) an AMRI when father absence occurs in childhood are not only influential in childhood and adolescence. It may be the case that these mechanisms are still at work in adulthood, and evoke long-term effects that persist into the third generation. Regardless, this unexpected finding provides an excellent starting point for research that examines familial influences on third-generation health and development, as it highlights the long-term effects of father absence from the home not only on his child’s health but also his grandchildren.

5. Future Directions

While the hypotheses for this study were only partially supported, evolutionary theories that examine the intergenerational transmission of health are still beneficial for addressing current public health issues. While this study utilized life history theory to assess the impact of an AMRI on quantity and quality of offspring, the next step should be to examine trade-offs between quantity and quality of offspring. That is, do those women with an AMRI and who have more offspring make a trade-off between the number of offspring and the quality of offspring? Future studies will need to examine whether an AMRI is advantageous for humans in terms of increasing the number of offspring, or if an AMRI is not actually advantageous because it may contribute to poor infant health outcomes.
Acknowledgements

This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (http://www.cpc.unc.edu/addhealth). No direct support was received from grant P01-HD31921 for this analysis.
References


Figure 1. Conceptual Model

**Individual, Family, and Socioeconomic Factors**
- Child (Target)
  - Low birth weight
  - Ethnicity
  - Father absence
  - Parent income/education level
  - Weight status
  - Access to health insurance
- Adult (Target)
  - Income/education level
  - Weight status
  - Access to health insurance
  - Utilized prenatal care
  - Age

**Maternal Reproductive Trajectory**
- Early menarche
- Early sexual debut
- Early first childbirth

**Outcomes**
- Number of offspring
- Premature birth
- Low birth weight

Offspring Quantity

Offspring Quality
Table 1.
Sample Descriptives ($n=2,797$)

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</tr>
<tr>
<td>Early sexual debut</td>
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<td>Early age at first birth</td>
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</tr>
<tr>
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</tr>
<tr>
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<tr>
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</tr>
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<td>Prenatal Care</td>
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<td>Health Insurance</td>
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Table 2.
Linear Regression Predicting Number of Offspring \((n=2,797)\)

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<td>Adulthood Variables</td>
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Notes: \(*p<.05, \***p<.001\); (a) Reference Group: College Degree Obtained; (b) Reference Group: Healthy Weight; (c) Reference Group: Whites.
Table 3. Logistic Regression Predicting Low Birth Weight ($n=2,797$)

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Notes: *p<.05; (a) Reference Group: College Degree Obtained; (b) Reference Group: Healthy Weight; (c) Reference Group: Whites.
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<td>0.37</td>
</tr>
<tr>
<td>Overweight</td>
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<td>0.19</td>
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<tr>
<td>Obese</td>
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<td>0.17</td>
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<tr>
<td>Prenatal Care</td>
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<tr>
<td>Health Insurance</td>
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<td>0.18</td>
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<tr>
<td><strong>Reproductive Index</strong></td>
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<tr>
<td>Accelerated Maternal Reproductive Index</td>
<td>1.00</td>
<td>0.08</td>
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Notes: *p<.05; (a) Reference Group: College Degree Obtained; (b) Reference Group: Healthy Weight; (c) Reference Group: Whites.
CHAPTER 3. THE INFLUENCE OF AN ACCELERATED MATERNAL REPRODUCTIVE INDEX AND INFANT BIRTH OUTCOMES ON SUBSEQUENT CHILD HEALTH OUTCOMES

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Abstract

Evolutionary theories of human health, development, and disease provide innovative and holistic perspectives on modern health. Additionally, scholars have found a relationship between a mother’s reproductive developmental index (timing of menarche, sexual debut, and first childbirth) and her offspring’s health. In this study, life history theory was used to examine the effect of an accelerated maternal reproductive index (AMRI) on her offspring’s health and development in light of numerous individual, familial, and socioeconomic influences. Using data from the *National Longitudinal Study of Adolescent Health* (Add Health), multiple linear regressions examined, in a step-wise fashion, the influences of lifetime maternal characteristics, offspring birth outcomes (low birth weight and premature birth), and offspring age on the number of health conditions (including physical, mental, and developmental conditions) the offspring had in childhood. Results show that an AMRI predicts the number of health conditions in

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childhood even after controlling for low birth weight and prematurity at birth, but an AMRI is not significant when the child’s age was included. However, post-hoc analyses revealed that an AMRI was significant when the offspring were four years or younger. Potential reasons for these findings as well as broader implications of this study are discussed.
Introduction

A call for utilization of evolutionary theories as applied to medicine and public health has been made (Gluckman et al. 2011; Belsky 2010) as evolutionary theories of human health, development, and disease can provide an innovative and holistic perspective on modern human health. Specifically, an evolutionary perspective calls attention to the ways in which humans respond to their environments, and provides a holistic framework for understanding the intergenerational cycle of health. An evolutionary perspective not only provides an explanation for proximate causes of diseases and conditions, but also an ultimate explanation for how and why these diseases or conditions emerge and persist throughout the life course and over generations. While many scholars have focused on individual health over the life span, few have focused on why the health and development of the parent influences infant and child health and development. But if public health issues such as the rise in childhood obesity, diabetes, and asthma and allergies, are to be remedied, a holistic, intergenerational perspective is needed. If researchers can pinpoint and understand the mechanisms through which poor health is transmitted to future generations, policy makers and intervention/prevention programs will be able to improve prevention and intervention programs.

One way in which poor health is transmitted from one generation to the next is from mother to child. Indeed, maternal characteristics and life events heavily influence the health and well-being of her children before they are even born. For example, early menarche and early age at first childbirth predicts a low birth weight (LBW) offspring (Basso et al. 2010; Kirchengast and Hartmann 2000) and preterm birth (Jolly et al. 2000; Fraser et al. 1995; Chen et al. 2007). In terms of weight status, scholars have found that
underweight women are more likely to have a LBW infant and preterm birth (Goldenberg et al. 2008; Hendler et al. 2005; Doherty et al. 2006; Annie E. Casey Foundation 2009). Overweight and obese mothers are more likely to give birth to a high birth weight infant and to have a preterm birth (Baeten et al. 2001).

Thus, this study utilized life history theory (an evolutionary theory used to explain growth, maintenance, and reproduction; Stearns 1992) to explore the intergenerational pathways through which patterns of growth, development, and health are transmitted from mother to child. Specifically, this study explored the impact of an accelerated maternal reproductive development (AMRI; experiencing early menarche, and/or early age at sexual debut, and/or early age at first childbirth; Belsky, Schlomer, and Ellis 2011; Quinlan 2003) on the health and well-being of the mother’s first child as measured by the prevalence of health conditions experienced by the child. The longitudinal nature of the analyses in this study present special issues with terminology; thus, each generation must be clearly defined and labeled. For the duration of the paper, the “target” is the mother, for whom information exists from infancy to adulthood. The target reported on the health of her first-born child who was still living at the time of the survey; this child is referred to as the “offspring.”

Life History Theory

The exploration of the effects of an AMRI on offspring health will be guided by life history theory. In general, this theory is used to explain the ways in which an organism allocates finite metabolic energy for growth, maintenance, or reproduction in order to maximize reproductive success (Ellis 2004). Since resources (e.g., food, energy, mates) are limited, organisms have to make energetic trade-offs to balance growth,
maintenance, or reproduction, with the goal of reproducing viable offspring. Life history theory suggests that humans have evolved flexible developmental patterns (i.e. variable timing of menarche) that vary based on their environment so as to maximize the ability to reproduce. Indeed, humans have evolved a reproductive strategy that favors quality over quantity; humans have few offspring but these offspring require significant parental investment (Gluckman et al. 2011; Gluckman and Hanson 2006).

Life history theory suggests that, when the environment is not optimal, natural selection favors earlier maturation and reproductive development over later maturation and reproductive development to ensure that genes are passed on, in light of the potential for an early death (Ellis 2004). That is, an adverse environment (e.g., father absence, stress) will signal to the human that his/her chances of mortality are high, so early maturation and reproduction may be the only way to pass on genes before death. An earlier maturation should confer a longer reproductive lifespan, allowing the parent more chances to have offspring that will survive to reproductive maturity. But a fundamental question that needs to be answered is whether or not an accelerated reproductive index is beneficial for humans. A stepping stone toward answering this question, this study explored the impact of an AMRI on the health and well-being of offspring in childhood.

**Accelerated Maternal Reproductive Index**

The maternal reproductive index refers to the timing of three correlated reproductive developments and life events: timing of menarche, timing of sexual debut (first vaginal/penile intercourse), and timing of first childbirth (Quinlan 2003). Women who experience one or all of these events earlier than the average woman have an accelerated reproductive index (Belsky, Schlomer, and Ellis 2011; Quinlan 2003). While
others have explored the singular effects of early menarche, early sexual debut, and early age at first childbirth on the health and well-being of offspring (see Kirchengast and Hartmann 2000; Basso et al. 2010; Jolly et al. 2000; Lenders, McElrath, and Scholl 2000; Jokela et al. 2007), none have, to our knowledge, examined the effect of a cumulative AMRI on the health and well-being of offspring into childhood.

The reproductive index begins with menarcheal timing, which has been shown to influence the timing of the other two variables: sexual debut and first childbirth. Undeniably, age at menarche has decreased over the past few generations. Between the 1850s and 1950s, average menarcheal age declined from 17 years to 12 years in the U.S. (Parent et al., 2003); similarly, the average age of menarche dropped from 14 years in 1900 (Chumlea et al. 2003) to 12 years in 2005 (Pinyerd and Zipf 2005). Girls now begin puberty, on average, between nine and 10 years of age in the United States (Herman-Giddens 2006).

An early age of menarche may often contribute to girls experiencing sexual debut and first childbirth at an earlier age. Girls who reach menarche early are more likely to experience early sexual debut (defined as first sexual intercourse before age 16; Draper and Harpending 1982; Neberich et al. 2010; Downing and Bellis 2009; Edgardh 2000; Chodick et al. 2005; Watson, Taft, and Lee 2007). Furthermore, girls who reach menarche early and/or sexual debut early are more likely to have their first child at an earlier age (Chisholm et al. 2005; Udry 1979).
Connecting the Maternal Reproductive Index to Infant and Child Health

The exploration of the relationship between an AMRI and childhood health outcomes (in terms of prevalence of health conditions) is fairly new; thus, little research exists. But the relationship between a mother’s reproductive development and her offspring’s health in childhood can be understood when infant health is taken into account. Due to fetal programming, adverse environments in utero and in early infancy permanently alter physical health and development that lead to chronic diseases later in life (De Boo and Harding 2006). Specifically, LBW infants are “programmed” to survive in a stressful environment, such that they will compensate for restricted growth in utero by rapidly accelerating growth in child- and adulthood. This compensation leads to changes in metabolism and overall physiology, contributing to adult diseases, such as hypertension, diabetes, and heart disease (Goldenberg and Culhane 2007).

Compensatory catch-up growth can also negatively affect children in terms of childhood obesity. Although mothers who had an early menarche tend to give birth to lower birth weight babies, Basso et al. (2010) found that children whose mothers had early menarche had a faster growth index than children whose mothers did not have early menarche; the children were taller and had a higher body mass index (BMI) at ages seven and eight. It is through this sequence of events (maternal early menarche, LBW, and compensatory catch-up growth) that an intergenerational growth pattern of accelerated maturity and childhood and adult obesity, hypertension, and cardiovascular disease occurs (Ong et al. 2007; Sloboda et al. 2007; Basso et al. 2010). Indeed, in the U.S., approximately one in every three children are overweight or obese (Anderson and Butcher 2006). Since 1976, childhood obesity has increased from about 5-6 percent to
18-19 percent in 2008 (Ogden and Carroll 2010). In addition, diabetes is now one of the most prevalent chronic diseases among children and adolescents; as of 2011, about 151,000 people below age 20 had diabetes, with approximately 13,000 youths diagnosed with Type 1 diabetes every year (Centers for Disease Control 2011a).

But metabolic conditions are not the only consequence of LBW/preterm birth. Among other consequences, LBW and/or preterm babies are more likely to experience intellectual disabilities, cerebral palsy, breathing and respiratory problems, and vision and hearing loss (Centers for Disease Control 2011b). In the year 2010 alone, 5 million children (ages 3-17 years) had a documented learning disability/Attention Deficit Hyperactive Disorder (ADHD; Bloom, Cohen, and Freeman 2011). According to the Centers for Disease Control (CDC), one in 303 eight-year-old children in the U.S. have cerebral palsy (Centers for Disease Control 2011c).

In addition, children in the U.S. today are facing a myriad of other health problems, including asthma and allergies, cancer, epilepsy, and chronic joint problems/arthritis. Over 10 million children have been diagnosed with asthma (Bloom, Cohen, and Freeman 2011). Of children aged 17 and under, 10 percent have suffered from hay fever, 12 percent from respiratory allergies, 5 percent from food allergies, and 13 percent from skin allergies (Bloom, Cohen, and Freeman 2011). As of 2007, approximately 10,400 children in the U.S. under 15 years of age were diagnosed with cancer; cancer is the leading cause of death by disease for U.S. children between one and 14 years of age (National Cancer Institute 2008). About 300,000 U.S. children under the age of 14 suffer from epilepsy (Epilepsy Foundation 2010) and children under age two are at the most risk for developing epilepsy (Centers for Disease Control 2011d). As for
chronic joint problems/arthritis, a 2007 CDC study estimated that approximately one in 250 children in the U.S. have been diagnosed with arthritis or another rheumatologic condition (Centers for Disease Control 2009).

**Individual, Socioeconomic, and Environmental Influences on Reproductive Development and Health Outcomes**

Life history theory suggests that humans are impacted not only by individual characteristics but also the social and physical environments. In addition, the theory suggests that environments and events early in life impact development and well-being in later life. Researchers have identified a plethora of individual, social, and environmental influences on reproductive development and offspring health outcomes; thus, they were considered in this study and are briefly delineated here.

*Individual influences.* Health disparities along ethnic lines are prevalent in the U.S., with minorities experiencing the highest burden of illness, disease, and death (Centers for Disease Control 2009). Since acquisition of appropriate health care is often bound by race, socioeconomic status, and gender, optimal health is more difficult for minorities to obtain (Centers for Disease Control 2007). Non-Hispanic Black children are more likely to have asthma, and White children are more likely to suffer from hay fever (Bloom, Cohen, and Freeman 2011). Bloom et al. (2011) found that both Black and White children were nearly equally likely to have a learning disability, while Hispanic children were least likely to develop a learning disability or ADHD. In addition, African American and Hispanic girls in the U.S. tend to reach menarche earlier than White girls do (Wu, Mendola, and Buck 2002; Anderson and Must 2005; Cavanagh 2004; McDowell, Brody, and Hughes 2007). African Americans tend to reach sexual debut
earlier than any other ethnic group in the U.S. (Cavazos-Rehg et al. 2009; Blum et al. 2000).

Girls who experience childhood obesity are more likely to start puberty (and menarche) earlier (Rosenfield, Lipton, and Drum 2009; Adair and Gordon-Larsen 2001; Anderson and Must 2005; Currie et al. 2012). BMI also influences timing of sexual activity; more obese or overweight adolescents (especially girls) are less likely to engage in early sexual behavior (Cheng and Landale 2011). Adolescent weight status is also an indicator of adult weight status; an obese adolescent is likely to be an obese adult (Guo et al. 2002). Indeed, overweight and obese children are more likely to have a mother with a high BMI (Lohman et al. 2009).

**Socioeconomic influences.** Parent income and education level influences reproductive development. For example, father education level covaries with menarcheal timing, sexual debut age, and age at first pregnancy, while mother’s education covaries with sexual debut age and age at first pregnancy, such that those whose parents with less education experience these events earlier (Quinlan 2003). Blum et al. (2000) found that children whose parents made more money were less likely to engage in sexual intercourse at a young age.

Adolescents who reach sexual debut early and “on time” were less likely to participate in postsecondary education than those who reached sexual debut late (Spriggs and Halpern 2008). Parkes et al. (2010) found that those girls who had an early sexual debut were less likely to participate in postsecondary education. Child health outcomes can also vary depending on parent income and education level: Children in poor families are more likely to have asthma, learning disabilities, or ADHD (Bloom et al. 2011).
Access to health insurance in adolescence and adulthood were included in the study. Health disparities exist between children who have health insurance and those who do not. As of 2007, nearly 9 million children in the U.S. did not have health insurance (Cuttler and Kenney 2007). Among other conditions, poor children, who are less likely to have health insurance, are more likely to have ADHD, asthma, and be obese (Currie 2009). In addition, receipt of prenatal care was included, despite the current debate regarding the effectiveness of prenatal care in improving birth outcomes (see Lantos and Lauderdale 2011; Fiscella 1995; Walford et al. 2011; MacDorman and Singh 1998; Hodnett, 2008).

Environmental influences. In terms of reproductive development, girls whose fathers are absent from the home during childhood are more likely to reach menarche early (Draper and Harpending, 1982; Belsky, Steinberg, and Draper, 1991; Ellis et al. 1999; Hoier, 2003; Maestripieri et al. 2004; Matchock and Susman 2006; Bogaert 2005). Girls whose fathers are absent from the home during childhood are more likely to experience sexual debut early and to have their first child at an early age (Chisholm et al. 2005; Quinlan 2003). According to life history theory, an absent father from the home may signal to the child that he/she lives in an unstable environment and that the future will be unstable too, thus prompting accelerated reproductive maturation.

Hypotheses

The primary aim of this paper was to be the first to assess the effect of an AMRI on offspring health into childhood in light of individual, socioeconomic, and environmental influences (see Figure 1). Thus, we hypothesized that:
Hypothesis 1: An AMRI would predict more incidences of health conditions for offspring in childhood.

Hypothesis 2: An AMRI would predict more incidences of health conditions for offspring in childhood even after controlling for LBW or preterm birth.

Hypothesis 3: An AMRI would predict more incidences of health conditions for offspring in childhood even after controlling for LBW or preterm birth and offspring’s age.

Methods

Sample

Restricted data from the National Longitudinal Study of Adolescent Health (Add Health), a nationally representative panel study of adolescents initially drawn from a random cluster sample of public, private, and parochial high schools (and their feeder schools) in the United States was used for this study. The first wave of the study (1994-1995) gathered information from students in grades 7-12, with a second wave following one year later. A third wave of the study took place six years later (2001-2002) when most respondents were ages 18-26, and a fourth wave of the study was completed in 2008, when the sample was between the ages of 24-34 (Harris et al. 2009). Data from the in-home interviews were collected via an interviewer-assisted survey and laptop computer. In Wave I, the adolescent’s caregiver participated in the study and in Waves III and IV, the targets answered questions regarding their offspring. For the purposes of this study, only target females who participated in, at minimum, Waves I and IV, whose caregiver participated in Wave I, and who reported on their first-born child’s health
conditions were utilized for analyses, yielding a sample size of 2,396. A description of the sample is provided in Table 1.

**Dependent Measures**

*Offspring health conditions in childhood.* In Wave IV, the target was asked, “Which of the following children’s health problems has a doctor told you that [your child] has?” The health problems were: hearing problems/deafness; delayed speech/problems speaking; a problem with sight even when wearing glasses; a developmental delay/ slowness in learning; allergies/hay fever; asthma; any other chronic lung/breathing condition; epilepsy/convulsions; chronic orthopedic, bone, or joint problems; cerebral palsy; cancer; obesity; diabetes; ADHD. The target reported 1 = yes or 0 = no for each health condition. This was transformed into a composite variable, wherein the number of conditions participants reported was summed. A higher score suggested more health conditions of the offspring. The number of health conditions was non-normally distributed, with skewness of 2.70 (SE = .05) and kurtosis of 10.63 (SE = .10). To reduce skewness, this variable was top-coded at two or more health conditions, such that the range of this variable was from zero, no health conditions to two or more health conditions.

**Independent Measures**

*Accelerated maternal reproductive index (target).* The following three variables were used to create the maternal reproductive index variable: early menarche; early sexual debut; early age at first childbirth. Each variable was dichotomized where 1 = early and 0 = not early and then summed to create a composite variable wherein a higher
value represented a more accelerated reproductive index. A description of each dichotomous variable follows.

*Early menarcheal age (target).* In Waves I and II, targets were asked, “How old were you when you had your very first menstrual period?” Wave II age was used for this measure, but Wave I age was used when Wave II age was not given. This variable was dichotomized where 1 = reached menarche at age 11 years or younger and 0 = reached menarche at age 12 or later, as consistent with previous methods (Ge et al. 2007; Watson, Taft, and Lee 2007; Ge et al. 2001; Cavanagh 2004; Foster et al. 2008; Manlove, Ryan, and Franzetta 2007).

*Early sexual debut age (target).* In Waves I and II, targets provided the year they first engaged in sexual intercourse; in Wave III, participants reported their age at first sexual intercourse. Sexual debut age in Waves I and II were calculated by subtracting the target’s birth year from the sexual debut year. Following Brückner and Bearman (2005), age of sexual debut was taken from Wave I if reported; from Wave II if no age at Wave I was reported; and Wave III if no age at Wave I or II was reported. This variable was dichotomized where 1 = reached sexual debut age 15 years or younger; and 0 = reached sexual debut age 16 years or older, as is consistent with previous studies (Neberich et al. 2010; Downing and Bellis 2009; Edgardh 2000; Chodick et al. 2005; Watson, Taft, and Lee 2007).

*Early age at first childbirth (target).* In Wave IV, the target answered questions about her first and, if applicable, subsequent births. The age of the first birth reported was subtracted from the target’s age, yielding mother’s age at first childbirth. This variable was dichotomized where 1 = had first child early (before age 21), or 0 = had first
child at 21 years or after (Boden, Fergusson, and Horwood 2008; Dupont and Page 1986; Chisholm et al. 2005).

*Infant (offspring) low birth weight.* In order to assess whether an infant had LBW, targets in Wave IV were asked, “Did [baby] weigh less than 5.5 pounds at birth?” They responded 1 = yes or 0 = no.

*Premature birth (offspring).* In Wave IV, targets reported whether their baby was born before, on, or after his/her due date. For those who reported their baby was born before the due date, the number of weeks early was recorded. The variable was coded as 1 = birth before 37 weeks and 0 = birth 37 weeks or later (Centers for Disease Control, 2012a).

**Control Variables**

*Low maternal (target) birth weight.* A parent questionnaire was completed by a caregiver of the target adolescent in Wave I, wherein the caregiver was asked to report the birth weight of the target in pounds and ounces. The variable was dichotomized where 1 = birth weight below 5.5 pounds and 0 = birth weight of 5.5 pounds or more (Centers for Disease Control 2013).

*Parent income during target adolescence.* In Wave I, the caregiver reported the total household income (in 1995 dollars). Following Blum et al.’s (2000) measure using Add Health data, income was rounded to the nearest thousand dollars. For those participants whose parents did not report income, the median value of the participant’s subgroup was provided (based on sex, race/ethnicity, marital status, welfare status, and education).
Parent education level during target childhood. In Wave I, the caregiver reported on the highest level of education he/she had attained. This was converted into four categories: less than high school diploma; high school diploma or GED; some postsecondary education; bachelor's degree or higher (Spriggs and Halpern 2008). Dummy variables were created for each of the categories where 1 = attained that education level and 0 = did not attain that education level; those who reported having a bachelor’s degree or higher comprised the referent group.

Target age. Age of the participant at Wave IV was included. Ages ranged between 24 and 32 years.

Target ethnicity. Race/ethnicity information was reported at Wave I. Targets were categorized as either Hispanic/Latino, White, Black or African-American, American Indian or Native American, and Asian, or Other (Add Health, n.d.), where 1 = part of that ethnic group and 0 = not part of that ethnic group. Those who identified as White comprised the referent group.

Father absence during target childhood. The caregiver reported, in Wave I, whether or not the adolescent’s father was living in the home or not. This variable was dichotomized where 1 = father was absent at any point during childhood (including those who never lived with the adolescent) and 0 = father was not absent during childhood.

Target access to health insurance in adolescence. In Wave I, the caregivers were asked, "What type of health insurance does [child] have?" Participants could specify what type of insurance they had, or could indicate they did not have any. This variable was dichotomized where 1 = had health insurance of some kind; or 0 = did not have health insurance.
Target weight status in adolescence. Body mass index (BMI) in Wave I was calculated using the adolescent's self-reported measurements of height and weight (kg/m$^2$). Up to age 20, BMI is age and gender-specific; thus, BMI percentages were used to categorize the targets into four weight categories based on the CDC's classification system: underweight (BMI <5$^{th}$ percentile); healthy weight (BMI 5$^{th}$ to 84$^{th}$ percentiles; overweight (BMI 85$^{th}$ to 94$^{th}$ percentile); and obese (BMI ≥ 95$^{th}$ percentile; Centers for Disease Control 2011). Dummy variables were created for each of the categories where 1 = part of that weight status and 0 = not part of that weight status; participants who were “healthy” comprised the referent group.

Target weight status in adulthood. In Wave IV, Add Health field interviewers collected weight and height measurements of the target; the Add Health team then created a BMI variable. Participants were categorized into the following four CDC BMI categories: underweight (<18.5); normal (18.5-24.9); overweight (25-29.9) and obese (30+; Centers for Disease Control 2011). Dummy variables were created for each of the categories where 1 = part of that weight status; or 0 = not part of that weight status; participants who were “healthy” comprised the referent group.

Target access to health insurance in adulthood. In Wave IV, participants were asked about their current health insurance situation. Participants reported either that they did not have health insurance, or the type of health insurance they did have (e.g., through work, school, Medicaid). This variable was dichotomized, where 1 = had health insurance of some kind; or 0 = did not have health insurance.

Target adult education level. Targets reported their highest education level they had achieved to date in Wave IV. This variable was categorized into four categories: less
than high school diploma; high school diploma or GED; some postsecondary education; Bachelor's degree or higher (Spriggs and Halpern 2008). Dummy variables were created for each of the categories where 1 = attained that education level and 0 = did not attain that education level; those who reported having a bachelor’s degree or higher comprised the referent group.

*Target visited a midwife/doctor for prenatal care.* In Wave IV, the targets were asked if they ever visited a doctor, nurse-midwife, or any other type of health care provider for prenatal check-ups. Participants answered 1 = yes or 0 = no.

*Offspring’s age.* The target reported her offspring’s age in years at Wave IV. The ages ranged from zero to 15 years old.

**Analytic Plan**

In order to explore the influence of maternal characteristics (most notably, the AMRI) and offspring characteristics on the number of health conditions the offspring had, multiple linear regressions were completed in SPSS 21.0. Dummy variables for ethnicity, education level, and weight status were included in the model in order to fully understand the differential impact that membership in each of the categories may have on the number of health conditions experienced by the offspring. In a stepwise fashion, maternal influences accumulated throughout her lifetime were regressed on the number of health conditions in the first model. Then, birth outcomes (LBW, premature birth) were included in Model 2. The age of the child (both as a continuous variable and a dichotomous variable to denote an age cut-off effect) was included in Models 3 and 4, respectively.
Results

Results of the multiple linear regressions are presented in Table 2. Significant findings for each regression are discussed in turn.

**Hypothesis 1: An AMRI predicts more incidences of health conditions for offspring in childhood**

The results of the first model revealed support for the first hypothesis, as an AMRI significantly predicted more health conditions experienced by her offspring ($\beta=.10, p<.001$). Other maternal characteristics also predicted a number of offspring health conditions. The older the target was, the more health conditions her offspring were likely to experience ($\beta=.06, p<.01$). Hispanic targets had less reported offspring health conditions than Whites ($\beta=-.06, p<.01$). Conversely, Native American targets had offspring with more health conditions than Whites ($\beta=.05, p<.05$). Those targets who, as adults, only had a high school degree ($\beta=.07, p<.01$) or some college education ($\beta=.08, p<.01$) had offspring with more health conditions than those targets who had a college degree. Those who received prenatal care during pregnancy had offspring with more health conditions ($\beta=.04, p<.05$; this seemingly counterintuitive finding is discussed later).

**Hypothesis 2: An AMRI would predict more incidences of health conditions for offspring in childhood even after controlling for offspring birth outcomes**

Results from the second model revealed the influential effects of the offspring’s birth outcomes in light of maternal characteristics, including the AMRI, on the number of offspring health conditions. An AMRI significantly predicted more offspring health conditions ($\beta=.10, p<.001$). The same maternal characteristics that were significant in
Model 1 were significant in Model 2 and thus are not explicated here. However, if the offspring had a LBW ($\beta=.08$, $p<.01$) or a premature birth ($\beta=.05$, $p<.05$), he/she had more health conditions in childhood than those who were not LBW or premature.

**Hypothesis 3: An AMRI would predict more incidences of health conditions for offspring in childhood even after controlling for offspring birth outcomes and offspring’s age**

When the offspring’s age was included in the third model, the predictive effect of the AMRI was no longer significant. The offspring’s age was significant in predicting the number of health conditions in childhood, wherein the older the offspring was, the more health conditions he/she was likely to experience ($\beta=.19$, $p<.001$). The significant influences of offspring birth outcomes, prenatal care, and Native American and Hispanic ethnicity were similarly significant as in the previous model. However, the target’s education level and age as an adult were no longer significant. Additionally, African Americans were less likely than Whites to have offspring with health conditions ($\beta=-.06$, $p<.05$), and those targets who were obese in adolescence were more likely to have offspring with health conditions compared to those who were healthy ($\beta=.05$, $p<.05$).

**Additional Analyses**

Because the offspring’s age was significant in predicting the number of health outcomes he/she experienced, thus nullifying the effect of an AMRI, we conducted a post-hoc analysis to explore how far into childhood an AMRI would influence the development of child health conditions. Thus, as displayed in the fourth model, we found that when controlling for the offspring’s age at four years and younger, an AMRI was still significant in predicting the number of offspring health conditions ($\beta=.05$, $p<.05$).
However, this effect disappeared when we controlled for the offspring’s age at five years and younger.

Discussion

Scholars have stated the need for utilizing evolutionary theories to examine modern human health. Guided by life history theory, this study was novel in that it examined the intergenerational influence of a mother’s reproductive development on the health of her offspring in childhood while simultaneously considering other individual, familial, and socioeconomic factors. According to the theory, those who have an AMRI would be more likely to have offspring with poor health. The results supported the theory, as an AMRI significantly predicted more health conditions for a target’s first child (up to age four).

Since an AMRI significantly predicts more health conditions only when children are age four or younger, this suggests a tipping point in regards to the duration of the effect of an AMRI on the offspring’s health. In other words, an offspring’s development and well-being is influenced by the timing and tempo of his/her mother’s reproductive development only up until age four, suggesting that other factors, potentially individual and/or environmental, become more influential for those children who are five years and older. Perhaps this tipping point reflects a change in the offspring’s world at age five; he/she will start kindergarten and will likely be required to receive a physical examination by a doctor, increasing the likelihood of a diagnosis of health conditions. Having to function in a classroom (i.e. sitting in a chair and reading the board) as well as exposure to teachers who may be more adept at detecting disabilities like ADHD, developmental delays, speech and sight problems, etc. may increase the likelihood a child
would be referred to see a doctor and be diagnosed with a health condition. However, further research is needed to confirm the reasons behind this tipping point.

One unpredicted finding was that prenatal care significantly predicted more health conditions for the offspring. The effectiveness of prenatal care on infant health is debated (as aforementioned) but its influence on health in childhood is unknown. However, we suggest that this finding may be due to the way in which the question about health conditions was asked: targets were asked if a doctor had told them their child had a certain health condition. Those who utilized prenatal care during pregnancy may be more oriented toward accessing medical care, and in turn more likely to take their children to the doctor, thus increasing the likelihood they would have reported health conditions in the survey. A second unpredicted finding was that Hispanic and African American mothers were less likely to have children with health conditions than White mothers. Given that health inequalities in the U.S. fall along ethnic and socioeconomic lines, African Americans and Hispanics are twice as likely to be uninsured (The White House n.d.) and thus may be less likely to take their children to the doctor, potentially decreasing the likelihood they would have reported health conditions in the survey.

**Conclusion**

This study provides support for the viability of using evolutionary theories to examine public health issues, as this study provides novel findings regarding the long-term, next-generation impact of the AMRI on offspring health. In addition, this study sheds light on the extent to which an AMRI impacts offspring health, contributing to our understanding of parental influences on child development and well-being. The results suggest that if the rise of debilitating health conditions in childhood is to be slowed or
reversed, one point of intervention may be with the mother before the child is even born.

Policies and programs that help to delay a mother’s reproductive development may contribute to positive, long-term outcomes for children.
Acknowledgements

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References


Centers for Disease Control. 2011. Healthy weight – it’s not a diet, it’s a lifestyle! About
BMI for children and teens. Accessed April 2014:
http://www.cdc.gov/healthyweight/assessing/bmi/childrens_bmi/about_childrens_bmi.html#How is BMI used with children and teens


http://www.cdc.gov/Features/PrematureBirth/


Centers for Disease Control. 2011d. Targeting Epilepsy. Accessed May 2012:
http://www.cdc.gov/chronicdisease/resources/publications/AAG/epilepsy.htm

http://www.cdc.gov/reproductivehealth/maternalinfanthealth/PretermBirth.htm

http://www.cdc.gov/nchs/fastats/birthwt.htm


http://www.whitehouse.gov/assets/documents/Pages_from_Health_Insurance_Reform_PDF-5.pdf


Figure 1. Conceptual Model

**Individual, Family, and Socioeconomic Factors**
- Child (Target)
  - Low birth weight
  - Ethnicity
  - Father absence
  - Parent income/education level
  - Weight status
  - Access to health insurance
- Adult (Target)
  - Income/education level
  - Weight status
  - Access to health insurance
  - Utilized prenatal care
  - Age

**Maternal Reproductive Trajectory**
- Early menarche
- Early sexual debut
- Early first childbirth

**Offspring Characteristics**
- Low birth weight
- Premature birth
- Age

**Child Health Conditions (Offspring Quality)**
- Hearing problems/deafness
- Delayed speech/problems speaking
- Problem with eyesight
- Developmental delay/slowness in learning
- Allergies/hay fever
- Asthma
- Chronic lung/breathing condition
- Epilepsy/convulsions
- Chronic orthopedic, bone, or joint problems
- Cerebral palsy
- Cancer
- Obesity
- Diabetes
- ADHD
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*Offspring Variables*

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