CHANGING CHILDHOOD HOUSEHOLD CONTEXTS AND INDIVIDUAL WELL-BEING IN MEXICO AND THE PHILIPPINES

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ABSTRACT

Kammi Schmeer: Changing Childhood Household Contexts and Individual Well-Being in Mexico and the Philippines
Under the direction of Barbara Entwisle

This dissertation studies the origins of social inequalities by analyzing the influence that childhood household contexts exert on individuals' health and education, two important measures of well-being and social status. The key relationships studied here are: (1) father absence due to migration and child illness in rural Mexico; (2) changes in sibsize during childhood and educational attainment in Cebu, Philippines; and, (3) household income effects on underweight and overweight status at the transition to adulthood in Cebu, Philippines. Using prospective, longitudinal data, I find that: (1) father absence due to migration increases child illness especially in the absence of social welfare programs; (2) changes in siblings living in the home tend to decrease educational attainment (although the effects depend on the stage of childhood when the change occurs); and, (3) childhood income affects individuals' weight status at age 19, with important differences across stages of childhood and by weight status. These findings, overall, suggest that childhood households, and changes in them over time, have important implications for the development of health and education disparities. Future research should pay more attention to measuring changes in household contexts (i.e., the movement of family members in and out, fluctuations in economic and other resources), and to identifying *when* social contexts matter most for individuals' well-being.

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CHAPTER 1: INTRODUCTION

Individuals' health and education are important aspects of their social status and life chances. Social structures during childhood begin to create disparities in health and education which can last a lifetime. Understanding inequalities in health and education at their origin during childhood, then, can be useful in beginning to understand the development of social inequalities over the life course.

My dissertation focuses on the role that childhood household contexts play in the development of inequalities in health and education. Household and family contexts are important for child outcomes because they are the main socializing environment for young children, buffering the effects of other more distal social contexts in children's lives (Bronfenbrenner 1989; Duncan, Boisjoly, and Harris 2001; Furstenberg, Cook, Eccles, Elder, and Sameroff 1999). The question of how childhood household contexts affect individuals' life chances has long been studied in status attainment literature under the rubric of "family background" (Blau 1992; Blau and Duncan 1967), with a particular emphasis on the development of inequalities in education (Blake 1989; Bond and Saunders 1999; Rumberger 1983). Recent sociological literature is also beginning to emphasize the importance of childhood contexts for health disparities(Foley 2000; Heaton, Forste, Hoffmann, and Flake 2005; Holland, Berney, Blane, Davey Smith, Gunnell, and S. M. Montgomery 2000; Makinen, Laaksonen, Lahelma, and Rahkonen 2006), as well as the importance of childhood

health for individuals' health and socioeconomic status later in the life course(Conley and Bennett 2000; Conley and Bennett 2001; Haas 2006; Palloni 2006).

Yet our understanding of how health and education inequalities develop is limited by our reliance on static theories and cross-sectional studies. This is reflected, for example, in the use of measures of family background in status attainment research, which often use household or family characteristics assessed late in adolescence to reflect one's access to resources during childhood (Warren, Sheridan, and Hauser 2002). Given the extent of change in families, households and individuals during childhood, dynamic, longitudinal perspectives are required to more fully understand how childhood household contexts influence social inequalities.

I build on current literature related to health and education inequalities by applying a life course perspective to my study of how household contexts during childhood affect individuals' life chances and social status. Life course is the study of individuals' lives and how they unfold over time and within changing social contexts (Elder 1985). Further, the life course concept of linked lives suggests that the lives of other household members are important for creating the household environment within which a child develops (Elder 1985). As such, there is no single household context. The social context of a household may change over time as a reflection of the life course development of its members. Children, then, may be exposed to multiple household contexts over time as dynamic characteristics of households, such as economic resources and residential status of family members, change as household members pass through different stage of the life course. A life course perspective, then, directs one to assess *change* in a child's household context, and to assess the impact of these changing contexts on the individuals' well being.

In conceptualizing households and their impact on individuals over time, a life course perspective also suggests that *timing* is important (Elder 1985). Timing refers to the stage in an individual's life course when they experience a particular social context or event. Household contexts at key developmental stages may be more important for health or education outcomes than household contexts at other times. For example, it may be that household contexts in early childhood are particularly important due to the cognitive and physical growth that happens during these stages, with the potential to influence subsequent development trajectories. It may be, however, that improved contexts and resources later in childhood can correct past deficiencies caused by early distress. In short, the impact of household contexts on individuals' health and education may depend on the stage of childhood when a given level of resources is experienced.

Research to date on social inequalities that has taken a life course approach provides evidence that *change* (Brown 2006; Cavanagh, Schiller, and Riegle-Crumb 2006; Macmillan, McMorris, and Kruttschnitt 2004; Strohschein 2005b) , and *time and timing* (Benzeval and Judge 2001; Chen, Matthews, and Boyce 2002; Currie and Stabile 2003; Duncan, Yeung, Brooks-Gunn, and Smith 1998; Guo 1998; McLeod and Shanahan 1996) need to be considered in our understanding of how childhood contexts affect individuals' life chances. My dissertation builds on this relatively recent and limited body of literature by investigating three stratification processes from a life course perspective are: (1) how changes in father residency status affect illness during childhood (Chapter 2); (2) how changes in sibsize during childhood affect individuals' educational attainment (Chapter 3); and, (3) how household income during childhood affects weight status (underweight and overweight) in

the transition to adulthood (Chapter 4). The first topic is explored in the context of rural Mexico, and the subsequent two studies are based in the Philippines.

In addition to offering new insights into the development of social inequalities that cannot be gleaned from static theories and cross-sectional models, my dissertation contributes to sociological research by applying life course and stratification models to developing country settings. I chose to study these processes in developing country contexts for several reasons. First, much of the stratification and life course research has been conducted in the U.S. and other developed country settings. Applying theories and empirical tests in *developing* country settings allows us to test theoretical predictions we have made based on *developed* country research. This increases our ability to generalize about stratification processes cross-culturally, and to consider the potential impact of the larger social context in determining how family and household contexts affect children's lives. It also provides researchers and policy makers with a better understanding of the development of social inequalities in developing countries, where research on the dynamics of families, households and social inequalities is limited.

Second, developing countries often provide the most relevant settings in which to study social inequalities. The rapid socioeconomic and cultural changes taking place in developing countries provide a context of differences both across households and over time that make it possible to consider the dynamics of household-level stratification processes. For example, the impact of father absence due to migration, an under-studied aspect of change in family structure, can best be studied in developing countries (like Mexico) where divorce rates are low and migration rates high. Developing countries are also particularly relevant for investigating specific types of family/household change, such as change in number of

residential siblings, since larger ranges in family size in developing countries provide a broader, more dynamic view of how changes in number of residential siblings can affect children's lives over time. The Philippines, like many developing countries, is also in the midst of the nutrition transition, when social, economic and cultural change produce a dual burden of under and over nutrition. In this setting it is possible to investigate the role of household contexts on both traditional (i.e., underweight) and modern (i.e., overweight) health problems at the societal level. In short, developing countries often provide the setting where interesting questions that are left unaddressed by developed country research can be posed and answered.

The third, and possibly most important, reason to study health and education inequalities in developing country contexts is that the low levels of health and human capital attained during childhood are serious social problems in these settings, affecting both individual lives and broader societal trends. Thus, my research informs not only sociological literature, but also our understanding of potential avenues through which policy makers can improve the lives of some of the most vulnerable individuals in the world.

In sum, this dissertation provides life course and developing country perspectives on the relationships between key and dynamic household resources (fathers, siblings, and income) during childhood, and how changes in these resources over time can affect individuals' health and education. I now turn to a brief description of each of the three studies (Chapters 2-4) included in this dissertation. I save the specific results of the studies to be read in the subsequent chapters, and to be reviewed in the conclusion section (Chapter 5) of this dissertation.

Summary of Three Studies

My first study (Chapter 2: Father Absence, Social Welfare, and Child Illness in Rural Mexico) provides a life course approach to studying health inequalities in childhood by emphasizing the importance of *change* in family/household contexts for individual well-being. In this study I am interested in change that occurs in family and household structure due to paternal migration, and how this affects the health of children 0-5 years old. Literature on absent fathers due to divorce or non-marital childbearing suggests potentially serious negative consequences of father absence for child well-being due to the loss of social and economic support. However, the migration literature theorizes potentially positive effects when fathers migrate, especially if they are able to find work and send money home. Given the conflicting predictions in the literature, my overall aim is to assess whether the average effect of paternal migration is positive or negative. I am also interested in how social safety nets might protect children from potential negative effects of father absence. I hypothesize that a negative effect of paternal migration on child health may be mitigated by the household receiving social welfare payments during his absence.

The data I utilize for this paper are from a 1997 longitudinal study of individuals in all households in 506 randomly-selected, poor, rural communities in Mexico. The purpose of the survey was to determine which households in these communities would qualify for a state-funded welfare program called PROGRESA. The PROGRESA program included a monthly cash transfer worth, on average, 20% of poor households' income to mothers, and some targeted preventive health services. The data I use come from the baseline and follow up PROGRESA surveys, and include information on health status, household resources, and parental residency assessed at 6 month intervals from October 1998-November 1999. Because PROGRESA was implemented following an experimental design, the selection of

households was not correlated with father absence, providing a unique opportunity to the study how the relationship between father absence and child illness may differ by whether the household is enrolled in this important welfare program.

In my statistical analyses I use logistic regression to test the average effects of father absence on child illness (reported as ill in the last 4 weeks). To assess the PROGRESA effects, I introduce interactions between father absence and PROGRESA receipt into these models. My results inform both family structure and migration literature, and provide insight into how household context may interact with social welfare in affecting child well-being. This study also supports the broader life course idea that changes in social contexts, in this case family structure, during childhood have important implications for individuals' well-being.

My second study (Chapter 3: Changing Sibsize and Educational Attainment) focuses on the lasting impact of childhood family structure dynamics for individuals' educational attainment. Specifically, I study the relationship between changes in the number of co-resident siblings (henceforth referred to as "sibsize") and education over time during childhood. I develop hypotheses based on resource dilution theory, the idea that individuals do worse when they have more siblings to compete with for resources. However, I provide a more dynamic, developmental view of the theory by suggesting that both changes in sibsize and timing of these changes may have serious implications for individuals' educational attainment. Changes in sibsize may affect individuals' education through changes in resource allocation and social organization that come with the addition or loss of a sibling. In terms of timing, most child development literature predicts stronger effects of family context on outcomes during early childhood due to the importance of cognitive development that occurs

at young ages. However, since schooling becomes more expensive and selective over time, competition for resources among siblings may be particularly detrimental to education in later stages of childhood. It is not clear a priori when changes in sibsize may matter the most for individuals' educational attainment.

I differentiate the processes of change by age of the siblings (younger vs older) and the type of change (adding vs losing siblings) to better capture the types of changes that occur in a child's household. Furthermore, consistent with a life course perspective, I investigate whether changes in sibsize differ by stage of childhood. The three main research questions posed by this study are: (1) How do changes in residential sibsize affect individuals' educational attainment; (2) Do these effects differ by stage of childhood when the changes occur; and (3) Do these effects differ by younger versus older siblings? I evaluate these hypotheses by assessing, prospectively, the effects of changing sibsize across various stages of childhood, including birth-age 2, age 2-9, age 9-12, and age 12-16, and their effects on individuals' education at age 19.

This study uses data from the Cebu Longitudinal Health and Nutrition Survey, which provides prospective data on residential sibsize during of childhood for a cohort of children born in 1983. Because the Philippines has higher fertility than most developed countries, it provides larger variation in family size due to births compared with the U.S. At the same time, as is common across cultures, Filipino children move out of parental homes to pursue education, to marry, for work, or for other reasons. Thus, over the 18 years of childhood, most children in the Philippines will have experienced both additions and reductions in sibsize. How these changes in family structure affect individuals' ability to pursue their own

education is an important question, with consequences for individuals' social status as they move into adulthood.

The findings from this study highlight the importance of changes in family structure, and particularly certain types of changes in sibsize. The results further reinforce the importance of considering timing in assessing the effects of family structure changes on individuals' education; and, that studies controlling for child age or utilizing cross sectional data may be missing potentially important information on how family and household contexts contribute to the development of social inequalities.

My final study (Chapter 4: Childhood Income and Weight Disparities in the Transition to Adulthood) considers how household income during childhood affects weight status as individuals transition to adulthood. Being underweight (a more traditional nutrition problem in poor countries) and being overweight (a relatively modern nutrition problem emerging in developing countries) are health problems that are associated with lower quality of life (Sach, Barton, Doherty, Muir, Jenkinson, and Avery 2007), decreases in economic productivity (Tunceli, Li, and Williams 2006), work place discrimination (Carr and Friedman 2005), and increases in the risk of illness and earlier mortality (Flegal, Graubard, Williamson, and Gail 2005; Katzmarzyk, Craig, and Bouchard 2001; Khongsdier 2002).

In this study I review theoretical and empirical literature that suggests the importance of childhood income for adult health, and that childhood income may affect individuals' health differently depending on when it was experienced and the nature of the health outcome. I pose the following research questions aimed to move forward our understanding of the development of health disparities: (1) Are there lasting effects of childhood household income on weight status as individuals transition to adulthood (at average age 19)? (2) If so,

does income affect the probability of being underweight and overweight equally? (3) Does income at different periods in childhood affect weight status at age 19 differently? (4) Are the patterns of timing effects similar for underweight and overweight outcomes?

This study is also set in the Philippines, which provides a developing country setting and opportunity to investigate weight outcomes in the context of the dual nutrition burden (continuing underweight and rapidly growing overweight populations) imposed by the nutrition transition. My data again come from the Cebu Longitudinal Health and Nutrition Survey, providing me with prospective, longitudinal information on household income at multiple stages of childhood. My outcomes, underweight and overweight, were assessed objectively by trained personnel for individuals in 2002, when the index children reached age 19 (on average).

I use logistic regression models to assess how income from birth to age 16 affects individuals' weight status (underweight and overweight) at age 19. I model both average childhood household income and income at specific stages of childhood (birth, age 2, age 9, age 12, and age 16) to investigate both average and timing effects. The results inform the growing body of health disparities literature by suggesting the lasting impact of childhood income on adult health; and, different effects by when income is experienced and which health outcome is considered.

The three studies described above together provide the substance of my dissertation. While I build on somewhat distinct bodies of literature in each, all three bring together stratification and life course research to provide dynamic view of the origins of social inequalities. I provide a conclusion section in Chapter 5 that highlights the multiple lessons learned across the three studies. In doing so, I suggest ways in which this dissertation informs

our current understanding of the development of social inequalities, as well as possible areas for future research.

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CHAPTER 2: FATHER ABSENCE, SOCIAL WELFARE, AND CHILD ILLNESS IN RURAL MEXICO

Introduction

Child illness remains a serious social problem that reduces societal and individual well-being around the world. In developing countries, common illnesses, such as diarrhea and respiratory infections, coupled with malnutrition lead to roughly 40% of deaths of children under 5 (Black, Morris, and Bryce 2003). Even when not leading to death, illness constrains play, learning, and social interactions, as well as parental work time. Repeated and/or severe child illness can affect children's cognitive skills (Caughy 1996), physical development, and susceptibility to disease (Martorell and Ho 1984), setting individuals up for a lifetime of disadvantage (Case, Fertig, and Paxson 2005).

Given the importance of child health to individuals, their families and society as a whole, a main challenge for social scientists and policy makers is to better understand the causes of child illness, especially among the poor. Social contexts, and particularly household and family contexts, have been shown to be closely tied to child well-being (Duncan, Boisjoly, and Harris 2001). Household and family contexts may be particularly important for the healthy development of young children, because children spend much of their daily life in the household/family context. Furthermore, parents and other family and household members affect how school, community or government resources (or the lack of them) translate into child health. One key to understanding, and promoting good health

during childhood, then, is to better understand how household and family contexts affect child illness.

This paper addresses a little studied aspect of children's household and family environment—father absence due to migration—and its implications for illness among young children. Family structure research highlights the importance of fathers to children's lives. Children in two-parent families do better on number of outcomes than those in one-parent (usually mother-only) or step families (Sigle-Rushton and McLanahan 2004). Further, recent research highlights the potential detriments of changes in family structure (parental marital status) for child well-being(Cavanagh and Huston 2006). However, family structure research tends to focus on father absence due to non-marital childbearing, divorce or death. In many settings around the world, fathers are absent for a different reason: they move out of their household and community of origin, leaving their children behind, to pursue economic or educational opportunities. This type of change in family structure has not been studied in the family structure literature to date.

Father absence due to migration is a part of the migration literature. Unlike the negative effect anticipated in family structure research, however, migration theory and empirical evidence suggests that migration of family members may improve household economic and social well-being, with the potential to improve child health (Frank and Hummer 2002). A limit to this research has been the focus on migrants or migration by any household member rather than on fathers per say.

Neither the family structure nor the migration literatures provide a clear picture of how father absence due to migration affects children. In fact, these two bodies of literature illustrate the contradictory and complex nature of father absence due to migration and its

potential to affect child health and illness. The scarcity of research on the topic to date motivates this empirical study of how father absence due to migration may affect illness among young children in rural Mexico. Given the importance of physical health to children's quality of life and future potential, and the serious consequences of illness for children living in extreme poverty, it is critical that we better understand how fathers, and particularly their absence, may affect child health in this setting.

A second contribution of this paper is to investigate the role that social welfare plays in buffering children from negative consequences of changes in family structure. Social welfare programs are instituted by governments around the world to assist children in poverty, and evaluations of programs in the U.S. and Mexico generally show success in terms of improving children's health and well-being (Frongillo, Jyoti, and Jones 2006; Gertler 2004). However, little research has been done on how social welfare, in any setting, may condition the relationship between family structure and child well-being. In this paper, I consider the possible interaction between social welfare and father absence on child illness to assess whether a social welfare program can mitigate the potential negative effects of fathers absence due to migration on child health.

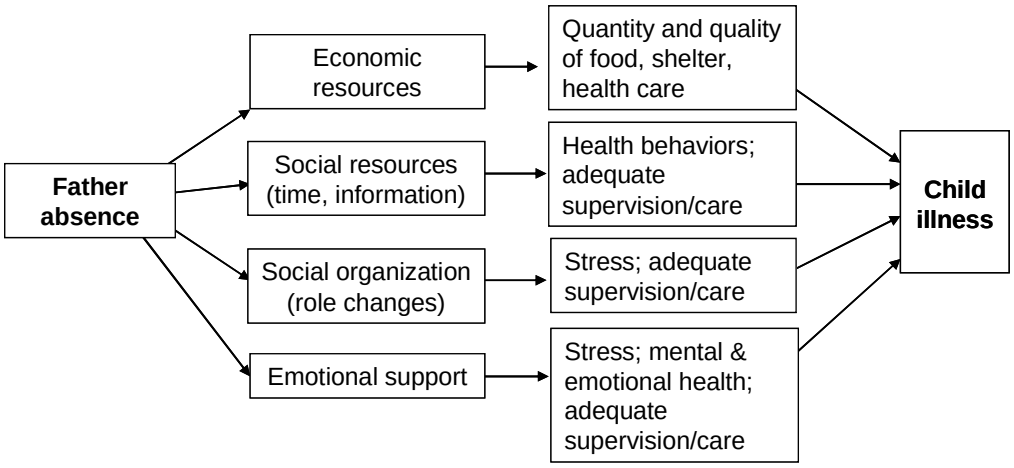
The research questions to be answered in this study are: (1) How does father absence due to migration affect child illness? (2) How does a household's enrollment in a social welfare program affect the relationship between father absence due to migration and child illness? I answer these questions using a sample of children living in households in poor, rural communities in Mexico. The households in this study, like many other poor households around the world, face a complex set of challenges to ensuring their children's healthy development. This study addresses one of these challenges: the temporary loss of a key

household and family member. In doing so, I highlight the importance of dynamics in family structure in understanding child illness. Further, the evidence presented here suggests that within the context of poor communities, social welfare programs may be critical for protecting children's health when fathers leave.

Theoretical Background

Research on family structure suggests that residential fathers are important members of children's households, and their absence may have serious consequences for the health of young children who are dependent on their household and its members for their well-being (Sigle-Rushton and McLanahan 2004). As Figure 2.0 shows, fathers' absence may affect their children's health through several mechanisms. First, fathers are often main economic earners and source for purchasing health-promoting goods and services. Paternal income may be critical in providing young children with adequate food, housing, and health care needed to prevent and treat illnesses. Research shows that paternal employment increases child well-being, indicating an important economic role for fathers (Landale and Oropesa 2001). When fathers do leave, substantial losses of income often result (Page and Stevens 2004), and that economic loss is one of the main pathways through which fathers absence affects child well-being (Clarke-Stewart, Vandell, McCartney, Owen, and Booth 2000; Hango and Houseknfcht 2005). The economic pathway seems particularly relevant for cognitive (and perhaps physical health) compared with emotional outcomes (Ram and Hou 2003). Further, the payment of child support has been linked to improved child outcomes, suggesting the importance of fathers' economic role even after they leave the household (Amato and Gilbreth 1999; Argys, Peters, Brooks-Gunn, and Smith 1998).

Figure 2.0: Theoretical links between father absence and child illness



Second, residential fathers provide households with social resources (mainly, knowledge and time), which are also important to child health. Fathers' knowledge may improve health behaviors and care of children, while their time may allow for more parental supervision and care giving of young children. Empirical evidence suggests that the presence of fathers improves child health even when controlling for household economic resources (Gage 1997; Gertler, Martinez, Levine, and Bertozzi 2003), and that father involvement with their children is a key mediator of father absence and child well-being (Carlson 2006). Further, fathers have been found to be important sources of advice and assistance for women managing child illness (Carter 2004). This research suggests that father absence may decrease key social resources needed for children's healthy development.

Third, fathers are a part of the household's social organization; that is, they have designated roles and responsibilities. Fathers leaving the household may leave these roles unattended, and other household members may have to pick up these additional duties (e.g., mothers or others may have to take on additional work or child care responsibilities). These changes in social organization may induce stress and reduce proper supervision and care of

children (Albrecht and Teachman 2003). Increased disorganization may be particularly evident in the short term before the household has had time to adjust to the change. Furthermore, the transitions and instability in home life experienced by children as their fathers come into and go out of their lives periodically may cause prolonged periods of stress (Albrecht and Teachman 2003). Empirical research is limited in this area, but recent work suggests that increased levels of caregiver stress increase with new roles and these higher levels of stress are linked to problem behaviors in children (Oburu 2005).

Finally, fathers provide emotional support to children and the children's mothers, which may be important in overall health of children. Poor parental mental and emotional health can decrease children's well-being (Conger and Donnellan 2007), potentially increasing their susceptibility to disease. The divorce literature shows worse child mental health after a divorce (Amato and Sobolewski 2001; Strohschein 2005a), and that these effects may be direct, or through increased parental emotional strain (Ram and Hou 2003). However, fathers are not always a source of emotional support, and may actually cause more distress to children and their mothers when living in the household. Empirical research suggests that the effects of father presence may be harmful to children if the father was a bad parent and role model (i.e., high antisocial behaviors) (Jaffee, Moffitt, A., and A. 2003), and the divorce literatures shows that the emotional health of children is also worsened by marital discord (when fathers are present) (Amato and Sobolewski 2001; Strohschein 2005a). In short, it is not clear whether father absence would increase or decrease the emotional health of the household, and through it, child health.

The above conceptual framework lays out the pathways through which father absence may affect child illness, based on the current theoretical literature related to family structure.

This literature focuses on father absence due to divorce or non-marital childbearing in developed countries and suggests that the loss of a father would increase child illness through decreased economic and social resources, increased social disorganization, and reduced emotional support in the household. However, father absence due to migration (rather than marital disruption or non-marital childbearing) has not been addressed in the family structure literature, and its links with child health have not been conceptualized.

To move forward research on family structure and child health, I utilize Figure 2.0 as my conceptual model of how father absence due to migration would be expected to affect child illness. However, I add the consideration of migration literature to illuminate potential positive effects of father absence that are not considered in the family structure literature. Theory suggests that migration is largely an economic strategy that is employed by either individuals (neoclassical economic theory) or households (new economics of labor migration) to improve their standard of living (Massey, Durand, and Malone 2002). This demographic phenomenon is particularly relevant in economically-deprived areas like rural Mexico where jobs are scarce and pay is low (although, parents relocating (with or without their families) for work opportunities also occurs in developed countries). Under these conditions, one option for improving individual and family well-being is to seek education and employment opportunities in other communities or countries.

In the case of migration, fathers who are absent may be able to provide more economic resources to for their children than those who remain in a poor household and community. These remittances, or money sent back to the household during their absence, may be particularly helpful for poor households to provide a healthy environment for their children (Frank and Hummer 2002; Kanaiaupuni and Donato 1999). Another potential

advantage of father migration may be to change the social organization of the household in ways that increases mothers' control over household decisions. This may be beneficial if, as research suggests, mothers, more than fathers, prioritize the spending of resources in support of child health (Case and Paxson 2001; Maitra 2004; Schmeer 2005). In households with migrating fathers, then, children may benefit from increased resources and maternal autonomy, which, in turn, may allow mothers to better provide for their children's needs (food, clothing, doctor visits, etc.) and reduce stress (without the strains of marital discord and decision making). One caveat to these positive effects is that they may occur only in cases of longer term migration, when fathers are more likely to find jobs and mothers' may have more time to change household resource distribution and health behaviors. Another more direct, short term effect may be the loss of a "bad" father, thereby reducing stress and violence (similar to what the divorce literature has found). One migration study highlighted that paternal migration benefited adolescents' emotional health by increasing the calmness and decreasing violence in the household (Aguilera-Guzman, de Snyder, Romero, and Medina-Mora 2004).

While these positive effects may occur, the negative effects identified by the family structure literature remain relevant and affect child health through the pathways identified in Figure 2.0. Households may experience increased economic deprivation even when fathers migrate to earn more money if fathers are not yet sending money back and there is a significant cost to supporting the migration trip. Furthermore, households that experience paternal migration are more likely to be socially disorganized that those where the father does not migrate. Similar to a marital separation, paternal migration requires changing roles to accommodate the loss of the father's time and attention to children, as well as other duties

around the home. Research shows that both wives (Snyder and Nelly 1993) and children (Aguilera-Guzman, de Snyder, Romero, and Medina-Mora 2004) of migrants experience stress related to the disorganization and feelings of vulnerability caused by the migration of a spouse or father.

Given the potential positive and negative effects of father absence due to migration on child illness, it is not clear, a priori, what the aggregate effect might be. Based on current theory, father absence due to migration may be hypothesized to decrease or increase child illness, or the opposing pathways may cancel each other out, resulting in a no overall effect of father absence due to migration on child illness. In this study I provide an empirical test of what the overall effect of father absence due to migration is on child illness to inform our understanding of how children fare when fathers leave to pursue work or educational opportunities.

I also consider whether the relationship between father absence due to migration and child illness differs by whether a household has an external source of financial and social support—e.g. social welfare. In these cases, households may be less dependent upon fathers for economic resources. Social welfare payments may provide households with enough resources to maintain an acceptable standard of living and make it possible for mothers not to work for pay when fathers are absent. Mothers also may have a better chance of exercising their newly found autonomy if they have their own source of income (such as welfare payments). Finally, some programs (such as the one studied here) may require that households adhere to certain guidelines (taking children to the doctor, continuing school enrollment, etc.) to receive welfare payments. This may ensure that healthful behaviors are maintained in the face of changing household roles and responsibilities. Thus, there is reason

to believe that social welfare programs, and the resources and structure they provide to poor households, may reduce the negative consequences of father absence on child illness.

I now turn to a review of studies that have assessed the effects of father absence on child health, which provides the empirical foundation for this study.

Father Absence & Child Health: Empirical Research

Empirical literature on father absence in the U.S. presents a generally consistent picture of the negative impact of father absence on children (McLanahan 1997; Sigle-Rushton and McLanahan 2004): children living in married, two-parent families have better academic achievement (Dawson 1991), behaviors (Albrecht and Teachman 2003; Flewelling and Bauman 1990), mental health (Garnefski and Diekstra 1997), and physical health outcomes (Dawson 1991; Guttmann, Dick, and To 2004; Page and Stevens 2004) compared with children living without their biological father. In developing country studies, children also have better health outcomes in married, two-parent families than in single mother families (Bronte-Tinkew and DeJong 2004; Bronte-Tinkew and Dejong 2005; Gage 1997). Some developing country evidence further suggests that father absence affects child well being through the loss of social as well as economic support (Carter 2004; Gage 1997; Gertler, Martinez, Levine, and Bertozzi 2003).

Turning to migration research, potential positive aspects of father absence due to migration are apparent: household migration experience benefits infant health in sending households and communities (Frank and Hummer 2002; Kanaiaupuni and Donato 1999; McKenzie and Hildebrandt 2005). Remittances, which provide economic support to the household in the absence of a key member, seem to be particularly important for this positive effect (Frank and Hummer 2002; Kanaiaupuni and Donato 1999); although one of the studies

suggests that migration experience in a household increases the health knowledge of mothers, in addition to household economic resources, which in turn, benefits infant health (McKenzie and Hildebrandt 2005). These studies also suggest that the positive effects may be felt more in the long term, after migrants have been gone longer (Kanaiaupuni and Donato 1999). There is some evidence that women have more decision making power in households that have experienced adult male migration overseas (Hadi 2001), and that, controlling for economic status, children in female headed households actually do better than those in male-headed, two-parent households (Johnson and Rogers 1993; Pryer, Rogers, and Rahman 2004; Shell-Duncan and Obiero 2000).

Few negative effects have been found in the migration literature. However, one of the above-cited studies provides evidence that despite improvements in infant health in migrant households, these infants are less likely to receive preventive care, such as breastfeeding and vaccinations than those living in non-migrant households (McKenzie and Hildebrandt 2005). This suggests the conflicting nature of migration and its effects on child health. The only study to focus on paternal migration (rather than household migration experience) finds no effect of the number of years a father is present in the household on their measures of child health (breastfeeding, timing of vaccinations, and caloric intake in the preceding day) (Fernandez 1998). The null effect may be due to the positive and negative effects being cancelled out, or due to the use of retrospective data that summarizes the migration experience of the father, rather than assessing child health during his absence.

In short, consistent with family structure theories, father absence in general seems to reduce child health in both developed and developing countries. Consistent with migration theories, however, empirical research on migration provides evidence of potential positive

effects of father absence due to migration on child health. The one study to address father absence due to migration provides suggests a null effect, although this is based on retrospective job histories rather than prospective father absence data. These contradicting theoretical predictions and the lack of conclusive empirical evidence on how father absence due to migration affects child health, motivate this study. I focus on prospectively assessing father absence due to migration and test whether this type of father absence has a positive, negative or null effect on child illness.

A second question to be answered here is whether father absence has the same effect on child illness in all poor households, or whether those receiving social welfare payments may be able to protect children's health from any negative consequences of father absence. In response to the higher illness burden among children in poor households, social welfare policies are often aimed at counteracting the unequal distribution of resources among families/households to provide a basic level of well-being for these children. Such programs may be beneficial in filling resource gaps left by absent fathers. Studying this issue is difficult in the U.S., where welfare receipt is highly correlated with single-parent family structures (usually due to non-marital childbearing or divorce). In the context of Mexico, however, welfare is less tied to family structure, since poor parents are more likely to marry, and stay married, than in the U.S. In Mexico, and other countries where the poor continue to raise children in married couple families, one can more feasibly study how social welfare programs may alter the effects of father absence on child well-being.

In this paper I further investigate the effects of father absence due to migration by assessing whether any negative child health effects may be reduced by enrollment in a national welfare program (*PROGRESA*) in Mexico (*PROGRESA* is described in detail in the

next section). Several program evaluations have suggested that *PROGRESA* has had a positive impact on child health (Behrman and Hoddinott 2005; Gertler 2004; Skoufias 2005), and has reduced specific illnesses such as diarrhea and acute respiratory infections among young children (Huerta 2006). No research to date has tested how this program may interact with family conditions to affect child health.

I now turn to a description of the setting of my study and the data I utilize to answer my research questions.

Setting & Data

Mexico is a middle-income country; however, in 2003 it was estimated that 40% of the population lived below the national poverty line (Central Intelligence Agency 2005). Poverty inevitably leads to poor health conditions for children. Moderate and severe stunting (low height-for-age), which reflects chronic malnutrition and illness, currently occurs in about 18% of Mexican children under 5, compared with 16% in the Latin American region as a whole (UNICEF 2005). Both child poverty and poor health are concentrated in rural areas: in the late 1990s it was estimated that almost 32% of children under 5 suffered from malnutrition in rural areas compared with 12% in urban areas (INSP 1999).

In such economic and social conditions, household migration is an important survival and risk reduction strategy for households (Massey, Durand, and Malone 2002). Mexico as a whole has a long history of international migration to the U.S. (Bean, Corona, Tuirán, and Woodrow-Lafield 1998), and the net annual international migration rate for the country is currently estimated at -4.57 migrant(s)/1,000 population (Central Intelligence Agency 2005). The high rate of out migration may include substantial repeat migration—it has been estimated that in a select group of rural Mexican communities almost 90% of migrants

returned home within one year (Massey, Alarcon, Durand, and Gonzalez 1987). Migration rates, however, vary across states, and the states included in my study include two traditional sending states (Michoacán and San Luis Potosí) where migration rates are highest (Bean, Corona, Tuirán, and Woodrow-Lafield 1998). In all communities included in my study, there is an annual outflow of international migrants.

Migration as an aspect of father absence has not been widely assessed. However, there is reason to suspect that migration is a main cause of father absence due to the low rates of non-marital childbearing, union dissolution, and parental separation. Current estimates suggest that the divorce rate is roughly 6% in Mexico(Frank and Wildsmith 2005), and that rates are even lower in rural areas and among women with low education (de Oliveira 2000). A recent unpublished paper suggests the importance of father migration to children's lives: 7% of Mexican children aged 0-14 live with their parents in union and their father absent due to migration; and, on average, 17% of children are likely to experience father absence due to migration at least once during their first 14 years of life (Nobles 2006).

An additional aspect of the setting of my study is the implementation of an important social welfare program, PROGRESA, in the late 1990s. The Mexican government created the Education, Health and Nutrition Program (*Programa de Educación, Salud y Alimentación*, or *PROGRESA*) to provide poor families in rural communities with resources to invest in their children's education and health. Under PROGRESA, women in qualifying households are given a cash transfer for food consumption of, on average, 99 pesos, which represents about 20% of a poor household's income. Mothers with children between the ages of 7 and 18 are also given education grants to support school enrollment, and those with children under 5 receive nutritional supplements and information to promote healthy child care and

development. These food assistance and education funds are tied to specific requirements, including regular medical visits for children, yearly medical visits for adults, attendance at health information meetings, and sustained school attendance for school-aged children (Skoufias 2001). The ongoing program is now called *Oportunidades*, and has expanded into urban communities and other states, and has been replicated in other countries in Latin America.

In rural Mexico, then, father absence due to migration is a salient aspect of children's lives that may have implications for their health. Given the existing conditions of poverty and child malnutrition, father absence due to migration may provide important economic resources needed to prevent illnesses, or it may induce more illness through the immediate loss of an income, parental time, and through increased social disorganization. Households' enrollment in the *PROGRESA* program may prevent some of these negative effects by providing economic resources and health guidelines for mothers in the absence of their spouse. I now turn to a description of the data and sample used to answer my research questions.

Data

The data utilized was collected by *PROGRESA*, and includes information from the baseline survey of household members and living conditions (1997) and three follow up surveys (October 1998, June 1999 and November 1999) intended to evaluate the success of *PROGRESA*. In 1997, all households in 506 poor, rural localities were surveyed, resulting in a sample size of over 24,000 households (World Bank 2004). The localities were selected from the states of Guerrero, Hidalgo, Michoacán, Puebla, Querétaro, San Luis Potosí, and Veracruz (the first seven states to be incorporated into *PROGRESA*) based on their relative

marginalization score as established from multiple indicators of socioeconomic development assessed in the 1995 census (Skoufias, Davis, and Behrman 1999). To implement the large-scale program incrementally and to better evaluate the program effects, eligible families in treatment communities (320 randomly-selected communities of the 506) received cash transfers and health information beginning in August 1998, while eligible households in control communities were delayed entry until 2000. The follow up surveys were conducted beginning in October 1998, after the experimental implementation of *PROGRESA* began (Gertler 2004).

The main strength of these data for considering this question of father absence and child health is the ability to assess the residential status of the father and child morbidity prospectively at three time points over a one-year period. The close spacing of the waves allows for short-term changes in both father residency and child health to be assessed, and the large sample size covering over 500 rural communities provides both statistical power and the ability to generalize the results to households in poor communities across rural Mexico. These data are also unique in allowing for the consideration of the effects of father absence in the context of a social welfare program that was being implemented following an experimental design. Since the benefits (both the payments and information) were given to the children's mothers, father absence during this time did not preclude the receipt or use of *PROGRESA* benefits. These data, then, provide the best available opportunity to evaluate both the effects of father absence due to migration on child health, and a consideration of whether welfare support may condition these effects.

The sample used in this study consists of children aged 0-5 living in the surveyed communities. Children in this age group are physiologically vulnerable, so that even the most

basic illnesses (like diarrhea or respiratory infections), if not handled properly can lead to repeated illnesses and increased risk of mortality. Under these conditions of poverty, illness in young children, together with malnutrition, can stymie cognitive and physical growth. Young children are also highly dependent on their household and its members for protecting their health, and thus would be most affected by changes in the household that ensue when their fathers migrate. This age group also closely corresponds to the age group utilized by official child mortality and morbidity measures (usually, 0-4).

The sample is restricted to children living with their parents in union at the baseline, as those who are at risk for father absence. I then follow the children who remain living with their mothers in 1998-1999, and add any new children who were born or moved into these households. To avoid bias through selective mortality, I also include children who died by categorizing them as "sick" in the wave in which they were listed as having died. The remaining missing data were dealt with through case-wise deletion.

My sample size is roughly 29,300 cases, which represent about 12,400 children who were observed, on average, 2.3 times out of the three waves. Consistent with the overall setting of rural Mexico, the sample children live in difficult conditions, with 30% of living without electricity, 46% without a toilet, 63% with a dirt floor, and 66% without piped water in 1998. About 89% of the sample children live in households that qualify for *PROGRESA* based on their means testing for poverty. Consistent with the experimental design, 60% of the sample children live in households enrolled in *PROGRESA* during this study period, while the other 40% live in control communities.

Consistent with the current nationally representative estimates of father absence due to migration, 6% of sample children who were observed in all waves had a father absent at

some point during the year. Since I observe children only over a year period, there are few cases of multiple absences. Further, almost all of the fathers return within a 6 month period. Thus, the father absence cases observed in these data is short term and occurs only once during the year. Had these children been observed over a longer period of time there might have been evidence of multiple and longer-term absences. In terms of illness, 25% of the sample children were reported as having been ill in the past four weeks in the fall of 1998. This dropped to 19% for the spring and fall in 1999.

Using these data, then, I provide statistical analyses that aim to estimate the relationship between father absence due to migration and child illness in rural Mexico. In the next section I describe the specific variables, statistical methods, and analyses I use to answer my research questions.

Methods

A main strength of the analysis provided in this study is the use of prospective, longitudinal data, which reduces recall bias and allows for closer temporal ordering (where child illness is assessed while father are gone). My dependent variable, *child illness*, is measured by whether the respondent (usually the child's mother) reported a child as being ill in the four weeks prior to the survey (assessed in October 1998, May 1999 and November 1999). This measure has been substantiated in the literature as being highly correlated with objective measures of child illness (Rousham, Northrop-Clewes, and Lunn 1998). However, this illness measure has the disadvantage of including some recall bias, being a subjective assessment, and only obtaining reports on one illness in the past four weeks rather than the number of times a child was ill.

My main independent variable of interest—*father absence due to migration*—was also assessed during these waves. I utilized the household census to determine whether a father was living at home or not during each wave, and combine this with his marital status to create a father residency status categorical variable. This categorical variable includes the following categories: *father absence due to migration* (where the father is no longer living in the household because he was studying, working or for another reason, and remains in union with the mother), *other father residency status* (the father had moved out and is no longer in union with the mother, died, did not exist, or was reported as living in the household regularly but was not there at the moment)[1]; and, *father present* (the father lives regularly in the household). In the regression analysis, then, *father absence due to migration* and *other father residency status* are entered as dummy variables with *father present* as the omitted category. My approach to measuring paternal migration provides a direct assessment of married fathers' absence at the time of the survey rather than an assumption based on the location of his work or retrospective histories of migration. However, a limitation of this measure is that I cannot assess further information about the purpose or conditions of his absence, nor whether he remits money during his absence.

To assess the potential interaction of father absence with *PROGRESA*, I include a measure of the effect of the *PROGRESA* program in my models. The *PROGRESA* program effect is assessed in the models by interacting two variables—whether the household qualified for *PROGRESA* (*poor*=1) and whether the household was located in a treatment community (*treat*=1). To estimate the effect of father absence for households who are

[1] This last residence category could be construed as absence due to migration, but is less clear than the first category, which specifies that the father has temporarily moved elsewhere. The results are not affected by including the latter category of residency status as father absence due to migration.

enrolled in *PROGRESA* and those who are not, I include an interaction term of *father absence due to migration x PROGRESA*.

The method I use to provide statistical tests of my research questions is a pooled logit model, which controls for clustering among individuals over time, within households, within communities, and within regions. This general model can be written as:

$$\log\left(\frac{(y_{tihcr}=1)}{(y_{tihcr}=0)}\right) = b_0+b_{1\ldots k}X_{it}+\square_r+u_{cr}+a_{hcr}+\varepsilon_{ihcr} \qquad (2.0)$$

where, y equals 1 if reference child "i" in household "h" in community "c" in region "r" is ill at time "t", 0 if not. The intercept is b_0, while the coefficients on the independent variables in the model (X) are designated by "$b_{1\ldots k}$", with k number of independent variables. The model includes a composite error term that controls for clustering at various levels, including error at the regional level (□), the community level (u), the household level (a), and the individual level (ε).

This model estimates the log odds of being ill utilizing all cases across children and survey waves, and accounts for multiple observations per child. The model uses maximum likelihood estimation, and the coefficients refer to changes in the natural log odds of sickness occurring or not (Long 1997). Maximum likelihood performs best with large samples, and thus should provide reliable results in this sample even if there are few events occurring. The multiple error components capture clustering of the data at multiple levels, from the individual child up to the region level. I account for the unobserved error clustering at the highest (regional) level, which adjusts the standard errors for clustering at the regional and lower levels, allowing for accurate hypothesis testing (Angeles, Guilkey, and Mroz 2005).

In answering my research questions, I estimate the following two specific models, the second which includes an interaction effect:

$$\log\left(\frac{(y_{tihcr}=1)}{(y_{tihcr}=0)}\right)=b_0+b_1FA_{it}+b_2PROGRESA_i+d_{1\ldots k}Z_{it}+\square_r+u_{cr}+a_{hcr}+\varepsilon_{ihcr} \quad (2.1)$$

$$\log\left(\frac{(y_{tihcr}=1)}{(y_{tihcr}=0)}\right)=b_0+b_1FA_{it}+b_2PROGRESA_i+b_3FA_{it}*PROGRESA_i+d_{1\ldots k}Z_{it}+\square_r+u_{cr}+a_{hcr}+\varepsilon_{ihcr} \quad (2.2)$$

where, FA=*father absence due to migration*, *PROGRESA*=*PROGRESA* program effect, Z=control variables. The first model estimates the average effect of father absence due to migration, while the second model adds an interaction term between *father absence due to migration* and *PROGRESA* to estimate whether the effect of father absence due to migration differs for households enrolled in *PROGRESA*. Because I do not hypothesize a specific direction of the father absence effect (and by extension its interaction with *PROGRESA*), I assess the significance of the coefficients through two-tailed tests of $p<.05$.

To obtain the best possible estimates of the effects of father absence due to migration and its interaction with *PROGRESA*, I include control variables that are theoretically related to father absence due to migration and child illness and can be obtained from the data. I utilize information from the baseline survey, which provides socioeconomic status, parental characteristics and other information on each child's household and community. All children in this sample have a baseline household, even if they were born after the baseline survey (i.e., children born after 1997 were added to existing households and thus all have baseline data). The following baseline controls are included in my statistical models: *child sex*; *maternal age, literacy* and *ethnicity*; and, community variables (*percent of households with a migrant, piped water,* and *electricity*). I also include the following time varying controls

assessed at the time of each survey: *child age* (a spline allowing for different effects for children under and over age 2); *lagged number of children under 10, number of teens* aged 10-16, and *number of adults* 16 years or older, in the household; *lagged asset score* (a summed score of household consumer durables lagged by one survey wave), and survey wave dummies (*wave 2* and *wave 3* with *wave 1* omitted) to control for trends in both migration and child health. I also include regional dummy variables to control for differences in migration rates and disease distribution across the seven regions included in this study.

As a check on the results, I conduct an individual-level fixed effects regression (using conditional logistic regression, which provides a consistent estimator) of the final model to assess whether time-invariant unobserved heterogeneity might be accounting for my results. The conditional logit model estimates the effect of father absence on the log odds of being ill conditional on illness status in the previous wave. This can be estimated for individuals who have gone from not being sick to being sick in a later wave, or from being sick in an earlier wave to not being sick in a later wave[2]. Since the coefficients are estimated conditional on having been not ill and then ill (or visa versa), the model can only be estimated for children who have changed illness status across the two or three waves when they are present (roughly 1/3 of the sample). Due to these sample limitations, I compare the conditional logit (fixed effects) model to a pooled logit model with a limited sample similar to that of the conditional model (i.e., limited to those who have been sick at least once during the year). The results of these supplementary analyses are summarized in the text, and the author may be contacted for the full tables.

[2] For statistical discussion of how a fixed effects logit model is estimated through a conditional logit model, see: Chamberlain, Gary. 1980. "Analysis of Covariance with Qualitative Data." *Review of Economic Studies* XLVII:225-238.

Results

Table 2.0 below shows the results from four logit models. The first model includes only the *father absence due to migration* variable, and the second model adds the control variables. This allows for a consideration of the correlation between father absence and child illness (Model 1) with the more causal relationship that includes variables controlling for other potential confounders (Model 2). The final model adds the *PROGRESA* variables and the interaction between *father absence due to migration* and *PROGRESA*. This interaction term accounts for potential differences in the effect of father absence by whether the household is enrolled in *PROGRESA*. This tests my hypothesis that receipt of social welfare will alter how father absence affects child illness.

To assess the effect of *father absence due to migration*, two of the three father residency status categories are entered in the mode (*father absence due to migration* and *other father residency status*), while the third category (*father present*) is the omitted category. The coefficient on *father absence due to migration* Model 1, then represents the log odds of illness when fathers are absent compared with when fathers are living in the household regularly. The 0.42 coefficient is statistically significant and illustrates a relatively large, positive effect of *father absence due to migration* on illness. The *other father residency status* coefficient, which represents the effects of fathers being absent for reasons other than migration or having an ambiguous residency status compared with fathers who are living in the household, is much smaller (0.07) and statistically insignificant.

The bivariate relationship between father absence due to migration and child illness is mitigated slightly by the control variables added in Model 2: the coefficient remains positive, statistically significant and almost reaches 0.40. This represents an increase in the risk of

illness of over 7 percentage points when the father is absent due to migration compared with when he is present. The control variables illustrate, among other things, that the *PROGRESA* program does indeed have a significant negative effect on child illness, with a coefficient of -0.21. This translates into a two percentage point reduction in the probability of illness for each case. The significant child and household-level control variables are: a) child age, which illustrates the reduction in the log odds of illness for older children; b) maternal age, which increases the log odds of illness; c) maternal ethnicity, with mothers who speak an ethnic dialect seeming to reduce illness; and, d) household size (number of kids and teens) also reducing illness. The maternal ethnicity and household size variables are a bit counterintuitive, and may be due to an underreporting of illness by indigenous mothers and those with more children. Several of the regional dummy variables are statistically significant, and are jointly significant as a group ($p<.01$), indicating important regional differences in the log odds of child illness.

Table 2.0: Pooled logistic models of child illness in rural Mexico, N=29368

	(1)	(2)	(3)
Father absence due to migration[a]	0.422	0.394	0.721
	(0.067)**	(0.090)**	(0.116)**
Other father residency status[a]	0.073	0.122	0.122
	(0.102)	(0.114)	(0.114)
Father absence due to migration x *PROGRESA*			-0.651
			(0.214)**
PROGRESA (*poor* x *treat)*		-0.213	-0.200
		(0.089)*	(0.093)*
Treat (household in treatment community)		0.109	0.108
		(0.092)	(0.094)
Poor (household qualified for welfare)		-0.008	-0.006
		(0.068)	(0.067)
Child control variables			
Child sex (male=1)		-0.015	-0.015
		(0.014)	(0.014)
Child age spline, under 2 years		-0.245	-0.245
		(0.025)**	(0.025)**

Child age spline, 2+ years		-0.132	-0.133
		(0.011)**	(0.011)**
Maternal control variables			
Maternal age		0.051	0.051
		(0.016)**	(0.016)**
Maternal age squared		-0.001	-0.001
		(0.000)**	(0.000)**
Mother speaks ethnic dialect		-0.129	-0.128
		(0.038)**	(0.038)**
Mother literate		0.058	0.057
		(0.058)	(0.058)
Household control variables			
# children, lagged		-0.098	-0.099
		(0.013)**	(0.012)**
# teens, lagged		-0.060	-0.061
		(0.008)**	(0.007)**
# adults, lagged		-0.031	-0.031
		(0.020)	(0.020)
Total HH assets, lagged		0.028	0.028
		(0.015)	(0.015)
Community control variables			
% HHs with a migrant		0.407	0.388
		(0.739)	(0.719)
% HHs with piped water		-0.178	-0.175
		(0.110)	(0.111)
% HHs with electricity		-0.151	-0.155
		(0.107)	(0.107)
Wave 2 dummy[b]		-0.220	-0.219
		(0.128)	(0.129)
Wave 3 dummy[b]		-0.306	-0.306
		(0.178)	(0.178)
Region 2[c]		-0.105	-0.104
		(0.006)**	(0.006)**
Region 3[c]		-0.191	-0.191
		(0.018)**	(0.018)**
Region 4[c]		0.003	0.005
		(0.032)	(0.032)
Region 5[c]		-0.499	-0.499
		(0.030)**	(0.028)**
Region 6[c]		0.097	0.100
		(0.011)**	(0.012)**
Region 7[c]		-0.100	-0.100
		(0.023)**	(0.023)**
Constant	-1.307	-0.653	-0.660
	(0.056)**	(0.316)*	(0.314)*
Log pseudo-likelihood	-15256	-14953	-14949

Robust standard errors in parentheses. *p<.05; **p<.01
[a]Omitted category: father present. [b]Omitted category: Wave 1 [c]Omitted category Region 1.

Model 3 includes the interaction of *father absence due to migration* with *PROGRESA* and all control variables. This model shows that the interaction term is statistically significant, negative and relatively large at -0.65. In this model, the coefficient on *father absence due to migration* represents the effect of father absence compared with fathers who are present when households are not enrolled in *PROGRESA*, and is a large positive effect at 0.72. The coefficient for father absence due to migration for households that are receiving *PROGRESA* is obtained by adding the main effect coefficient to the interaction coefficient (0.72-0.65), and results in a small and insignificant effect (0.07). This suggests that the increased log odds of illness when fathers are absent compared to present is wiped out by *PROGRESA* benefits.

To provide an idea of the magnitude of the effects of father absence due to migration on child illness, Table 2.1 below shows predicted probabilities calculated for key coefficients from Models 2 and 3. For ease of interpretation, these probabilities were calculated setting all households as qualifying for *PROGRESA* (*poor*=1), which is similar to the sample values for this variable since almost 90% of the sample cases were in households that qualified for *PROGRESA*. The rest of the control variables were held at their actual values while varying the father absence and *PROGRESA* variables as appropriate for each scenario.

Table 2.1: Predicted probabilities and marginal effects of father absence due to migration

	Average Effect (Model 2)	When NOT enrolled in *PROGRESA* (Model 3)	When enrolled in *PROGRESA* (Model 3)
Predicted Probabilities of Child Illness:			
Father present	21.3%	22.1%	20.6%
Father absent	28.5%	36.5%	21.8%
Marginal effect:	7.2**	14.4**	1.2

*p< .05 **p< .01

Table 2.1 shows that the average effect of *father absence due to migration* (based on Model 2) is to increase the probability of child illness by over 7 percentage points--from 21.3% when fathers are present to 28.5% when fathers are absent. This effect is doubled in households that are not receiving *PROGRESA*, where father absence due to migration increased the chance of illness by over 14 percentage points (based on Model 3). However, *father absence due to migration* has only a small and insignificant effect on the probability of illness in households enrolled in *PROGRESA* (just over 1 percentage point). The predicted probabilities also illustrate the estimated magnitude of the *PROGRESA* program on child illness. Here we see the effect is generally small, reducing the probability of illness by about 1.5 percentage points when fathers are present (the vast majority of the cases). However, in cases where fathers are absent, the *PROGRESA* effect may reduce the chance of illness by 13.7 percentage points. This indicates the potential importance of social welfare in these cases where children are experiencing both poverty and the temporary loss of their father.

The robustness of the relationship between father absence due to migration and child illness can be assessed in several ways. First, the addition of control variables in the model ensures that observed differences across households and communities are not accounting for the relationship. Second, by including regional dummy variables in the models, I account for any unobserved regional differences that might be causing the relationships of interest. I also tested for community-level fixed effects (entering dummy variables for each community), but these did not change the results. Time invariant unobserved characteristics of communities and regions are apparently not causing the relationship between father absence and child illness. However, it may be that something unobserved at the family or household level affects both whether a father is migrating and whether his child is ill. One possibility is that a

household where the father is not a productive or supportive member has both more child illness and the father is more likely to migrate to find work or to get away from a difficult situation. I first test this possibility by including paternal education in the model (omitting maternal education due to multicollinearity), but it does not affect the results. Second, I consider an individual-level fixed effects model using conditional logit as a consistent estimator. As previously mentioned, however, conducting an individual-level fixed effects model with a binary outcome drops those children who were not ill during the year. Thus, the model is not preferred for sample bias reasons, but may be compared to a similarly sample-limited logit model (i.e., that included only children who had been sick at least once) to get an idea of whether unobserved differences at the individual or family level might be related to father absence. Comparing the two estimation techniques for the final model (which includes the interaction term) yields very similar coefficients for *father absence due to migration* (conditional logit: 0.55** and logit: 0.58**) and the interaction term (*father absence x PROGRESA*) (conditional logit: -0.56 and logit: -0.54**)[3]. This suggests that unobserved, time invariant heterogeneity among fathers, households or children may not play a major role in the relationships of interest.

In sum, the results suggest that father absence due to migration contributes to increasing probability of illness of children aged 0-5 living in a sample of rural Mexican households. The significant and relatively large interaction effect between father absence due to migration and *PROGRESA* further illustrates that the social welfare program is able to alter how father absence affects child illness: the effect of father absence due to migration on child illness is virtually zero when households are enrolled in the program. These results are robust to child, household, community and regional-level controls, and there is some

[3] Full results can be obtained by contacting the author.

evidence that the relationship is not caused by time-invariant unobserved differences among households or children.

Conclusions

Illness during childhood is an important social problem that affects both individuals' lives and societal well-being. Although child illness is produced by a complex interacting set of social contexts at multiple levels, the family/household context is particularly important for understanding the development of child health and illness. In this study, I advance our knowledge of how the household context affects child illness by focusing on the dynamics of family structure, and in particular, changes in father residency status due to migration.

Father absence due to migration is a relatively understudied aspect of children's family/household context that has the potential to improve or hinder child health. Research on family structure, and father absence due to non-marital childbearing, divorce or death, suggests that father absence due to migration may increase child illness due to decreased economic resources, increased social disorganization, and decreased time and social support when fathers leave. However, the migration literature provides contradictory ideas, suggesting that especially within poor communities fathers may provide more economic resources for children by moving away. Furthermore, migrating fathers may engage social networks (family and friends) to support their households when they are gone. Finally, when fathers leave, mothers may have more power to direct scarce resources towards child well-being. Thus, migration research, although lacking in empirical findings directly related to father migration, suggests potential benefits of father absence due to migration. Given these contradictory predictions in the literature, my basic research question aimed to answer whether father absence due to migration has an overall positive, negative or null effect on

child illness. I also asked whether the effect of father absence on child illness may be affected by a social welfare program, which may provide families with needed resources and structure while fathers are absent. The question of how father absence effects may differ by social welfare receipt has not been posed or tested in the sociological literature to date.

Using data from the large scale *PROGRESA* social welfare program, this study provides a prospective, longitudinal view of the short term child health impact of married father absence in the context of Mexico, where both marriage and migration rates are high. This allows for an investigation of how child health fares when fathers are absent due to migration, as well as a test of whether a key social welfare program conditions this effect. The results illustrate consistent evidence that, in the context of poor, rural Mexican communities, short term father absence due to migration increases the likelihood of child illness. In this sample, father absence due to migration, on average, increases the probability of illness by over 7 percentage points. When viewed within the context of children's lives in rural Mexico, this effect is significant, since increasing the chance of illness may result in increased malnutrition and subsequent illnesses for an individual child. Furthermore, if a father migrates repeatedly, a child may be subject to repeated illnesses, further exacerbating their precarious health situation.

The findings also contribute support for the mounting evidence that the *PROGRESA* welfare program does improve child health, in this case by decreasing the chance of illness in young children. The main program effect, however, seems modest in this study (decreasing the change of illness, on average, by 1.7 percentage points). The study goal related to *PROGRESA*, however, was not to determine its main effects, but rather, whether it can buffer children from potentially negative consequences of father absence. The interaction effect

between *PROGRESA* and father absence due to migration provides insight into this critical and overlooked role of social welfare. Given that the marginal effect of father absence due to migration on illness decreases from 14 percentage points when households are not enrolled in *PROGRESA* to 1 percentage point when they are enrolled in *PROGRESA*, this study highlights that the important social welfare program seems to protect child health during father absences due to migration.

There are several limitations of this study that should be considered in interpreting the results and informing future research and policy in this area. First, the father absences assessed in this study were largely short term, lasting 6 months or less. Further, the consequences of these absences were assessed concurrently, focusing on short term effects of the absences. Thus, it cannot be concluded from this study whether or not child health is permanently impacted by paternal migration or the effects of more permanent, long term migration. Second, the findings are based on a relatively small number of father absence events. This may be because the study considered only one year of children's lives, and because of the relative marginality of the communities in this sample. Further, due to the sample limitations, the findings are generalizable to married-couple households with children in poor, rural communities.

These limitations not withstanding, the findings can inform both subsequent research and policy development. In terms of research, this study provides further evidence of the importance of fathers and their presence in the household for child well-being. Similar to what the divorce literature postulates, when fathers leave their origin households, even temporarily, children are worse off than when they are present. Although I did not test for mechanisms, the increase in child illness with father absence is most likely due to the loss of

economic and social resources, as well as increasing social disorganization. This is supported by the fact that the *PROGRESA* program, which provides economic and social resources, as well as social organization (through program guidelines), buffers all of the father absence effects. Increased child illness through emotional distress may be a less important mechanism, since if that were the main mechanism *PROGRESA* would be unable to equalize child health among households with fathers absent and present.

The findings go against what the general migration literature predicts, i.e., that migration is a benefit for poor households. In capturing the status of households and their children's health when fathers have recently left, my study suggests that origin households bear significant costs to migration in the short term. It may be that, in the short term, paternal migration may not increase household economic well-being, and/or that the social consequences of migration for the origin household outweigh economic benefits. In focusing on the short term effects, my results are consistent with the migration literature that suggests that the benefits of migration may be felt only in the long term when migrants are established and sending money home and origin households have had time to adapt.

In terms of advancing sociological research, this study informs family structure research by demonstrating the potential importance of considering short term father absences and how they affect children's health. Fathers, and other key family members, may be absent multiple times in a child's life course, even if more formal aspects of family structure remain the same (i.e., the parents remain married). Although this study focused on poor, rural households, it may be that fathers temporarily move out of urban households and poor households in the U.S. and other developed countries to find work as well. More research is needed on migration and residential mobility as a process of changes in family structure, and

how these changes affect individuals' well-being in multiple settings. Furthermore, this study highlights that family status should be considered, both conceptually and empirically, as fluid, with potential to change over time. Assessing family structure at one point in time reduces the validity of family structure measures during childhood, and misses the opportunity to assess how changes in family structure over time affect individuals' lives. In fact, as other recent research has suggested, it may be that changes in family structure are as important for child well-being as the family structure a child is born into (Brown 2006; Cavanagh and Huston 2006).

This study informs a second body of sociological literature as well—that related to migration—in two ways: (1) the need to consider who specifically is migrating; and, (2) identifying the effects of different aspects of the migration process. First, the negative effects found here may be due to the fact that fathers are important providers in the context of rural Mexico and short term absences may be difficult for poor households to absorb. It is not clear whether migration of siblings or other members of the household would have similar effects. Future research should test whether the effects of migration on child well-being depends on the household/family member who migrates. Second, migration is a long, complex process that includes the decision to migrate, preparation for the migration, the loss/absence of the household member, communication (or lack of) between the migrant and the household, and the rejoining of the household with the migrant (either in the origin or destination household, or not at all). Migration research to date often assesses migration retrospectively, in terms of whether a household has experienced migration. This summary measure misses the potential conflicting effects of different parts of the migration process on child well-being. This paper focused on the loss/absence aspect of the migration process. The negative effects suggest not

that migration as a whole is negative, but rather, the absence of a key family member harms children in the short term. It may be that the loss or absence part of the process has negative effects, while the return of a migrant has positive effects (especially if they bring money back) on children in the origin household. In future research, it may be useful to disaggregate the migration process and the effects of different parts of this process on children in the sending household.

These findings also inform social policy related to improving child well-being. First, as past studies of *PROGRESA* and other programs have suggested, targeted programs such as this one can improve child well-being. This study, however, unlike other suggests that social welfare programs may provide important buffering effects to protect children in the midst of changes in family structure. This means, that, in poor households, although changes in family structure may be inevitable, welfare programs may alter how these changes impact children. Further, social welfare programs may be particularly beneficial to children experiencing multiple risk factors—i.e., poverty and father absence. Where funds are tight, then, it may be feasible to further target social welfare programs towards those children with multiple household risk factors.

It is hoped that future research can build on these findings to further explore how the mobility of family members may affect child well being, as well as how social policies and programs may buffer child well-being from the potential negative effects of family and household change.

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CHAPTER 3: CHANGING SIBSIZE DURING CHILDHOOD AND EDUCATIONAL ATTAINMENT

Introduction

Social stratification research suggests that family and household characteristics during childhood may have strong effects on individuals' educational attainment (Blau and Duncan 1967; Featherman and Hauser 1978; Sewell and Hauser 1975). Educational attainment, in turn, affects adult earnings and occupation and is a measure of where an individual, and their subsequent children, will end up in the social hierarchy of society (Blau and Duncan 1967). This paper focuses on one aspect of the childhood household/family that has been of interest to social scientists since the 1800's—siblings—and its relationship with educational attainment. Number of siblings during childhood has been found to be an important stratifying force (Steelman, Powell, Werum, and Carter 2002). However, research analyzing the effect of sibsize on educational has been limited by its reliance on static theories, cross-sectional data and developed country experiences to try to understand what is perhaps a quite dynamic, time sensitive, and context-specific relationship.

This paper aims to move forward our understanding of the importance of sibsize by applying a life course perspective to conceptualizing and analyzing how number of siblings affects educational attainment. I define sibsize based on siblings' residency in the index child's household, and assess how changes in the number of residential siblings during childhood impacts individuals' educational attainment. I focus on residential siblings as those most likely to compete with the index child for educational resources, and those who interact

with the index child the most during childhood. Further, by assessing the number of residential siblings over time, I can capture changes in the index child's household context as these siblings come into and leave their household through birth, death, migration and residential mobility. In doing so, I assess how individuals' educational attainment is affected by their changing number of residential siblings during childhood.

I hypothesize that changes in residential sibsize may be meaningful to children's lives because: (a) increasing or decreasing the number of siblings living with the child may significantly alter the resources (of all types) allocated to the index child for education; and, (b) change in and of itself is a stressful process that requires reorganization of the household. Adapting to changes in residential sibsize may take additional resources that may not be required in times of stability (i.e., parental and index child time, stress, and economic resources needed to establish a new household environment). In assessing changes in residential sibsize for individuals throughout childhood, sibsize is no longer a single measure of family background; but rather, a measure of the childhood environment that reflects a series of demographic changes that occur in the household from birth through adolescence.

Based on this perspective, I ask the following questions about the relationship between sibsize and education: (1) How do changes in residential sibsize during childhood affect individuals' educational attainment; (2) Do these effects differ by stage of childhood when the changes occur; and (3) Do these effects differ by younger versus older siblings? These questions, and the life-course approach that spurs them, are critical to our understanding of the relationship between sibsize and education. Too often social stratification research focuses on individuals at the end of childhood (i.e., the Wisconsin study of high school seniors) or in adulthood. Childhood is considered a background measure

rather than an important time period in which to understand the development of stratification forces. In this study, however, the childhood household, and in particular its dynamics over time, are the focus in understanding individuals' educational attainment. Sibsize may vary quite dramatically over time during an individual childhood and these changes may have important implications for children's lives. Recent family structure research shows that experiencing change in parental marital status matters as much or more than living in a given family structure (Brown 2006; Cavanagh and Huston 2006). This indicates that adapting to changes in the childhood household environment may pose additional challenges to children not captured in static measures of children's household and family context.

I also assess potential differences in the effects of changing sibsize depending on when they are experienced during childhood, and whether the siblings are younger or older than the index child. Changes in sibsize may affect education through changes in cognitive stimulation during early childhood, through changes in parental time and support with school during middle childhood, and through the changes in the index child's non-school responsibilities (i.e., work inside and outside the home) during late childhood. There is no theoretical reason to believe that the effects of changing sibsize should be the same throughout childhood, but no study provides evidence as to how the effects of sibsize may differ throughout childhood. Changes in younger versus older sibsize incorporates the idea of birth order, but in a dynamic way, considering how changes in the index child's position in the family (with respect to work and school roles in particular) may be affected by adding or losing younger versus older siblings. The effects of changing sibsize may be a combination of these two aspects, with changes in younger sibsize mattering at different stages of childhood than changes in older sibsize.

A further contribution of this paper is to provide a developing country perspective on this stratification process, based on longitudinal data from the Philippines. Globalization and modernization forces are making education an increasingly important aspect of success in life for developing countries. Given the importance of education for obtaining a well-paid job in the Philippines, as in most countries around the world, truncated educational attainment can have serious consequences for individuals' socioeconomic status and other aspects of their adult lives. Further, higher fertility, mortality and migration rates in developing countries result in a context of potentially large changes in sibsize during an individual's childhood. No study to date has assessed how these changes may affect individuals' educational attainment in a developing country context.

In sum, this paper provides a life course and developing country view of the relationship between changes in sibsize during childhood and educational attainment. With a focus on the dynamic relationship between sibsize and education over time, the conceptual approach and empirical findings contribute to sibling research, as well as to the broader social stratification theories about how family and household contexts during childhood affect individuals' position in society later in the life course. Only by taking such a dynamic perspective can we advance our understanding of the complex way that childhood conditions affect individuals' life chances and contribute to the development and reproduction of social inequalities.

Theoretical Background

The theoretical foundation of my study is the resource dilution hypothesis, which was initially proposed by Judith Blake to explain why individuals from larger families did worse on educational and socioeconomic status outcomes than those from smaller families (Blake

1989). According to this theory, families and households provide important resources (economic, time, emotional support and cognitive stimulation) that are needed to support individuals' educational attainment. Having a larger family size means that household/family resources would be divided among more siblings, decreasing those allocated to any one child (Blake 1989). This "dilution" of resources, then, results in individuals from larger families not getting the money, time, or attention needed to succeed in the educational system. The resource dilution hypothesis guides current thinking on the effects of sibsize, with most work aiming to test whether sibsize does indeed have a negative effect on individuals' education in developed countries (Steelman, Powell, Werum, and Carter 2002).

In this study I advance our understanding of the effects of sibsize on individuals' lives by incorporating the life course perspective into the ideas proposed in the resource dilution hypothesis. Life course is the study of individuals' lives and how they unfold over time and within changing social contexts (Elder 1995; Elder 1985). I apply this perspective to the resource dilution hypothesis by incorporating residency status, change over time, and a consideration of the life course stage of the index child in the conceptualization of childhood sibsize and its effects on educational attainment. My first contribution to the resource dilution hypothesis is to define sibsize prospectively and throughout childhood based on the number of siblings living in the index child's household at a given time during childhood. In doing so, I focus on sibsize as a dynamic aspect of children's household environment. The use of residential siblings differs from the resource dilution hypothesis and most existing sibsize literature that defines siblings based on biological ties and relative to a single point in time (Steelman, Powell, Werum, and Carter 2002). This standard definition does not provide a full view of sibsize, but rather a cross-sectional view at a given moment in time, and does not

assess whether and when the siblings shared or competed for household resources. Residential sibsize, on the other hand, incorporates multiple demographic processes that shape how a child's household grows and changes over time. Residential sibsize may be increased through births or return migration of siblings previously living elsewhere, or decreased through deaths, residential mobility, and migration of siblings out of the household. When assessed over time, residential sibsize allows for a more dynamic characterization of the resource dilution process; where, individuals experience multiple sibsizes and, by extension, changing shares of household resources over time. Although siblings no longer living in the household may also compete for some resources, competition is likely most pronounced for those sharing a roof.

Building on this definition of residential sibsize, I emphasize changes in sibsize during childhood and their effects on individuals' educational attainment. Changes in sibsize during childhood reflect the dynamics of a child's position in the family. These changes in number of siblings over time may have important implications for individuals' education by altering resource competition, and causing social upheaval in the household, both of which may affect whether a given child is allocated sufficient time, money and attention needed to succeed in the education system. Increasing sibsize during childhood may reduce individuals' education if, as resource dilution theory would predict, increasing the number of siblings living in the household decreases the resources allotted towards each child's education. Not considered by the resource dilution theory, however, is the idea that siblings are a source of income and help around the household, and that resources may be lost if they leave the household. When siblings move out, the child left behind may need to drop out of school to help with housework, home businesses, or to provide extra income by working outside the

home, particularly in poor households where resources are scarce. I propose in this study, then, that both the addition and loss of siblings may have the potential to decrease educational attainment. Further, child development research suggests that changes in the home environment can negatively affect individuals if it is harder for parents to provide attention and other resources to their children in times of change and instability (Baydar, Greek, and Brooks-Gunn 1997; Baydar, Hyle, and Brooks-Gunn 1997). When sibsize changes competition for educational resources may become stronger as current resources are stretched to accommodate the immediate addition or loss of a sibling.

At the same time, there are potential positive effects of change in sibsize on individuals' educational attainment. Extending the resource dilution hypothesis, the loss of a sibling may lead to less resource competition with the household, increasing the educational possibilities of those left behind. However, it may also be that the addition of siblings through moving back into the household has a positive effect on educational attainment, if these siblings bring with them resources (time, money or knowledge) or provide needed labor to the household. Finally, adding a sibling either through birth or immigration may improve children's social skills (Downey and Condron 2004), or provide more opportunities for tutoring (Zajonc 1976; Zajonc 2001), which may be beneficial to schooling and learning processes.

These theoretical predictions suggests that change in sibsize could have multiple and competing effects on educational attainment. Further complicating the question of sibsize effects is the issue of timing. It may be that changes in residential sibsize have different effects depending on when the changes occur in a child's life course. This may be due to competition for resources working differently at different stages of childhood. One thought is

that early social context matters most to individuals' educational life course because it encompasses key developmental and cognitive milestones (Alwin and Thornton 1984; Guo 1998; Ou 2005). The formation of cognitive skills may influence individuals' school trajectories and ultimate educational attainment. Further, given the cumulative nature of the educational system, in early childhood individuals are learning the most basic skills that may set them on educational pathways that are difficult to change (Entwisle and Alexander 1989; Entwisle, Alexander, and Olson 2005). It may be, then, that early social contexts, and in this case changes in sibsize, set individuals on paths of cumulative advantage or disadvantage due to cognitive and schooling experiences produced early on. According to this perspective, changes in sibsize that occur in early childhood are important to educational attainment at age 19.

On the other hand, changes in sibsize may be more important later in children's educational life course when school becomes more selective due to increased real and opportunity costs. Most children attend primary school, but fewer and fewer attend beyond that as children drop out temporarily or permanently to work, start a family, or because they feel they cannot keep up with the rigors of school at these higher levels. The change in the number of siblings one has may reduce education in later stages due to resource dilution if the household cannot provide the economic and time resources needed to support a child in meeting the challenges of each educational stage.

Finally, in studying the effects of changing sibsize on educational attainment, I consider how the effects may differ by changes in number of younger versus number of older siblings. Although not explicitly considered in the resource dilution hypothesis, it may be that siblings who are younger use more resources than older siblings. This may happen if

younger siblings may need more attention, and take on fewer household or work responsibilities. Younger siblings may also reduce cognitive stimulation in the household, especially while they are in infancy and early childhood (Zajonc 1976; Zajonc 2001). Under these conditions, one would predict that adding younger siblings to the household during childhood would decrease educational attainment, and losing younger siblings would increase educational attainment. However, if younger siblings leave the household to pursue educational opportunities, they may require more parental support (money and time) living elsewhere, to the detriment of the index child's education. Furthermore, there is some evidence to suggest that younger siblings may provide an opportunity for the index child to engage in tutoring, with potentially beneficial effects on schooling (Zajonc 1976; Zajonc 2001). This positive effect would be expected to occur when the index child's younger siblings are of school age, and the index child is old enough to teach them (mid- to late childhood).

Changes in number of older siblings may have similarly complex effects on a child's education. Older siblings may require more resources than younger siblings because they are further along in school; so that, increasing the number of older siblings in the household (through return migration) increases competition for schooling resources. Or, it may be that older siblings work and provide additional labor to the household that can be used to support the index child's education. If that is the case, losing older siblings may leave the index child in charge of additional work, to the detriment of their schooling. Again, the changes in younger and older sibsize may depend on timing, reflecting both the index child's and their own life course stage.

These complex aspects of sibsize are incorporated in my study of the relationship between changes in sibsize and educational attainment in Cebu, Philippines. The developing country context allows me to test these dynamic sibsize effects because family size is generally higher, there are fewer cases of only-children, and there is more movement of siblings in and out of the household compared with developed country settings. Further, in the context of poverty (40% of Filipino households are below the national poverty line), household resources may not be sufficient to support all children in obtaining high levels of education. The movement of siblings into and out of a child's household may thus mean changing access to resources with important implications for their progress in the educational system. Finally, testing theoretical ideas formed based on developed country research (i.e., resource dilution and life course theories) in a developing country context provides additional evidence as to the generalizability of these perspectives to developing country settings. Thus, this study advances our current thinking on how childhood households affect individuals' educational attainment by delineating multiple aspects of the dynamics of sibsize during childhood and their affects on educational attainment in a developing country context.

I now turn to a review of the empirical literature that informs this study.

Empirical Evidence

A literature review on the effects of siblings suggests evidence supporting the resource dilution hypothesis (Steelman, Powell, Werum, and Carter 2002), and a more recent comparative study finds negative effects of number of siblings on academic achievement across 30 countries (Marks 2006). Some research, however, casts doubt on the negative effects of sibsize: research controlling for time invariant unobserved parental differences have found no effect of sibling size on children's cognitive skills (Guo and VanWey 1999;

Rogers, Cleveland, van den Oord, and Rowe 2000). Thus, there is still some debate in the literature about the causal effects of sibsize on individual outcomes. This may be due, in part, to the vast majority of empirical studies using cross-sectional and biologically-based assessments of sibsize. It may be that adding and losing siblings have different effects; or, that the effects of changing sibsize depend on the stage of childhood. The studies that find no effect of sibsize focus on cognitive ability (as opposed to educational attainment), a specific stage of childhood, and do not assess the full extent of changes in sibsize (in terms of both losses and additions of younger and older siblings). It may be that the causal effect of sibsize on individuals' lives is more complex than indicated in the current body of sibsize literature.

Few studies have assessed whether and when siblings are present in the index child's household, or the dynamic effects of sibsize over time. Three studies have looked at change in sibsize and its effects on cognitive development. Two of the papers use longitudinal data to test the effects of adding a sibling through birth on a child's behavior and verbal and reading test scores in within the first 6 years of childhood (Baydar, Greek, and Brooks-Gunn 1997; Baydar, Hyle, and Brooks-Gunn 1997). They find negative effects of adding a sibling on test scores, with the strongest effects in poor households. These papers suggest that adding a sibling early in childhood may have negative effects on educational outcomes, particularly when household economic resources are scarce. The third study assessed change in sibsize by the addition of a birth between average ages of 6 and 12, and found no effect on change in cognitive skills during these ages (Guo and VanWey 1999). Although the authors attribute the null effect to their ability to control for time invariant unobserved child and family differences, their results may suggest that adding a sibling during mid-childhood may be less important for cognitive development than adding a sibling earlier in childhood. Thus, these

three studies suggest that change in sibsize may be important (although they only assess additions through birth); and, that when these changes occur may matter for children's development.

Another study provides further evidence of timing effects, although it focuses on level rather than change in sibsize. Alwin & Thornton (1984) find that sibsize measured early (between birth and age 4) and late (between ages 15 and 18) in childhood are independently and negatively related to children's GPA, curriculum placement, and years of schooling (but not verbal ability) at age 19. In conducting further, indirect analyses they infer that early sibsize is more important than late sibsize for an individual's education, although this cannot be directly tested due to the high correlation between the sibsize measures over time (Alwin and Thornton 1984). Their paper suggests that sibsize at different stages of childhood may have independent and lasting effects on education, as well as the possibility that sibsize in early childhood exerts stronger effects on educational attainment than sibsize established later in adolscence. The main limitations of the study are the lack of sibsize measures between ages 4 and 15 (when a lot of changes may take place), and the measurement of sibsize as the number of live births to a woman. Changes in sibsize across childhood, and in particular whether siblings are living in the household at the later ages is not assessed.

Research investigating how the effects of sibsize differ by younger and older siblings is similarly limited. Research on birth order is one source of research that takes into account the age of the siblings relative to the index child. However, the measure (usually assessed at one point in time) combines the effects of number of younger and older siblings, making it difficult to disentangle whether it is the number of younger or older siblings that are driving the results. For example, one study found that being higher in the birth order (interpreted as

having more older siblings) had a large negative impact on education (Black, Devereux, and Salvanes 2004), while another found that later born children (interpreted as those with fewer younger siblings) are more likely to attend college and than those who were born earlier (i.e., had more younger siblings) (Steelman and Powell 1991). While the overall birth order relationship with education may be negative in both cases, one suggests that this is due to having more older siblings, while the other suggests that it is due to having fewer younger siblings.

A few studies have tested the effects of the number of younger and older siblings directly. Two studies find that number of younger and number of older siblings reduce schooling in the Philippines (DeGraff, Bilsborrow, and Herrin 1996) and China (Costello and Casterline 2002). Another study found a negative effect of number of younger siblings and a positive effect of number of older siblings on schooling outcomes. However, number of siblings was assessed by biological ties in a setting (Ghana) where less than half of school-aged children lived with both parents (Lloyd, 1993). Thus, many siblings may not have been living together. It is not clear whether increasing the number of older siblings residing in the same household as the index child would have similar positive effects. This limited research on younger versus older sibsize effects on education likely underestimates the effects of older siblings, if sibsize is assessed at one point in time after a sibling has moved out. The current research in this area does not assess how changing the number of younger versus older residential siblings affects individuals' education.

In sum, neither theory nor empirical studies have adequately defined the potentially complex aspects of changing residential sibsize during childhood and their effects on educational attainment. This is, in part, due to defining sibsize as the number of siblings an

individual has at a particular point in life (two time points at most). One problem with this operationalization of sibsize is that it lacks an identification of which siblings share or shared the individual's childhood household. Second, it characterizes sibsize as a static individual trait, missing changes in sibsize and potential differences in the effects of sibsize depending on when it is experienced. Another gap in the literature is the identification of differences in resource competition by younger and older siblings. Since the type of resource dilution may be different for changes in the number of younger versus older siblings (index children may receive less cognitive stimulation with younger siblings and fewer economic resources with more older siblings), it may be that differences in the effects of younger versus older sibsize depends on the stage of childhood.

My study furthers our current understanding of how childhood sibsize relates to individuals' social status by answering the following research questions: (1) How do changes in residential sibsize during childhood affect individuals' educational attainment; (2) Do these effects differ by stage of childhood when the changes occur; and (3) Do these effects differ by younger versus older siblings? The use of change in sibsize (rather than level of sibsize) is not only interesting conceptually, but also allows for a more direct comparison of the timing effects since change in sibsize is less correlated over time than level of sibsize. The effects of changes in sibsize at a particular stage of childhood, controlling for those changes at other stages, can be assessed directly in empirical models. I also separate out potential differences in timing effects by whether younger or older siblings were added to or left the household during different stages of childhood.

I now turn to an overview of the methodological approach I use to answer my research questions.

Methodological Approach

I provide a prospective, longitudinal approach to assessing how changes in sibsize over time during childhood affect individuals' education in age 19. To answer my research questions I use data from *Cebu Longitudinal Health and Nutrition Survey* (CLHNS), a cohort study that follows a group of randomly-selected individuals born in Cebu, Philippines in the same year (1983-84) over time into adulthood. The cohort data provide longitudinal information on children's household members and educational status from birth to age 19. The unit of analysis in this research is the child born between 1983 and 1984, referred to as the index child. I am able to construct measures of change in sibsize using household roster of siblings present in the index child's house at the following stages of the index child's life: birth (1983/84), age 2 (1986), age 8.5 (1991), age 11.5 (1994), age 16.5 (1998). The use of this prospective data provides an opportunity to investigate the dynamics of residential sibsize at regular intervals during childhood, and to assess how these changes in sibsize over time affect individuals' educational attainment. I use multivariate regression analysis to assess the statistical relationship between changes in sibsize during childhood and the index children's education at age 19.

The use of a single cohort followed over time is advantageous because it allows for the macro environment (including educational policies and general cultural value of education) to be in a sense "controlled" across households because all index children experience the same national context over time. This is important, since some research suggests that the macro setting may affect the relationship between sibsize and education (Pong 1989; Razzaque, Streatfield, and Evans 2007; Sudha 1997). However, using a single cohort does not allow me to disentangle the way that the larger macro context might affect

the relationship between sibsize and education. This is particularly a concern with the timing results, which aim to differentiate effects of changing sibsize at different stages of childhood. With a single cohort, I cannot determine whether differences in effects across stages are due to the age of the index child when the changes occur, or to unobserved differences in the macro contexts at different stages of childhood. For example, a recent study suggests that sibsize has stronger effects in countries as they move from high to low fertility status lower (Razzaque, Streatfield, and Evans 2007). The Philippines has experienced a decline in the total fertility rate from 5.0 (in 1980-85) to 3.5 (in 2003) during the index children's childhood (National Statistics Office Region 5 2005; United Nations 2004). If I find that changes in sibsize have stronger effects over time, I can not determine whether this is due to the increasing importance of resource dilution in later stages to individuals' educational attainment; or, to the decreasing fertility rate and thus increasing importance of sibsize in later years.

Although I cannot determine the role that the larger context plays in this relationship, researching the dynamics sibsize effects on educational attainment in the Philippines further adds to our understanding of the importance of this aspect of childhood for stratification processes. Findings from a developing country setting increases our ability to generalize theories based on U.S. and other developed country research to less developed contexts. This setting also provides the changes in sibsize needed to answer my research questions. Assessing determinants of individuals' education is an important issue in the Philippines, where educational attainment is relatively low, and individuals often arrive at the end of childhood less than a high school-level education. In the next section I provide more specifics about the setting and data utilized in this study.

Setting & Data

The Philippines is classified by the World Bank as a lower middle-income country. Although experiencing some periods of economic growth, the Philippines has remained stagnated economically in the past 20 years, in part due to various economic crises that have kept long run economic growth to a minimum (Lim 2000). The latest poverty figures show that almost 40% of the population lives below the national poverty line, and almost 15% lives on less than $1 per day (The World Bank 2004). Competition for resources among siblings is likely to be a reality in this setting, both for economic reasons, and because of the relatively large family size in the Philippines. Although fertility has been declining in the Philippines, during the childhood of the cohort under study, fertility rates were relatively high compared with rates in other Asian countries and the developed world (National Statistics Office 1999): the 1980-1985 total fertility rate (TFR) averaged almost 5.0, 4.6 in 1985-1990 (United Nations 2004), and to 3.5 in 2003 (National Statistics Office Region 5 2005).

There is also a strong cultural orientation towards education in the Philippines, with 95% of school-aged children completing primary school in 2002 and a 95% literacy rate in 1999 (The World Bank 2004). The provision of education is relatively equal across genders (The World Bank 2004), reflecting, in part, that Filipino households tend to be more gender-egalitarian than households in other developing countries (Haddad, Peña, Nishida, Quisumbing, and Slack 1996). Although cross-sectional studies suggest that more siblings mean less education in the Philippines, there is not yet a widespread belief that keeping families to two or fewer children is a prerequisite for higher family and individual achievement (Costello and Casterline 2002). In the Philippines, having multiple children, and educating them, is highly valued.

Although education is culturally supported, the ability of individuals to complete high school (or go on to college) in the formal education system is limited, and is, in part, reflected in the time it takes the average Filipino to get through school. The current public schooling structure in the Philippines consists of six years of elementary schooling (entry age 6, exit age 12) and four years of secondary school (entry age 12, exit age 16) (Commission on Higher Education 2001). Elementary schooling is further divided between primary and intermediate schooling, the former designed to take four years, and the latter two years. However, the actual entry and exit ages for elementary and secondary school do not necessarily coincide with those designated by the Department of Education. This is in part due to later initiation of schooling, as well as students taking longer than one year per grade to complete both elementary and secondary school (Department of Education 2003b). In 1983/84, the birth year of this study cohort, the average age of primary school entrance was seven, the average age of secondary school entrance was thirteen, and there was substantial variation in continuation and completion rates (King and Lillard 1983). Currently, students take, on average, almost seven and a half years to complete 6 years of elementary school and more than five and half years to complete four years of secondary school; and, boys take more than a year longer than girls to get through high school (Department of Education, 2003b).

Relatively few changes were made to the public school system during the lives of the children in this study. Any major changes felt during this time were at the upper level of education when, in 1994, the Commission on Higher Education (CHED) was established to supervise tertiary degree programs (Department of Education 2003a). Since this is a study of

a single cohort, any changes in the education system during the 18 years covered by this study were experienced by all of the children.

Thus far I have described the Philippines as a whole. The specific setting of this study is Metropolitan Cebu, which is located in the center of Cebu Island and consists of 270 administrative units—207 urban and 63 rural. In general, Cebu resembles the socio-economic and household patterns as the Philippines (Flieger 1994), and the most recent total fertility rate for the Cebu region (3.6) is very similar to the national TFR (3.5) (National Statistics Office Region 5 2005). The extent of poverty is evident in that 25% of households lacked electricity and 21.4% had no toilet facility in 1990 (Flieger 1994). In terms of educational infrastructure, Metropolitan Cebu offers both public and private schools from kindergarten to the university level.

The data used in this study are from the *Cebu Longitudinal Health and Nutrition Survey* (CLHNS). The CLHNS is a sample of children born in 1983-84 to all pregnant women from thirty-three randomly selected communities (17 urban and 16 rural) in the Metropolitan Cebu area (Adair and Popkin 2001). About 2,800 infants were followed-up bi-monthly during a 2-year period (1984-1986), and then again in 1991, 1994, 1998, and 2002 (Adair and Popkin 2001). Household, parental and individual child information was gathered consistently in all rounds of the survey. This allows for the development of a panel dataset, in which each child has multiple measures over time, at birth, infancy (mean age 2 years), early childhood (mean age 8.5 years), mid childhood (mean age 11.5 years), and late childhood/adolescence (mean age 15.5 years).

My study sample is 2117 children with valid educational attainment data, 69% of the original births. The children present in my sample represent a slightly lower level of maternal

education (7.0 vs 7.3) and higher number of siblings at birth (2.1 compared with 1.7) than the means for those excluded. No other child or household measures at birth differed in terms of their means at birth between my sample and those missing education data. My achieved sample size is further reduced by missing data on siblings, due to the child missing household information in one or more waves. To obtain the largest and most representative samples possible, I kept all children in the dataset so that those with valid data in some stages but not others would be included in the analysis. Those with missing sibling data at a given stage are dropped from that particular model, but their information is included in other analyses. Thus, the sample size depends on the stage of childhood being analyzed. My largest sample, 2023, is when changes in sibsize are averaged across stages, allowing for changes in sibsize to be assessed for all children who have at least two consecutive survey waves of valid sibsize data. The stage-specific samples range from 1999 in infancy (the birth-2 stage) to 1863 when all stages are included in the analysis.

A final issue in arriving at the achieved sample is dealing with missing data on the control variables. To reduce missing control variable data, I calculate variables such as parental education and age based on information in other waves of the survey. Missing data on household economic resources is more difficult to estimate, since it is likely to vary significantly over time. I replaced missing household economic data with data from the immediate preceding or subsequent survey. The remaining missing data were dealt with through case-wise deletion. This resulted in very few cases being deleted due to missing control variable data (about 20-30 cases per wave on average).

It should be noted that there is a concern that the missing data across stages of childhood may account for the results found, particularly since there are distinct sample sizes

for models based on different stages of childhood. To check this, I ran the models using the sample of children who were present in all waves. The results with this more limited sample are consistent with these results found using the stage-specific samples; thus, missing sibling data across waves does not seem to affect the results found (the author can be contacted for full results from the all-waves sample).

Table 3.0 below provides descriptive statistics for this sample of children.

Table 3.0: Descriptive statistics of sample

Variable	Mean	Std. Dev.	Min	Max
Years of education by age 19	9.54	2.70	0	14
% male	0.52	0.50	0	1
Mean # sibs (birth-age 16)	2.87	1.66	0	10.2
% first born	0.22	0.41	0	1
Maternal education at birth	7.03	3.30	0	18
Maternal age at birth	26.10	6.02	14	46
% moms married to same spouse at age 11/12	0.93	0.26	0	1
% in extended family at birth	0.39	0.49	0	1
% in extended family at age 11/12	0.20	0.40	0	1
# of household assets at birth	2.53	1.92	0	10
# of household assets at age 19	5.18	2.09	0	11

These statistics show that this sample is about half boys and girls, and 22% are first born children. Family size is variable across childhood, but averages almost 3 siblings per index child. Maternal education is low, averaging 7 years, and the index children show some intergenerational mobility, averaging 9.5 years of education by age 19. Most households in

this sample were poor at the birth of the index child, with an average number of household assets (e.g., TV, refrigerator, vehicle, etc.) of 2.5. The asset score also reflects some upward mobility, as the sample average number of assets by age 19 of the index child is 5. In terms of family structure, the vast majority of mothers remain married to the index child's father. Although 40% of the sample children were born into an extended family household, only 20% remain in an extended family by age 11 or 12. Thus, this sample consists of poor, relatively low educated, married parent households. Reflecting the national averages, education is lower and sibsize is higher for these children than those growing up in developed country settings. I now turn to a more detailed description of the variables of interest in this study: the index children's educational attainment at age 19 and changes in sibsize from birth to age 16.

Measurements

The outcome of interest in this study is children's educational attainment measured by grade attained by the last survey, when the children were on average 18.5 years old. At this point, most children have either finished their educational career (graduating high school or dropping out prior to finishing), or graduated and gone on to college. To reduce missing data, highest grade achieved (the dependent variable) was assessed using the latest data available from 1998-2002. A tracking survey was conducted in 2000, which collected limited information on the children, including last grade completed. Over 95% of the sample is based on highest grade completed by 2002, 4% on highest grade reported in 2000, and .04% on highest grade completed in 1998. A control variable accounting for the year when educational attainment was assessed is included in all analyses. Mean educational attainment for my sample (N=2117) is 9.5 years (median=11), with a range from 0 to 14 years. Roughly

90% of the sample children completed 6 years of elementary school, and 55% of completed high school by average age 18.6 years. Thus, almost half of the children in this sample arrived at age 19 with less than a high school degree. 17% of the sample went on to some form of higher education.

Change in sibsize is based on the difference in number of siblings in the index child's household at each time point. I created sibsize variables at each time point using a variable that defined the relationship of each household member to the index child. After identifying the siblings (and their ages) who were present in the household, I summed the number of younger and older siblings living in the index child's household at birth, age 2, and average ages 9, 12, and 16. My sibsize measures potentially include half-siblings, since no distinction was made between full and half siblings. However, in this setting there is only a limited occurrence of step-families; thus, it is assumed that most siblings identified here are full siblings. Using the number of siblings identified at each time point, I then developed the following sibsize change variables by differencing sibsize for the following pairs of stages of childhood: birth-age2, age 2-9, age 9-12, and age 12-16. This resulted in the following stage-specific linear variables used in the statistical analyses: number of younger siblings that entered the household, number of older siblings that entered the household, number of younger siblings that left the household, number of older siblings that left the household.

To obtain a view of the extent of change across childhood for each childhood, I created childhood average change variables by averaging the number of siblings lost or gained across the four stages of childhood. The resulting variables, childhood change in each type of sibsize, provide holistic measures of childhood changes in sibsize that include the fluctuations in sibsize across stages of childhood. I prefer this average measure to a straight

change variable (# of sibs at age 16 minus # of sibs at birth) because it picks up changes that occur throughout childhood that may be missed in a change measure that only uses number of siblings at birth and age 16. I also chose to average rather than sum the number of siblings added or lost across stages because it allows me to consider children who are present in some but not all stages, increasing my sample size and allowing for more of the original sample to be included in the analysis.

Changes in sibsize during childhood are quite common in this sample of Filipino children (see Table 3.1 below). Since there are only 28 sample children who do not have siblings, virtually all children have experienced some change in sibsize during childhood[4]. The percentages in Table 3.1 represent the percent of the sample children who experienced each type of change in sibsize, for all of childhood and by stage of childhood[5]. Although my variables of interest are number of younger/older siblings added or lost (linear variables), to provide an overview of the extent of change in the sample, Table 3.1 provides the percent of children experiencing any change (i.e. 1 or more siblings lost or gained) by childhood stage.

The summary statistics for change in sibsize during all of childhood (Table 3.1, column 1) illustrate that gaining a younger sibling was the most common sibsize change experience, with 78% of the sample children gaining at least one younger sibling between birth and age 16. Although less common, also present in this sample are older siblings moving back into the index child's household: 20% of index children gained an older sibling at some point during childhood. The loss of siblings, which is ignored in other sibling

[4] This is due, in part, to the wide-held belief in the Philippines that growing up with no siblings is unhealthy Costello, Marilou P. and John B. Casterline. 2002. "Fertility Decline in the Philippines: Current Status, Future Prospects." New York.

[5] The variables presented in Table 3.0 are dummy variables measuring whether a child had at least one sibling (separated by younger and older) enter or leave the household.

research, is an important component of sibsize change as well. In this sample, 47% of children lost an older sibling (presumably due to residential mobility or migration) and 13% lost a younger sibling at some point during childhood.

Table 3.1: Percent of sample children experiencing changes in sibsize during childhood

Type of change in sibsize	**% of sample experiencing the change**				
	All of childhood	By Stage of Childhood			
	Birth-Age 16	Birth-Age 2	Age 2-Age 9	Age 9-Age 12	Age 12-Age 16
At least one younger sib gained	79%	23.6%	65.5%	26.2%	20.2%
At least one older sib gained	20%	3.2%	2.9%	4.4%	3.3%
At least one younger sib lost	13%	0%	1.4%	1.8%	2.7%
At least one older sib lost	47%	6.5%	17.0%	14.5%	25.4%

Table 3.1 also shows the distribution of these changes over time. Early childhood is a time of extensive change for these children, which is mainly due to the addition of a younger siblings—almost 90% of the sample children experience the addition of at least one younger sibling before age 9. There is also a surprising amount of change in sibsize during later stages of childhood, from age 9-16, when 46% of children gain at least one younger siblings, and 40% lose at least one older sibling. Although the nature of sibling change is not assessed here, when the changes occur indicates possible reasons for the change. For example, since the majority of younger siblings enter the household in the earlier stages of childhood, it is assumed that most of these are due to births. The loss of older and younger siblings is concentrated in the later stages of childhood, and is assumed to represent mainly residential mobility, migration, and new household formation, since siblings in the later years of the index child's life may be of the age to be leaving home for school, marriage or work. The

loss of a sibling may be due to death as well, especially in the case of the loss of a younger sibling.

These extensive changes in sibsize, with differences across children and for individual children over time, make it possible to answer my research questions. Next, I describe the specific models and regression analyses utilized to assess how these changes affect children's educational attainment.

Models & Statistical Analysis

To estimate the effects of changes in sibsize on educational attainment, I conduct multivariate linear regression analyses of the effects of the various aspects of change in sibsize on educational attainment. The basic model I estimate is:

$$y_i = B_0 + B_{1\ldots k}X_{i1\ldots k} + \theta_{1\ldots k}Z_{i1\ldots k} + \varepsilon_i \qquad (3.0)$$

where y=years of education; X=changes in sibsize; and, Z=control variables.

To isolate the effects of these changes in sibsize on educational attainment, I control for the variables that may affect sibsize and educational attainment. Child-specific controls include sex of the child and whether he/she is first born or not, and whether the child is living in his/her mother's household at ages 9 and 12. Household level controls assessed at birth include maternal education, maternal age, and whether the household speaks an ethnic languate. Several household-level controls are assessed at the beginning of each stage of childhood, including: number of siblings (controls for larger families having more changes in sibsize and potentially lower education), logged total household income, household asset index (sum of number of assets including: electricity, house, material of house, air conditioning, TV, tape recorder/CD player, refrigerator, fan, and car or jeepney); number of non-sibling children (<18); and adults in the household, and whether the child lives in an

extended or nuclear family[6]. I also control for whether the mother remains married to the same spouse as in earlier waves (assessed at ages 9 and 12). Finally, I include dummy variables at the community level to control for all differences among communities, such as fertility and education norms.

In conducting these analyses, I estimate models with the "standard" control variables, and then add controls for sibsize at the beginning of each stage and first born status in subsequent models. This allows me to assess whether changes in sibsize matter independent of the total number of siblings and one's birth order. In all models, I adjust the standard errors for clustering at the community level and for heteroskedasticity.

To gain an overall view of the effects of changes in sibsize during on educational attainment I regress educational attainment on average number of siblings lost or gained from birth to age 16 using my average change measures (and including controls). Then, to allow for difference in effects across stages, I conduct separate analyses of educational attainment on all changes in sibsize for each stage of childhood: infancy (birth-age 2), early childhood (ages 2-9), mid-childhood (ages 9-12), and late childhood (ages 12-16). The control variables are assessed at the initial time period for each stage. These models address both which types of change matter, and during which stage, allowing for cumulative effects of changes that have previously taken place. To further assess the timing effects, I run a final model of educational attainment on all changes across all stages. This final model includes control variables mainly from baseline, with select variables (e.g., whether the mother is married to the same spouse and whether child is still in maternal household) assessed later in childhood. This final model illustrates the effects of changes at each stage, controlling for changes

[6] In the average childhood models, sibsize, household income, and assets are measured as an average from birth to age 12.

occurring in the previous stages, and thus provides a view of the timing effects, taking out potential cumulative effects from previous changes.

Results

Change in sibsize and education

The first question to be answered from my results is whether change in sibsize matters for educational attainment, and if so, which types of change. Table 3.2 shows the effects of the average number of siblings that have been added or lost from birth to age 16[7]. The results reviewed here are from Model 3, which has the full set of controls, including average number of siblings from birth to age 12[8] and birth order. Although average sibsize and first born are somewhat correlated with the changes that occur throughout childhood, adding these controls does not significantly alter the results (see Model 1 and 2 compared with Model 3) and provides a view of how changes in sibsize affect individuals' education independent of sibsize and birth order.

The first result from this table is that clearly changes in sibsize from birth to age 16 negatively affect educational attainment.[9] The one potential positive effect (adding older siblings to the household) is statistically insignificant, due, in part, to the relative infrequency of this type of change in sibsize. In terms of types of change that matter, adding younger siblings and losing a younger or older siblings during childhood significantly reduce a child's educational attainment. Model 3 suggests that each additional younger sibling that is added to

[7] Significant coefficients on independent variables of interest are bolded to highlight the results discussed in the text.

[8] Average sibsize is calculated up to age 12, excluding sibsize at age 16, since sibsize at age 16 cannot theoretically influence changes that occurred up to age 16. The results were not affected by including number of siblings at birth rather than average number of siblings from birth to age 12.

[9] Interactions between sex of the child and change in sibsize were tested and found statistically insignificant for all changes.

the household during childhood (through births or moving back home) reduces the index child's education by almost a half of a grade (-0.49). The loss of a younger sibling during childhood (presumably through residential mobility or migration rather than death) has an even stronger effect than adding a younger sib, reducing the index child's education by almost a grade and a half (-1.47). Each older sibling lost, in turn, reduces a child's education by more than half a grade (-0.59).

It should be noted that the coefficients across the types of changes in sibsize are statistically different (assessed through an F-test), with the loss of younger siblings mattering the most, followed by the loss of older siblings. Further, these effects are additive, since the effect of each type of change is estimated controlling for the other types of sibsize changes. Based on the full sample estimates, this means that children who experience, for example, a younger sibling birth and a then a younger sibling moving out may have their education reduced by almost 2 full grades (1.47+.49). Further, these coefficients represent linear effects, or the unit change in education when adding or losing one sibling. The effect on education may be more severe if children lose more than one sibling. For example, the loss of two younger siblings at some point between birth and age 16 results in a potential loss of almost 3 grades (1.47+1.47).

It is interesting to note that the effects of change in sibsize remain statistically significant even when adding controls for average sibsize and birth order. I also tested for interactions between each type of change with sibsize and first born, which were not statistically significant and the model fit did not noticeably improved by including interaction terms. This suggests that change and level of siblings are additive rather than interactive. In other words, changing the number of siblings living in a child's household has important

effects on their education independent of the number of siblings a child had during childhood, and independent of his/her birth order.[10]

Table 3.2: Effects of average childhood changes in sibsize on educational attainment

		With average sibs	With first born
	(1)	(2)	(3)
Change in Sibsize			
# of younger sibs gained	**-0.458**	**-0.380**	**-0.486**
	(0.119)**	**(0.135)****	**(0.139)****
# of older sibs gained	0.177	0.167	0.249
	(0.412)	(0.418)	(0.413)
# of younger sibs lost	**-1.437**	**-1.463**	**-1.469**
	(0.330)**	**(0.332)****	**(0.324)****
# of older sibs lost	**-0.587**	**-0.499**	**-0.586**
	(0.214)**	**(0.214)***	**(0.217)***
Child Controls[+]			
Average number of sibs (birth-age 12)	--	-0.057	0.000
	--	(0.045)	(0.052)
First born (vs other parity)	--	--	0.510
	--	--	(0.114)**
Age when education assessed	0.435	0.435	0.442
	(0.084)**	(0.085)**	(0.083)**
Male	-1.218	-1.220	-1.217
	(0.125)**	(0.125)**	(0.123)**
Living in mom's HH (ages 9 & 12)	0.814	0.825	0.824
	(0.295)**	(0.294)**	(0.298)**
Household controls[+]			
Log ave. HH income (birth-age 12)	0.074	0.096	0.078
	(0.069)	(0.071)	(0.073)
Average assets (birth-age 12)	0.317	0.314	0.317
	(0.041)**	(0.041)**	(0.043)**
Maternal education	0.162	0.158	0.155
	(0.024)**	(0.024)**	(0.025)**
Maternal age	-0.013	-0.006	-0.002
	(0.012)	(0.014)	(0.014)
Ethnic household	-0.174	-0.172	-0.194
	(0.273)	(0.274)	(0.270)
Extended family (vs nuclear)	0.133	0.115	0.090
	(0.145)	(0.145)	(0.146)
# non-sib kids (<18) in household	-0.006	-0.009	-0.034
	(0.043)	(0.031)	(0.042)
# adults in household	-0.046	-0.047	-0.048
	(0.031)	(0.043)	(0.031)
Mother married to same spouse (ages 9&12)	0.230	0.255	0.264

[10] There is some multicollinearity—the highest correlations are between the average number of older siblings lost and average sibsize (r=0.57) and between average sibsize and first born (r=-0.48). All other correlations among change in sibsize and average sibsize or firstborn are less than 0.25.

	(0.170)	(0.173)	(0.172)
Community dummy variables	*Coefficients not shown here for brevity.*		
Constant	-0.683	-0.880	-1.146
	(1.773)	(1.814)	(1.787)
Observations	2023	2023	2023
R-squared	0.30	0.30	0.30

+Measured at birth unless otherwise noted.

Robust standard errors in parentheses. * significant at 5%; ** significant at 1%

Change in sibsize and education—Does timing matter?

The second question to be answered whether there are different effects of these changes on education by stage of childhood. Table 3.3 provides initial assessment of this question by providing the regression results from stage-specific models, which estimated the effects of changes in sibsize during each stage of childhood—infancy, early childhood, mid childhood or late childhood—on education attained by age 19. It is clear that in terms of statistical significance, when modeled separately, the effects of change in sibsize differ across stage of childhood. Changes in sibsize during the first 2 years of life, for example, have no lasting consequences for educational attainment (Table 3.3, Model 1), while younger siblings added in early (Model 2) and late (Model 4) childhood reduce education by 0.18 and 0.38 grades, respectively. Losing younger siblings, in turn, is most important for educational attainment when these siblings leave the household during the index child's mid-childhood (between ages 9-12), with a -0.43 coefficient (Table 3.3, Model 3), and in late childhood (between ages 12-16) with a coefficient of -0.57 (Model 4). Finally, effect of the loss of older siblings is exerted mainly in mid childhood, when the index child's educational attainment is reduced by 0.35 grades for every older sibling lost (Table 3.3, Model 3).[11]

[11] The number of older siblings lost during late childhood (age 12-16) is the only change effect that significantly differs by gender. For boys, the effect is -0.158, while for girls the effect is +0.196; however, neither of these effects is statistically different from zero.

In the full sample, then, it seems that the negative effects of sibling additions and losses are concentrated mainly in the later years of childhood, between ages 9 and 16. In the early years, birth-age 9 only adding siblings to the household between the ages of 2 and 9 affect the index child's education later in life.

Table 3.3: Effects of changes in sibsize by stage of childhood on educational attainment

	Birth-Age 2 (1)	Age 2-9 (2)	Age 9-12 (3)	Age 12-16 (4)
Change in Sibsize				
# of younger sibs gained	-0.100	**-0.182**	-0.183	**-0.377**
	(0.118)	**(0.048)****	(0.114)	**(0.107)****
# of older sibs gained	-0.165	-0.085	0.113	0.063
	(0.220)	(0.140)	(0.239)	(0.240)
# of younger sibs lost	0.000	0.349	**-0.434**	**-0.573**
	(0.000)	(0.336)	**(0.198)***	**(0.201)****
# of older sibs lost	-0.099	-0.063	**-0.351**	-0.035
	(0.116)	(0.089)	**(0.108)****	(0.100)
Child Controls[+]				
Average number of sibs	-0.083	-0.084	-0.113	-0.119
	(0.050)	(0.051)	(0.051)*	(0.047)*
First born (vs other parity)	0.301	0.314	0.335	0.264
	(0.146)*	(0.135)*	(0.121)**	(0.120)*
Age when education assessed	0.454	0.461	0.489	0.537
	(0.107)**	(0.111)**	(0.072)**	(0.075)**
Male	-1.311	-1.333	-1.241	-1.228
	(0.133)**	(0.136)**	(0.131)**	(0.133)**
Living in mom's household (ages 9 & 12)	--	--	0.346	0.603
	--	--	(0.481)	(0.564)
Household controls[+]				
Log tot HH income	0.152	0.222	0.158	0.087
	(0.059)*	(0.060)**	(0.112)	(0.072)
Household assets	0.139	0.145	0.267	0.287
	(0.032)**	(0.033)**	(0.035)**	(0.033)**
Maternal education	0.224	0.208	0.153	0.153
	(0.023)**	(0.024)**	(0.024)**	(0.022)**
Maternal age	0.018	0.009	0.022	0.010
	(0.016)	(0.016)	(0.013)	(0.013)
Ethnic household	-0.533	-0.509	-0.133	-0.033
	(0.416)	(0.405)	(0.329)	(0.266)
Extended family (vs nuclear)	0.088	-0.136	0.108	0.147
	(0.152)	(0.133)	(0.142)	(0.127)
# non-sib kids (<18) in household	-0.065	-0.043	-0.065	-0.099
	(0.038)	(0.066)	(0.118)	(0.128)
# adults in household	-0.070	-0.088	-0.123	-0.018
	(0.046)	(0.030)	(0.050)*	(0.064)

Mother married to same spouse (ages 9 & 12)	--	--	0.353	0.122
	--	--	(0.286)	(0.178)
Community dummy variables	*Coefficients not shown here for brevity.*			
Constant	-1.019	-0.998	-2.586	-2.945
	(1.904)	(2.201)	(1.651)	(1.588)
Observations	1999	1969	1983	1912
R-squared	0.25	0.26	0.35	0.36

+Measured at beginning of interval unless otherwise noted.

Robust standard errors in parentheses. * significant at 5%; ** significant at 1%

Another aspect of the timing question is whether these changes in sibsize across the different stages of childhood have independent effects on educational attainment. Of particular interest is whether the effects of sibsize change in the later stages of childhood are independent of changes that occurred earlier; and, whether sibsize changes in early childhood work through changes in later stages to affect educational attainment. I address these questions by conducting regression models with changes from multiple stages of childhood included in one model. The results, provided in Table 3.4, illustrate the effects of changes in sibsize when educational attainment is regressed on: infancy-early childhood changes in sibsize (Model 1); infancy-mid-childhood changes in sibsize (Model 2); and, infancy-late childhood changes in sibsize (Model 3).

Table 3.4 suggests that most of the stage-specific effects hold, even when controlling for changes that occurred in other stages. The effects that remain strong and significant, even when changes in all other stages are controlled (Table 3.4, Model 3) are: (1) gaining younger siblings in early childhood; (2) losing older siblings in mid-childhood; and, (3) losing younger siblings in late childhood. Further proof of the timing effects is that all of the significant effects are statistically different from the coefficients in other stages, suggesting that change does affect children differently depending on when it occurs during childhood.

The two effects that change size and significance with the inclusion of changes in other stages are: (1) the effect of losing younger siblings in mid childhood (age 9-12); and, (2) the effect of adding younger siblings in late childhood (age 12-16). Both of these effects become insignificant in the full model. In the case of losing younger sibs in mid-childhood, the effect decreases from -0.43 to -0.33 and becomes insignificant when lagged sibling changes are included (Table 3.4, Model 2). The effect drops further to -0.16 when late childhood changes are added (Model 3). It seems that losing siblings in mid childhood might be mediated out by the inclusion of subsequent changes in sibsize, perhaps if losing a younger sibling in mid childhood affects education by increasing the chance of losing another in late childhood. This may occur, for example, if the younger sibling who leaves earlier on establishes social networks (a place to live near school, or work opportunities) that ease subsequent migration by other siblings. The effect of adding a younger sibling in late childhood, the other effect that becomes insignificant, remains somewhat strong in size when past changes are included. The resulting insignificance may be due in part to the effect representing the cumulative effect of past additions of younger siblings. Or, it may be due to correlation with changes in the previous stages. For example, gaining younger sibs during early childhood is correlated with gaining younger sibs in mid and late childhood ($r = 0.3$ for each).

In sum, the results from Table 3.4 provide us with evidence that some sibsize changes over time are additive, and that children's educational attainment may be affected by multiple changes across childhood. When thinking about how the dynamics of sibsize during childhood affect one's education, we need to consider that children may experience the addition or loss of multiple siblings, and these changes at multiple times during childhood.

For example, in this sample, 7.7% of children gain at least one younger sibling in early childhood (age 2-9) and lose at least one older sibling in mid-childhood (age 9-12); 17.3% gain siblings in both early and late childhood (age 12-16); and, 9% gain both younger siblings in early childhood and the loss of a younger sibling in adolescence. Over the entire period of childhood, multiple changes may occur, with potentially compounding effects on education. For example, the loss of two older siblings in mid childhood (-0.45x2=-0.9) and the loss of a younger sibling in late childhood (-0.74) can reduce a child's education by almost two grades (based on coefficients from Table 3.4, Model 3).

Table 3.4: Effects of changes in sibsize by stage of childhood, including lagged effects

	Partial Effects		
	(1)	(2)	(3)
Change in Sibsize Birth-Age 2			
# of younger sibs gained	-0.193	-0.166	-0.161
	(0.128)	(0.130)	(0.127)
# of older sibs gained	-0.066	-0.049	-0.050
	(0.217)	(0.223)	(0.230)
# of younger sibs lost	0.000	0.000	0.000
	(0.000)	(0.000)	(0.000)
# of older sibs lost	-0.092	-0.157	-0.177
	(0.133)	(0.131)	(0.139)
Change in Sibsize Age 2-9			
# of younger sibs gained	**-0.221**	**-0.187**	**-0.146**
	(0.044)**	**(0.051)****	**(0.054)***
# of older sibs gained	-0.041	0.018	0.013
	(0.162)	(0.157)	(0.141)
# of younger sibs lost	0.324	0.359	0.358
	(0.347)	(0.381)	(0.371)
# of older sibs lost	-0.032	-0.076	-0.088
	(0.102)	(0.109)	(0.119)
Change in Sibsize Age 9-12			
# of younger sibs gained		-0.219	-0.177
		(0.119)	(0.114)
# of older sibs gained		-0.053	-0.012
		(0.338)	(0.331)
# of younger sibs lost		-0.294	-0.150
		(0.244)	(0.234)
# of older sibs lost		**-0.464**	**-0.492**
		(0.126)**	**(0.125)****
Change in Sibsize Age 12-16			
# of younger sibs gained			-0.206

			(0.107)
# of older sibs gained			0.452
			(0.240)
# of younger sibs lost			**-0.540**
			(0.202)*
# of older sibs lost			-0.136
			(0.097)
Child Controls[+]			
Number of sibs	-0.067	-0.009	0.009
	(0.054)	(0.057)	(0.059)
First born (vs other parity)	0.435	0.505	0.562
	(0.134)**	(0.131)**	(0.137)**
Age when education assessed	0.407	0.406	0.458
	(0.100)**	(0.096)**	(0.091)**
Male	-1.264	-1.288	-1.274
	(0.135)**	(0.134)**	(0.139)**
Living in mom's HH (ages 9 & 12)	0.958	0.817	0.750
	(0.303)**	(0.294)**	(0.290)*
Household controls[+]			
Log tot HH income	0.115	0.137	0.132
	(0.060)	(0.060)*	(0.066)
Household assets	0.121	0.116	0.108
	(0.030)**	(0.030)**	(0.029)**
Maternal education	0.218	0.208	0.202
	(0.024)**	(0.024)**	(0.024)**
Maternal age	0.003	0.006	0.010
	(0.016)	(0.016)	(0.017)
Ethnic household	-0.318	-0.281	-0.372
	(0.361)	(0.340)	(0.342)
Extended family (vs nuclear)	0.138	0.156	0.148
	(0.159)	(0.160)	(0.159)
# non-sib kids (<18) in household	-0.031	-0.073	-0.066
	(0.047)	(0.034)*	(0.047)
# adults in household	-0.071	-0.030	-0.036
	(0.034)*	(0.046)	(0.034)
Mother married to same spouse (ages 9 &12)	0.271	0.215	0.137
	(0.191)	(0.185)	(0.181)
Community dummy variables	*Coefficients not shown here for brevity.*		
Constant	-0.474	-0.385	-1.188
	(1.945)	(1.936)	(1.868)
Observations	1902	1899	1863
R-squared	0.27	0.28	0.28

+Measured at birth unless otherwise noted.

Robust standard errors in parentheses. * significant at 5%; ** significant at 1%

Table 3.5 summarizes the results, showing the effects of average childhood changes in sibsize and stage-specific models that have significant effects (all but infancy). The stage-

specific models are separated into the total effects versus direct effects when changes in sibsize from the other stages are present. As Table 3.5 illustrates, this study found that the effects of sibsize on education are entirely negative and strongest when considering the average number of siblings lost or gained throughout childhood. This indicates support for the idea that changes over time are additive and cumulate into major disadvantages in educational attainment.

Table 3.5: Comparison of effects of changes in sibsize by stage of childhood (from Tables 3.2-3.4)

	Average changes birth-age 16	Stages with Significant Effects					
		Age 2-9		Age 9-12		Age 12-16	
		Total effects	Partial effects	Total effects	Partial effects	Total effects	Partial effects
Change in Sibsize							
# of younger sibs gained	**-0.486**	**-0.182**	**-0.146**	-0.183	-0.177	**-0.377**	-0.206
	(0.139)**	**(0.048)****	**(0.054)***	(0.114)	(0.114)	**(0.107)****	(0.107)
# of older sibs gained	0.249	-0.085	0.013	0.113	-0.012	0.063	0.452
	(0.413)	(0.140)	(0.141)	(0.239)	(0.331)	(0.240)	(0.240)
# of younger sibs lost	**-1.469**	0.349	0.358	**-0.434**	-0.150	**-0.573**	**-0.540**
	(0.324)**	(0.336)	(0.371)	**(0.198)***	(0.234)	**(0.201)****	**(0.202)***
# of older sibs lost	**-0.586**	-0.063	-0.088	**-0.351**	**-0.492**	-0.035	-0.136
	(0.217)*	(0.089)	(0.119)	**(0.108)****	**(0.125)****	(0.100)	(0.097)
Observations	2005	1969	1863	1983	1863	1912	1863
R-squared	0.30	0.26	0.28	0.35	0.28	0.36	0.28

Table 3.5 also shows that the loss of younger siblings seems to exert the strongest negative effect on the index child's educational attainment. Much of this effect occurs when younger siblings are lost during late childhood, when important decisions are made about continuing on to high school. The negative effect of losing an older sibling (the second largest effect), however, seems to be accounted for mainly by those older siblings lost during mid-childhood. Comparing these two effects, it may be that losing a sibling in mid-childhood means delaying progress in elementary school, while losing a sibling in late childhood means

delaying high school, which may be harder to return to and thus have more serious consequences.

In addition to highlighting the fact that timing of changes in sibsize seem to matter for educational attainment, Table 3.5 also reminds us that age of the siblings matters. There is a difference between losing an older sibling and losing a younger sibling, which is reflected in the different coefficients in the average childhood model as well as in comparing stage effects. The large negative impact of losing younger siblings may be due to the fact that they move out to pursue educational opportunities at the cost of schooling for the index child.

When older siblings move out, they also seem to take resources with them that reduce the index child's educational progress. The loss of an older sibling may result in new non-school responsibilities for the index child and less support for the index child's education due to limited economic or social (especially time) resources that remain in the household. The loss of older siblings may not be quite as harmful as the loss of younger siblings because older siblings take fewer resources with them when they leave (if they are going to work or marry, say, rather than attend school). Thus, it may be that the stage effects reflect both the life course of the siblings, as well as that of the index child. If older siblings leave when the index child is between 9 and 12 years old, they may be school age and perhaps pursuing educational opportunities. Several years later, when the index child is between ages 12 and 16, the older siblings who leave at that point may be past their schooling years, and thus have no negative effect on the index child's education.

It is important to note another possible explanation for the difference in effects of the loss of younger and older siblings found here. Since relatively few index children experience the loss of a younger sibling (13%) compared with the loss of an older sibling (47%) during

childhood, the large effect of the loss of a younger sibling may reflect a serious family issue that required the younger sibling to move out, or that the sibling died. It is possible, then, that the loss of a younger sibling may have such a strong negative effect on educational attainment because it reflects a major family problem or shock. The effect of losing an older sibling, however, is more likely related to normal family growth/aging transitions, and the changes in resource distribution accompanying those transitions.

Discussion

Household and family contexts during childhood affect individuals' cognitive and social development, and access to economic resources, with potentially important effects on educational attainment. Educational attainment, in turn, has important implications for individuals' well-being in childhood and success later in life. Childhood household/family contexts, then, can create lasting social inequalities. This paper focuses on a dynamic aspect of the childhood household/family context—number of residential siblings—and how changes in this context throughout childhood can affect individuals' education attained by age 19. Most research on sibsize provides only a snap-shot of children's lives and misses both changes in sibsize as well as differences in the effects of these changes depending on when they occur during childhood.

To advance sibsize literature and our understanding of the larger stratification process, I apply a life course perspective to the question of how sibsize affects education, giving attention to change and timing in estimating the relationship between residential sibsize and educational attainment. I answer three main research questions: (1) How do changes in residential sibsize during childhood affect individuals' educational attainment; (2)

Do these effects differ by stage of childhood when the changes occur; and (3) Do these effects differ by younger versus older siblings?

The results suggest that in this sample of individuals during childhood, changes in sibsize are extensive and, overall, reduce children's educational attainment. Changes in sibsize, both adding and losing siblings, have negative effects that are independent of number of siblings already existing in the household and a child's birth order. The negative effect of the addition of a younger sibling is consistent with the resource dilution hypothesis and empirical literature to date, which suggests that the changing environment and reduction in resources of all types that come with the birth of a sibling reduces a child's educational attainment. The effects of change in sibsize net of sibsize at the beginning of the stage further suggest that resources may be particularly stretched during times of change. This supports existing research on the negative effects of a sibling birth (Baydar, Greek, and Brooks-Gunn 1997; Baydar, Hyle, and Brooks-Gunn 1997).

The negative effects of decreasing residential sibsize, however, are not entirely consistent with the resource dilution hypothesis, and provide new evidence that siblings' movement out of the household may also strain resources. The static version of resource dilution theory suggests that fewer children in the household would increase the resources directed towards a given child's education. However, the dynamic approach to sibsize taken in this study suggests that reducing the number of siblings living at home may reduce resources provided to an index child. This may be due to the loss of siblings who were providing resource to the household (time or income), or due to siblings who leave taking parental resources (namely, financial support) with them. The negative effects of decreasing sibsize also may reflect increased household social disorganization in times of change, and

that this disorganization reduces parental support for the index child's education, at least temporarily. Given the cumulative nature of the education system, these temporary set backs may then lead to reduced levels of education attained by age 19.

The results presented here also show the importance of timing, as the effects of changes in sibsize differ across the stages of childhood. The addition of younger siblings, although generally negative for an individual's throughout childhood, is only significant in early childhood (age 2-9) when controlling for all changes across all stages (i.e., Table 3.4, Model 3). This finding is consistent with the idea that key cognitive development and school preparation (social and learning) is happening during this stage and reducing resources and the cognitive learning environment that comes with a younger sibling during this time can have lasting effects. More specifically, this stage includes children's entrance into school, which may be delayed with the addition of a new sibling. Delayed school entry, in turn, can lead to lower levels of educational attainment. Gaining younger siblings may reduce the cognitive stimulation, social/learning support, and economic resources adults can give to a child during this critical period.

The negative effects of the loss of siblings are mainly concentrated in the later stages of childhood, ages 9-16. These may be the stages when losing a sibling means a child must work to support the family, resulting in delays or early termination in schooling. This is particularly likely in poor households (i.e., the majority of this sample). Losing older siblings in middle childhood or younger siblings in late childhood have important negative effects on an index child's education. Given the common occurrence of losing an older sibling, these changes likely reflect normal household and family transitions as siblings age and more out to pursue educational or other opportunities. The effects of older siblings in mid-childhood

may be that these siblings are leaving to pursue school opportunities at the detriment of the index child's schooling. When an index child reaches adolescence, however, his/her *younger* siblings are of prime school age and may be leaving to pursue their education. As previously mentioned, though, the large effect of the relatively infrequent event of losing a younger sibling may be due, in part, to some sort of family shock, perhaps even the death of the sibling, which requires the index child to leave school.

The results provided here highlight new and important findings in relation to both sibsize and the broader family structure literature. First, the results show that *changes* in family structure may be as or more important than family structure itself. This is supported by the findings here that neither number of residential siblings nor birth order has an independent effect on educational attainment. Rather, the addition and loss of siblings over time seems to be the most significant for children's educational attainment. This not to say that sibsize does not matter, since those with more siblings are more likely to experience these changes. The findings emphasize that family structure, and sibling structure in particular, cannot be viewed statically. The movement of siblings into and out of the household is an aspect of children's lives that has been neglected in the literature to date, but, as the findings here suggest, may have important consequences for their well-being during childhood and social status as adults.

Second, when assessing change in sibsize, it is important to delineate the types of changes. If one were to view sibsize, and its change, linearly, the negative effects of both increasing and decreasing sibsize would not be adequately captured. Further, the focus on one type of change, namely births, leaves out a key demographic process that affects children's lives: sibling migration/residential mobility. In fact, decreasing sibsize (mainly due

to migration or residential mobility) seems to have stronger negative effects on a child's education than changes that ensue with additions to the family. Out migration of siblings is common in virtually all cultures for education, marriage, work and other reasons. Thus, more attention needs to be paid to how siblings leaving the household affect children who are left behind, and more generally to how siblings' life courses intersect with an individual's life course.

Finally, timing matters. These effects cannot be viewed accurately without taking into account when they occur in a child's life course. Delineating timing issues may be particularly important in understanding why some studies find negative effects of sibsize, and others do not. When viewed cross-sectionally or during one stage of childhood, one must be careful about interpreting the effects of sibsize and change in sibsize during childhood. This study suggests that in the case of educational attainment, family dynamics during multiple stages of childhood matter in different ways. The effects of adding a younger sibling seem most pronounced in early childhood, and most likely work through decreasing cognitive ability, with lasting consequences for educational attainment. Changes in sibsize later in childhood also matter, although more likely through straining economic and social resources needed to support a child in their schooling years. Changes in sibsize may affect individuals' educational attainment both through decreased cognitive ability and fewer resources needed to achieve higher levels of education. The other aspect of timing to consider is when changes occur in the life course of other members of the family. In this case, older and younger siblings have different effects on individuals' education over time, not only because of the age of the index child, but also due to the age of the sibling (in this case assessed in relative to the age of the index child) and their life course transitions.

Taken together, these findings illustrate the need for dynamic theories of family structure and their effects on individuals' lives. In terms of the sibsize literature, a dynamic resource dilution model may be particularly useful. Since changes in sibsize are generally negatively related to educational attainment, the results here support the continued use of a resource dilution theory. However, this theory needs to be adapted to consider the changing effects of siblings over time, and how siblings may take resources out of the household when they leave. Subsequent research should also build on the timing issues highlighted here to further conceptualize when and how household and family contexts most affect individuals' lives. As this study shows, early childhood contexts, although key to certain outcomes, are not the only contexts that matter. Given the past emphasis on early childhood, more studies of mid and late childhood, and those that compare contexts across stages, may yield further insights into how social contexts, and family structure in particular, may affect individuals' lives.

It should be noted that these conclusions are based on a sample that is limited in terms of size, historical context, household social status, and geographic location. This study provides a view of the effects of changes in sibsize for a sample of children who were born in Cebu, Philippines in 1983/84. Thus, the historical context that provides a backdrop for this study is one of a high but generally declining fertility rates, and relatively poor socioeconomic conditions. Of particular concern for these findings is that recent papers have compared the effects of sibsize across cohorts in developing countries, finding the effects to be stronger for later cohorts when fertility rates were lower (Razzaque, Streatfield, and Evans 2007). In the study of change in sibsize, however, it may be that higher fertility contexts during early childhood mean more sibsize transitions over time, and perhaps larger

reductions in educational attainment later on. Because this is a single cohort study, I am not able to determine whether the change and timing effects found here are somehow related to the initial or changing historical contexts as the children age.

A second issue of generalizability is that the households in this study tended to be poor, married couple households. Thus the results can only be generalized to children living in such households. It may be the changes in sibsize are particularly relevant for children in poor households because they experience more extensive change than those in better off households, perhaps as a reflection of family survival strategies. Further, in poor households, these changes may have more meaning for their educational attainment due to resource constraints. This will be an important idea to test in future research by comparing effects of changing sibsize on children in poor households across settings, and in samples with a larger range of socioeconomic conditions. The final generalizability issue is that this study was conducted in Cebu, Philippines. Thus, technically, the findings cannot be generalized beyond Cebu. However, because the households included in the sample have similar conditions as other poor households in the Philippines, the study may be somewhat generalized beyond Cebu to poor, married couple households in the Philippines.

The other limitation of this study is that although I was able to control for some unobserved effects at the community level, I do not account for unobserved differences among children and their families that may be causing the relationships found. Although it has been posited that unobserved parental attitudes and abilities affect both family size and children's education, it is not clear that the same factors would be related to *change* in sibsize and education nor the timing patterns found here.

Given the limitations of this study, then, empirical work remains to more fully understand the dynamics of childhood social contexts and the effects on individuals' education. The findings here suggest the potential importance of the dynamics of sibsize, and the need for subsequent research to further test the importance and timing of different aspects of change in sibsize across different settings, for different outcomes, and for households with a broader range of socioeconomic conditions.

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CHAPTER 4: CHILDHOOD INCOME & WEIGHT DISPARITIES IN THE TRANSITION TO ADULTHOOD

Introduction

The importance of childhood conditions to individuals' health is of interest to sociologists aiming to better understand the development of social inequalities over the life course (Conley and Bennett 2000; Conley and Bennett 2001; Haas 2006; Palloni 2006). Although there is a developing body of literature that highlights the importance of childhood resources for health later in the life course (Case, Fertig, and Paxson 2005; Foley 2000; Holland et al. 2000; Makinen, Laaksonen, Lahelma, and Rahkonen 2006), two recent reviews of the literature suggest the relative lack of research in this area given the importance of the topic (Conger and Donnellan 2007; Palloni 2006). Particularly lacking is a developing country view of these stratification processes that begin in childhood and cumulated over the life course.

The purpose of this paper is to provide new insights into the role household economic resources during childhood play in the development of health disparities, measured here by weight status. I investigate the role of childhood income in affecting individuals' BMI at average age 19 in the Philippines. At age 19, individuals are biologically adults, while socially they are beginning the transition to adulthood. This transition is a time when individuals make choices about jobs, further education, marriage and other aspects of life that will affect their future health and socioeconomic status. Young adults with exceedingly low

or high BMI may be forced into worse jobs or job conditions (Carr and Friedman 2005; Tunceli, Li, and Williams 2006), have lower health-related quality of life (Sach et al. 2007), and suffer from illnesses and earlier mortality (Flegal, Graubard, Williamson, and Gail 2005; Katzmarzyk, Craig, and Bouchard 2001; Khongsdier 2002), compared with individuals with normal BMI. In developing countries, the literature on cumulative disadvantage related to BMI finds that high BMI contributes to further health risks, such as increased blood pressure, in adulthood (Adair 2007; Mishra, Arnold, Semenov, Hong, and Mukuria 2006; Tesfaye, Nawi, Van Minh, Byass, Berhane, Bonita, and Wall 2007). Research across settings, then, suggests that abnormal BMI in early adulthood could put individuals on trajectories of socioeconomic and further health disadvantage with lasting effects throughout adulthood.

In this study I am particularly interested in comparing the different effects of childhood income on overweight and underweight status for the same sample of individuals. BMI at these two ends of the spectrum may represent distinct social and developmental processes. Underweight is often caused by lack of sufficient, quality food and repeated illnesses during childhood, which can lead to severe stunting and wasting during childhood and low BMI in adulthood. Overweight, in turn, is induced largely by changing eating and exercise patterns over the life course, and encompasses health behaviors that may be developed during childhood with lasting consequences for adult BMI. Given the potential difference in the processes that lead to being under-and overweight in the transition to adulthood, I investigate whether income during childhood has similar effects on these two weight outcomes.

Second, this research tests whether income at different periods of childhood has distinct effects on individuals' weight status at the transition to adulthood. Developmental

and early origins research would suggest the importance of income in infancy and early childhood, since social and health conditions early in life set have long term implications for adult health (Alderman, Hoddinott, and Kinsey 2006; Barker 1990; Gigante, Horta, Lima, Barros, and Victora 2006). However, it may be that income in later stages of childhood has more of an effect on individuals' BMI in adulthood by influencing behaviors in mid and late childhood when individuals begin to make more decisions about what to eat and the extent and type of physical activity (Chen, Martin, and Matthews 2006; Chen, Matthews, and Boyce 2002). Assessing childhood income at multiple points in time for underweight and overweight status may provide important insights into the role that income plays at different points in childhood in determining individuals' health as they are poised to enter adulthood.

I measure underweight and overweight status through objective measures of BMI, an indicator of body fat (weight/height^2) that is used cross-culturally to assess both underweight and overweight/obesity problems among children and adults. Overweight and obesity have become serious health problems in the developed world, and is increasingly relevant in developing country settings (Popkin and Gordon-Larsen 2004; Prentice 2006). At the same time, low BMI, or thinness, is still prevalent in developing countries where the poorest populations lack resources to provide their children with basic nutrition (The World Bank 2004), with lasting consequences for adult health (Khongsdier 2002)Hadden, 2003 #232}. In developing countries, then, both exceedingly high and low BMI are sources of health disparities that begin during childhood and can last a lifetime. Utilizing the Philippines as the setting of my study provides both a developing country view of the origins of health disparities, as well as a context of the dual nutritional health burden (FAO and Nations 2001). This context makes it possible to assess, with consistent measures and comparable

statistical models, whether and when economic resources during childhood influence the development of both underweight (undernourished) and overweight (overnourished) adults.

Theoretical Background

Although individuals experience multiple contexts during childhood, the household is one of the key contexts that influence individuals' growth and development due to the dependence of children on their households for social, economic and emotional resources needed for healthy development (Duncan, Boisjoly, and Harris 2001). Schools, communities and work places may become important contextual influences as individuals' age; however, during childhood (birth-teen years) the influence of these contexts is filtered through the household, which is largely responsible for the allocation of resources (of all types) to individuals during childhood.

One aspect of the household that plays an important role in children's healthy development is access to economic resources. Household economic resources have been theorized to affect health through the ability to afford the goods, services, and time required for providing adequate nutrition and health care to members of the household (Conger and Donnellan 2007; Mayer 1997). Health-promoting resources include sufficient amounts and quality of food, shelter, clothing, health care, water, or sanitation services. Children's health may also be tied to parental time, which is constrained in lower income households, needed to provide these material goods and care for their children. Furthermore, low levels of economic resources may put stress on parents, reducing their ability to provide proper physical and mental support to their children (Conger and Donnellan 2007; Mayer 1997). Childhood poverty may also pose risks to health during childhood and adulthood through the lack of good health behaviors learned earlier on due to time, money and knowledge

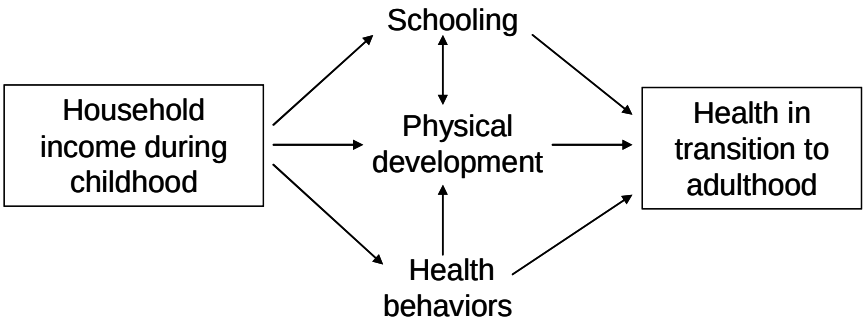
constraints. Less healthy parental behaviors may directly harm children's health, and socialize children into using poor health behaviors themselves with lasting consequences for their health in adulthood (Singh-Manoux and Marmot 2005). Finally, the lack of income poses structural constraints to households, reducing their access to safe and healthy communities. Children in poor households, for example, may have increased exposure to pollution, crime, and risky health behaviors (drug and alcohol use, early initiation of sexual activity, etc,) through household surroundings and schools in poorer areas (Leventhal and Brooks-Gunn 2000).

These theories have been developed mainly to explain how childhood circumstances affect child outcomes. Less work has been done to theorize about the pathways that lead from childhood economic conditions to adult health outcomes, the focus of this study. Although the purpose of this study is not to test pathways, conceptualizing how income during childhood may affect health later in the life course provides an important theoretical base for my study hypotheses.

I theorize three main pathways through which economic conditions may work to influence health in adulthood, including weight status (see Figure 4.0 below). First, household economic resource during childhood may induce health problems in children that cumulate and/or last into adulthood. For instance childhood poverty may lead to lack of quality food, which leads to undernutrition (stunting or wasting) and low growth rates during childhood and short stature or thinness as an adult. This is a biological pathway (labeled as "physical development" in Figure 4.0), where social conditions influence child health, and child health influences adult health. In the social science literature, this pathway is often referred to as "selection", where disadvantaged children become disadvantaged adults

(Conger and Donnellan 2007; Palloni 2006). In the case of weight disparities, income during childhood may set children on poor growth trajectories, which may result in either underweight (Walker, Chang, and Powell 2007) or overweight (Popkin, Richards, and Montiero 1996) status later in life.

Figure 4.0: Theoretical model of how childhood household income affects adult health



Second, poverty and poor health in childhood may lead to less schooling, which increases health risk further during childhood and as adults (Wickrama, Conger, Wallace, and Elder 2003). This is related to the life course process of cumulative disadvantage (DiPrete and Eirich 2006), where children who are sicker are less involved in school or get fewer years of schooling, and become even less healthy as they move through childhood. Thus, less schooling may induce poorer health during childhood, producing a cumulative disadvantage of sicker, less educated children arriving in adulthood with underweight or overweight problems. Although research is limited, there is some evidence to support this cumulative disadvantage process during childhood (Daniels and Adair 2004) and from childhood to adulthood (O'Rand and Hamil-Luker 2005). Schooling may be particularly important to nutrition-related outcomes, since schools provide physical activity and examples of healthy behaviors that may not be taught at home.

Finally, household income may be related to risky parental and child health behaviors. Unhealthy parental behaviors may include insufficient illness prevention or

treatment (i.e., not getting children vaccinated, not attending preventive visits, not following medical advice), purchasing and preparing unhealthy foods, and providing role models and home environments that promote sedentary behavior. These behaviors, and health problems that develop in children as a results, may lead to individual children's developing their own risky behaviors related to diet, exercise, substance abuse, and general care of their own health. These behaviors during childhood may affect adult health directly by producing risky health behaviors that last into adulthood, and through their influence on children's physical development, which then affects adult health (see Figure 4.0). The direct effects through individuals' own behaviors is related to the process of socialization during childhood, in which behaviors learned early on in childhood set individuals on paths of unhealthy behaviors into adulthood (Singh-Manoux and Marmot 2005). Socialization during later stages of childhood may also occur outside the household; however, household income may be an important determinant of this external socialization process by affecting the types of social networks and activities in which children are involved.

Although there is little research comparing the effects of childhood economic resources on different types of health outcomes in adulthood, I hypothesize that some health outcomes may be more affected by economic resources than others; and, that the theoretical pathways may work differently across health outcomes. In terms of weight outcomes, it could be hypothesized that underweight status in adulthood is more heavily influenced by household resources early in childhood, since its origins are often through early physical growth patterns (Alderman, Hoddinott, and Kinsey 2006; Martorell and Habicht 1986). Household income during childhood may influence adult underweight status through physical development and perhaps schooling mechanisms, which keep children on a low

growth path into adulthood. Overweight, to the extent that it is developed later during childhood may be less sensitive to household income during early childhood, and more affected by resources later in childhood and into adulthood. Although there are biological origins of adult obesity in childhood, the strongest pathway from childhood income to overweight status in adulthood is likely to be in conditioning individuals' health behaviors as they move through childhood. Childhood health behaviors, in turn, may be influenced by parental health behaviors (through socialization) that may differ by economic status.

Another issue related to how childhood economic conditions affect adult health is the consideration of timing, i.e., when individuals experience different economic conditions. Life course and development research suggests that social contexts grow and change over time as individuals age (Elder 1985), and empirical evidence suggests that household economic conditions in both developed and developing countries can vary substantially over time (Baulch and McCulloch 2002; Berthoud 2001; Dearing, McCartney, and Taylor 2001; Duncan, Yeung, Brooks-Gunn, and Smith 1998; Strohschein 2005b; Yaqub 2002). Thus, the household an individual is born into may differ dramatically from that in which he/she lives during mid or late childhood.

Literature related to "timing" of childhood contexts generally posits differences between "early" and "late" effects. Health deficiencies caused by the lack of resources early in childhood may have more serious consequences for childhood and adult status, since much of the cognitive and physical growth patterns of later life are set in infancy and early childhood (Martorell and Habicht 1986). Household resources may have the strongest effects during early childhood also because that is when individuals depend so much on the household context for their survival and well being (West. 1997). Other research suggests

that income at later stages of childhood may further affect adult health through cumulative health insults (Case, Lubotsky, and Paxson 2002) or by influencing health behaviors later in childhood and adolescence (Chen, Martin, and Matthews 2006; Chen, Matthews, and Boyce 2002).

Developmental theory highlights more specific stages in childhood that may be particularly sensitive to contextual influences (Bornstein 1989); and BMI-related research in particular suggests that infancy, early childhood (between ages 5 and 7), and mid childhood (puberty, around age 11 or 12) are key biological periods when changes in body composition may have lasting effects on individuals' risk of obesity as adults (Dietz 1994; Lawlor and Chaturvedi 2006). Thus, it may be that childhood household contexts matter more at these particular stages for adult overweight status, due to the nature of individual physical growth patterns.

In considering the potential timing effects of childhood household contexts on adult health, I hypothesize that, not only will timing matter, but that the timing effects may differ by whether the outcome is underweight or overweight status. Broader sociological studies that consider the timing of household contexts during childhood on social status outcomes (education and non-marital childbearing, mainly) suggest the importance in distinguishing "ability" from behavioral outcomes. In his study on the timing of poverty for educational outcomes, Guo (1998) distinguished outcomes related to individuals' ability (stable individual trait related to the rate of learning) and achievement (measure of what has been learned and performance). A key element of the distinction is that achievement is more behavioral in its orientation, while ability is a trait that is developed early on before individuals being to shape their own lives with decisions and motivation. The paper

suggested that early childhood environments, poverty in this case, may have more to do with setting individuals' on stable cognitive ability trajectories, while later childhood environments may alter individuals' behaviors related to school achievement (Guo 1998). Two other papers suggest similar ideas when interpreting the timing of childhood conditions on individuals' education and non-marital births in early adulthood (Duncan, Yeung, Brooks-Gunn, and Smith 1998; Hill, Yeung, and Duncan 2001). Both papers find that lower income in early childhood matters most for educational outcomes (perhaps representing mostly an effect on ability), while only household income in later childhood influences non-marital childbearing (a behavioral outcome).

The three papers taken together provide additional insight into the potential timing effects of childhood income for health outcomes. Early childhood income and other social contexts may be particularly important for individuals' cognitive and physical growth, with lasting consequences for adult health measures that are relate to "health potential." This includes measures that may be determined largely in the early years of life and carried on to adulthood through physical development pathways. Being underweight in adulthood may represent a measures of health potential to some degree, since those who arrive in adulthood with very low BMI (thin adults) may be those whose low height or weight in early childhood could not be made up later on. At the same time, childhood household contexts later in childhood may be more important for adult health outcomes that are more malleable to individuals' behaviors and decision making as they move through childhood. Being overweight as an adult may reflect more behavioral influences as individuals begin to make decisions about what to eat and physical activity, and thus be more affected by household income in later childhood periods. Thus, it is possible that income during childhood will have

distinct timing effects on the risk of being underweight and overweight due to the nature of the distinct BMI outcomes, one which represents more health potential established early in childhood (underweight), while the other (overweight) reflects individuals' behaviors as they move through childhood.

In considering these theoretical ideas, I pose multiple research questions aimed to move forward our understanding of the development of health disparities: (1) Are there lasting effects of childhood household income on individuals' weight status as they transition to adulthood (at average age 19)? (2) If so, does childhood household income affect the probability of being underweight and overweight equally? (3) Does household income at different periods in childhood differently affect weight status at age 19? (4) Are the patterns of timing effects similar for underweight and overweight outcomes? I now turn to a summary of the existing empirical research related to these questions.

Empirical Literature

Research on health disparities across settings points to the importance of the childhood contexts, in particular socioeconomic status, for health in both childhood and adulthood (Case, Fertig, and Paxson 2005; Foley 2000; Heaton, Forste, Hoffmann, and Flake 2005; Holland et al. 2000; Makinen, Laaksonen, Lahelma, and Rahkonen 2006). In developed countries, multiple studies show support for a positive relationship between economic resources during childhood and adult physical health (Case, Fertig, and Paxson 2005; Foley 2000; Li, Manor, and Power 2004; Lundberg 1993; O'Rand and Hamil-Luker 2005; Poulton, Caspi, Milne, Thomson, Taylor, Sears, and Moffitt 2002; Wickrama, Conger, Wallace, and Elder 2003). In terms of weight outcomes, the main findings in developed countries are related to overweight problems in adulthood. In these settings, being poor is

related to being overweight due to the consumption of low cost fast food and other unhealthy diet items and less physical activity among individuals in lower socioeconomic groups. The higher income groups are able to afford lower fat, healthier food, and engage in recreational physical activity (Popkin 2001; Popkin and Gordon-Larsen 2004). Much research has been conducted in developed country settings, including life-course oriented research, which has found a negative relationship between household socioeconomic status during childhood and adult overweight/obesity (James, Fowler-Brown, Raghunathan, and Van Hoewyk 2006; Parsons, Power, Logan, and Summerbell 1999; Power and Parsons 2000; Power, Graham, Due, Hallqvist, Joung, Kuh, and Lynch 2005).

In developing countries, there are fewer studies of childhood economic conditions on adult outcomes. Limited research on the effects of childhood economic resources on undernutrition (stunting) at the transition to adulthood suggests that household income at birth may help increase individuals' height in early adulthood in Brazil. Some of this effect may have been mediated by the inclusion of birthweight and gains in height by age 4, but even with these variables in the model, income at birth remained positively related to height at age 19 (Gigante et al. 2006). This suggests the lasting importance of household income at birth for low height at age 19, with some effects working through physical growth in early childhood.

Another relevant study considered the effects of childhood socioeconomic contexts on high BMI in Cebu, Philippines. The study found that socioeconomic status (an index including per capita income, assets and maternal education) at birth and changes between birth and age 18 increased BMI in males but reduced BMI in females at average age 21[12]

[12] The negative effect for girls may have been related to urban, high-class girls dieting to fit the thin western "movie star" body image (Adair, 2007)

(Adair 2007). This paper, suggests the importance of household economic resources during childhood for BMI at the other end of the spectrum; however, it does not explore the effects of household income per say, timing effects, or effects for underweight versus overweight status in adulthood.

Empirical research related to the timing of household income effects point to the importance of household economic resources during early childhood (Berthoud 2001; Cooper, Arber, and Smaje 1998; Duncan, Yeung, Brooks-Gunn, and Smith 1998; Guo 1998; Korenman and Miller 1997; Miller and Korenman 1994). However, there is also evidence to suggest that increasing economic resources later in a child's life may be important for putting children back on a normal health path (Adair 1999; Eckhardt 2004; Martorell, Khan, and Schroeder 1994; Tanner 1981); and that economic deficits later in childhood can further harm children's health (Case, Lubotsky, and Paxson 2002; Currie and Stabile 2003). One study to date has considered timing of socioeconomic status during childhood for adult health using the British Cohort Study. This study finds that social class at age 7 is a significant predictor of obesity among women, and social class at birth and age 23 to predict obesity among men (Power, Manor, and Matthews 2003). This provides evidence that, in a developed country setting, there may be timing differences in the effects of family socioeconomic status during childhood on adult overweight status. However, this study does not test for household income effects. Compared to the static measure of social class, household income may be even more likely to have different effects on BMI over time, to the extent that it changes more and may alter consumption and activity patterns throughout childhood more than social class.

In short, recent research on health disparities suggests the importance of childhood household socioeconomic contexts for adult health across settings. The developing country setting, however, provides an additional challenge of investigating the role of childhood economic resources on both traditional (i.e., underweight) and modern (i.e., overweight) health problems in adulthood. There is insufficient evidence to date to determine whether childhood economic conditions are important for adult health in developing countries, and in particular how the effects may differ for different types of health problems. Literature on the effects of household income at different points in childhood suggest potential for both early and late, and even stage-specific effects on health in the transition to adulthood. However, no clear picture has emerged regarding the relative importance of the timing of childhood income on health in early adulthood, nor whether these effects may differ by whether the health outcome reflects physical development versus behavioral influences.

My study builds on this body of research related to childhood contexts and adult health by investigating the role of childhood economic contexts from birth to age 16 in determining BMI-related weight status in early adulthood (age 19) in the Philippines. My first contribution, then, is to assess whether early adult health is affected by childhood household income in a developing country setting, where longitudinal research on health disparities is limited. Secondly, I also consider whether childhood household income has a similar effect for both underweight (traditional) and overweight (modern) status in adulthood. This provides insight into income effects for different health problems for the same sample of individuals. Finally, and most importantly, I consider how childhood household income at different points from birth to age 16 affect individuals' likelihood of being underweight and overweight when they reach age 19. In doing so, I assess whether there are key stages of

childhood when income matters most for later health outcomes, and whether the timing patterns are similar for underweight and overweight health problems.

To answer my research questions I use prospective, longitudinal data on a cohort of children born in 1983 in Cebu, Philippines. The Philippines context of the growing problem of overweight and the continued presence of underweight populations provides a dual nutrition burden that allows for investigation of whether childhood economic resources affect both traditional, development-related health problems (underweight) and modern, behavioral health problems (overweight). Below I describe in more detail the setting and data utilized to answer my research questions.

Setting & Data

The Philippines is a lower middle-income country in the East Asia-Pacific region. During the years that cover the study children's childhood (1983-1998) various economic crises have kept long run economic growth to a minimum (Lim 2000).The latest poverty figures (estimated sometime between 1997 and 2003) show that almost 40% of the population remain living below the poverty line and 15% live on less than $1 per day (The World Bank 2004). At the same time, however, the country has experienced some periods of economic growth and development that allowed it to meet its year 2000 goal of $1000 per capita ahead of time (National Statistical Coordination Board 2006), and other indicators (such as cell phone usage and consumption of processed and fast foods) suggest rapid development and modernization among some sectors of the population.

The dropping fertility and mortality rates and increasing non-communicable diseases since the late 1980s reflect the ongoing demographic transitions and economic and cultural changes, which have produced a "nutritional transition" in the Philippines. This transition can

be broadly characterized as rapid economic development and cultural changes institutionalizing new consumption patterns and ways of spending work and leisure time, leading to increased consumption of high fat, high sugar diets, reduced physical activity, and, ultimately, obesity and related illnesses (Popkin 2001; Popkin and Gordon-Larsen 2004). Unlike in developed countries, in poorer countries childhood household income at the *upper* level rather than lower level is more likely to lead to overweight and obesity status in adulthood. This is due to relatively better off households acquiring sufficient resources to purchase previously unattainable goods, such as oils and meat, sugary and processed foods, which lead to worse diets among those who can afford such "luxuries". In the Philippines (and most developing countries), then, overweight problems are developing in the higher income groups, while underweight problems continue to prevail among the poor.

Evidence of the nutritional transition and its resulting dual nutrition burden in the Philippines is clear: national survey data find that 36% of individuals aged 13-19 were underweight and 5.8% of individuals aged 13-19 were overweight or obese by 1998 (FAO and Nations 2001). This historical context provides the diversity and change in socioeconomic status during childhood (1983-1998) and the unique health context of the nutrition transition that allows me to compare the effects of income across stages of childhood for both traditional (i.e., underweight) and modern (i.e., overweight) health risks.

The specific setting of this study is Metropolitan Cebu. Metro Cebu is located in the center of Cebu Island and consists of 270 administrative units—207 urban and 63 rural—called baranguays, which are similar to census tracts. In general, Cebu follows the same socio-economic and household patterns as the Philippines as a whole, although it is characterized as one of the fastest economic growth areas in the country. At the same time, a

substantial fraction of the Cebu population lives in economic deprivation: in 1994 25% of households lacked electricity and 21.4% had no toilet facility in 1990 (Flieger 1994). The nutrition transition is evident in Cebu, which has both undernourished and overweight populations (Adair 2004; FAO and Nations 2001; Ricci and Becker 1996).

The data used in this study are from the *Cebu Longitudinal Health and Nutrition Study* (CLHNS), a study following a children born in thirty-three randomly selected communities (17 urban and 16 rural) in Metropolitan Cebu in 1983-84 (Adair and Popkin 2001). Within each community, all pregnant women were selected to participate, which resulted in 3080 singleton, live births (i.e., index children) enrolled in the study (Adair and Popkin 2001). Community, household, and index child information was gathered at multiple points during the index child's life: at birth, and averages ages two, 8.5 (referred to as 9), 11.5 (referred to as 12), 15.6 (referred to as 16) and 18.5 (referred to as 19) years of age. The majority of the sample children were present during all waves, although there was some attrition, as would be expected, during the 19 years. Of the initial births, roughly 2800 were followed bi-monthly through age 2 (Adair and Popkin 2001). Of the CLHNS children who were lost to follow up between 1983 and the 1994 survey, the 12-month anthropometrics of those who were lost to follow up or had missing data after 12 months did not differ significantly from those with valid data through 1994 (Adair 1999).

This high quality, prospective information on household resources and health status of children over the entire period of childhood and into adulthood provides an excellent opportunity to investigate the relationship between childhood income and health status in the transition to adulthood. The base sample for this study consists of all children who have valid

BMI data and who are not pregnant in 2002: 1966 children. The actual sample for each analysis depends on the waves of income data included in the analysis.

My dependent variables are based on measures of body mass index (BMI) in 2002 (average age 19). BMI, calculated as weight/height^2, is an objective measure of health assessed in the CLHNS by trained personnel. I construct two dependent variables based on BMI using international standard cutoffs for underweight and overweight (Cole, Bellizzi, Flegal, and Dietz 2000): (1) underweight dummy variable=1 if the individual has a 2002 BMI<18.5; and, (2) overweight dummy variable=1 if BMI>=25. Although adult BMI>=23 has been associated with risk of chronic disease among Asian populations (Inoue and Zimmet 2000), using the international standards for categorizing under- and overweight status makes the findings from this study more comparable to those conducted in other countries.

As can be seen in Table 4.0 below, by 2002 (age 19) the sample included both underweight and overweight individuals, although majority of individuals were considered to have "normal" BMI by these standards. Consistent with the nutrition transition, by 2002 roughly 28% of the individuals in the sample are underweight, while 5.5% enter adulthood as overweight or obese (Table 4.0). The sex-distribution of overweight status is relatively equal (5.8% of girls and 5.4% of boys), while a slightly higher percent of the girls are underweight than boys (31% and 25%, respectively) at age 19.

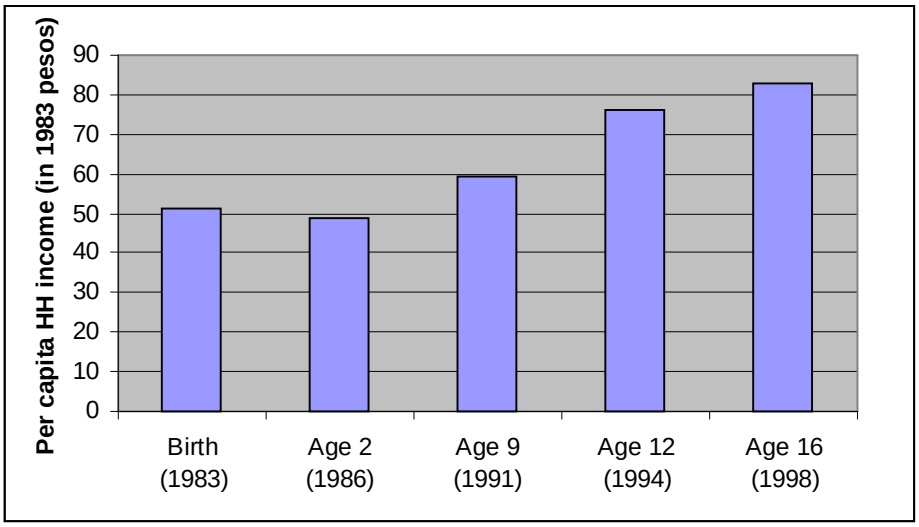
Table 4.0: Percent of sample by weight status at average age 19

	% underweight (BMI>=25)	**% overweight** (BMI<18.5)	**N**
Full sample	27.7%	5.5%	1966
Girls	30.5%	5.8%	892
Boys	25.4%	5.4%	1065

The key independent variable of interest is household income during multiple periods of childhood. Household income was collected consistently throughout the multiple survey waves by asking the respondent (usually mother of the index child) to enumerate income earned for different types of economic activities (wage labor, piece work, agricultural production, fishery, own business, unearned income) for each relative living in the household over age 6. Income for each of these activities was recorded somewhat differently depending on the activity, but all were adjusted and summed to represent each individual's weekly income, which was summed to produce weekly household income. For my analyses I create per capita weekly household income, dividing total household income (deflated to 1983 pesos) by the number of household members for each survey wave (birth-age 16). I also averaged these per capita income measures from birth to age 16 to produce an average childhood household income measure. All income measures are logged to adjust for their skewed distributions.

The mean household income across childhood for this sample is 46 pesos, or about $4, per person per week; thus, this is a sample of relatively poor households. Figure 4.1 shows how these households have fared over time, by illustrating mean household income levels from 1983 (at birth of the index children) to 1998 (index children at average age 16). The increase in per capita household income over time reflects, in part, the improving economic situation as households (and their earners) age. The increasing income over time also represents the changing historical context, as the Cebu area experiences development leading to increasing average income levels for this sample of households during the study period.

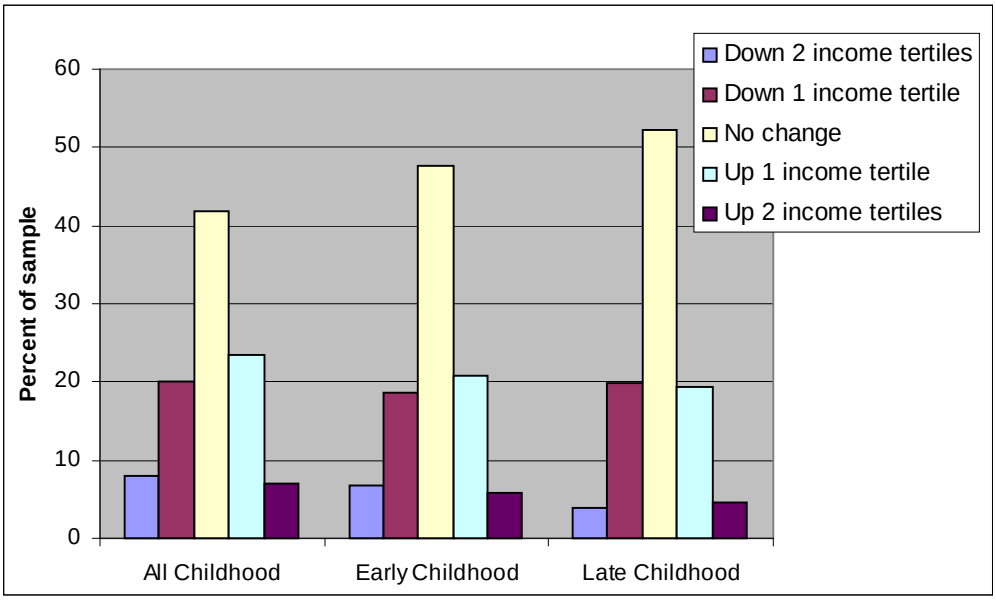
Figure 4.1: Mean per capita household income during childhood (for children with valid BMI data at age 19)



To provide a view of relative change over time, or how individual households fare compared to the rest of the sample, I calculated income tertiles, dividing the sample into thirds according to household income at each time period[13]. Figure 4.2 below highlights the extent of individual-level change in these tertiles during all of childhood, and early (birth-age9) and late (age 9-16) childhood. The bars represent the percent of the sample that experienced no change, moved up one tertile, moved up two tertiles, moved down one tertile, or moved down two tertiles during the given period (all, early or late childhood). Over the long term, between birth and age 16, almost 60% of the sample changed tertiles (moved up or down at least one terile). During early childhood 52% of households changed tertiles, and during late childhood 47% of households changed tertiles. Both upward and downward mobility were experienced in this sample: 28% moved up one or two tertiles, and 30% moved down one or two tertiles between birth and age 16.

[13] These change variables represent changes in income of the index child's household. Some of these cases may represent the index child moving to another (non-maternal) household, but the vast majority of changes represent mother-index child households increasing or decreasing in income over time.

Figure 4.2: Relative change in per capital household income during childhood



Children in this sample experience both real and relative changes in their household income from birth to age 16. Given the dynamic childhood household economic environment in this sample, I aim to test whether overall childhood income, and that assessed across the different stages, continue to affect individuals' BMI by the time they reach the transition to adulthood. I now turn to the specific statistical models and methods used in this study.

Methods

I use multivariate logistic regression analysis to provide statistical tests of my research questions. Logistic regression uses maximum likelihood estimation, and the coefficients refer to changes in the natural log odds of being in the abnormal weight category (under- or overweight) (Long 1997). Maximum likelihood performs best with large samples, and thus should provide reliable results in this sample even if there are relatively few overweight individuals. The following represents the general regression model estimated here (Long 1997):

$$\log\left(\frac{(y_i=1)}{(y_i=0)}\right) = \beta_0 + \beta_{1\ldots k}\, X_{1i\ldots ki} + \delta_{1\ldots k} Z_{1i..ki} \qquad (4.0)$$

where, y_i=weight status of child "i" in 2002; $X_{1\ldots k}$=deflated per capita household income variables; $\beta_{1\ldots k}$=average effect of income variables on log odds of being under/overweight; $Z_{1\ldots k}$= control variables; and, $\delta_{1\ldots k}$=average effect of control variables on log odds of being under/overweight.

I estimate robust standard errors and account for the unobserved error clustering at the community level[14] to allow for accurate hypothesis testing (Angeles, Guilkey, and Mroz 2005). The independent variable of interest ($X_{1\ldots k}$) are various forms of childhood income (average during all childhood or stage-specific income variables) depending on the particular model. I first estimate average childhood income effects to assess the overall relationship between childhood income and adult weight status. I then estimate models with one or more stage specific income variables to test for any timing effects. I assess the significance of the coefficients through two-tailed tests of $p<.05$.

I include several variables in the models to control for characteristics of the child, his/her household and community that may account for the relationship between income and BMI. Child age in 2002 (small differences in age of the child range: 17.9-19.8 years) and sex are included to account for age and sex differences that may affect household income (perhaps determining whether the child is providing some of that income) and weight status in 2002. I also included a measure of whether the child was ever living in a household other

[14] The index child's community was assessed in 2002, with 1998 or 1994 values used if 2002 community data were missing.

than his/her mother's household from age 9-16[15]. Research shows that mothers tend to prioritize child health over fathers (Thomas 2000), and that step-mothers do not contribute to the health of children as much as biological mothers do (Case and Paxson 2001). Children living with their biological mothers may be exposed to different resource distribution patterns and other decision-making processes than children living with other caregivers. These different living conditions may affect both household economic status and child health. Finally, I control for whether the child was the first born or not, to account for first born children being more likely to contribute to household income at some point and to have higher levels of BMI due to resource allocation favoring them.

Maternal controls include whether the mother changes spouses at some point between 1991 and 1998 (index child's age 9-16)[16]; and, maternal education (a 2-part spline allowing for differences in education effect for those women with 6 or fewer and those with greater than 6 years of education), maternal age, and maternal height all assessed at the index child's birth. These maternal characteristics may affect the extent of household income (through the mom's own or her spouse's earning potential), and the child's BMI in 2002 (through maternal care and genetics). It is important to note that I use maternal height rather than maternal BMI because maternal height was established prior to 1983. Although maternal BMI is probably more highly related to child BMI at age 19, it changes from 1983-1998 and is most likely affected by household income during this study period. Maternal BMI would be an endogenous variable in these statistical models. Maternal height provides an exogenous

[15] In the total effects models, this control variable is measured as whether the index child was living in a household other than the maternal household during the particular wave when income was measured, and as ever living in a non-maternal household in the average income and partial effects models.

[16] In the total effects models, this control variable is measured as whether the index child's mother had changed spouses during the particular wave when income was measured, and as ever changing spouses from index child age 9-16 in the average income and partial effects models.

measure and is thus the preferred control for unmeasured genetic potential that can affect both household earnings during childhood and BMI at age 19.

The household-level controls are: type of family (extended versus nuclear) and family size, both of which are measured at the same time as the income variables in the total effects models, and averaged across childhood due to correlation over time in the average childhood income and partial effects models. I also control for ethnicity of the household (dummy variable of whether the household spoke an ethnic dialect in the baseline year), which may affect both earnings and genetics differences in BMI.

Finally, controls for community development are important for isolating the effects of family resources on health because family resources have been found to have different effects in communities with more or less access to resources and infrastructure (Dargent-Molina, James, Strogatz, and Savitz 1994; Desai and Alva 1998). An urbanicity index, which has been found to perform better than an urban/rural dichotomy and categorical urbanicity variables (Dahly and Adair 2007), is used in the model to account for differences in access to community resources (including food sources, housing, job opportunities, health and other services). Since this measure is highly correlated over time, I create an average of this index from birth through age 16 to represent the index child's community during childhood for the average childhood income and partial effects models.

Because the coefficients from logistic regression models are difficult to interpret (in log odds), I calculate predicted probabilities to describe the effects of childhood income on the risk of being underweight or overweight in the transition to adulthood. To provide a view of how being poor versus well-off in childhood affect one's health in adulthood, I compare predicted probabilities of being underweight and overweight for childhood income at two

levels, at the mean of the lowest income tertile and at the mean of the highest income tertile, for each income period being considered. The table in Appendix A provides the values associated with mean income for each tertile by stage of childhood, and averaged across early, late, and all of childhood.

Results

Table 4.1 shows the regression results for underweight and overweight status at age 19 on average childhood income (birth to age 16). Model 1 illustrates that average childhood income has a negative effect on the log odds of being underweight by average age 19[17]. This average childhood income effect (-0.19) is marginally significant at $p<.052$. I tested for a sex interaction with household income, which was insignificant; thus, no sex-specific models were needed for underweight status. Models 2-4 provide the results of the effect of average childhood income on overweight status, which did have significant differences in the effect of household income by sex of the index child. The results from the full sample (Model 2) illustrate that average childhood household income influences the log odds of being overweight or obese at the beginning of adulthood, with a significant coefficient of 0.59. This represents a 6 percentage point increase in the probability of being overweight at age 19 when childhood income is at the mean of the highest compared with lowest tertile (i.e. the effect of increasing income by 132 pesos). For the boys (Model 6), the effect is stronger at 0.93, which represents a 7 percentage point increase in the probability of being overweight at age 19 for the same increase in income. Both of these effects are statistically significant. Average childhood income does not seem to affect girls' log odds of being overweight at age 19 (Model 4, Table 4.1).

[17] Because there were no significant differences in the income effect on the log odds of being underweight by sex, only the full sample model is shown for underweight status.

Table 4.1: Effects of average childhood household income on underweight & overweight status at age 19

	Underweight	Overweight		
	All	All	Boys	Girls
	(1)	(2)	(3)	(4)
Income during childhood				
Average household income (birth-age 16)	-0.194	**0.594**	**0.930**	0.235
	(0.100)	**(0.207)****	**(0.300)****	(0.276)
	(p<.052)			
Controls				
Child age in 2002	-0.067	-0.037	0.020	-0.193
	(0.139)	(0.289)	(0.411)	(0.419)
Male	-0.261	-0.128	--	--
	(0.092)**	(0.173)	--	--
First born	0.057	-0.225	0.018	-0.436
	(0.131)	(0.329)	(0.360)	(0.433)
d not in mom's HH[1]	-0.030	-0.610	-0.333	-0.636
	(0.179)	(0.354)	(0.556)	(0.521)
Mom's height	-0.024	0.018	0.053	-0.021
	(0.009)**	(0.017)	(0.026)*	(0.028)
Mom changed spouse[1]	-0.323	0.289	0.040	0.439
	(0.173)	(0.232)	(0.338)	(0.426)
Mom educ<6 yrs spline	0.037	-0.136	-0.165	-0.117
	(0.039)	(0.087)	(0.126)	(0.121)
Mom educ>6 yrs spline	0.015	0.088	0.143	-0.003
	(0.021)	(0.045)	(0.059)*	(0.077)
Mom age	0.011	-0.023	-0.008	-0.041
	(0.009)	(0.024)	(0.029)	(0.035)
Ethnic household	0.485	-0.855	-0.640	--
	(0.265)	(0.710)	(0.684)	--
Total HH size[2]	-0.014	-0.149	-0.279	-0.048
	(0.025)	(0.075)*	(0.151)	(0.084)
Extended family (vs nuclear)[2]	-0.073	1.235	0.920	1.587
	(0.194)	(0.395)**	(0.435)*	(0.574)**
Community urbanicity[2]	-0.003	0.027	0.032	0.022
	(0.005)	(0.009)**	(0.013)*	(0.013)
Constant	4.571	-6.456	-14.211	3.919
	(2.919)	(5.887)	(7.062)*	(8.938)
Observations	1957	1957	1065	871

Robust standard errors in parentheses. Bold=effects of interest. * p<.05; ** p<.01

[1]Defined as ever occurring 1991-98.

[2]Averaged 1983-98.

In exploring the potential timing effects, Table 4.2 provides models of the log odds of being underweight on logged per capita household income at different stages of childhood,

while Table 4.3 shows results from the same models for the log odds of being overweight. Both tables provide the total effect of income at each stage of childhood, followed by partial effects that include income variables across multiple stages. The total effects results provide insight into the cumulative effect of income at that age, not controlling for past or subsequent income. The partial effects, in turn, illustrate how important each stage is in and of itself (controlling for income at other time periods), and allow a more direct test of early versus late income effects.

In terms of income effects at different stages on underweight at age 19, the largest effect seems to be income at birth. The coefficient on logged per capita household income is -0.14 when income at other stages is not included (Model 1, Table 4.2). When income at all other stages are included in the model (Model 6, Table 4.2), the coefficient remains about the same at -0.16. Because of the high correlation among income at ages 9, 12 and 16 (r=.55-.60), I provide a final model that includes income averaged from age 9-16 (mid to late childhood). In this model, the effect of income at birth remains strong at -0.14, while no effect of later income is discernable. The partial effects models, then, indicate that income at birth has lasting effect on the log odds of being underweight at age 19 holding income at other stages constant. The -0.14 coefficient means that decreasing household income at birth by 86 pesos (the difference between mean of the lowest and highest in 1983) results in a 5.3% increase in the chance of being underweight at age 19. (See Table 4.4 below for predicted probabilities.) Since income at other stages of childhood makes no difference to individuals' chances of being underweight by age 19, it seems that an economic deficit at birth can set individuals on track for health disadvantage into adulthood.

Table 4.2: Effects of household income by stage of childhood on log odds of being *underweight* at age 19

	Total Effects					Partial Effects	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Income by stage of childhood							
Income at birth (1983)	**-0.136**					**-0.163**	**-0.144**
	(0.055)*					**(0.068)***	**(0.064)***
Income at age 2 (1986)		0.010				0.058	0.051
		(0.054)				(0.061)	(0.060)
Income at age 9 (1991)			-0.039			0.020	
			(0.060)			(0.087)	
Income at age 12 (1994)				-0.013		0.022	
				(0.059)		(0.088)	
Income at age 16 (1998)					-0.132	-0.131	
					(0.081)	(0.113)	
Income mid-late childhood							-0.095
(average from age 9-16)							(0.088)
Controls							
Child age in 2002	-0.061	-0.096	-0.050	-0.082	-0.158	-0.125	-0.100
	(0.141)	(0.133)	(0.142)	(0.146)	(0.160)	(0.153)	(0.141)
Male	-0.251	-0.229	-0.311	-0.312	-0.269	-0.244	-0.224
	(0.090)**	(0.103)*	(0.085)**	(0.085)**	(0.100)**	(0.105)*	(0.101)*
First born	0.083	0.042	-0.021	-0.021	-0.061	0.083	0.095
	(0.131)	(0.122)	(0.128)	(0.132)	(0.137)	(0.139)	(0.133)
Child not in mom's HH[1]	--	--	0.670	0.178	0.139	0.159	0.063
	--	--	(0.469)	(0.485)	(0.388)	(0.187)	(0.188)
Mom's height	-0.023	-0.023	-0.023	-0.023	-0.018	-0.018	-0.021
	(0.009)**	(0.009)*	(0.009)*	(0.009)*	(0.009)*	(0.010)	(0.009)*
Mom changed spouse[1]	--	--	-0.319	-0.283	-0.660	-0.279	-0.247
	--	--	(0.264)	(0.214)	(0.216)**	(0.178)	(0.177)
Mom educ<6 yrs spline	0.033	0.044	0.040	0.030	0.027	0.044	0.056
	(0.039)	(0.040)	(0.040)	(0.040)	(0.035)	(0.041)	(0.041)
Mom educ>6 yrs spline	0.013	-0.012	0.009	0.004	-0.003	0.010	0.005
	(0.021)	(0.022)	(0.022)	(0.023)	(0.025)	(0.026)	(0.024)
Mom age	0.007	0.011	0.009	0.009	0.004	0.010	0.012
	(0.010)	(0.010)	(0.010)	(0.010)	(0.010)	(0.009)	(0.009)
Ethnic household	0.444	0.663	0.595	0.654	0.743	0.638	0.561
	(0.268)	(0.274)*	(0.252)*	(0.293)*	(0.312)*	(0.297)*	(0.285)*
Total HH size[2]	-0.009	0.001	0.012	0.007	-0.041	-0.009	0.001
	(0.022)	(0.017)	(0.019)	(0.022)	(0.023)	(0.022)	(0.017)
Extended family[2]	-0.096	-0.072	-0.142	0.231	-0.189	-0.084	-0.087
	(0.165)	(0.111)	(0.130)	(0.125)	(0.146)	(0.179)	(0.177)
Community urbanicity[2]	-0.003	-0.006	-0.007	-0.003	-0.005	-0.005	-0.004
	(0.005)	(0.005)	(0.005)	(0.006)	(0.005)	(0.006)	(0.006)
Constant	3.994	4.036	3.535	3.985	5.584	4.605	4.496
	(2.981)	(2.847)	(3.008)	(3.133)	(3.366)	(3.194)	(2.947)
Observations	1945	1827	1846	1814	1762	1765	1819

Robust standard errors in parentheses. Bold=effects of interest. * p<.05; ** p<.01

[1]Measured when income assessed in total effects models. In partial effects models, ever occurring 1991-98.

[2]Measured when income assessed in total effects models. In partial effects models, averaged 1983-98.

Turning to the timing effects at the other end of the BMI spectrum, Table 4.3 illustrates the effects of income at different periods of childhood on the log odds of being overweight at age 19. Income at ages 9 and 12 is large and significant when not controlling for income at other stage of childhood. When controlling for other stages of childhood income, the age 9 effect decreases to half the size and becomes insignificant. The effect at age 12, however, increases in size and remains significant (Table 4.3, Model 6). The loss of significance of income at age 9 when income at subsequent stages is taken into account may be due to correlation between income at age 9 and that at 12 (r=.56), or it may be that income at age 12 mediates the effect at age 9. Model 7, then, provides the results when income at ages 9, 12 and 16 are averaged, avoiding the problem of correlation across income at later stages of childhood. The significant effect of 0.71 is larger than any of the stage specific effects, suggesting that income at age 9 and 12 may be additive in their effect on individuals' overweight status at age 19. As Table 4.4 below shows, increasing average mid-late childhood income by 110 deflated pesos (the difference between means of the lowest and highest income tertiles) increases the risk of becoming overweight at age 19 by 5.3%.

Due to some significant sex differences in income effects, I also assessed these timing effects of childhood income on overweight status at age 19 for males and females separately. The sex-specific models are presented in Appendix B. Generally, the results hold by sex, although age 9 income seems to matter more for boys while age 12 income matters more for girls. Age 12 income, however, is equally as large in the boys' model, but is insignificant, perhaps due to the small sample size and correlation among income at ages 9 and 12. The sex-specific results, however, highlight additional findings that were not evident in the full sample results. Both the girls' and boys' models suggest that early childhood income may

have some importance for the development of overweight status in early adulthood. For boys, the positive effect of income is evident for income at age 2, which remains significant when income at birth and average childhood income in mid-late childhood are included in the model (Table 4.3A, Model 7). For girls, a negative effect of income at birth emerges when controlling for income at later stages of childhood (Table 4.3B, Model 7). The effect size rivals that of income in mid to late childhood, although they work in opposite directions, which is why average childhood income seemed to have no effect on overweight status for girls (Table 4.1, Model 4). The effect of income at birth on girls' overweight status at age 19 may illustrate that increasing income at birth prevents girls from needing rapid catch up growth later in infancy. Small size at birth followed by more rapid growth may be linked to obesity in adulthood (Adair 2007), and thus infant girls in high income households may be better able to develop normally rather than at the riskier rapid, catch up growth during infancy of poor girls. In short, the sex-specific results suggest that while mid to late childhood income exerts the largest effects on both boys and girls, income during early childhood may have some important sex-specific effects on individuals' risk of being overweight at age 19. These results must be viewed with caution due to the small sample sizes and number of cases of overweight by sex (about 51 boys and 57 girls are overweight at age 19).

Table 4.3: Effects of household income by stage of childhood on log odds of being *overweight* at age 19

	Total Effects					Partial Effects	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Income by stage of childhood							
Income at birth (1983)	0.020					-0.209	-0.218
	(0.130)					(0.132)	(0.131)
Income at age 2 (1986)		0.176				0.122	0.154
		(0.128)				(0.149)	(0.141)
Income at age 9 (1991)			**0.400**			0.166	
			(0.150)**			(0.207)	
Income at age 12 (1994)				**0.466**		**0.571**	
				(0.186)*		**(0.146)****	
Income at age 16 (1998)					0.184	-0.044	
					(0.216)	(0.199)	
Income mid-late childhood							**0.709**
(average from age 9-16)							**(0.167)***
Controls							
Child age in 2002	0.018	-0.029	0.021	-0.022	0.019	-0.069	-0.033
	(0.296)	(0.282)	(0.306)	(0.334)	(0.337)	(0.317)	(0.295)
Male	-0.153	-0.127	-0.085	-0.062	-0.172	-0.159	-0.125
	(0.167)	(0.169)	(0.182)	(0.189)	(0.183)	(0.175)	(0.178)
First born	-0.026	-0.073	0.000	-0.020	-0.063	-0.150	-0.148
	(0.327)	(0.308)	(0.334)	(0.344)	(0.386)	(0.341)	(0.331)
Child not in mom's HH[1]	--	--	--	--	-0.704	-0.527	-0.612
(1991-1998)	--	--	--	--	(1.025)	(0.374)	(0.376)
Mom's height	0.026	0.022	0.020	0.021	0.017	0.018	0.017
	(0.016)	(0.019)	(0.017)	(0.019)	(0.019)	(0.020)	(0.019)
Mom changed spouse[1]	--	--	-0.480	0.272	0.510	0.262	0.282
	--	--	(0.577)	(0.335)	(0.260)*	(0.234)	(0.221)
Mom educ<6 yrs spline	-0.098	-0.118	-0.150	-0.134	-0.123	-0.168	-0.155
	(0.090)	(0.083)	(0.094)	(0.084)	(0.088)	(0.083)*	(0.080)
Mom educ>6 yrs spline	0.154	0.141	0.125	0.137	0.159	0.098	0.088
	(0.042)**	(0.043)**	(0.041)**	(0.043)**	(0.046)**	(0.049)*	(0.046)
Mom age	-0.013	-0.035	-0.021	-0.031	-0.041	-0.054	-0.049
	(0.025)	(0.025)	(0.025)	(0.025)	(0.024)	(0.023)*	(0.025)*
Ethnic household	-0.852	-0.664	-1.500	-1.504	-1.386	-0.482	-0.592
	(0.708)	(0.723)	(1.005)	(1.001)	(1.006)	(0.719)	(0.759)
Total HH size[2]	-0.090	-0.042	-0.105	-0.053	-0.064	-0.101	-0.108
	(0.058)	(0.039)	(0.061)	(0.057)	(0.053)	(0.071)	(0.068)
Extended family [2]	0.235	0.315	0.561	0.821	0.632	0.937	1.027
	(0.237)	(0.227)	(0.270)*	(0.243)**	(0.274)*	(0.457)*	(0.440)*
Community urbanicity[2]	0.035	0.025	0.020	0.018	0.024	0.030	0.030
	(0.009)**	(0.008)**	(0.008)**	(0.009)	(0.010)*	(0.010)**	(0.010)**
Constant	-7.294	-5.524	-6.864	-6.869	-5.807	-5.328	-6.258
	(6.101)	(6.009)	(6.029)	(6.587)	(6.677)	(6.451)	(6.071)
Observations	1945	1827	1813	1790	1762	1765	1819

Robust standard errors in parentheses. Bold=effects of interest. * p<.05; ** p<.01

[1]Measured when income assessed in total effects models.In partial effects models defined as ever occurring 1991-98.

[2]Measured when income assessed in total effects models. In partial effects models, averaged 1983-98.

Table 4.4 below summarizes the results discussed in terms of predicted probabilities of being underweight and overweight at age 19. The probabilities are calculated for income values at the mean income of households in the lowest tertile (i.e., poor households) and the mean income of households in the highest tertile (i.e, "rich" households). This is generally equivalent to increasing household income by 100 deflated pesos, although the exact difference depends on the stages of childhood. The marginal effects, then, represent the difference in probabilities of being underweight or overweight for those individuals living in relatively rich compared with very poor households during childhood.

Table 4.4: Predicted probabilities of weight outcome at 19 by childhood economic status[1]

	Weight Outcome at Age 19	
	Underweight	*Overweight*
Average Childhood Income[2]		
Predicted probability if poor	32.2%	2.6%
Predicted probability if "rich"	23.9%	8.5%
Effect of being "rich"	-8.3%	5.8%
Income at birth[3]		
Predicted probability if poor	29.6%	no
Predicted probability if "rich"	24.3%	significant
Effect of being "rich"	-5.3%	effect
Income ages 9-16[4]		
Predicted probability if poor	No	2.9%
Predicted probability if "rich"	Significant	8.2%
Effect of being "rich"	Effect	5.3%

[1]Predictions made for poor by setting household income=mean income of tertile1, for "rich" by setting household income=mean income tertile 3. All other variables held at actual values.
[2]Based on Table 4.1 coefficients
[3]Based on Table 4.2, Model 7
[4]Based on Table 4.3, Model 7

Tables 4.1-4.4, then provide the results that inform my research questions. Clearly childhood income is important for individuals' BMI-based weight status in the transition to adulthood, although it works differently for my distinct health outcomes. Average childhood

income reduces the odds of being underweight and increasing the odds of becoming overweight in the transition to adulthood, and the effects are slightly stronger for the likelihood of being underweight (-8% effect of being "rich") than overweight (6% effect of being "rich") (see Table 4.4). In this setting, while income generates a health advantage in terms of the more traditional under nutrition problem, it seems to exacerbate the modern health problem of becoming overweight.

In terms of the research questions relating to timing, I find that household income at birth significantly affects the log odds of being underweight at age 19. Income at other stages of childhood does not have any significant effect on this health outcome at age 19. For overweight status, however, household income in mid and late childhood seems particularly important for the likelihood of being overweight at age 19. For boys, early income (age 2) may also be important. For girls, income at birth may have an important benefit, reducing the likelihood of being overweight at age 19 controlling for income at all other stages of childhood (see Tables 4.3A & 4.3B in Appendix B). However, across the full sample and sex-specific models, income at ages 9 and 12 stand out childhood periods when income increases the risk of becoming overweight at age 19. Further, when averaged together, household income during mid and late childhood have the only positive effect for girls, and a much stronger positive effect than early (age 2) income for boys. The implications of these findings are discussed below.

Conclusions

Childhood is an important period of life when economic resources may have large and lasting impacts on individuals' health status into adulthood. Given the important decisions that are made during early adulthood, it is critical that we better understand how

health inequalities evident at this stage are influenced by socioeconomic conditions earlier in the life course.

This paper provides insight into this question by investigating how childhood economic resources affect individuals' weight status at age 19. Both underweight and overweight can lead to subsequent health problems, lower socioeconomic status and other disadvantages during adulthood. This study considers the relationship between household income, an important and dynamic measure of socioeconomic status, during childhood (birth-age 16) and BMI at age 19 in the context of the Philippines. The presence of both underweight and overweight health problems allows for an assessment of the effects of income at both ends of the BMI spectrum using comparable data and models.

The first main conclusion from this study is that childhood income can work in multiple ways, reducing traditional health problems (underweight) while also inducing modern diseases (such as overweight/obesity). The negative effect of income on the likelihood of being underweight as an adult suggests that childhood income puts individuals on healthier growth trajectories. Households with higher income are likely providing their children with better nutrition, and possibly more schooling, which lead to better physical growth and less chance of becoming an underweight adult. The risk of becoming an overweight adult, however, increases with childhood household income. This suggests that richer households are providing an unhealthy childhood environment, most likely through unhealthy behaviors such as overfeeding, high fat diets, and/or little physical exercise. Parental behaviors in richer households may influence the child's physical development, as well as their own health behaviors as they enter adulthood.

The effects of income are stronger for the risk of being overweight in terms of coefficient size, although the predicted probabilities are of similar magnitude for underweight and overweight outcomes. In terms of populations at risk, more children are entering adulthood underweight (27% in this sample) and thus increasing household income during childhood may be considered a more pressing health issue. Fewer children arrive in adulthood overweight (5.5%) based on western standards. However, another 6% of the sample has a BMI>=23, which has been linked to chronic disease in Asian adults (Inoue and Zimmet 2000). Considering this lower BMI cut off results in almost 12% of the sample children transitioning to adulthood with the potential for health problems related to high BMI. Given the trend of increasing prevalence of overweight populations, the harmful effects of increasing income on the probability of being overweight must also be taken seriously by researchers and policy makers.

The results from the timing models suggest the importance of considering individual development in the relationship between childhood income and weight status at the beginning of adulthood. The main finding from the underweight models is that higher household income at birth reduces the risk of being underweight as an adult, and household income during subsequent stages of childhood does not alter this effect. This supports the early origins hypothesis, which suggests that early environments set individuals on lasting physical growth trajectories (Alderman, Hoddinott, and Kinsey 2006; Barker 1990; Gigante et al. 2006).

At the same time, having higher household income later in childhood seems particularly important for increasing the risk of being overweight in the transition to adulthood. Although difficult to discern exact differences in stage effects from age 9 – 16 due

to correlation of household income across these ages, the total effects models suggest the importance of household income at ages 9 and 12. Mid to late childhood (ages 9-12) may be a time in children's lives when they are making more food choices and possibly changing their physical activity patterns, while still highly dependent on household resources. Increasing resources during this stage in childhood, particularly in the context of the nutrition transition, may mean increasingly unhealthy food choices and limited physical activity due to the lack of physical labor required by better off households. It is interesting that the effects of income at age 16 do not seem as important as those at ages 9 and 12. It may be that household income in adolescence has less of an effect on individuals' BMI if they are earning their own income or beginning to spend more time outside of the household. It may also be that at this stage in their lives individuals begin to worry more about being thin (as the data suggest high income, urban girls tend to begin dieting at this age), and use their household income to reduce their weight (going to a gym, or buying more expensive, healthier foods). There are also some early childhood effects, only found in the sex-specific models. This indicates that early childhood income may have some effects on overweight status in early adulthood through physical growth patterns (e.g., negative effect of income at birth for girls), or through early health behaviors (e.g. positive effect of income at age 2 for boys).

In sum, the findings here provide additional evidence that childhood conditions influence individuals' health in adulthood, and specifically their weight status as measured by BMI. New evidence is provided that suggests that household income may work differently depending on which end of the BMI spectrum is considered. Further, the findings illustrate the importance of considering age and stage of childhood in assessing the effects of

childhood income on adult health. Particularly noteworthy is that the stage of childhood when income matters most for adult health depends on the outcome considered. This is somewhat consistent with the broader sociological literature, which suggests that childhood income may have different effects on ability (when early income is most important) and behavioral (when late income is most important) outcomes (Duncan, Yeung, Brooks-Gunn, and Smith 1998; Guo 1998; Hill, Yeung, and Duncan 2001). To the extent that underweight adults are influenced exclusively by income at birth (and not income at other stages) suggests that underweight status is likely established early in life, perhaps setting up "health potential" akin to "cognitive ability," and later income has little effect on it. This is not to say that all poor children who are underweight at birth continue to be underweight in adulthood. Rather, it suggests that the benefits of childhood income for preventing health problems related to health potential are strongest at birth.

The effects of childhood income on overweight status in adulthood may represent more health behavior mechanisms at work; and that household income matters more for this health outcome when individuals are beginning to make decisions about their own behavior while still being tied to their parental household income. However, the influence of childhood income on becoming overweight may begin in early childhood, as the sex-specific models suggest. These early effects of income may work through both socialization (i.e., health behavior) and establishing key growth patterns (i.e., physical development) in increasing the risk of being overweight in the transition to adulthood. Overweight status in adulthood, then, seems to be a complex measure of health, which includes aspects of health potential (i.e., height and other growth patterns) developed early in childhood, as well as health behaviors developed in early and later stages.

Finally, this research suggests that household income early in life is critical for individuals' healthy development, since increasing income in later stages does not help reduce the chance of being underweight and may promote overweight problems in early adulthood. This suggests the household that are upwardly mobile during individuals' childhood (were poor at birth and better off later in childhood) may pose significant risks to individuals' healthy development, particularly within the context of the nutrition transition.

In interpreting these findings and conclusions it is important to consider the changing macro context being experienced by this cohort of children from 1983-2002. During these years the Philippines was experiencing the nutrition transition, where overweight and obesity problems were beginning to emerge, side by side with under-nutrition problems that continued to exist. This dual nutrition burden provides the context necessary to most effectively compare income effects on BMI issues at both the low and upper ends of the distribution. Furthermore, following a birth cohort over 19 years of their lives provides important benefits in being able to test for timing differences in the income effects on BMI. However, in using a cohort of children who all experience the same macro context it is not possible to empirically distinguish whether the timing effects are due to individual development periods or to changes in the macro context during this time.

Although the timing pattern is consistent with the changing role of income during this period being related to the changing macro context, several things suggest that changes in the macro context alone cannot account for the timing patterns found here. First, if these effects were entirely due to the changing macro context, one would not expect to find the positive effects of income in 1986 (at age 2) for boys. Further, one would expect increasing positive effects of income in 1998 on the risk for being overweight in 2002. The overweight models,

instead, show relatively small and insignificant effects of 1998 income relative to 1991 and 1994 effects. Thus, it is likely that, although the larger social context plays a part in explaining how childhood income affects weight disparities in 2002, individual stage of development seems to be a more plausible explanation for the importance of income across childhood for determining individuals' overweight status in the transition to adulthood. Subsequent studies that can provide longitudinal data for multiple cohorts would be useful in separating the effects of individual development from the larger historical context in interpreting the timing of childhood income effects on health disparities in early adulthood; and, in determining how the household context may affect when overweight/obesity issues emerge during childhood.

This study has several limitations that should be considered. First, this is not a representative sample of the Philippines and thus can only be generalized to married couple households with children. These are also generally poor households, with the average income for the highest tertile reaching about 13.5 dollars per week per person in 1994 and 1998. It is not clear whether similar effects and timing patters would be found in other settings with richer households or a different income distribution. Second, the overweight findings are based on roughly 108 cases of overweight status in 2002 in the final timing models, and the sex-specific models are based on 58 boys and 57 girls who are overweight. Thus, the results found should be viewed with caution in light of the small number of cases upon which they are based. More substantively, the specific mechanisms through which childhood income affects adult health status were not tested here. Future research that measures the pathways through which income at different stage of childhood influence various adult health outcomes would provide further tests of the conclusions posed here. Finally, research in this area would

benefit from assessing the timing effects of other aspects of the childhood household environment and their effects on multiple health outcomes in the transition to adulthood.

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CHAPTER 5: CONCLUSION

This dissertation has explored how childhood household contexts affect well-being in childhood and the transition to adulthood. In doing so, I have contributed to the current body of research on the origins of social inequalities by providing a life course view of traditional stratification questions. My three studies assessed how one's childhood household economic resources and family structures affect health and education, two key indicators of social status and well-being. Each of my three studies (Chapters 2-4) focused on a distinct set of research questions, dataset and methods. However, several broader conclusions can be pulled from the somewhat distinct findings.

All of the results point to the importance of childhood household contexts for well-being. The household conditions explored here were father absence, change in sibling residency, and economic resources, and all three turn out to be important to individuals' social status (i.e., health, education). For example, Chapter 2 finds that father absence due to migration increased illness among children under 6, and Chapter 3 highlights the role that changing number of residential siblings during childhood plays in the number of years of education individuals obtain. These two aspects of household contexts during childhood have not been explored in past research. Thus, their importance to individuals' lives informs our understanding of how health and educational inequalities develop, and that we need to pay further attention to the changing residency status of family members over time.

The importance of changing contexts, and particularly the movement of family members into and out of the household, directly relates to the life course tenet of linked lives. This tenet highlights the importance of the life course trajectories of other members of one's family and household to an individual's own life course. This is particularly highlighted in Chapter 3, in which siblings following their life courses, moving out of the household for work, education, marriage or other reasons, had serious negative consequences for individuals' educational attainment.

An extension of the idea that childhood contexts matter, is that these contexts may have long term implications, affecting social inequalities into adulthood. Connecting two stages of the life course, childhood and the transition to adulthood, is an important application of the life course perspective to stratification questions. Two of the three papers (Chapters 3 & 4) utilized longer-term longitudinal data to illustrate the importance of childhood households for outcomes later in life as individuals are poised to enter adulthood. In Chapter 3 I found that sibling changes between age 2 and 16 affected individuals' educational attainment at age 19, and Chapter 4 results suggested that children born into higher income households have less risk of being underweight in the transition to adulthood.

Another contribution of these papers is the emphasis on the *change* in childhood social contexts, based on the life course perspective that both individuals and their social contexts change over time. All three studies pointed to the dynamics of childhood contexts: fathers move out and back in, siblings are born into, leave, and return to the household, and household income changes in absolute and relative terms during the periods of childhood under study. The impact of these changes was the focus of the first two papers, which considered dynamics in childhood family structure and emphasized the importance of

changes in residence status of household members for individuals' lives. Chapter 2 found that child illness was higher in households where fathers were absent due to migration, than in households where the father was present; and Chapter 3 illustrated how the movement of siblings in and out of households affected individuals' education. Both father absence due to migration and changes in sibsize, especially movement of siblings out of the household, proved to be important for creating social inequalities. These aspects of family structure have been neglected in past research, in part due to the lack of attention to change over time in children's social contexts. The third paper focused less on change per say, but did incorporate household income at different periods in childhood to capture any changes that occurred.

Another clear application of the life course perspective is attention to the importance of timing in the relationship between social context and individual outcomes. Chapters 3 and 4 considered how childhood household conditions (sibsize and income, respectively) affected individuals' outcomes at the transition to adulthood differently depending on the stage of childhood when they were experienced. For example, the loss of siblings in mid and late childhood had particularly strong effects on individuals' education compared with the same changes in other stages of childhood. Changes in sibsize before age 9 had less of an impact, and changes in infancy had no impact on education later in the transition to adulthood. This suggests that change in sibsize and the household resources it requires (time and money) may be more detrimental to children during their school years (ages 9-16) rather than during their key cognitive development stages (birth-age 9). A second example of the importance of timing of resources in childhood is that household income in mid to late childhood seemed to most strongly affect the likelihood of becoming overweight by the transition to adulthood. Both changes in sibsize and income experienced during mid-late childhood were important

for education and health outcomes (overweight) at age 19. This contrasted with the importance of income at birth for the risk of being underweight at the same age. It may be that household contexts early in childhood affect outcomes most closely related to "ability" or "potential", while later resources and contexts work to affect outcomes more closely related to "achievement" and "healthy behaviors" (Duncan, Yeung, Brooks-Gunn, and Smith 1998; Guo 1998; Hill, Yeung, and Duncan 2001). Thus, these results suggest that given the fact that childhood households change over time: (1) *when* certain household conditions occur in individuals' lives during childhood is important to understanding the development of social inequalities at the transition to adulthood; and, (2) which stages of childhood are most important for individuals' development may depend on the nature of the outcome considered.

In addition to the above contributions to the literature on the development of social inequalities, this dissertation also provides insights into these issues within developing country contexts. As outlined in the introduction, it is important to study social inequalities in these contexts, in part, because they provide the necessary setting within which these complex questions can be addressed. Mexico, for example, proved important in providing a setting where migration is relevant to children's lives, and where a large-scale social welfare was being implemented following an experimental design. This allowed me to test how an exogenously-determined welfare program affected the relationship between father absence and child illness (turning out to be a critical element in the relationship). The Philippines, the setting of the other two papers, presented an environment with fertility and residential mobility rates that were high enough to detect differences in the effects of changing sibsize on education. Further, the Philippines, as a country in the midst of the nutrition transition,

allowed for an investigation into the comparative effects of income on underweight and overweight status for the same sample.

Overall, then, the following points can be taken from this dissertation to apply to the current body of research related to social inequalities (i.e., health and education):

(1) Childhood household conditions have important, and lasting, consequences for individuals' health and education in developing country contexts.

(2) Childhood household contexts are diverse and change over time during childhood; and, taking into account these changes may be key to understanding the development of health and education disparities.

(3) Residential status of family members is an under-studied aspect of the dynamics of childhood households, with important implications for individual well-being.

(4) Stages of individual development should be considered when posing and answering questions about how childhood contexts affect individuals' health and education.

(5) The timing of the effects of childhood resources may depend on the nature of the outcome studied, with a potentially important distinction between outcomes determined largely by ability and those more affected by individuals' own behavior.

(6) Developing country settings should be explored further because they provide interesting, relevant, and socially-important grounds for the study of social inequalities.

Although not the main goal of this dissertation, some aspects of these studies also inform social policy. First, the findings from Chapter 2 suggest that social welfare payments may interact with household contexts in important ways to protect the most vulnerable children. In this case, children with absent fathers were particularly helped by social welfare in the

context of rural poverty. Thus, it may be possible to target households with young children and migrating fathers in protecting public health and reducing the risk of child morbidity and, by extension, child mortality.

A second social policy implication of this work is that households (or individuals) may be targeted according to their age, with a broader view to critical ages or stage of childhood. Social policy often focuses on young children, due to early childhood encompassing critical stages of cognitive and physical growth that influences both health and education later in childhood and adulthood. All three studies illustrated that early childhood contexts do matter for health during childhood and adulthood and educational attainment. However, Chapters 3 and 4 found that that household conditions later in childhood may also determine individuals' well-being. Further, the age at which to target interventions, and the type of intervention, may depend on the outcome of interest.

Finally, the development of social inequalities, and the role of household resources in that, largely depends on the social-historical context within which they occur. For example, in most setting poor children are targeted to improve health and education outcomes. However, in the context of the nutrition transition, higher income households with children should be targeted to try to improve food and physical activity choices. Thus, social policy makers should be sure to take into account the specific setting of their social problems in determining what kind of solutions may be most effective.

Limitations from this dissertation are many, and the results presented here only begin to address the complexities inherent in the development of social inequalities over the life course. The topics under consideration are clearly complex processes that need to be explored further. The results here revealed the potential importance of interaction and timing

effects. These effects may be specific to the samples or settings of these studies and thus should be tested further with other datasets. Furthermore, this dissertation did not explore the specific mechanisms through which these contexts affected individuals' lives. Subsequent research may effectively build on the findings here by testing some of the mechanisms proposed and theorizing about other potential ways these stratification processes work.

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APPENDIX A

Table 4.2A: Sample mean per capita household income in pesos and dollars by income tertile

Age	Income Tertile	Mean per cap income in Php	Mean per cap income in U.S.$
Birth	1	15.0	$1.4
	2	36.9	$3.3
	3	101.7	$9.2
Age 2	1	12.4	$1.1
	2	31.5	$2.8
	3	98.3	$8.8
Age 9	1	21.0	$1.9
	2	44.8	$4.0
	3	114.5	$10.3
Age 12	1	27.3	$2.5
	2	56.8	$5.1
	3	148.7	$13.4
Age 16	1	33.5	$3.0
	2	63.7	$5.7
	3	147.5	$13.3
Averages			
Birth-Age 9	1	16.1	$1.5
	2	37.7	$3.4
	3	104.8	$9.4
Age 9-16	1	27.3	$2.5
	2	55.1	$5.0
	3	136.9	$12.3
Birth-Age 16	1	17.4	$1.6
	2	52.3	$4.7
	3	149.5	$13.5

APPENDIX B: Overweight tables by sex

Table 4.3A: Effects of household income by stage of childhood on log odds of being *overweight* for males

	Total Effects					Partial Effects	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Income by stage of childhood							
Income at birth (1983)	0.214					-0.079	-0.087
	(0.156)					(0.175)	(0.176)
Income at age 2 (1986)		**0.437**				0.305	**0.351**
		(0.151)**				(0.170)	**(0.162)***
Income at age 9 (1991)			**0.718**			0.492	
			(0.212)**			(0.266)	
Income at age 12 (1994)				0.442		0.517	
				(0.315)		(0.331)	
Income at age 16 (1998)					0.083	-0.035	
					(0.285)	(0.252)	
Income mid-late childhood							**0.887**
(average from age 9-16)							**(0.322)****
Controls							
Child age in 2002	0.192	0.185	0.097	0.158	0.144	0.012	0.077
	(0.405)	(0.405)	(0.443)	(0.460)	(0.476)	(0.454)	(0.437)
First born	0.303	0.113	0.285	0.350	0.299	0.095	0.033
	(0.369)	(0.366)	(0.401)	(0.403)	(0.454)	(0.360)	(0.356)
Child not in mom's HH[1]	--	--	--	--	0.538	-0.202	-0.366
(1991-1998)	--	--	--	--	(1.021)	(0.552)	(0.562)
Mom's height	0.062	0.063	0.044	0.054	0.058	0.053	0.053
	(0.024)*	(0.025)*	(0.027)	(0.027)*	(0.027)*	(0.029)	(0.027)*
Mom changed spouse[1]	--	--	0.155	-0.318	-0.042	0.150	0.143
	--	--	(0.715)	(0.612)	(0.416)	(0.358)	(0.317)
Mom educ<6 yrs spline	-0.111	-0.110	-0.173	-0.165	-0.207	-0.180	-0.152
	(0.140)	(0.134)	(0.134)	(0.119)	(0.124)	(0.123)	(0.122)
Mom educ>6 yrs spline	0.224	0.190	0.176	0.226	0.272	0.110	0.108
	(0.054)**	(0.048)**	(0.056)**	(0.060)**	(0.065)**	(0.061)	(0.059)
Mom age	-0.005	-0.012	0.000	-0.013	-0.029	-0.022	-0.019
	(0.032)	(0.031)	(0.029)	(0.031)	(0.031)	(0.031)	(0.030)
Ethnic household	-0.538	-0.273	-1.354	-1.311	-1.304	-0.186	-0.253
	(0.713)	(0.740)	(0.952)	(0.860)	(0.878)	(0.709)	(0.735)
Total HH size[2]	-0.143	-0.084	-0.155	-0.083	-0.117	-0.178	-0.197
	(0.114)	(0.086)	(0.116)	(0.098)	(0.073)	(0.137)	(0.136)
Extended family[2]	-0.061	0.122	0.384	0.696	0.637	0.446	0.680
	(0.327)	(0.278)	(0.377)	(0.347)*	(0.349)	(0.428)	(0.444)
Community urbanicity[2]	0.033	0.030	0.021	0.029	0.044	0.036	0.037
	(0.012)**	(0.011)**	(0.011)	(0.013)*	(0.014)**	(0.014)*	(0.013)**
Constant	-16.994	-17.652	-13.646	-16.039	-14.959	-15.252	-16.538
	(7.315)*	(7.766)*	(7.484)	(7.961)*	(8.202)	(7.922)	(7.692)*
Observations	1060	996	978	960	944	963	993

Robust standard errors in parentheses. Bold=effects of interest. * p<.05; ** p<.01

[1]Measured when income assessed in total effects models.In partial effects models defined as ever occurring 91-98.

[2]Measured when income assessed in total effects models. In partial effects models, averaged 1983-98.

Table 4.3B: Effects of household income by stage of childhood on log odds of being *overweight* for females

	Total Effects					Partial Effects	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Income by stage of childhood							
Income at birth (1983)	-0.216					**-0.379**	**-0.396**
	(0.200)					**(0.183)***	**(0.191)***
Income at age 2 (1986)		-0.092				-0.084	-0.056
		(0.136)				(0.182)	(0.171)
Income at age 9 (1991)			0.115			-0.044	
			(0.197)			(0.253)	
Income at age 12 (1994)				**0.468**		**0.524**	
				(0.201)*		**(0.188)****	
Income at age 16 (1998)					0.294	0.005	
					(0.250)	(0.310)	
Income mid-late childhood							**0.500**
(average from age 9-16)							**(0.215)***
Controls							
Child age in 2002	-0.147	-0.381	-0.147	-0.237	-0.220	-0.296	-0.287
	(0.422)	(0.414)	(0.433)	(0.441)	(0.421)	(0.454)	(0.426)
First born	-0.288	-0.267	-0.334	-0.438	-0.410	-0.340	-0.308
	-0.147	-0.381	-0.147	-0.237	-0.220	-0.296	-0.287
Child not in mom's HH[1]	--	--	--	--	--	-0.527	-0.596
(1991-1998)	--	--	--	--	--	(0.517)	(0.549)
Mom's height	-0.016	-0.025	-0.021	-0.023	-0.031	-0.028	-0.024
	(0.026)	(0.030)	(0.028)	(0.030)	(0.029)	(0.032)	(0.033)
Mom changed spouse[1]	--	--	-1.036	0.655	0.958	0.433	0.427
	--	--	(1.008)	(0.442)	(0.394)*	(0.405)	(0.407)
Mom educ<6 yrs spline	-0.079	-0.117	-0.124	-0.107	-0.084	-0.163	-0.164
	(0.117)	(0.120)	(0.122)	(0.122)	(0.121)	(0.113)	(0.114)
Mom educ>6 yrs spline	0.044	0.038	0.028	0.016	0.025	0.021	0.010
	(0.075)	(0.085)	(0.069)	(0.078)	(0.077)	(0.093)	(0.087)
Mom age	-0.030	-0.067	-0.046	-0.048	-0.056	-0.083	-0.084
	(0.040)	(0.038)	(0.042)	(0.040)	(0.038)	(0.038)*	(0.037)*
Ethnic household	--	--	--	--	--	--	--
	--	--	--	--	--	--	--
Total HH size[2]	-0.045	-0.005	-0.063	-0.029	-0.007	-0.052	-0.041
	(0.065)	(0.048)	(0.071)	(0.063)	(0.075)	(0.096)	(0.093)
Extended family[2]	0.513	0.429	0.806	0.989	0.675	1.427	1.422
	(0.313)	(0.348)	(0.389)*	(0.312)**	(0.363)	(0.682)*	(0.677)*
Community urbanicity[2]	0.034	0.021	0.019	0.007	0.010	0.030	0.028
	(0.014)*	(0.011)	(0.012)	(0.013)	(0.013)	(0.015)*	(0.015)
Constant	3.275	9.965	4.178	4.717	6.196	8.403	7.580
	(8.599)	(9.028)	(9.016)	(9.250)	(8.991)	(10.200)	(9.808)
Observations	865	814	816	810	789	788	810

Robust standard errors in parentheses. Bold=effects of interest. * p<.05; ** p<.01

[1]Measured when income assessed in total effects models.In partial effects models defined as ever occurring 91-98
[2]Measured when income assessed in total effects models. In partial effects models, averaged 1983-98.