ABSTRACT

Title of dissertation: The Effect of Stress on Developmental Trajectories: Empirical Evidence from Peru

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In this dissertation I examine the relationship between early childhood development (ECD) and stress. In the first chapter I conduct a comprehensive and systematic analysis of research on human development to compose a holistic portrayal of development that takes place during early life, analyze the role of quality parental care and stimulation as enabler or inhibitor of ECD, and discuss how stress can set children, particularly those growing up in poverty, on sub-optimal developmental trajectories. The second chapter investigates empirically the link between maternal depression and children's physical growth during early life in Peru. I present evidence of a negative effect of maternal depression on childhood growth. Evidence in this essay suggests that maternal depression hinders maternal engagement, which in turn could lead to sub-optimal care practices that lead to worse nutritional outcomes. The third chapter investigates the relationship between maternal depression and child cognitive development in Peru. Results indicate that while the effect of temporary cases of maternal depression in the sample is negligible, the effect of chronic cases of depression is sizable and statistically significant, and persists over time. When the impact of maternal depression is analyzed separately by gender and

maternal education level, there is evidence of worse effects for boys, as well as for children of mothers with incomplete primary school. The three chapters discuss the policy implications of the current knowledge of the effect of stress on ECD, which, even if incomplete, is compelling enough to warrant intensifying efforts to shelter children from stress, particularly during early childhood and in low-income settings.

THE EFFECT OF STRESS ON DEVELOPMETAL TRAJECTORIES: EMPIRICAL EVIDENCE FROM PERU

by

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Dedication

To my family

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1. Introduction

In this dissertation, I study the relationship between stress and early childhood development. The point of departure for this study is the new wave of multidisciplinary knowledge that has emerged over the last two decades, which is prompting a paradigm shift in the understanding of how human development occurs. The convergence of findings from multiple and diverse streams of research around the hierarchical and interdependent nature of human development on the one hand, and the high malleability of developing systems during early childhood on the other, serve to highlight the crucial role of early experiences and environmental inputs at this extraordinarily significant stage of children's development.

The consistency of findings across disciplines that study early childhood development implies that exposure to toxic stress, i.e., excessive or prolonged activation of the physiologic stress response systems without the buffering support of stable and responsive relationships, has a profoundly negative impact in developmental outcomes. In particular, toxic stress constitutes a developmental risk factor for disadvantaged children, who tend to live in highly stressful environments that affect them, as well as their parents.

I conduct the study of the relationship between toxic stress and early childhood development in three separate but highly related essays. The first essay conceptually addresses the dissertation's central question regarding the relationship between stress and early childhood development, with a particular focus on children growing up in disadvantaged circumstances. This essay provides the conceptual framework for the overall dissertation. Its analysis of the current literature on early childhood development provides a multidimensional platform for the research in the second and third essays; it motivates and informs them.

In the second and third essays, I narrow the focus of research and empirically investigate the effect of mental health problems associated with acute maternal stress on two particular developmental outcomes, child nutrition and cognitive development in Peru, using data from the Young Lives survey. Despite differences in methodological approaches and the sub-samples analyzed, the empirical findings in both essays support the main argument laid out in the first: that severe stress and associated maternal common mental disorders undermine children's development. My empirical findings along with evidence cited in the first essay of the disproportionately high prevalence rates of anxiety and depression among households with low socio-economic status, suggests that stress in general, and associated maternal mental illness in particular, constitute a pathway from poverty to substandard developmental trajectories and potentially worse outcomes later in life. Maternal mental illness, the evidence suggests, constitutes yet another contributor to the intergenerational transmission of poverty.

Together, the three essays of this dissertation further the understanding of the effect of stress on the developmental outcomes of children. In addition, the analysis points to the need for further research that provides the necessary context to achieve a more integrated understanding of how maternal depression affects the whole child, as well as the role that factors such as maternal characteristics and behavior, child attributes, and poverty play in influencing the effect of stress on child outcomes.

Nevertheless, the essays conclude that the current knowledge of the effect of stress on early childhood development warrants intensifying efforts to shelter poor children from toxic stress, particularly during early childhood, by arming families with the tools and knowledge to protect children, tackling maternal common mental disorders associated with stress, and/or providing alternative ways to safeguard children's development.

2. Does Stress Affect Children's Developmental Trajectories, Particularly for Those Growing Up in Poverty?

2.1 Introduction

Multiple streams of research from diverse fields that have emerged over the last two decades are prompting an important paradigm shift in the understanding of how human development occurs – a shift that suggests stress may have a profound impact in developmental outcomes, particularly those of disadvantaged children.

At the core of the new wave of knowledge is the significance of development that takes place early in life, captured in four contributions of the emerging literature: i) development that takes place during early childhood (EC) deeply influences developmental trajectories that tend to be difficult to change later in life; ii) the period of crucial development spans from the time of conception through the fifth birthday, much earlier than previously considered; iii) environmental inputs continually interact and integrate with genetic endowments to determine a person's biological state, particularly during early life; and iv) early parent-child interactions are essential inputs for human development because they shape the architecture of the brain (Shonkoff and Phillips 2000; Knudsen et al 2006).

In addition to emphasizing the importance of early life for human development, the findings above highlight the impact of early environments in children's development and suggest that parental influence is more complex and pervasive than previously understood. Parents directly condition children's developmental outcomes not only via genetic endowments and material conditions, but also by shaping their environments and determining the quality of the inputs they provide for their health, welfare, maintenance and protection during early life. In fact, the latter factors may play as critical a role in determining children's developmental trajectories as the first two (Hertzman 2000; Shonkoff and Phillips 2000; Cunha et al 2005; Heckman 2007).

The fact that early experiences and environmental inputs shape the architecture of the brain implies that stress during early life becomes embedded in foundational neural circuits. Stress can affect children's biological systems directly, or indirectly by affecting parents and their ability to provide quality inputs for Early Childhood Development (ECD). Because stress is endemic in low-income settings, poor children's development is particularly vulnerable to its effects, both direct and indirect. Moreover, parental stress combined with low levels of parental capability (i.e., the combination of attributes such as health, cognitive and socio-emotional abilities) that are typically associated with poverty is likely to exacerbate the suboptimal quality of parental inputs poor children receive during early life, which compounds on the effects of direct stress, material deprivation and exposure to external shocks on these children's developing systems, and increases the chances that they fall into a poverty trap.

The issue of how stress affects children's human capital accumulation has been under-explored, with limited evidence that systematically quantifies the consequences of stress on child development, the channels of effect, and how to mitigate its impact on children. This essay contributes to furthering the understanding of the determinants of ECD by studying the relationship between ECD and stress, the understanding of which is crucial to designing sensible policy interventions to protect children's welfare, particularly those growing up in poverty.

To do this, I conduct a comprehensive and systematic analysis of the various streams of research that have pushed the frontier of knowledge on the formation of human development in recent decades. Because it aims to capture the multidimensional nature of human development, the scope of this analysis is unprecedented: it weaves together findings from conceptual and empirical work from a broad range of disciplines, including neurobiology, genetics, developmental psychology, as well as sociology and economics to compose a holistic portrayal

of development that takes place during early life (section 2.2), analyze the role of quality parental care and stimulation as enabler or inhibitor of such development (section 2.3), and discuss why poor children's developmental trajectories are particularly vulnerable to the effect of stress (section 2.4). Finally, the essay discusses the implications of the new science on human development for the design of effective ECD interventions and the imperative of advancing the understanding of the ways in which poverty and stress undermine human development (section 2.5). The analysis here provides a multidimensional platform for the research in the second and third essays of the dissertation. Together, these three essays further the understanding of the effect of stress on the developmental outcomes of children.

The essay concludes that exposure to toxic stress in early life constitutes a developmental risk factor for children, especially those in poor households. Further, the analysis suggests that the combination of parental stress and low levels of capability may help explain why policy initiatives to improve poor children's outcomes may have not been more effective. Although in principle increased access to quality and quantity of inputs should lead to better outcomes, in practice parents in poor households may not be able to take advantage of increased access to resources, services and opportunities made available by public interventions given their own overwhelming circumstances, which exacerbates developmental deficiencies and/or inadequate understanding of the significance of EC development or important inputs it entails.

The analysis in the essay suggests that, to increase effectiveness, efforts directed at improving early developmental outcomes should address the effect of stress that affect young children, as well as their parents, on early developmental outcomes. The current policy focus on the provision of income support for poor families should be complemented with initiatives to improve parental understanding of the importance of early childhood development and how best

to foster it, not only through effective child rearing and parenting, but also by sheltering from toxic stress. In addition, support for parents to minimize or deal with their own stress may greatly benefit young children's development. Parental health, involvement and endorsement are necessary conditions to increase the effectiveness of ECD interventions. And when circumstances are such that parents are not able to shield children from toxic stress, effective alternatives, such as increasing the availability of high quality childcare, can protect children's development while at the same time reducing stressors in parents' lives.

Importantly, focusing on the earlier years of life, and thus curbing developmental shortfalls, has a much greater chance (and is cheaper) of leading to better developmental outcomes than attempting later remediation. As much as the high malleability of developing systems during EC represents a source of vulnerability, it also constitutes a window of opportunity; human capability can be shaped and improved to unleash the potential embodied in initial genetic endowments, regardless of material conditions.

2.2 A More Comprehensive Understanding of Early Human Development

Over the last two decades, research from the fields of neuroscience, biology, psychology, and the economics of human capital formation has significantly furthered the understanding of human development – an integrated, multidimensional process that takes place over the life cycle. Findings from this research converges around two aspects of human development that highlight the significance of early childhood (EC) for developmental trajectories: one, development occurs in a hierarchical and integrated fashion, causing what happens earlier in life to set the foundations for all future development; and two, the formation of developing systems stems from the interaction of genetics and environmental influences, which are highly malleable early in life.

2.2.1 Hierarchical & Interdependent Nature of Human Development

The notion that biological systems form in a hierarchical, bottom-up fashion represents a significant contribution to the understanding of human development. This is particularly the case for brain development, which constitutes one of the fundamental dimensions of overall development because of the central role that the organ plays in the functioning and survival of the human body (OECD 2007). Although the full development of the brain spans between a few days after conception and early adulthood, it is now understood that the time between conception and a child's third birthday is the most critical because this is when foundational neural circuits are set up (Shonkoff and Phillips 2000).

The neurobiology literature explains that the process by which neural circuits form is sequential and cumulative, with simpler networks developing first and more complex ones building on them later (Knudsen 2004). The fast pace of neuronal maturation during EC is accompanied by the highest growth of synaptic connections in a person's life, a process that facilitates the increasing need for inter-connectivity between neurons and among networks that marks this period of rapid growth (OECD 2007). This is crucial for early development given that the areas of the brain associated with the different dimensions of ECD are highly integrated and the brain's multiple functions tightly coordinated (Knudsen et al 2006).

Recent findings from research in the behavioral and social sciences both converge with and complement those from neurobiology, finding the nature of skills and behavior development to be sequential, cumulative, and highly integrated, with later attainments building on earlier ones. Of particular interest is a series of widely cited papers written by James Heckman and several co-authors over the last decade, which propose that ECD has a greater effect on human capital formation than development at any other time in a person's life (Heckman 2000; Heckman and

Carneiro 2003; Heckman, Stixrud and Urzua 2006; Cunha et al 2005; Cunha and Heckman 2007; Heckman 2007). This work proposes a more comprehensive concept of human capital than the traditional definition, one that forms in a dynamic, multi-stage, and multi-generation process.

Heckman proposes that a conceptualization of human capital that explicitly takes into account a person's multidimensional nature is best suited for the analysis of human development (Cunha et al 2005; Cunha and Heckman 2007). Hence, his human capability framework considers the many components of human development –i.e., health, sensory-motor, cognitive and socio-emotional skills — in unison and assumes that all of them can be cultivated to improve life outcomes. Moreover, there is a high level of integration and interdependency across all developmental outcomes, essentially constituting a web of reciprocal links that ultimately determines a person's ability to thrive.

Human capability formation

Heckman and co-authors propose a model in which capabilities are formed throughout life in a multistage, dynamic process that begins with the health of the mother before the time of conception. In this model, childhood is comprised of multiple periods, each of which is related to a stage of capability formation. These economists assume agents have a vector of capabilities at each stage in life that consists of skills, as well as health stocks that include the propensity for mortality and morbidity (Cunha et al 2005; Cunha and Heckman 2007; Heckman 2007). Inputs and investments at each stage produce outputs in the next stage; outputs consist of the level of each capability achieved at a given stage.

Two important implications of this multistage technology are the self-productivity and complementarity of the capability formation process. Self-productivity entails that capabilities developed in one stage persist into future stages and promote the production of further

capabilities in later periods (e.g., early skills provide the basis to master later skills). This is of particular significance when human capital is considered to be multidimensional, as it implies 'cross-fertilizing' effects among health, sensory-motor, cognition, and social and emotional capacities: higher levels of health promote learning; emotional security fosters child exploration and more vigorous learning; individuals with higher IQs tend to be more farsighted because they envision future scenarios more clearly; and higher levels of self-regulation and conscientiousness reduce health risks and avoid accidents (Knudsen et al 2006; Heckman 2007).

The other feature of the multistage capability formation technology, complementarity, implies that capabilities formed in one period increase the productivity of investments at later periods. It also means that levels of investments in capability formation at different ages reinforce each other. Consequently, for earlier investments to be productive, they have to be followed by more investments later in life. The combination of complementarity and self-productivity results in multiplier effects for investments in capability formation during early childhood (Cunha 2005), which capture the hierarchical and interdependent nature of the development of the neural networks underlying capability formation.

This multiplier effect provides a strong incentive to ensure investing in skill formation during children's earliest years. In addition, as Schady (2005) argues, it also implies that, while there are no significant equity-efficiency tradeoffs to investing in poor or well off children during early childhood, once abilities are formed, such tradeoffs become substantial given that the returns are higher from investments in the healthiest, most able and most motivated (Cunha et al 2005; Heckman 2007).

2.2.2 High Plasticity During Early Childhood

The expansion of scientific knowledge of human development over the last two decades also highlights the high plasticity of developing systems during EC, that is, how sensitive they are to the influence of environmental factors during the first years of life. The 'nature versus nurture' debate has been replaced by the understanding that genetic endowments (nature) and experiences and environmental inputs (nurture) work in tandem, and that their interaction and integration determine early developmental outcomes.

To be sure, genetic factors are still considered crucial to development. However, it is now understood that environmental inputs that range from nutrition, physical, chemical and social environments, and experiences affect how such development plays out, influencing the selection of synaptic connections that shape the fundamental organization of biological systems and structures that mediate sensory-motor, cognitive, and socio-emotional capacities, conditioning both their development and subsequent functioning (Shonkoff and Phillips 2000; OECD 2007; Center on the Developing Child 2010).

Though the interaction between genes and external inputs occurs throughout life, the body's sensitivity to such inputs is particularly acute during early life, the time of fastest growth of its biological systems. Among environmental inputs, early experiences play a central role in the formation of the brain. This is because the brain is designed to recruit and incorporate experience into its developing architecture and neurochemistry, allowing it to change in response to environmental demands (Shonkoff and Phillips 2000; Knudsen 2006; Center on the Developing Child 2010). Genes determine when specific brain circuits are formed and experiences then shape how that formation unfolds by customizing circuits' information processing capabilities according to the demands of the experience (Knudsen 2004).

In fact, developmental processes of brain growth rely on specific experiences that have been selected through the process of evolution (e.g., infants' stable and responsive interactions with caregivers, auditory stimulation, exposure to patterned light) to organize and structure essential behavioral systems (Greenough and Black 1992; Knudsen et al 2006). Other early, more idiosyncratic experiences that are not deemed essential help trigger new brain growth and refine existing brain structures, affecting the trajectory of the individual's brain development. Such inputs include factors that expand the developmental horizons, such as perceptual stimulation, social interaction and opportunity for varied activities (Greenough and Black 1992).

Across species, experience is essential to the unfolding of brain development; the more adaptable the species, the more experience plays a role in its development. In fact, the human brain is especially susceptible to experience, not only because of its high adaptability, but also because it develops at a slower pace than other species, expanding its window of malleability (Greenough and Black 1992).

Gene sensitivity to environmental influences is particularly acute during sensitive periods (Johnson 2005; Knudsen et al 2006). For neural circuits lower in the circuit hierarchy, sensitive periods tend to take place earlier in life, while higher circuit sensitivity can occur as late as adolescence (Center on the Developing Child 2011). Because neural circuit malleability diminishes once a sensitive period ends, earlier experiences have a disproportionate effect on foundational circuits. As the sequential development of neural circuits entails that circuits at higher levels in the hierarchy are dependent on information provided by lower-level circuits, early experiences are also likely to affect the configuration of higher level circuits (Knudsen et al 2006).

The fact that most neural connections are not configured by the time of birth means that early experience can instruct neural connection patterns without interference from patterns that have already been established (Knudsen 2004). In this way, experiences determine which neural connections survive, with those connections that are used more often growing stronger and becoming permanent, and those that are used less gradually disappearing through a normal process called pruning (Glaser 2000; Grantham-McGregor 2007; Center on the Developing Child 2010).

The pruning factor works in tandem with the plasticity mechanisms described above, helping explain the strong shaping effect of early experience for development. Experiences later in life can alter neural circuit configuration, but the process tends to get less efficient and require more intensity beyond EC (Knudsen et al 2006). Although it increases the receptivity to external input and the opportunity for adaptation, plasticity also prolongs the developing brain's vulnerability to the surrounding environment and events in a child's life (Vegas and Santibáñez 2010).

2.3 Parental Inputs: Key Determinants of Children's Developmental Trajectories

The hierarchical and interdependent nature of human development, along with the high plasticity of biological systems during early life, and the fact that children rely on their parents for the provision of the inputs that shape all aspects of development, highlight the extent to which the quality of parental care and stimulation received during early life is vital for their development. Further, because human development is the result of the interaction and integration of genetic and environmental inputs, but the quality of genetic factors cannot be altered after conception, the availability and quality of environmental inputs that parents provide constitutes the variable term of the development equation; they largely determine the trajectory of human development.

This section first explores parental contributions that are vital for development. The focus then shifts to the determinants of parental inputs, and the consequences of substandard quality parental contributions during early life.

2.3.1 Parental Inputs: Early Care & Stimulation

The fact that parents provide essential inputs for their children's development has been established for some time. Parents shape the environment in which children grow up and provide essential care, i.e., the provision of what children need for their health, welfare, maintenance, and protection. However, evidence on the extent to which early experiences shape foundational brain circuits has motivated increasing research interest in the effects of parental stimulation and parent-child interactions during early life on development outcomes. This research shows that, in better and worse circumstances alike, parent-child interactions leave a significant imprint in young children.

Some of the channels through which parent-child interactions influence children's development are apparent. For instance, parental stimulation has consistently been found to be an important determinant of better cognitive and behavioral outcomes (Shonkoff and Phillips 2000; Walker et al 2007). Studies find that parental behavior that encourages exploration and provides a rich verbal environment, as well as opportunities and resources for learning early in life, is strongly associated with better developmental outcomes (Ginsberg et al. 1998, Griffin and Morrison 1997, Beals et al. 1994, Gallimore and Goldenberg 1993, Saxe et al.1987 and Starkey and Klein 1992 in Shonkoff and Phillips 2000).

New evidence from the fields of neurobiology, developmental psychology, as well as behavioral sciences sheds light on an alternative, less apparent channel of influence: emotions. Emotions are biologically basic features of human functioning. Due to the high plasticity that

characterizes developing systems during EC, emotions have the potential to shape neural tissue and become embedded in the architecture of the brain (OECD 2007). Consistent, sensitive and responsive care, and affect (nurturance and warmth) that trigger positive emotions facilitate the formation of secure attachments between young children and their parents. Secure attachment in turn fuels the creation of neural pathways that set the foundation for the normal development and functioning of healthy brains and other biological systems[1] (Glaser 2000, Shonkoff and Phillips 2000, Farah et al 2006; Walker et al 2007), and facilitate physiological, cognitive and socio-emotional development (Heckman 2007). On the other hand, negative emotions associated with parental neglect or maltreatment can be enormously harmful to ECD. I discuss this issue in section 2.4 below.

2.3.2 Parental Capability Determines Parental Inputs

Although parental characteristics have long been suspected to play a causal role in children's outcomes, until relatively recently the empirical research on the channels through which parents influence children's development focused mainly on hereditary factors conditioning gene endowments and household income/credit constraints that limit how much parents could invest in their children (Heckman 2007; Almond and Currie 2010).

However, the focus of this research has expanded to the study of the ways in which parental capability (i.e., the combination of attributes such as health, cognitive and socio-emotional abilities) shapes children's early environments beyond budgetary constrains. Recent findings suggest that parental sensitivity, responsiveness, availability, and affect, as well as childrearing practices and decisions parents make on their children's behalf –collectively referred to here as *parental engagement*- are equally important mediators between parental

[1] These include the neuroendocrine system, which is pivotal for the regulation of physiological processes of the human body, as well as immune and inflammatory responses, which are essential for defending against disease

capability and children's developmental outcomes, complementing and compounding the effect of gene endowments and availability of material resources. (Shonkoff and Phillips 2000; Farah et al 2006; Cunha et al 2005; Center on the Developing Child 2010). These factors have a lasting effect on children's developmental outcomes; they interact and accumulate differently within different families to determine the quality of care and stimulation children receive, starting before conception and continuing throughout childhood (Hertzman 2000; Shonkoff and Phillips 2000; Carneiro and Heckman 2002).

Levels of parental engagement may vary widely depending on parents' own attributes, such as their ability to be present in their children's lives in the face of health issues or income constraints, their capacity to make informed choices regarding educational or health inputs based on their own cognitive and socio-emotional skills (related imperfectly to their educational background) or ability to access new information, and more. Indeed, by influencing the degree of engagement, these parental characteristics determine the quality of the care and stimulation – and thus, their children's developmental trajectories (Shonkoff and Phillips 2000, Cunha et al 2005; Farah et al 2006; Walker et al 2007).

The fact that parental capability largely determines the extent to which parents can offer consistent care and stimulation to their children helps explain the emergence of differentials in health, cognitive, and socio-emotional outcomes along socio-economic lines early in life that tend to widen with age. Aside from capturing genetic endowments and limited access to quality resources, the steep correlation between parental socio-economic status (SES) and their children's developmental outcomes is a function of differentials in parents' own developmental outcomes and associated knowledge about their children's development that determine differentials in the quality of early circumstances and inputs they can provide (Cunha et al 2005).

Low parental capability in low-SES households increases the likelihood of suboptimal childrearing practices and decisions, less parental availability, responsiveness, sensitivity and affect, potentially leading to physiological maladaptations that undermine children's future outcomes (Hertzman 2000; McEwen 2000; Shonkoff and Phillips 2000; National Scientific Center for the Developing Child 2005; Center for the Developing Child 2010). What is more, sub-optimal levels of parental care and stimulation exacerbate the other channels through which low parental capability undermines children's development, i.e., material deprivation and exposure to intense and frequent shocks (Almond and Currie 2010). These factors combine and accumulate to undermine parental efforts to provide the inputs necessary for their children's development.

The SES-developmental outcome gradient may also reflect the effect that the acute stress associated with living in poverty has on children's early developing systems and parent's ability to provide adequate care for them. In light of the new understanding of the extent to which early environments in general, and parent-child interactions in particular, shape foundational brain circuits, stress represents a potential, if under-analyzed, pathway from poverty to worse parenting and hence sub-optimal children developmental outcomes. I explore this connection in the next section.

2.4 The Effect of Stress in Poor Children's Developmental Trajectories

Living in poverty is highly stressful for children and parents alike. Poor parents cannot make ends meet or plan ahead, and have worse health, as well as general life satisfaction levels (Graham 2015). Poor children are more likely to be exposed to poor nutrition, infections, environmental toxins and endemic substance abuse beginning early in the prenatal period, as well as chaotic and violent environments throughout the early childhood years and beyond

(McLloyd 1998; Shonkoff and Phillips 2000; Center on the Developing Child 2010). Although each one of these factors is sufficiently stressful and detrimental to children's early development, evidence suggests that their co-occurrence rate is high in low-income settings (Walker et al 2007; Horton et al 2008; Center on the Developing Child 2010).

Poor children's disproportionate and cumulative exposure to multiple risk factors subjects their developing systems to a level of wear and tear that, aside from being detrimental in itself, is also enormously stressful (Center for the Developing Child 2010). In fact, children who grow up in deep and persistent poverty are more likely to present higher allostactic loads, a physical symptom of persistent stress, as well as heightened activation of stress response systems (Lupien et al. 2001). Parents can be an important source of buffering protection; a wealth of evidence suggests the bodies of securely attached children who grow up in adverse environments are better able to regulate the physiological response to stress, generally exhibiting lower levels of allostatic load (Glaser 2000) and more adaptive responses to stress (Gunnar and Quevedo 2007).

However, parents in poor households are themselves overwhelmed by the stressful circumstances in which they live, which hinder the quality of parenting they can provide. Parental stress has been associated with insufficient stimulation and affect at best and maltreatment and neglect at worse. Far from fostering the secure attachments between young children and their parents that are so crucial for robust and timely development, parental stress triggers further stress, fear, and anxiety in their children, negative emotions that can disrupt the architecture of the brain and increase the odds of permanent deficits across early outcomes (McEwen 1998; Hertzman 2000; McEwen 2000; Shonkoff and Phillips 2000; National Scientific Center for the Developing Child 2005; Center for the Developing Child 2010).

2.4.1 Toxic Stress

Exposure to toxic stress during early life, meaning "the excessive or prolonged activation of the physiologic stress response systems in the absence of the buffering protection afforded by stable, responsive relationships" (Garner and Shonkoff 2012), puts the body in a continual state of alert. This state can permanently alter the neurobiological systems that guide physiological and behavioral responses to stress and lower the threshold for the activation of the stress response system for life (Shonkoff and Phillips 2000; National Scientific Council on the Developing Child 2005; National Scientific Council on the Developing Child 2010; OECD 2007).

On the physiological front, persistent elevation of the hormone cortisol, which is associated with stress, is related to lower growth hormone level in children, which can lead to reduced stature and even stunted growth (Stratakis 2006). Repeated activation of the stress response system can weaken the immune system and other metabolic regulatory mechanisms and permanently increase the individual's susceptibility to acute and chronic illness both during childhood and well into the adult years (McEwen 2007; National Scientific Council on the Developing Child 2007). Ample research documents how repeated exposure to adverse circumstances during childhood increases the risk of common adult chronic conditions, including diabetes, respiratory and cardiovascular diseases, stroke, and hypertension (Felitti et al. 1998; Edwards et al 2003; Anda et al. 2006), heart disease (Dong et al 2004; Caspi et al 2006), and life-threatening psychiatric disorders (Center for the Developing Child 2010). The greater the exposure to adverse experiences early in life, the greater the risk of physical and mental problems during adulthood, with longer exposure to trauma consistently associated with more severe physiological changes in the brain and worse sensory-motor, and cognitive outcomes

(Rutter et al. 1998, De Bellis and Putnam, 1994 and De Bellis et al. 1999b in Shonkoff and Phillips 2000).

Toxic stress during early life also undermines the development of socio-emotional skills because it disrupts the consolidation of neural circuits that connect the multiple central nervous system regions in which emotions are rooted (National Scientific Council on the Developing Child 2004). Evidence suggests that young children in stressful environments tend to exhibit higher levels of aggressive behavior, attention problems, depression, decreased social competence and empathy for others, and difficulty recognizing others' emotions (Pynoos and Eth 1985 and Scheeringa et al. 1995 in Shonkoff and Phillips 2000; Walker et al 2007).

In addition, growing up in a constant state of stress can interfere with the development of executive function, a theorized system of higher-level brain functions that is essential for early development of cognitive and socio-emotional capacities,[2] which assist individuals in the self-regulation of goal-directed behavior required to participate in school, work and other social settings.[3] Executive function systems both influence and are affected by early emotions because the brain circuits associated with executive functions are extensively interconnected with brain structures related to emotions. Evidence suggests that growing up in a constant state of stress or fear can lead to the consolidation of neural pathways associated with the flight-fight defense mechanisms to the detriment of the development and deployment of executive function capacities. Conversely, well-developed executive skills can help manage stress and adversity more effectively (Center on the Developing Child 2011).

[2] Executive function includes working memory, inhibitory control, and cognitive or mental flexibility that activate, organize, integrate and manage other, more basic cognitive and non-cognitive processes such as attention, memory, problem solving, production and understanding of language, inhibition, and initiation and monitoring of actions (Center on the Developing Child 2011).

[3] Recent research has shown that executive function affect how children learn, which is a greater predictor of later educational outcomes (e.g., math and reading achievement at age 21; college completion by age 25) than what they learn (e.g., earlier math or reading ability) (Welsh et al 2010; McClelland and 2011).

2.4.2 Maternal Depression and Anxiety

Severe and/or chronic stress can lead to common mental disorders (CMD) such as anxiety and depression in both parents and children. Although these health problems affect parenting across the income strata, evidence suggests that this is disproportionally so in low-SES households due to the high levels of adversity and deprivation parents are subject to (Walker et al 2007; National Scientific Council on the Developing Child 2010).

Effects on ECD

It has been suggested that CMD constitute a pathway from poverty-related stress to compromised child development, particularly when it affects mothers, who typically play a more prominent role in childrearing and are more prone to depression in the post-partum period due to hormonal changes linked to childbirth and stressors associated to childrearing (Shonkoff and Phillips 2000; Walker et al 2007).

However, there is surprisingly limited conclusive evidence demonstrating that the relationship between CMD and deficient developmental outcomes is indeed causal and what the channels of effect are, perhaps the result of data limitations. Research from developed countries consistently reports that young children of depressed mothers have lower quality interpersonal functioning and attachment, and more prevalent behavioral problems. A number of studies from high-income countries report evidence that suggests that maternal CMD leads to worse socio-emotional outcomes (Lyons-Ruth 1986; Stein et al 1991) and behavioral outcomes (Peterson and Albers 2001; Kim-Cohen, Muffin et al. 2005; Frank and Meara 2009).

Studies of the effect of CMD on physical development have been less conclusive: for instance, while some studies find negative (Surkan et al 2011) and persistent effects (Wojcicky et al 2011; Surkan et al 2012; Surkan et al 2014), others find none or transient effects (Surkan et al

2007; Wright et al 2006; Santos et al 2010). The second essay of this dissertation, which investigates whether maternal mental health undermines offspring's growth during early childhood in Peru, finds negative, sizable and statistically significant effects, even when excluding mothers with more severe cases of depression from the estimations. As Surkan et al (2012) suggest, the heterogeneity of findings across studies may be the result of differences in measurements of both physical growth impairment and maternal depression, length of follow-up, sample characteristics, and sample sizes across studies, as well as varying level of social resources, caregiving norms, and prevalence of both malnutrition and maternal depression in the populations under study.

Research on the effect of CMD on cognition also reports mixed results, with Cogill et al (1986) and Petterson and Albers (2001) reporting lower cognitive outcomes for children exposed to depression in the United Kingdom and the United States, respectively, and Frank and Meara (2009) finding no effect in the United States. In the last essay of the dissertation, I also find that temporary cases of maternal depression have a negligible effect on Peruvian children's cognitive development over the first eight years of life, except for boys and children of mothers with lower education and or those who were chronically depressed, for all of whom the negative effect on child cognition is sizable and statistically significant.

Mechanisms

The mechanisms through which CMD undermine ECD are not well understood, as evidence is limited. Moreover, it is not known whether these mechanisms vary between countries and regions, as factors such as cultural differences in caregiving and feeding, and the degree of food (or other inputs) insecurity may all play a role (Surkan et al 2011). Maternal depression may interfere with mother-child interactions and lead to an increased stress response in children and

associated elevated levels of cortisol, which, as the section on toxic stress details, can have a permanent effect on developmental outcomes. Alternatively, maternal depression may undermine maternal behavior, potentially hindering the effectiveness of parental skills and the quality of maternal childrearing practices, including general child care, feeding, and child supervision (Lovejoy et al 2000, McLearn et al 2007, Hurley et al 2008, Melchior et al 2009). My study of the effect of maternal depression on child nutrition and cognitive development in this dissertation find that, while depressed mothers appear to be more likely to disengage, when they do engage, their responsiveness may mediate the effect of depression on child cognition.

CMD can also reduce the productivity of parental investments (Frank and Meara 2010), which may already be sub-optimal in low-SES households given low levels of parental capability. Because, as section 2.2 makes abundantly clear, it is very difficult and costly to compensate with investments later on once the foundation of development is compromised, inadequate investments due to maternal CMD during a child's early life (both in terms of levels and choices) have lasting consequences for the accumulation of a child's own capability, and thus outcomes later in life (Heckman and Carneiro 2002; 2003). Furthermore, for less than perfect substitutability between earlier and later childhood, parental investment in children during EC determines the impact of exogenous shocks on their outcomes (Almond and Currie 2010). Lower levels of baseline investments for children in poorer households, who tend to be subject to more frequent and intense shocks, increase their vulnerability to such shocks and the probability of long-term damage and compromised developmental outcomes.

CMD Moderators

What are the factors that either prevent the damaging effects of parenting associated with depression from occurring or attenuate its effects? Research on the topic suggests that SES status

helps moderate the effect of maternal depression on children's development, with income level and maternal education typically mitigating the effect of the shock. Yet my empirical research in Peru suggests that the role of SES as a moderator of maternal depression is not straightforward. For instance, my regression results indicate that the cognitive outcomes of children whose mothers have lower levels of education (primary school incomplete) are on average more vulnerable to the effects of depression, and the physical growth of children of indigenous mothers (who on average are poorer) is disproportionately affected by depression. However, there do not appear to be heterogeneous effects of depression by average household wealth level.

Similarly, there is also some evidence that the effect of CMD on child development varies by child gender. Studies have found that boys' cognitive functioning may be more sensitive than girls' to the effect of mothers' mental illness (Sharp et al 1995; Hay et al 2001; Kurstjens and Wolke 2001). Although I also find that maternal depression affects the vocabulary accumulation of Peruvian boys but not that of girls (essay 3), I find no differences in the effect of maternal depression on child stature along gender lines (essay 2). Identifying the factors behind these heterogeneous effects of depression may help articulate more effective interventions to protect vulnerable children's welfare from the effect of maternal depression.

2.4.3 Parental Maltreatment

Severe parental stress and related CMD are associated with parental maltreatment, that is, intense and repeated child abuse and/or neglect, which is extremely detrimental to early development (Shonkoff and Phillips 2000). Aside from its direct consequences, maltreatment can cause irreversible damage to children's physical, cognitive, and socio-emotional development and, if not addressed, set them on a sub-optimal trajectory for life (Center on the Developing Child 2010). Although child maltreatment represents an unusual and extreme disruption in parenting,

incidents of child abuse and neglect are highest in low-income families (McLloyd 1998) and have been shown to have an impact on learning abilities, IQ scores, language ability, and school performance (National Scientific Council on the Developing Child 2010).

Longer-term effects of abuse during EC include a greater risk of being involved in both familial and non-familial violence later in life (Malinosky-Rummell and Hansen 1993), as well as a higher probability of deviant behavior, such as adolescent pregnancy and smoking (Center for the Developing Child 2010). In terms of labor market performance, a study by Currie and Widom (2009) found that, by the time they reached 40 years old, individuals who had been abused as children were less likely to be employed and had lower earnings and fewer assets, patterns that were particularly pronounced among women.

Evidence on the effects of neglect is more limited, but suggests serious repercussions on health and socio-emotional outcomes (Erickson & Egeland, 1996 in Shonkoff and Phillips 2000), including high levels of behavioral problems (Walker et al 2007). These developmental deficits may be partly explained by the fact that, because the infant brain is unable to learn self-regulation of affect, young children rely on sensitive caregivers to modulate their arousal. Neglected children tend to lack these sensitive interactions, resulting in developmental deficits that may only become apparent later in life, manifesting themselves as aggression or hyper-vigilance (Glaser 2000).

Although individually both abuse and neglect are sufficiently harmful, evidence suggests that they tend to coexist, particularly in low-income settings, where they are the most prevalent (Trickett et al., 1991 and Waldfogel, 1998 in Shonkoff and Phillips 2000; Ricketts and Anderson 2008 in Vegas and Santibáñez 2010). Differences in co-occurrence patterns, as well as children's age and gender, all influence the extent of the impact of maltreatment on early development

(McLeod and Shanahan 1993; Aber, 1994; National Research Council 1993 and Watson et al. 1996 as cited in Shonkoff and Phillips 2000).

2.5 Public Policy Implications

The evidence just presented suggests that for children growing up in disadvantaged circumstances, the damage of early exposure to toxic stress compounds on the insults caused by suboptimal care and stimulation, material deprivation, and exposure to shocks, increasing the likelihood of lower developmental trajectories. The fact that, in the face of such levels of adversity, poor children still grow up to be relatively productive members of society is a testament to their incredible resilience, as well as their parents' effort and sacrifices. Yet the quality of life these children may lead, and that which they will eventually provide to their own offspring, is more likely to be substandard, a reflection of their own limited access to opportunities as children due to factors over which they had no control. What is more, these children's own levels of parental engagement when they grow up, and consequently the quality of care and stimulation they provide to their own children, is likely to be substandard, thus perpetuating the transmission of developmental deficits, sub-optimal life quality, and limited opportunities.

The loss of developmental potential that results from severe or chronic insults during childhood is a private tragedy for poor households and their children. But it also represents an enormous loss for society, as developmental deficits are associated with lower productivity and income, risky behavior, and crime (Grantham-McGregor et al 2007; Heckman 2007). This dynamic has considerable implications for the design of social policy; it represents a rare opportunity where efforts to improve people's lives and reduce inequality can be pursued while raising productivity both at the individual as well as societal levels (Cunha et al 2005).

The increased appreciation of development that takes place during early life and the role of parental care and stimulation in such development has important implications for the design of public policy aimed at improving the outcomes of children growing up in disadvantaged circumstances. On the one hand, it suggests that focusing on the earlier years of life, and consequently on the prevention of developmental shortfalls, has a much greater chance (and is cheaper) of leading to better developmental outcomes than attempting later remediation. On the other hand, such efforts should be targeted not only to young children, but also to addressing issues that undermine parental care and stimulation.

Broad support for ECD initiatives has emerged in academic and policy circles in recent years, prompting myriad initiatives to improve the lives of young children growing up in disadvantageous circumstances. The results of these EC interventions have been considerably less uniform and conclusive than the sound conceptual foundation underlying them or the empirical evidence on the foundational nature of early development from the neuroscience and developmental biology fields described in section 2.2.

Although at first glance these mixed results appear to question the relevance of ECD for long-term outcomes, two possible alternative -and mutually complementary- explanations emerge. First, the diversity of results is a function of the enormous variations across ECD interventions (Nores and Barnett 2010). These programs range from childcare or early childhood education to targeted interventions for specific vulnerabilities (e.g., child maltreatment). Their scale, intensity and duration vary greatly. Interventions target children at varying stages of early development, their parents, or both, and focus on health, cognitive, or socio-emotional outcomes, or a combination of them.

Format and service modality also varies considerably, involving material support (conditional or unconditioned, cash or near-cash transfers), childcare and/or parental education. Programs can be home-based or center-based; carried out in formal schools or in informal settings, conducted by professional staff with extensive training and supervision or by volunteers with considerably less training who are not closely monitored. These program features are combined differently in different interventions and implemented in diverse contexts (Baker-Henningham and Lopez Boo 2010; Nores and Barnett 2010). The quality of their implementation, which is a key determinant of program success, varies widely across interventions (Shonkoff and Phillips 2000; Engle et al 2007; Almond and Currie 2010; Baker-Henningham and Lopez Boo 2010; Nores and Barnett 2010).

This diversity notwithstanding, the evaluation literature consistently reports a few findings which suggest that the heterogeneity of results are more a reflection of what is (or not) known about how to best support ECD, than the extent to which early life influences developmental trajectories. Among these findings, two stand out: i) interventions that target children from very early on in life are the most effective (i.e., lead to better outcomes) and efficient (i.e., higher economic returns) (Schady 2005; Almond and Currie 2010; Baker-Henningham and Lopez Boo 2010; Nores and Barnett 2010); ii) multiple-outcome interventions yield stronger results: for all outcomes, programs that offer a combination of interventions report larger average effects than single-focused ones (Grantham-McGregor et al 1991; Engle et al 2007, Nores and Barnett 2010).

The consistency of these two findings with those reported in section 2.2 about the hierarchical and interdependent nature of human development is significant; it points to specific areas where public policy interventions can focus to improve the prospects of children born in

disadvantaged households, even if the knowledge on what determines optimal design and implementation for effective ECD interventions remains incomplete.

Second, the mixed results reported by the ECD intervention evaluation literature may reflect deficits in parental engagement in poor households. Although, in principle, increased access to quality and quantity of inputs should lead to better outcomes, in practice, children in low-income households may not fully benefit from the additional resources, services and opportunities made available by interventions because of their parents' own overwhelming circumstances, developmental deficiencies, or inadequate understanding of the significance of EC development or important inputs it entails.

Given the central role that parents play in children's early development, addressing the issues that undermine parental engagement is a necessary condition to increase the effectiveness of ECD interventions. The growing policy focus on the earlier years of life should be complemented with an expansion of programs that support parents in low-income households so they can foster the development that takes place during their children's early life.

Current policy initiatives that aim to support parents still largely focus on relaxing budget constraints or increasing the availability of resources. While these interventions are helpful in somewhat alleviating the material constraints and lower parental stress of disadvantaged families, evidence suggests that, in and of themselves, they do not significantly improve poor children's developmental trajectories (Baker-Henningham and Lopez Boo 2010). Rather, the evidence I review in this essay and the results of the empirical research I conduct in this dissertation suggest that improving the quality of parental inputs during early life should be given its due attention. In this regard, two main issues need addressing: i. heightening parental awareness of ways to

increase the effectiveness of their contributions to their children's early development, and ii. acknowledging and addressing the impact of stress on low-income households.

Explicitly focusing on increasing parental awareness about the high returns on investments during early childhood and the most influential environmental inputs can go a long way toward improving the quality of care and stimulation that parents in disadvantaged households provide to their children (Center for the Developing Child 2010), even in the face of material deprivation. In particular, educating parents on childrearing best-practices and promoting parental sensitivity, responsiveness, and availability can help boost the quality of early experiences and minimize the effect of toxic stress that poor children are disproportionally exposed to. The limited evidence from EC interventions supports this point: programs that offer child-focused educational activities with explicit attention to parent-child interaction patterns and relationship building improve the quality of these interactions, while simultaneously boosting children's developmental outcomes (Baker-Henningham and Lopez Boo 2010).[4]

As important as it is to provide resources and information for the improvement of the quality of care and stimulation that poor parents provide their children, these efforts may not be sufficient to close the parental engagement gap. This is because, as section 2.4 makes clear, poverty undermines parenting in more ways than material deprivation and inadequate knowledge about ECD. Without a doubt, much remains to be learnt about how poverty and stress interact to undermine poor children's development. And given the intricate and multidimensional nature of poverty, empirically establishing such channels of influence is not straightforward. The abundance of confounding factors makes it very difficult to disentangle cause from consequence, as well as isolating the effects of intermediate from fundamental channels.

[4] In addition, the extent to which parents perceive that they or their children need help and accept a program's design, requirements, and conditions has been consistently found to be an important determinant of its effectiveness at both affecting parenting behavior and children's outcomes (Almond and Currie 2010).

However, without the buffering support of available, responsive, sensitive, well-informed parents and other adults in their lives, toxic stress can derail children's developmental trajectories. Policy actions in two fronts could help to shield vulnerable children's outcomes from the noxious consequences of the stress associated with growing up in poverty: 1. addressing mothers' CMD, and 2. building support systems at the household, community, or institutional level that either improve the quality of the home environment or provide alternative nurturing environments for children outside the home. Chapters 2 and 3 of the dissertation provide an extensive discussion of these issues.

The evidence presented in this essay, along with the empirical findings in the other essays of this dissertation, highlight the pervasive effect of stress on children's development. The fact that low-SES households are disproportionately vulnerable to stress and associated CMD due to the high levels of adversity and deprivation to which they are subject to (Walker et al 2007; National Scientific Council on the Developing Child 2010) suggests that stress and associated mental health problems constitute a channel through which poverty undermines parents' ability to care for their children and result in worse development outcomes.

However, my research raises nearly as many questions as it answers. Further research on this topic is necessary to better understand how stress interacts with poverty to undermine parenting and ECD. More evidence that systematically quantifies the consequences of stress on child development, the channels of effect, and how to mitigate its impact on children could go a long way in the quest to improve the developmental outcomes of children growing up in disadvantaged circumstances, giving them a chance to live a better life and, in turn, be able to provide higher quality care and stimulation to their own children. In addition to benefiting poor

children, a better understanding of the relationship between ECD, stress and poverty may lead to more efficient and effective applications of social policy that are valuable for society at large.

If better care and stimulation during early life improves the odds of higher developmental trajectories, ECD interventions that address parenting deficits can lead to a healthier population and more productive workforce that make a greater contribution to economic growth and pay more taxes while reducing the need for public expenditures on special education, grade retention, welfare assistance, and incarceration (Grantham-McGregor et al 2007; Heckman 2007). In the face of competing demands for limited resources, investments in the development of the most vulnerable young children (and their families) offer the greatest returns for both beneficiaries and society at large.

2.6 Conclusions

My exploration of the relationship between early childhood development and stress in this essay suggests that stress represents a considerable developmental risk factor, particularly for children growing up in poverty. Poor children are not only disproportionally subject to levels of stress that on their own are highly detrimental to their early development, but are also less likely to have the buffering support their parents could provide because parents themselves are victims of such stressful environments. Because robust and timely development during early childhood relies on sound environmental inputs, the acute level of deprivation that results from parental stress and low capability may compromise developmental foundations, further conditioning children's later outcomes and undermining their ability to reach their full potential later in life. Moreover, because parental engagement is a function of parental capability, poor children's developmental deficits are likely to undermine their own parenting, thus perpetuating the transmission of poverty.

This essay provides motivation for public investments in ECD for disadvantaged families. The evidence suggests that timing matters. Although developing systems are resilient and exposure to shocks during early life need not lead to permanent developmental deficits, post-EC adjustments are both difficult and costly (and usually incomplete). Consequently, efforts that focus on building strong foundations instead of remediating ECD deficits are not only more likely to lead to better developmental outcomes, but also to do so more effectively and efficiently.

In the end, however, parental involvement and endorsement may be the critical factors for increasing the effectiveness of initiatives aimed at improving the developmental outcomes of children growing up in disadvantaged circumstances. Although limited, the current evidence from early childhood interventions suggests that by educating parents about children's developmental stages, inputs and capabilities, programs can improve parental practices, sensitivity and responsiveness, and hence remove some crucial obstacles that hinder poor households' investments in their children's capabilities, even when household income levels remain unchanged. In addition, maternal mental health problems should be recognized as disorders of public health significance and integrated as such into maternal and infant health policies.

The confluence of knowledge on ECD from fields ranging from neurobiology to developmental psychology and the economics of human capital formation has added enormously to the appreciation of development that takes place during early life and the role of parental care and stimulation in such development. Still, my analysis in this essay suggests that there is much more to learn: further research is warranted to deepen the understanding of how poverty and stress interact to undermine children's development directly and contribute to the parenting gap. Better understanding of these two questions is essential for the design of effective policies that

protect poor children's developmental trajectories, which is critical in and of itself, as well as for the broader goal of disrupting the inter-generational transmission of poverty.

3. Does Maternal Mental Health Significantly Determine Early Childhood Nutritional Outcomes?

3.1 Introduction

Maternal mental health problems have been singled out in the early childhood developmental (ECD) literature as a developmental risk factor (Shonkoff and Phillips 2000; Knudsen et al 2006; Walker et al 2007; Center on the Developing Child 2009). Numerous studies have shown a strong association between maternal Common Mental Disorders (CMD), such as depression and anxiety, and sub-optimal children's physical, cognitive and socio-emotional outcomes (Patel et al 2003; Rahman et al 2004; Harpham et al 2005; Walker et al 2007). Beyond these strong associations, there is limited conclusive evidence demonstrating that CMDs lead to deficient developmental outcomes. This essay aims to address this gap in knowledge.

Maternal mental health problems can undermine maternal competence in childcare and negatively affect the quality of mother-child interactions (Cohn & Tronick 1989 in Glaser 2000; Lovejoy et al 2000; Shonkoff and Phillips 2000; Farah et al 2006). Theoretically, children's development can be conceptualized as the result of the actions of utility-maximizing parents, who invest in a child's human capital subject to initial endowment (i.e., health, innate abilities of the child), parental preferences for the quality of children's outcomes, and parental budget constraints (Behrman and Lavy 1994). If CMDs constrain parents' ability to turn inputs into outcomes, a key aspect of this utility-maximization process, the resulting outcomes can be compromised.

Of particular interest are episodes of CMD affecting mothers (who typically play a more prominent role in childrearing and are more prone to depression in the post-partum period due to hormonal changes linked to childbirth and stressors associated with childrearing), during early childhood (up to age five), a time when development systems are particularly malleable

(Shonkoff and Phillips 2000). Because maternal competence in childcare is likely to play a greater role in a child's physical wellbeing and survival in developing countries where environments can be more hostile (poor sanitation, environmental toxins, violence) (Patel et al 2004) and both CMD prevalence rates and developmental deficits tend to be higher (Walker et al 2007, Fisher et al 2012), studying the effect of maternal depression on ECD in these countries is particularly important. However, the identification of a causal link between CMD and ECD outcomes in developing countries is particularly difficult given that: i) economic deprivation and low levels of education are among the most common risk factors for both children developmental deficits and maternal CMD (Wachs et al 2009); and ii) restricted availability of longitudinal studies that collect data on both CMD and children's indicators has limited research on this topic to cross-sectional studies that fail to address the potential endogeneity of maternal mental health (Harpham et al 2005).

This essay seeks to establish whether CMD lead to worse developmental outcomes for children in Peru, providing much needed empirical evidence of this important issue in a region where the causal nexus between CMD and ECD outcomes has been only scantily studied despite CMD prevalence rates that range between 35 and 50% (Wolf et al 2002).[5] To address the evidence gap on the link between CMD and ECD outcomes, and the potential endogeneity associated with it, this essay exploits a rich longitudinal study, the Young Lives survey, to study the effect of maternal depression and anxiety on children's nutritional status in Peru.

Determining whether maternal mental problems constitute a developmental risk factor is relevant for the effective design of public policy aiming to tackle children's developmental deficits, particularly those growing up in disadvantaged circumstances. Because poverty entails a higher likelihood of CMD (Walker et al 2007; National Scientific Council on the Developing

[5] Maternal depression in Africa and Asia has been estimated at 15%-28% (Husain, Creed, and Tomenson 2000).

Child 2010), if these problems significantly hamper the quality of maternal care and stimulation, this would increase the chances of a poverty trap: low household socio-economic status leads to maternal depression that intensifies the negative effects of material deprivation and exposure to exogenous shocks associated with poverty, confining children to substandard developmental trajectories and hence worse outcomes later in life for themselves and their offspring. Indeed, the substantiation of a causal link between CMD episodes during early life and children's outcomes in low-income settings would suggest that maternal mental health problems contribute to the intergenerational transmission of poverty and thus its long-lasting impact. Policy interventions targeting CMDs may therefore help substantially dampen the transmission and persistence of poverty across generations by improving children's developmental trajectories, along with the direct benefits they would provide to mothers.

This essay contributes to the literature that studies how parents influence children's developmental outcomes in several ways. First, it analyzes the relationship between maternal mental health problems and ECD outcomes, taking advantage of the Young Lives survey, which allows to better capture the complex and dynamic nature of early development missed in cross-sectional studies to date. Moreover, the availability of siblings' nutritional outcomes in the study, combined with its longitudinal design and the wealth of information included in the survey allows for addressing the issue of causality in the relationship between CMDs and ECD.

Second, while most research on the effect of maternal mental health on children's outcomes focuses on antenatal and postnatal episodes of CMD, I investigate the effect of maternal depression beyond what is usually considered the post-partum period (the first six months after birth). Although women are particularly susceptible to CMD during pregnancy and immediately after a child's birth, episodes of depression or anxiety can occur beyond the first

few months of giving birth (National Scientific Council on the Developing Child 2010), particularly in lower income settings. Because development that takes place during the first five years of life shapes developmental trajectories (Knudsen et al 2006), it is crucial to expand the understanding of the CMD-ECD nexus beyond the post-partum period. The results of my study contribute to this understanding.

An additional contribution of this essay is its focus on children's long-run nutritional status, a key determinant of human capital (Glewwe and Jacoby 1995; Strauss and Thomas 1998; Shonkoff and Phillips 2000; Walker 2007; Black et al 2008; Horton et al 2008; Center on the Developing Child 2010), which has received insufficient attention in studies of the impact of maternal mental health on ECD outcomes.

Finally, I investigate the potential channels through which CMD affect mothering to not only answer the question of *whether* maternal mental health problems lead to worse developmental outcomes, but also *how* this happens, a subject that has received limited attention in the literature, partly due to data limitations.

Results suggest that maternal mental health problems negatively affect child growth, and that this effect is sizable and statistically significant, despite the fact that the analysis in this study (based on maternal fixed effects) excludes cases of depression that span both rounds of data collection. Because the excluded cases are likely to represent more severe or chronic cases, this essay's results probably represent a lower bound of the true impact of maternal depression on child growth. Results are not statistically different depending on maternal or household characteristics. In addition, evidence in the essay suggests that a potential channel of effect entails maternal depression hindering the level or quality of maternal engagement, which in turn could lead to sub-optimal care practices that undermine children's growth. Estimates are robust

to alternative specifications, including an instrumental variable strategy, as well as to excluding migrant households.

3.2 Maternal Mental Health and Early Childhood Development

This essay is broadly related to the literature that studies how parents influence children's developmental outcomes. This literature has exploded over the last two decades and benefits from contributions from an array of disciplines. For instance, research from the fields of neuroscience and developmental biology establishes that i) early parent-child interactions and stimulation during early life are crucial for human development because the brain is wired to rely on such interactions for its development; and ii) the quality of environmental inputs during early life matters most due to the hierarchical and interdependent nature of human development (Shonkoff and Phillips 2000; Knudsen et al 2006).

The first essay of this dissertation concludes that, because early experiences and environmental inputs shape the architecture of the brain, acute stress during early life can be highly detrimental to early development. Without the buffering protection afforded by stable, responsive relationships, stress can become embedded in foundational neural circuits. Although parents can be an important source of protection in the face of acute stress, they can also be severely affected by it. As the essay highlights, this is particularly the case for parents living in poor households, who are themselves overwhelmed by the stressful circumstances in which they live, which hinder the quality of parent-child interactions, as well as the care they provide to their children. In fact, as highlighted in the first essay of the dissertation, CMD associated with stress, such as depression and anxiety, are disproportionally higher in lower-income settings.

A number of studies from psychology, psychiatry, and sociology present evidence on how CMD disrupt mothers' caregiving, as well as the environment in which children grow up. For

instance, the seminal study on child development edited by Shonkoff and Phillips (2000) makes the case that maternal mental health undermines maternal availability, sensitivity, responsiveness, nurturance and warmth, all essential determinants of quality interactions between mothers and their children. Cohn and Tronick (1989 in Glaser 2000) and Lovejoy et al (2000) report that CMD increases the probability of hostile environments. Farah et al (2006) present evidence that CMD are the most common factors behind child maltreatment, especially neglect. The literature on human capital formation provides evidence that mental disorders affect productivity (Frank and Koss 2005), which is in line with evidence that CMD inhibit mothers' ability to provide optimum care for their children (Center on the Developing Child 2009).

Do lower quality caregiving environments and reduced maternal productivity affect children's developmental outcomes? Maternal depression undermines infant-mother attachment (Martins and Gaffan 2000), which is instrumental for robust and timely development, and leads to lower quality interpersonal functioning, as well as more prevalent behavioral problems. Several studies from high-income countries report evidence of a causal relation between maternal CMD and worse socio-emotional outcomes (Lyons-Ruth 1986; Stein et al 1991) and behavioral outcomes (Peterson and Albers 2001; Kim-Cohen, Muffin et al. 2005; Frank and Meara 2009). Research on the effect on cognition has been less conclusive: for instance, while Cogill et al (1986) and Petterson and Albers (2001) report lower cognitive outcomes for children exposed to depression, Frank and Meara (2009) find no effect on cognition.

Findings on the effect of CMD on physical development are constrained by the fact that most studies are cross-sectional and only follow-up through the first 2 of life. Studies report mixed results, perhaps the result of differences in measurements of both physical growth impairment and maternal depression, length of follow-up, sample characteristics, and sample

sizes across studies, as well as varying level of social resources, caregiving norms, and prevalence of both malnutrition and maternal depression in the populations under study (Surkan et al 2012). For instance, Wright et al (2006) study the relationship between post-natal depression and weight gain for infants in northeast England, finding a strong but transient effect after adjusting for confounding factors.

Wojcicky et al (2011) compare growth curves given exposure to maternal depression for the children of Latino women born in California and report reduced weight gain in the first two years of life and greater risk for failure to thrive among children consistently exposed to depression in the pre- and postnatal period compared to those unexposed or exposed intermittently. Using US nationally representative longitudinal data, studies by Surkan and co-authors (2012 and 2014) report that mothers with moderate to severe depressive symptoms at 9 months post-partum had children with significantly shorter stature than children's of mothers without depressive symptoms, effects that persisted throughout the child's first 6 years.

Harpham et al (2005) study the association between CMD and child nutritional status for four developing countries (Ethiopia, India, Peru and Vietnam) using cross-sectional data. The authors find a positive association between maternal depression and poor child nutritional status for all countries, but once confounding factors are controlled for, the relationship remains significant only in India and Vietnam. These results are in line with previous cross-sectional studies in South Asia showing an association between maternal depression and impaired child growth (Patel et al 2002, Chandran et al 2002, Rahman et al 2004, Patel et al 2004). Cross-sectional (Surkan et al 2007) and longitudinal studies (Santos et al 2010) found no effect of maternal depression on child anthropometric measures in Piaui, Northeast Brazil and Pelotas, Southern Brazil, respectively. Also using longitudinal data, Adewuya et al (2007) analyze the

effect of major post-partum depression on child malnutrition in Nigeria. The authors report that depressed mothers were more likely to stop breastfeeding early and their infants were more likely to have episodes of diarrhea or other infectious illnesses. Finally, a systematic review and meta-analysis in developing countries found an association between maternal depression and early childhood underweight and stunting (Surkan et al 2011).

3.3 Conceptual Framework & Identification Strategy

This section presents a simple conceptualization of the relationship between maternal mental health problems and ECD outcomes drawing from work by Frank and Meara (2009), which motivates the empirical identification strategy described below. Consider two stages of early childhood (EC): the first stage (*t-1*) comprises the first 3 years of a child's life, a time that has been touted as a critical window for development (Shonkoff and Phillips 2000). The second stage (*t*) spans from the third through fifth birthday. Frank and Meara (2010) model the effect of maternal depression on the formation of children's skill by building on James Heckman and co-authors' inter-generational model of human capability formation (Cunha, Heckman et al 2005; Cunha and Heckman 2007): [6]

$$DO_t = F\,(PS, DO_{t-1}, I_{t-1}; M_{t-1}) \qquad (1)$$

where *DO* is the level of developmental outcomes (including physical health accumulation up to age five), PS represents parental skill attributes (education, cognitive abilities, etc.), I investments (both monetary and non-monetary) in child capabilities at time *t-1*, and M is maternal mental health status at time *t-1*. Investments are assumed to be a function of maternal preferences, budget constraints, and initial conditions (Outes-Leon et al 2011). Mental health

[6] Heckman's model encapsulates the advances of knowledge regarding the effects of the home environment on children's early development. In it, children's capability is a function of their parents', along with genes, initial conditions, and investments. The authors define capability as a more comprehensive conceptualization of human capital that includes health attributes, as well as cognitive and socio-emotional skills.

problems that interfere with mother-child interactions or undermine maternal behavior during *t-1* could potentially undercut the effectiveness of parental skills and/or reduce the productivity of investments and hinder the accumulation of children's physical health.[7]

The challenge of estimating the effect of maternal depression on child nutritional status is that characteristics that determine parental investments in children's outcomes (e.g., child's or mother's age, parental health knowledge, child genetic endowments or innate healthiness; Glewwe and Miguel 2008) could also be associated with maternal mental health problems. In this context, if such observable and unobservable factors are not controlled for, ordinary least squares estimates of the effect of maternal depression on child nutrition are likely to be biased (Behrman 1996). Consequently, I take advantage of information on nutritional outcomes for pairs of siblings to conduct maternal fixed-effects estimations, thus eliminating bias due to household factors that are common across siblings (Alderman et al 2006).

Empirical Strategy

Figure 1, which depicts the non-parametric relationship between height-for-age Z-scores and age in months by maternal depression, motivates the identification strategy. The figure illustrates the results of kernel-weighted local polynomial regressions of height-for-age Z-scores on age in months using a Epanechnikov kernel.[8] The red line indicates children whose mothers were depressed during their first stage of life, while the blue line indicates children whose mothers were not depressed during the same period. The figure presents suggestive evidence that the height-for-age Z-scores for children of depressed mothers during early childhood's first stage is

[7] Given data collection issues described in section 3.4.3, the age of children in stage 1 actually ranges from 6 to 60 months, well beyond the window proposed in the conceptual framework. To the extent that: i. all children in the sample are observed during early childhood in stage 1; ii. controls are included in the empirical strategy to account for critical windows; and iii. the production function represents a process in which outcomes accumulated at the end of the second stage are assumed to capture investments made in the child during the first stage, the fact that children's age profile does not exactly match that proposed in the concept framework should not have significant implications for the empirical strategy.

[8] The zero line represents the WHO standard.

lower by the time they reach the second stage than that of those whose mother was not depressed during EC stage 1.

The empirical identification strategy exploits information on nutritional outcomes for 359 pairs of siblings and relies on the fact that the mental health status of 29 percent of mothers in this sub-sample changed between the first and second rounds of the survey (i.e., they were depressed in 2002 but not depressed in 2006/7, and vice-versa). Identification comes from comparing the nutritional status measured when siblings were of similar age but at different points in time, where one was exposed to maternal depression during early childhood while the other was not. The implicit assumption is that differences across children in average height-for-age Z-scores would be similar between siblings of depressed and non-depressed mothers in the absence of maternal mental health problems.

Using mother fixed effects to compare the effect of exposure to maternal CMD between siblings' outcomes, the econometric specification is:

$$HAZ_{tij} = \beta D_{t-1ij} + X'_{tij}\,\Theta + BW'_{t-1ij}\,\gamma + Year\ 2006_t + \lambda_j + \varepsilon_{tij} \qquad (2)$$

where *HAZ* denotes height-for-age Z-score for child *i* in household *j* in EC stage 2 or *t* (i.e., 2006/7 or 2009/10). $D_{i,t-1}$ captures whether the mother suffered from depression in EC stage 1 or *t-1* (i.e., 2002 or 2006/7). The parameter of interest, β, measures the impact of maternal depression on children's nutritional status**.** Because the identification strategy relies on changes in maternal depression between the first and second rounds of the survey, in this model β only captures the effect of cases of depression that do not span both rounds of data collection, which are likely to represent more temporary, perhaps milder cases of depression.

Year 2006 is a binary variable that controls for the average changes in HAZ Z-scores between 2002/2006-07 and 2006-7/2009-10. To capture potential time-varying factors that lead

to differences in health status between birth cohorts, all regressions incorporate a vector of demographic controls, $X_{tij,}$ that may influence parental investments in children (wealth, exposure to shock, and urban/rural location), whether the mother is a teenager, as well as community controls, including access to social assistance programs and violent crimes in the community, both of which have been documented to affect children nutritional status (for a review, see Shonkoff and Phillips 2000). [9]

To explore whether the effect of CMD on child's health differs depending on children's characteristics, the baseline specification also includes a range of child-specific controls, such as gender, birth order, and sibling difference in age in *t-1* and *t* to address the fact that the baseline information and outcome measurements were collected at different time intervals for index children and their younger sibling (see section 3.4.3). λ_j represents unobserved maternal characteristics that are fixed over time that affect children's nutritional status. ε_{tij} is a random, idiosyncratic error term.

The fact that the specification uses measures of maternal mental health and child's health taken at different points in time (*t-1* and *t*, respectively) addresses to a large extent the possibility of reverse causality, i.e., that a child's poor health triggers maternal depression. Nevertheless, I include additional indicators for children's birth weight, long term health conditions and disability (BW_{t-1ij}) in the specification to allay concerns related to reverse causality.

The probability that there are unobserved factors that have affected the household between rounds that influence maternal CMD and children's outcomes. Consequently, I consider an alternative specification that incorporates an instrumental variable (IV) approach to address the possibility of omitted variable bias. This method hinges on finding observable covariates that are

[9] Although the survey includes a rich set of community indicators, comparability issues between rounds of data restrict the community controls I include in the estimations to the two mentioned above.

correlated with maternal mental health but which do not affect child nutritional status or other possible omitted variables. These covariates are then used in a two stage least squares (2SLS) estimation of the effect of maternal CMD on children height-for-age.

As the instrument, I use the proportion of depressed mothers in the community, which is both relevant and exogenous to maternal depression/anxiety in the household. There are two possible mechanisms through which the proportion of maternal CMD at the community level may influence a particular mother's mental health. First, higher levels of community CMD may increase the probability of individual CMD. Evidence from studies from the field of psychology suggest that depressive symptoms (such as cognitive vulnerability to depression, i.e., a person's tendency to focus on their negative mood and to ruminate about it) can be contagious (Haeffel and Hames 2014). Second, the percentage of depression in a community may be capturing the effect of an external factor that affects the community at large, such as environmental factors like weather, which in turn influences mothers' mental health (e.g., Rosenthal et al 1984). Regardless of the mechanisms, it is highly unlikely that the proportion of depressed mothers in the community has an impact on children's health status other than through the impact on maternal mental health. The instrumental variable specification (FEIV) controls for the vector of household, child, and community characteristics in equation (2). For all regressions in the essay I use robust standard errors.

3.4 Data

3.4.1 Young Lives Peru Survey

The source of data for this study is the Young Lives Peru Survey (YL), conducted by the University of Oxford and core-funded by the UK Department for International Development.[10]

[10] The YL survey is also being conducted in Vietnam, Ethiopia, and India (Andra Pradesh).

The study consists of a longitudinal sample of children born in 2000-2001 (henceforth, index children) who are being followed from infancy until they reach their mid-teens.

The baseline sample of YL is cluster stratified, with 20 districts randomly selected across the country (seven on the coast, ten in the highlands and three in the jungle). Because the YL project is particularly interested in children living in poorer households (Wilson et al., 2006), the sampling frame excluded the top 5 percent of districts as measured by a district poverty ranking. Despite excluding the least poor, it has been documented that the data reflects the Peruvian population in a broad range of indicators (Young Lives-GRADE, 2008).

Within each of the selected districts, 100 households with at least one child born between 2001 and 2002 were chosen randomly to participate in the project. The survey collects a rich set of information at the child, household and community levels, including early developmental, economic and demographic indicators, as well as information about social assistance programs in every community. The attrition rate between rounds of data collection is approximately 4 percent, which is low by international standards (Outes-Leon et al 2011). In addition, attrited households are not systematically different from non-attrited households based on observable characteristics. While differences in unobservable characteristics cannot be ruled out a priori, this low attrition rate suggests that potential biases in the results due to attrition are likely to be small (Dercon and Outes-Leon 2008).

There are currently three waves of available data: the baseline round, conducted in 2002 when the index children were aged 6-20 months, the first follow-up conducted in 2006-7, when they were between 4 and 6 years old, and the last round in 2009, when they were between 7 and 8 years of age. In addition to that of the index child, in 2009 data were also collected for the sibling born immediately after the index child (hereafter, the younger sibling) if they were at

least four years of age at the time of the survey. Most of these younger siblings were between four and six years old when the data were collected, a very close match to the index children's age when the survey's second wave was conducted.

This essay uses all three waves of data. Of the 2,000 index children in the baseline round, the analysis here focuses on the sample of 718 siblings in 359 households present in all three waves who were matched and for both of whom data on child nutrition and maternal mental health is available.

3.4.2 Measurement Variables

I use height as a measure of child health status, the outcome of interest for this study, which is widely considered a correlate of long-run investments in child nutrition and the best indicator of nutritional conditions and disease environment of childhood (Schultz 2010). I compute height-for-age Z-scores in stage 2 for each child. The Z-score is defined as the difference between the child's height and the mean height of the same-aged international reference population, divided by the standard deviation of the reference population.[11]

The explanatory variable is constructed using information on maternal common mental disorders from the Self Reporting Questionnaire 20 items (SRQ20), in the first and second round of data collection, which correspond to stage 1 for index children and their siblings, respectively. The SRQ20 is a screening (case-finding) tool included in the YL survey and is recommended by the World Health Organization and has acceptable levels of reliability and validity in developing countries. The SRQ20 consists of 20 yes/no questions with a reference period of the previous 30 days. It is not diagnostic and cannot separate out anxiety from depression, but to the extent that

[11] Height-for-Age Z-score estimates are based on WHO Child Growth Standards for children age 0-60 months. A z score of -1 indicates that given age and sex, the child's height is one standard deviation below the median child in that age/sex group. Following guidelines from the Centers for Decease Control, height-for age Z-scores smaller than -5 or greater than 3 were considered outliers and excluded from the analysis.

these conditions are closely related and both of them can undermine the quality of care mothers provide to their children, the information gathered from the questionnaire is very valuable (henceforth, depression is used to referred to both cases of depression and/or anxiety).

Using the responses to the questionnaire, I use available standards validated against clinical assessments in Peru to determine whether the mother experienced anxiety/depression while the index children and/or their younger sibling were young. The cut off score of 7/8 yes answers to the SRQ20 separates probable non-cases/cases.

3.4.3 Preliminary Descriptive Statistics

Table 3.1 presents descriptive characteristics for children in the sample of paired siblings for the first and second stages. As Panel A shows, index children were on average considerably younger than their sibling in stage 1 (almost 12 months old and 27 months old, respectively). Panel B, however, shows that index children were slightly older than their siblings in stage 2. These differences in age reflect the fact that rounds of data collection were not conducted on regular intervals (round 1: 2002, round 2: 2006-7, and round 3: 2009-10). Consequently, the gap of time that elapsed between the collection of baseline information and outcomes measurements is different for both groups of children (between 4 and 5 years for the index child, and 2 to 4 for their siblings).

In terms of gender, there are five percent more boys among index children than siblings. On average, index children had slightly higher weights in stage 1 and were less likely to be underweight at birth (Panel A). However, their siblings appear to be better nourished in stage 2 (Panel B). Siblings' better nutritional status could be the result of being born at a later stage of the household's life cycle, improved economic conditions, or increase access to programs in the community (Outes-Leon 2011).

As Table A.3.1 indicates, children characteristics for the sample of paired siblings at the baseline are statistically indistinguishable to those in the rest of the sample. Children's age, height-for-age z-scores, and the probability of being stunted in stage 2, however, are significantly different from the rest of the sample. Maternal and household characteristics also differ in these two sub-samples: on average, mothers in the sample of paired siblings are less educated and more likely to be of indigenous origin than mothers in the rest of the sample, while households in the former are less wealthy, less likely to have experienced a shock and to be in urban areas than the latter.

To allay concerns of sample selection, I estimated the effect of depression on nutrition using cross-section OLS regressions for each round of data, as well as by pooling the data (not reported) and in all cases reject the null hypothesis that the results are significantly different between the paired-sibling group and the rest of the sample. These results suggest that, although the insights of the analysis in this study are representative of relatively poor families that have at least two children, sample selection should not be of particular concern and results could be potentially extrapolated to the larger sample.

Table 3.2 compares depressed mothers and their children with non-depressed mothers and their children in the paired-sibling group. Depressed mothers are less likely to be teenagers, although the difference is not statistically significant, and more likely to be of indigenous origin (significant). On average, children of depressed mothers are younger and more likely to have long-term health problems. In addition, nutritional outcomes of children whose mothers were depressed are significantly worse: their average height-for-age z-score is 31 percent of a standard deviation lower and almost 9 percent more likely to be stunted than children of mothers who are not depressed.

Finally, Table A.3.2 depicts maternal mental health status during children's first stage of life. Of the 718 observations, 210 –or 29 percent— appear to be depressed in one round of the survey but not in the other, while 64 mothers are depressed in both rounds.

3.5 Results

3.5.1 Main Findings

Table 3.3 reports the main results of the essay. Column 1 presents results for pooled OLS estimations that provide a benchmark for the other specifications, while column 2 shows results for the baseline mother fixed effects model as described in equation (2). As expected, maternal depression has a negative impact on child nutrition, even if estimations are restricted to the effect of shorter episodes of maternal depression.[12] The size of the effect of maternal depression using pooled OLS is -0.154, a result that is statistically significant. Once time-invariant unobserved household characteristics are controlled for, the magnitude of the coefficient for maternal depression is -0.197, also statistically significant. The fact that the FE coefficient is larger than the Pooled OLS one, in spite of the FE estimation not including mothers who were identified as depressed in both rounds of data collection, suggests that omitted household or maternal fixed attributes (e.g., a maternal personality trait, such as dutifulness) may be moderating the effect of maternal depression on child nutrition.[13]

The effects on child nutrition of demographic controls in the table are as expected. When comparing across households (Pooled OLS), Z-scores of children whose mothers are teenagers are 29 percent of a standard deviation lower (statistically significant at the 5 percent level) than those of children whose mothers are not. Maternal ethnicity and education also have a sizable

[12] Pooled OLS results (not reported) suggest that, not surprisingly, the effect of exposure to maternal depression in both 2002 and 2006 is larger than exposure during one round only.

[13] Results in Table 3.3 are robust to the inclusion of outliers, as well as to the inclusion of controls for the number of adults or female adults in the household.

and significant effect on children's nutritional status, as does living in urban areas, although having experienced a shock does not necessarily do so. The coefficient for wealth is positive, statistically significant, and among the highest in Table 3.3. Z-scores of children who are the oldest sibling are 31 percent of a standard deviation higher (statistically significant at the 1 percent level) than those of children who are not. As expected, living in a violent community is associated with worse nutritional outcomes, both when comparing across households, as well as within households overtime.

Does the coefficient for maternal depression in Table 3.3 reflect the detrimental effect of maternal depression on early growth or is it capturing the effect of other factors associated with maternal depression that undermine early child growth? I address this question by examining whether the effects of exposure to depression during a child's young age varies depending on maternal or household characteristics. I estimate the baseline fixed effects model separately by maternal indigenous origin and education level (primary complete or lower level of education versus secondary incomplete or higher level of education). Results in Panel A of Table A.3.3 suggest that exposure to maternal depression affects children of indigenous mothers almost twice as much as those whose mothers are not indigenous. However, the equality of these coefficients cannot be rejected (not shown).[14] Panel B presents results by maternal education level. The coefficient for depression for the group of mothers who have attended secondary school or beyond is -.481 and statistically significant at 5 percent, while that for mothers who are less educated is -0.076 and not significant. However, again, the equality of coefficients cannot be rejected (not reported).

[14] Throughout section 3.5 the equality of coefficients is tested with F-test using restricted and unrestricted specifications.

To analyze whether household characteristics influence the results, I interact maternal depression with area of residency (urban/rural), as well as the wealth index (using both the actual index and dummy variables for levels of wealth index) in separate regressions (not reported). Coefficients for all interaction terms are insignificant. These results, along with those by maternal characteristics above, suggest that for the sample under study maternal mental health problems matter in and of themselves and not due to their correlation with other factors that affect the household and child growth.

3.5.2 Alternative Estimations

To address the possibility that the indicator for maternal depression may be correlated with unobserved determinants of child growth, I use the percentage of depressed women in the community as an instrumental variable. Column 1 of Table A.3.4 in the appendix reports first stage regression of the IV 2SLS estimation. The table shows that the instrument is a highly significant predictor of the maternal depression measure at the household level. The F statistic for the model (31) is well above the critical threshold that would indicate a potential weak instrument bias (10), as suggested by Stock and Yogo (2005).[15]

Results for the model specification using the fixed effects IV approach are presented in Table 3.4 (column 3), along with results from Table 3.3 (columns 1 and 2) for comparability purposes. Height-for-age Z-scores of children whose mothers were depressed before their fifth birthday are .619 standard deviations lower than children whose mothers did not experience a depressive episode when they were younger. This is a sizable effect: it represents a 40 percent reduction of the Z-score of children of depressed mothers vis-à-vis the average Z-score for children of non-depressed mothers (-1.59).

[15] F statistic here is Kleibergen-Paap Wald rk.

I next investigate a potential source of selectivity that could undermine the essay's results: migration. I re-estimate the baseline fixed effects model excluding all households that reported having migrated between rounds of data collection to investigate whether household migration between survey rounds biases the results.[16] Migration across communities in the sample of paired siblings may not be random: assuming households decide to move to maximize their wellbeing, a household will migrate if conditions (economic factors, access to services, weather, etc.) are better in the destination community then their community of origin. Because factors associated with increased wellbeing tend to be inversely correlated with maternal depression, if households with healthy mothers choose to live in places with better conditions –and, likely, a lower average level of maternal depression—the impact of maternal depression would be over-estimated.

Table 3.5 shows results for the fixed-effect baseline and the FEIV specifications for the sub-sample paired siblings who did not migrate between survey rounds.[17] Although the estimations exclude 8 percent of the sample in the baseline estimations, there is no indication that migration may be a source of bias, as both the magnitude and significance level of the results are in line with estimations for the unrestricted sample.

3.5.3 Possible mechanisms

How do maternal mental health problems undermine children's nutritional status during early childhood? As discussed in the conceptual framework (section 3.3), maternal depression can impair maternal childrearing practices, including general child care, feeding, and child supervision (Lovejoy et al 2000, McLearn et al 2007, Hurley et al 2008, Melchior et al 2009), which is detrimental to ECD outcomes given young children's reliance on their mothers for

[16] I also conducted these estimations by excluding households for whom the community of residence changed between rounds of data collection, even if they had not reported having moved. Results (not reported) are consistent with those in Table 4.6.

[17] Column 2 of Table A.3.4 in the appendix reports first stage regression of the FEIV 2SLS estimation.

survival, especially in lower-income settings, where conditions (physical, chemical, and social) can be particularly hostile (Patel et al 2004). In addition, maternal mental health problems can undermine mother-child interactions and stimulation, both crucial during early life because they shape brain development, and lead mothers to be less attentive and responsive (Glaser 2000, Shonkoff and Phillips 2000, Farah et al. 2006; Walker et al. 2007).

I explore whether there is evidence of compromised maternal competence for depressed mothers in the sample under consideration by exploiting indicators of general maternal behavior and maternal responses to children's persistent crying available in the first round of the Young Lives survey. The limitation of data on potential mechanisms to index children restricts the analysis considerably, as sibling comparisons cannot be conducted. Nevertheless, I tabulate these indicators by maternal mental health status and present the results in Table 3.6.[18]

In general, maternal behavior as captured in the table does not appear to be significantly different for depressed mothers than for mothers who were not depressed. Of the broad spectrum of behaviors in Table 3.6, the only indicator for which the difference between columns 1 and 2 is larger in magnitude and statistically significant is that for injuries, which captures whether children had a serious fall, burn, or broken bone. This difference, which is substantial in size and statistically significant at the 1 percent level, is consistent with the literature on the effect of mental health problems on maternal caregiving, which reports that maternal depression can lead to mothers being less engaged, thus increasing the likelihood of children experiencing serious injuries (Shonkoff and Phillips 2000).

However, the overall picture that emerges from Table 3.6 is that caregiving behaviors are not particularly different between depressed and non-depressed mothers in the sample under

[18] I also tabulated the indicators for maternal behavior for the sub-sample of mothers who were depressed both in 2002 and 2006/7. Results (available upon request) are very similar both in terms of magnitude and significance to those in Table 3.6.

study, which is somewhat surprising given that maternal depression has generally been found to impact caregiving behaviors (McLearn et al 2007). That caregiving behaviors do not significantly differ by depression status could suggest that depressed mothers are striving to fulfill their parenting responsibilities, even if they occasionally disengaged. It is also possible that the cases of depression captured in the data are not sufficiently debilitating to impair mothers' ability to provide basic care for their children. Or perhaps the indicators available do not capture the specific childcare behaviors that are affected by maternal depression for the women in the sample. For instance, although Table 3.6 suggests that depressed mothers are as likely to breastfeed as non-depressed mothers, there is no information on other feeding practices, which is more relevant for the children under study, given that the large majority of these children are older than 6 months old, and consequently less likely to rely exclusively on breast milk. Because the provision of nourishing food is arguably more involved than breastfeeding, it is possible that depression interferes with its provision to children in a way that it does not with breastfeeding.

Recent research provides evidence to this effect. Melchior et al (2009) suggest that depressed mothers may be less likely to cook healthy foods or shop for groceries. Maternal depressive symptoms, anxiety and stress have also been associated with less responsive feeding practices, which can hinder children's nutritional status (Hurley et al 2008, Black and Aboud 2011). In addition, research on adult stress and eating behaviors suggests that stress may lead to both over and under-eating in adults, depending on the nature of the stressor (Torres and Nowson 2007), which could affect maternal feeding patterns if, as Surkan (unpublished work) suggests, the way adults feed their children reflects how they eat themselves.

It is also possible that other mechanisms are at play, including compromised mother-child interactions resultant from maternal stress or anxiety. As I extensively document in the first essay of this dissertation, early mother-child interactions are key for childhood development. Consistent, sensitive and responsive interactions and affect (nurturance and warmth) facilitate the formation of secure attachments between young children and their parents, which in turn facilitates the creation of neural pathways that set the foundation for the normal development and functioning of healthy brains and other biological systems (Glaser 2000, Shonkoff and Phillips 2000, Farah et al 2006; Walker et al 2007).

However, maternal stress has been associated with insufficient stimulation and affect at best, and maltreatment and neglect at worse, both of which triggers stress, fear, and anxiety in children. These negative emotions can disrupt the architecture of the brain, impact regulation of the stress response system, and increase the odds of permanent deficits across life outcomes (McEwen 1998; Hertzman 2000; Martins and Gaffa 2000; McEwen 2000; Shonkoff and Phillips 2000; National Scientific Center for the Developing Child 2005; Center for the Developing Child 2010).

Exposure to toxic stress in the absence of the buffering protection afforded by stable, responsive relationships during early life put the body in a continual state of alert (Garner and Shonkoff 2012). Chronically elevated levels of cortisol, the stress hormone, have been associated with lower levels of growth hormone, which can seriously compromise physical outcomes, and potentially lead to reduced stature and even stunted growth (Charmandari et al 2003, Stratakis 2006). In addition, constant activation of stress response systems during early life can permanently increase an individual's susceptibility to acute and chronic illness, including

depression and anxiety, both during childhood and well into the adult years (McEwen 2007; National Scientific Council on the Developing Child 2007).

The indicators on maternal behavior when children cry persistently captured in Table 3.6 provide a glimpse of the quality of mother-child interactions for the sample under study. The table presents no evidence that depressed mothers maltreat or are more hostile toward their children than non-depressed mothers.[19] In addition, a number of behaviors, including whether mothers carry, sooth/sing, or rock the child, suggest that depressed mothers in the sample are as responsive and nurturing as non-depressed mothers. However, the survey provides no explicit information on the quality of interactions when children are not crying, and in particular, no indication of the extent of stimulation that children of depressed mothers receive compared to those of non-depressed mothers.

Given the limited scope of available indicators and also given that the analysis is restricted to a single observation in time for one child per family, the extent to which the quality of mother-child interactions constitutes a mechanism through which maternal depression affects children's physical health cannot be evaluated in a comprehensive way. However, the table does provide suggestive evidence that, as much as depressed mothers in the sample appear to be able to provide basic care for their children, as well as the same amount of comfort when they are upset as non-depressed mothers, depressed mothers are also more likely to disengage or to simply go through the motions, which could result not only in more serious injuries to children, but also reduced stimulation and suboptimal feeding practices, both of which are essential for sturdy development during early life.

[19] Studies have found that depressed mothers in developed countries are more likely to have a negative perception of children behavior (Foreman and Henshaw 2002) and perceive their infant as temperamentally difficult (Edhborg et al. 2000), which can exacerbate sub-optimal mother-child interactions (Hart et al. 1999). However, I find no significant evidence that depressed mothers in this study perceive their children's behavior differently than non-depressed mothers.

3.6 Policy Implications

Results in section 3.5 provide suggestive evidence that maternal depression undermines child growth and therefore may represent a developmental risk factor. Because of the hierarchical and interdependent nature of human development, the consequences of growth impairment during early childhood due to maternal depression are likely to be very costly, particularly if such faltering has permanent effects. There is a wealth of evidence associating stature during early childhood not only with physical outcomes, such as attained body size during adulthood (Thomas, Lavy, and Strauss 1996) and premature mortality (Martorell 1999), but also with a host of later life outcomes, including cognitive ability and school outcomes, earnings, productivity and marital outcomes (Strauss and Thomas 2008).

Further, because the analysis in this study excludes cases of depression that span both rounds of data collection, which probably represent more severe or chronic cases, this essay's main results could represent a lower bound of the true impact of maternal depression on child growth. Even so, the magnitude and statistical significance of estimates in section 3.5 are comparable to the impact of large, aggregate and generally unpreventable shocks that produce complex and very costly consequences (destroyed infrastructure, displacement, etc.). For instance, Bustelo et al. (2012) analyses the effect of a devastating earthquake in Colombia in 2000 on child nutrition and finds that the height-for-age Z-score for children residing in affected departments is significantly lower by 0.182 standard deviations than children residing in not affected areas. Akresh et al. (2011) report that Z-scores for children exposed to the Eritrean-Ethiopic war are -0.244 standard deviations lower than children who were not exposed. CMD, on the other hand, are easier and cheaper to prevent and treat.

Indeed, evidence from developed countries points to cost-effective treatments for women of childrearing ages at various income levels that ameliorate CMD, including counseling, group therapy, as well as medication for women who require more intensive intervention (Harpham et al 2003; Center on the Developing Child 2009). Psychological interventions delivered by supervised, non-specialist health and community workers in developing countries have also been found to be effective for the treatment of maternal depression, both in low and middle-income countries. A number of key features shared by successful interventions include: delivery provided within the context of routine maternal and child health care beginning in the antenatal period and extending postnatally; focus of the intervention beyond the mother to include the child and involving other family members; and attention to social problems that trigger maternal CMD and a focus on empowerment of women (Chowdhari et al 2014). Benefits of interventions that extended to children include improved mother-infant interaction, better cognitive development and, relevant to this essay, growth, reduced diarrheal episodes and increased immunization rates (Rahman et al 2013). Taking into account cultural differences and local sensitivities, these treatments could prove effective and efficient to improve maternal mental health in Peru, and in so doing improve the livelihoods of children whose early development is hindered by maternal depression.

Improved access to treatment alternatives in and of itself will not completely address the threat of maternal mental health problems to child development, as recognition of depression and awareness of the potentially serious nature of mental illness is low in developing countries (Wachs et al 2009). Consequently, there is need to improve the diagnostic capacity of health workers to identify women who could benefit from treatment, as well as increase the appreciation of maternal depression as a serious public health problem, which negatively affects

not only child development, but also mothers' own outcomes and families' economic productivity. In addition, public awareness campaigns that increase the recognition of symptoms and consequences of maternal depression by family and community members, as well as address the social stigma associated with a family member being diagnosed with a mental illness (Rahman et al 1998) could also improve reporting and associated treatment of mental health issues.

However, given that residual consequences to children's outcomes often persist even if maternal depression can be successfully treated (Forman et al 2007; Nylen et al 2006), the most effective way to minimize the effect of maternal depression on child development is to prevent it whenever possible. Alleviating environmental stressors associated with maternal depression, which women in developing countries are disproportionally exposed to (Broadhead and Abas 1998), can help. Although depression-related risk factors include macro-level issues such as poverty, conflicts, disasters, violence, migration, and higher prevalence of HIV/AIDS (Wachs et al 2009) that are difficult to tackle with single, smaller scale interventions, other depression-related risk factors such as domestic violence, women's lack of control over household resources or reproductive health, maternal iron-deficiency anemia, large family size and low birth weight, could be addressed comparatively more easily.

Children's bodies and brains are very resilient, and post-EC adjustments are possible (Greenough and Black 1992). However, reversing the effects of exposure to adverse environmental inputs during EC is by and large very difficult, entailing high-quality and costly interventions that typically need to be conducted at a sufficiently young age to be effective. Moreover, when prevention is not possible, addressing the effects of maternal depression on children's growth while children are younger is likely to be more effective in minimizing the

impact of exposure given the high malleability of developing systems earlier in life, both a time of opportunity and vulnerability. Further, improving access to 'buffering' factors, such as maternal education or social support (Wachs et al 2009), can help minimize the impact of both stressors and depression.

Aside from addressing maternal depression directly, policy initiatives could alleviate its effect by targeting the channels through which maternal mental illness threatens children outcomes. Although data limitations restrict the analysis of possible mechanisms in this essay, it has been documented elsewhere that both increasing maternal awareness of the importance of early interactions and stimulation (Baker-Henningham and Lopez Boo 2010) and incorporating mother-child interactions in treatment programs for depressed mothers in developed countries has led to substantially better child functioning (Nylen et al 2006 in Wachs 2009). Policy makers in developing countries should take note of the potential that targeting mother-child interactions offers to improve developmental outcomes of children of depressed mothers.

In addition, policy initiatives that make quality care available outside the home, particularly to lower income households, may prove effective to protect the early development of children of depressed mothers. Evidence from early intervention programs that provide high-quality center-based childcare for children living in poverty and for children in the child welfare system in the United States suggests that these centers can help protect children from family-based risk associated with poverty, including maternal depression (Cohn et al 1986, 1991), as well as address some of the stressors that mothers in lower income households face (NICHD Early Child Care Research Network 1997 in Shonkoff and Phillips 2000).

Importantly, the evidence from existing programs cited above highlights the extent to which treating CMD does not necessitate development of a new wave of standalone, singularly

focused CMD interventions. Rather, a successful strategy to facilitate engagement can entail adding maternal mental health components to existing interventions that deal with issues such as ECD or gender violence. In addition, improving coordination across government agencies that deal with these currently recognized issues can lead to productive synergies across programs that directly or indirectly address maternal depression. For any of the above to be successfully undertaken, however, developing countries should recognize maternal mental health problems as disorders of public health significance and integrate them into existing maternal and infant health policies with the goal of increasing the public health commitment to this issue (Patel 2002).

These considerations notwithstanding, further research is necessary to better understand the ways in which maternal mental health problems undermine mothering and child growth, which is essential for the design of effective interventions. Increasing the availability of longitudinal studies that collect data on both CMD and ECD is imperative to further the understanding of how depression affects mothers' abilities to care for their children, the specific impacts of such effect on children's developing systems, as well as the extent to which such effects persist over time and ultimately what constitutes the most effective interventions. All of these factors are essential for shaping policy choices to curtail the impact of maternal depression on child outcomes.

3.7 Conclusion

This essay contributes to the growing literature on how parents influence children's outcomes by producing the first empirical evidence that maternal mental health undermines the growth of offspring during early childhood in Peru. The empirical strategy uniquely exploits data on siblings' nutritional outcomes and variation in their mothers' mental health status between rounds of data collection to investigate the effect of maternal depression on child growth by using maternal fixed-effects. The measure of maternal mental health in this study refers to cases

of depression and anxiety after the first six months post-partum, helping to expand the understanding of the CMD-ECD nexus beyond the postnatal period.

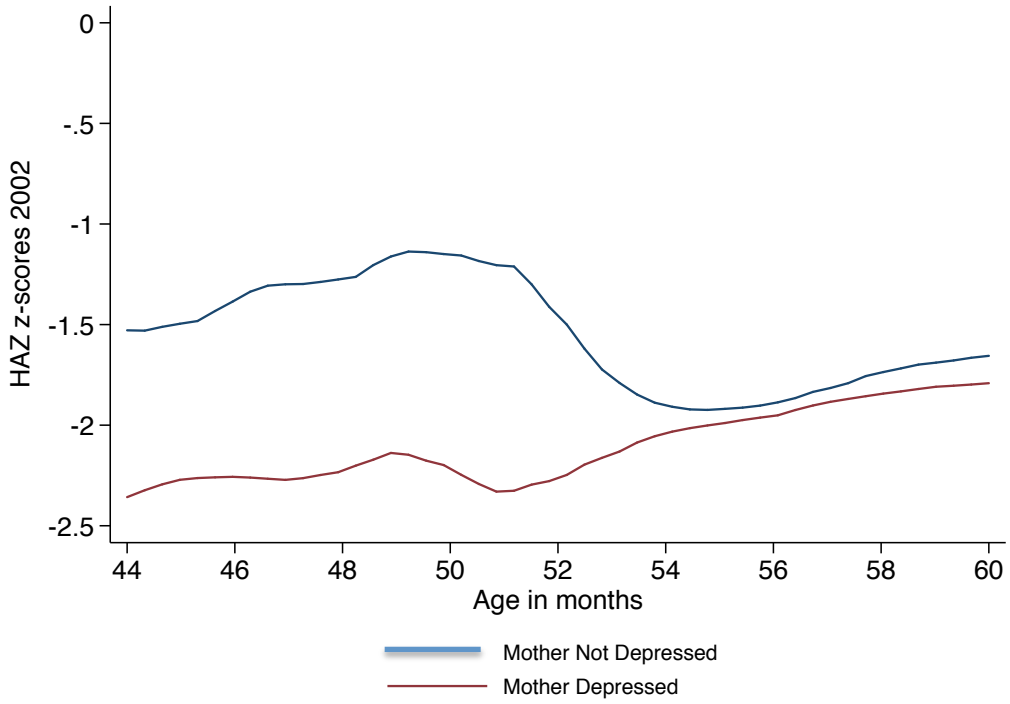
Estimates point to a negative effect of maternal depression on child growth. Results are not statistically different depending on maternal or household characteristics. In addition, evidence suggests that a potential channel of effect entails maternal depression hindering the level or quality of maternal engagement, which in turn could lead to sub-optimal care practices that undermine child growth. Estimates are robust to alternative specifications, including an instrumental variable strategy, as well as to excluding migrant households.

The results of this essay are noteworthy because, even if episodes of mental health problems captured in this study are not long-term, the consequences of poor growth during early childhood are likely to be, potentially affecting future health, education and economic outcomes. Despite its disease burden and the associated deleterious effects of child maltreatment and neglect, the commitment to maternal mental health has been minimal in developing countries (Patel et al 2006). The limited attention this issue has received is not entirely surprising given competing priorities for scarce resources and the limited evidence on the causal effect of depression on child health in these countries to date. Ironically, however, the poorer the setting, the higher the probability of maternal mental problems (Shonkoff and Phillips 2000). Although there is a lot more to understand about the poverty-maternal depression-child health connection, the results in this essay suggest that the cost of not addressing maternal depression can be very high, and thus contributes to the transmission of poverty across generations.

There is widespread agreement among health experts of the need to address maternal depression. The Consensus Statement developed by a UN-WHO expert group concluded that "political will, concerted action by global stakeholders and resources are needed now to integrate

maternal mental health in endeavors to achieve the Millennium Development Goals" (WHO-UNFPA 2007, p. 3). The additional evidence uncovered here for Peru adds further weight to the argument for policymakers to consider maternal mental health issues among the issues to address to make a positive impact on childhood development.

Figure 1: Height-for-Age Z-score by age in months and mother's depression



Source: Peruvian Young Lives survey, 2006/07 and 2009/10.
Note: Kernel-weighted local polynomial regression (using Epanechnikov kernel) of height-for-age Z-score. Red line indicates children of depressed mothers. Blue line indicates children of mothers who are not depressed. The zero line represents the WHO standard.

Table 3.1: Descriptive Statistics by Year

Variables						
Panel A –	Year 2002			Year 2006		
Early Childhood Stage 1	Mean	S.D	N	Mean	S.D	N
Age of the child (in months)	11.88	0.20	359	27.28	0.65	359
Eldest (%)	35.38	0.25	359	0.00	0.00	0
Male (%)	49.58	0.26	359	44.57	0.26	359
Birth weight	3.16	0.03	300	3.06	0.03	312
Low birth weight (%)	4.33	0.12	300	6.13	0.13	359
Long-term health problems (%)[a]	18.66	0.21	359	0.00	0.00	0
Panel B -	Year 2006			Year 2009		
Early Childhood Stage 2	Mean	S.D	N	Mean	S.D	N
Age of the child (in years)	4.72	0.03	359	4.58	0.06	359
Height for age z-score	-1.75	0.06	359	-1.57	0.06	359
Stunted (%)	43.73	0.26	359	36.49	0.25	359

Source: Own estimations using Peruvian Young Lives survey, 1st, 2nd and 3rd round.
Note: Sample of paired-siblings with available information on maternal depression in 2002 and 2006 and HAZ scores in 2006 and 2009.
[a] Sample of siblings exclude children with long-term health conditions.

Table 3.2: Descriptive Statistics by Maternal Depression

Variables	Mother depressed	Mother not depressed	Difference	
Panel A - Early Childhood Stage 1				
Child characteristics				
Age of the child (in months)	17.95	20.09	-2.14	**
Eldest (%)	15.98	18.22	-2.24	
Male (%)	49.11	46.45	2.66	
Birth weight	3.09	3.12	-0.03	
Low birth weight (%)	3.55	5.28	-1.73	
Long-term health problems (%)[a]	14.79	7.65	7.14	**
Maternal characteristics				
Teenage mother	4.73	7.10	-2.37	
Maternal indigenous ethnic group (%)	31.95	24.41	7.55	*
Mother with primary complete (%)	49.70	49.55	0.16	
Panel B - Early Childhood Stage 2				
Child characteristics				
Age of the child (in years)	4.71	4.63	0.08	
Height for age z-score	-1.90	-1.59	-0.31	***
Stunted (%)	46.75	38.07	8.68	**
Number of observations	169	549		

Source: Own estimations using Peruvian Young Lives survey, 1st, 2nd and 3rd round.

Note: Differences in characteristics between both groups are statistically significant *** at 1%, ** at 5%, and * at 10%. Sample of paired-siblings with available information on maternal depression in 2002 and 2006 and HAZ scores in 2006 and 2009.

[a] Sample of siblings exclude children with long-term health conditions.

Table 3.3: Impact of Maternal Depression on Child Nutritional Status

Dependent variable: Height-for-age Z-scores	Pooled OLS [1]	Fixed Effects [2]
Depressed mother	-0.154*	-0.197*
	(0.0867)	(0.109)
Teenage mother	-0.288**	-0.135
	(0.141)	(0.169)
Mother is indigenous	-0.219**	
	(0.0967)	
Mother with primary complete	-0.401***	
	(0.0982)	
Household experienced shock	0.0852	-0.0502
	(0.0765)	(0.0855)
Wealth index	1.082***	0.482
	(0.271)	(0.448)
Live in urban area	0.654***	-0.0499
	(0.107)	(0.145)
Year 2006	0.490***	0.272
	(0.188)	(0.195)
Child age in months S1	-0.00930	-0.0118
	(0.00984)	(0.0103)
Child age in years S2	0.109	0.175*
	(0.106)	(0.103)
Low birth weight	-0.199	-0.0511
	(0.173)	(0.211)
Oldest sibling	0.307***	-0.0264
	(0.113)	(0.142)
Child is male	-0.0165	0.0539
	(0.0724)	(0.0877)
Long-term health problems in	-0.00194	-0.152
	(0.123)	(0.137)
Violent crime in community	-0.184**	-0.357**
	(0.0877)	(0.153)
Social assistance (health) in	-0.407**	-0.0686
	(0.185)	(0.218)
Constant	-2.177***	-2.283***
	(0.433)	(0.465)
Observations	718	718

Source: Peruvian Young Lives survey 2002, 2006, and 2009.
Note: Robust standard errors in parenthesis. * significant at 10%, ** significant at 5%, and *** significant at 1%.

Table 3.4: Impact of Maternal Depression on Child Nutritional Status

Dependent variable: Height-for-age Z-scores	Pooled OLS [1]	Fixed Effects [2]	FEIV [3]
Depressed mother	-0.154*	-0.197*	-0.619**
	(0.0867)	(0.109)	(0.300)
Teenage mother	-0.288**	-0.135	-0.187
	(0.141)	(0.169)	(0.169)
Mother is indigenous	-0.219**		
	(0.0967)		
Mother with primary complete	-0.401***		
	(0.0982)		
Household experienced shock	0.0852	-0.0502	-0.0583
	(0.0765)	(0.0855)	(0.0872)
Wealth index	1.082***	0.482	0.417
	(0.271)	(0.448)	(0.447)
Live in urban area	0.654***	-0.0499	-0.0885
	(0.107)	(0.145)	(0.145)
Year 2006	0.490***	0.272	0.275
	(0.188)	(0.195)	(0.196)
Child age in months S1	-0.00930	-0.0118	-0.0150
	(0.00984)	(0.0103)	(0.0106)
Child age in years S2	0.109	0.175*	0.214**
	(0.106)	(0.103)	(0.105)
Low birth weight	-0.199	-0.0511	-0.0986
	(0.173)	(0.211)	(0.221)
Oldest sibling	0.307***	-0.0264	-0.0417
	(0.113)	(0.142)	(0.146)
Child is male	-0.0165	0.0539	0.0749
	(0.0724)	(0.0877)	(0.0893)
Long-term health problems in	-0.00194	-0.152	-0.0980
	(0.123)	(0.137)	(0.151)
Violent crime in community	-0.184**	-0.357**	-0.399**
	(0.0877)	(0.153)	(0.164)
Social assistance (health)	-0.407**	-0.0686	-0.0810
	(0.185)	(0.218)	(0.237)
Constant	-2.177***	-2.283***	
	(0.433)	(0.465)	
Observations	718	718	718

Source: Peruvian Young Lives survey 2002, 2006, and 2009.
Note: Robust standard errors in parenthesis. * significant at 10%, ** significant at 5%, and *** significant at 1%.

Table 3.5: Impact of Maternal Depression on Child Nutritional Status
Excluding Migrant Households

Dependent variable: Height-for-age Z-scores	Fixed Effects	Fixed Effects with IV
	[1]	[2]
Depressed mother	-0.197*	-0.452*
	(0.117)	(0.398)
Demographic controls	Yes	Yes
Year 2006	Yes	Yes
Mother fixed effects	Yes	Yes
Observations	660	660

Source: Peruvian Young Lives survey 2002, 2006 and 2009.

Note: Robust standard errors in parenthesis. * significant at 10%, ** significant at 5%, and *** significant at 1%. Demographic controls are the same as in Table 3.

Table 3.6: Maternal Behavior by Depression - Year 2002

	Mother depressed	Mother not depressed	Difference	
Panel A - General Childcare Behaviors				
Attended antenatal control	95.15	90.63	4.52	
Child covered by health insurance	81.55	78.52	3.04	
Child has received vaccines[a]	98.04	98.03	0.01	
Duration of breastfeeding	67.99	68.61	-0.62	
Mother is still breastfeeding	86.41	87.45	-1.04	
Child cared by young children[b]	4.85	2.73	2.12	
Child has had serious injuries[c]	26.21	10.55	15.67	***
Panel B - Maternal response to persistent crying				
Carries child	24.27	22.27	2.01	
Soothes/Sings to child	5.83	5.86	-0.03	
Rocks child	11.65	17.58	-5.93	
Gives child water	1.94	2.73	-0.79	
Smacks child	0.97	1.17	-0.20	
Shakes child	0.00	0.00	0.00	
Pinch/squeezes child tightly	0.00	0.00	0.00	
Threatens Child	0.00	0.39	-0.39	
Takes child to doctor	7.77	3.52	4.25	
Gives child medicine	4.85	3.13	1.73	
Bottle/breastfeed child	54.37	54.69	-0.32	
Puts child face down in crib	0.00	0.00	0.00	
Swaddles child tightly	0.00	0.00	0.00	
Lets child cry	0.97	1.95	-0.98	
Other	45.63	46.09	-0.46	
Observations	103	256		

Source: Own estimations based on Peruvian Young Lives survey, first round.
Note: Differences in characteristics between both groups are statistically significant *** at 1%, ** at 5%, and * at 10%. Sample of observations with children and sibling HAZ scores and available information on maternal depression in 2002 and 2006.

a. Vaccines include: polio, BCG, and measles
b. Child regularly left alone with children under 5yrs
c. Injuries include: serious fall or burn, and broken bone.

Appendix

Table A.3.1: Paired-Sibling vs Rest of Sample

Variables	Paired-Sibling	Rest of Sample	Difference	
Panel A - 2002				
Age of the child (in months)	11.88	11.48	0.4	*
Eldest (%)	0.35	0.4	-0.05	
Male (%)	0.5	0.51	-0.01	
Birth weight	3.16	3.22	-0.05	*
Low birth weight (%)	0.04	0.05	-0.01	
Long-term health problems (%)	0.19	0.21	-0.03	
Age of the child (in years)	4.72	4.86	-0.14	***
Height for age z-score	-1.75	-1.41	-0.34	***
Stunted (%)	0.44	0.28	0.16	***
Mom's edu: primary or less (%)	0.62	0.4	0.22	***
Mom indigenous (%)	0.26	0.12	0.14	***
Wealth index	0.35	0.45	-0.1	***
HH expirienced shock (%)	0.34	0.4	-0.06	**
Urban (%)	0.54	0.7	-0.16	***
Number of observations	359	1,475		
Panel B - 2006				
Age of the child (in months)	27.28	26.98	0.3	
Eldest (%)	0	0	0	
Male (%)	0.45	0.48	-0.03	
Birth weight	3.06	3.14	-0.08	
Low birth weight (%)	0.06	0.07	-0.01	
Long-term health problems (%)	0	0	0	
Age of the child (in years)	4.58	3.66	0.91	***
Height for age z-score	-1.57	-1.33	-0.23	**
Stunted (%)	0.36	0.29	0.08	*
Mom's edu: primary or less (%)	0.62	0.52	0.1	**
Mom indigenous (%)	0.26	0.12	0.14	***
Wealth index	0.37	0.42	-0.05	***
HH expirienced shock (%)	0.6	0.52	0.08	*
Urban (%)	0.34	0.52	-0.18	***
Number of observations	359	163		

Source: Own estimations using Peruvian Young Lives survey, 1st, 2nd and 3rd round.
Note: Sample of paired-siblings with available information on maternal depression in 2002 and 2006 and HAZ scores in 2006 and 2009.

Table A.3.2: Maternal Depression in Stage 1 –
Comparison across YL rounds

	Round 2		
Round 1	No Depression	Depression	Total
No Depression	444	68	512
Depression	142	64	206
Total	586	132	718

Source: Peruvian Young Lives survey, 1st and 2nd rounds.
Note: Sample of paired-siblings with available information on maternal depression in 2002 and 2006 and HAZ scores in 2006 and 2009.

Table A.3.3: Impact of Maternal Depression on Child Nutritional Status - Heterogeneous effects by maternal characteristics

Dependent variable: Height-for-age Z-scores	[1]	[2]
Panel A - Maternal ethnicity	Non-indigenous	Indigenous
Depressed mother	-0.191	-0.326*
	(0.14)	(0.193)
Demographic controls	Yes	Yes
Year 2006	Yes	Yes
Mother fixed effects	Yes	Yes
Observations	530	188
R-squared	0.061	0.182
Panel B - Maternal education level	Primary or lower	Secondary or higher
Depressed mother	-0.481**	-0.0759
	(0.194)	(0.123)
Demographic controls	Yes	Yes
Year 2006	Yes	Yes
Mother fixed effects	Yes	Yes
Observations	276	442
R-squared	0.153	0.112

Source: Peruvian Young Lives survey 2002, 2006 and 2009.

Note: Robust standard errors in parenthesis. * significant at 10%, ** significant at 5%, and *** significant at 1%. Demographic controls are the same as in Table 3.

Table A.3.4: IV Estimations - First Stage Results

Dependent Variable: Maternal Depression	Sample of Paired Siblings	Sample of Paired Siblings - non-migrants
Depressed women in community (%)	**0.869*** **	**0.809*** **
	(0.156)	**(0.179)**
Teenage mother	-0.0680	-0.0312
	(0.0774)	(0.0890)
Wealth index	-0.167	-0.200
	(0.226)	(0.274)
Household experienced shock	-0.00853	-0.00949
	(0.0376)	(0.0396)
Live in urban area	-0.000808	0.0164
	(0.0802)	(0.0849)
Year 2006/7	0.134	0.147
	(0.0930)	(0.0980)
Child age in months S1	-0.00454	-0.00541
	(0.00439)	(0.00483)
Child age in months S2	0.0598	0.0734
	(0.0456)	(0.0503)
Oldest Sibling	-0.0430	-0.0265
	(0.0608)	(0.0658)
Long-term health problems in 2002	0.175**	0.122*
	(0.0687)	(0.0723)
Low birth weight	-0.0982	-0.139
	(0.0867)	(0.102)
Child is male	0.0260	0.0197
	(0.0385)	(0.0407)
Violent Crime in Community	-0.0605	-0.0375
	(0.0754)	(0.0769)
Social Assistance (Health) in Community	-0.114	-0.112
	(0.111)	(0.114)
Observations	716	660
R-squared	0.1716	0.141
F statistic	31.04	20.41

Source: Peruvian Young Lives survey 2002, 2006, and 2009. Note: Robust standard errors in parenthesis. * significant at 10%, ** significant at 5%, and *** significant at 1%.

4. Does Maternal Depression Undermine Childhood Cognitive Development?

4.1 Introduction

As the first and second chapters of this dissertation document, parental depression affects not only the parents who are ill, but also the children in their care. When children grow up in an environment of mental illness, the development of their brains may be seriously weakened, with implications for their ability to learn, as well as for their own later physical and mental health (Center on the Developing Child at Harvard University 2009). Of particular interest are episodes of depression or anxiety that affect mothers, who traditionally play a more prominent role in childrearing, especially while children are younger. Although there is an increasing awareness, at least in developed countries, of the potentially far-reaching harmful effects of maternal depression on child welfare, there remains a limited amount of rigorous evidence that quantifies its consequences on child development, the channels through which it acts, and how to mitigate its impact on children, particularly in developing countries.

Understanding how maternal depression affects children's human capital accumulation is an important first step in recommending sensible interventions to protect children's welfare. The present essay aims to contribute to such an understanding by studying the under-explored relationship between maternal depression and child cognition, a dimension of child development that has been extensively documented as a crucial determinant of life outcomes (Becker 1964; Currie and Thomas 1999; Feinstein 2003; Cunha et al 2005).

Maternal depression is characterized by sadness, negative affect, loss of interest in daily activities, fatigue, difficulty thinking clearly, and bouts of withdrawal and intrusiveness – all of which may interfere with the consistent, attentive, and responsive caregiving associated with effective parenting (Paulson, Dauber, & Leiferman 2006). Because mother-child interactions

during early life shape foundational neural circuits (Shonkoff and Phillips 2000; Knudsen et al. 2006; OECD 2007), neglect or maltreatment associated with maternal depression can undermine children's brain development and lead to worse cognitive outcomes (Stratakis 2006; Gunnar and Quevedo 2007; Center for the Developing Child 2010).

The previous essays of this dissertation present ample evidence suggesting that maternal CMDs disrupt mothers' caregiving and the environment in which children grow up, as well as mother-child attachment, socio-emotional and nutritional outcomes. Findings from previous research of the effect of maternal depression on cognitive development are mixed. In a study in England, Cogill et al (1986) found that children of mothers who were depressed in the first year had reliably lower cognitive skills as measured by a test score at age 4 than children whose mothers had not been ill. Petterson and Albers (2001) also report lower cognitive outcomes for children exposed to depression in the U.S. In contrast, Kurstjens and Wolke (2001) conclude that maternal depression *per se* has a negligible effect on German children's cognitive development over the first seven years of life, although they report a higher probability of long-term effects for boys and neonatal risk born, chronic cases of depression, or in cases in which the family is exposed to other social risks.

Using the National Longitudinal Survey of Youth in the U.S., Frank and Meara (2009) do not find evidence that maternal symptoms of depression affect contemporaneous cognitive scores in school-aged children, as measured by the Peabody Individual Achievement Test in Math and Reading Comprehension, either. Along the lines of Surkan et al (2012)'s argument for nutritional studies (see chapter 2), it is possible that this heterogeneity of results across studies originates from differences in measurements of both cognitive development and maternal depression, length of follow-up, sample characteristics, and sample sizes across studies, as well as varying

levels of social resources, caregiving norms, and both levels of cognition, as well as the prevalence of maternal depression in the populations under study.

Nevertheless, what limited evidence is available warrants concern about the economic and human costs of maternal depression, because the costs may extend to the next generation (Wachs et al 2005), affecting children's health, development, and behavior. Given that it often 'goes hand in hand with poverty' (Shonkoff and Phillips 2000), a major concern about maternal depression is that it may increase poverty and contribute to its intergenerational transmission by intensifying the negative effects of material deprivation and exposure to exogenous shocks associated with poverty, and confining children to substandard developmental trajectories—and hence worse outcomes later in life. Further, beyond its direct effects on children, maternal depression can be a major barrier to the effectiveness of interventions aimed at protecting and/or promoting early childhood development given emerging evidence that depression can significantly deter enrollment and full participation in intervention programs (Teti 1999).

This essay aims to address the evidence gap on the link between maternal depression and child cognitive development by taking advantage of the Young Lives survey, a rich set of household data from Peru. In so doing, it complements the analysis conducted in the second chapter of this dissertation, which investigates whether maternal mental health undermines children's growth during early childhood in Peru using the same data. The second chapter finds negative, sizable and statistically significant effects, even when excluding mothers with more severe cases of depression from the estimations.

The current essay also makes several contributions to the literature that studies how parents influence children's developmental outcomes. First, it uniquely identifies the impact of maternal depression on child cognition in a developing country, which, to my knowledge, has not been

done before. In particular, my study focuses on Peru, providing much needed empirical evidence of this important issue in a region where the causal nexus between maternal depression and child cognition has not been studied before despite maternal depression prevalence rates that range between 35 and 50% (Wolf et al 2002).

Second, the essay focuses on an important marker of early cognition, the accumulation of vocabulary, which has been extensively shown to predict reading comprehension throughout school and into early adulthood (Powell and Diamond 2012). To capture vocabulary competence, I use performance in the Peabody Picture Vocabulary Test (PPVT), a test of receptive vocabulary, which has been widely used and found to be predictive not only of vocabulary later in life (Cunha and Heckman 2007), but also of performance in school, including grade repetition and scores on tests of math and reading in primary school in Ecuador (Schady 2011), as well as labor market outcomes, such as wages in adulthood in the United States (Case and Paxson 2008).

Lastly, the analysis in the essay takes advantage of the longitudinal nature of the Young Lives survey. The inclusion of questions related to maternal mental health in a relatively large, longitudinal household survey that also collects a wealth of information on child, family and community characteristics is rare. I exploit the richness and longitudinal nature of the data to better capture the dynamic nature of children's development and strengthen the robustness of the analysis by considering variations in the indicator used for child cognition, exploring heterogeneous effects of maternal depression, as well as adding controls for household composition. The analysis performed in this essay sheds some light on the connection between the chronicity of depression and child cognition, and explores mechanisms through which maternal depression might affect children's cognitive development. Finally, the analysis on the impact of maternal depression on child cognition is complemented by considering the effect of

two alternative measures of maternal well-being, life satisfaction and optimism, on child performance on the PPVT.

Results indicate that, while the effect of more temporary or milder cases of maternal depression in the sample is negligible and statistically insignificant, the effect of cases of depression that span both rounds of data collection are sizable and statistically significant, and persist over time. Estimations using alternative measures of maternal well-being point to no discernible effects of life satisfaction or optimism on child cognition, which complements the results on maternal depression and lends weight to the argument that only severely low maternal well-being may be associated with worse child cognition. When the impact of maternal depression is analyzed separately by gender and maternal education level, there is evidence of worse effects for boys, as well as for children of mothers with incomplete primary school. Finally, the analysis of possible mechanisms in this essay both confirms and complements the findings in chapter 2, suggesting that, although depressed mothers appear to be more likely to disengage, when they do engage, their responsiveness may mediate the effect of depression on child cognition.

4.2 Conceptual Framework & Identification Strategy

What are the ways in which maternal mental health problems can undermine children's cognitive outcomes? Frank and Meara (2010) model the effect of maternal depression on the formation of children's skill by building on James Heckman and co-authors' inter-generational model of human capability formation (Cunha, Heckman et al 2005; Cunha and Heckman 2007):

$$S_t = F(PS, S_{t-1}, I_{t-1}; M_{t-1}) \qquad (1)$$

where S is the level of skill formation, PS represents parental skill attributes (education, cognitive abilities, etc.), I investments (both monetary and non-monetary) in child capabilities at

time t-1, and M is maternal mental health status at time t-1. Mental health problems that interfere with mother-child interactions or undermine maternal behavior during *t-1* could potentially undercut the effectiveness of parental skills and/or reduce the productivity of investments and result in deficient children's cognitive ability later in life.

The empirical identification strategy in this essay exploits information on cognitive outcomes for 1113 children and relies on a comparison of PPVT Z-scores from the second and third rounds of data collection (2006/7 and 2009/10, respectively) of similar children, some of who were exposed to maternal depression during the period prior to taking the exam (*t-1*), while others were not. The implicit assumption is that differences in average PPVT Z-scores at *t* would be similar between children of depressed and non-depressed mothers in the absence of maternal mental health problems at *t-1*.

Using child fixed effects to estimate the effect of exposure to lagged maternal depression on cognitive outcomes, the econometric specification is:

$$PPVT_{it} = \beta D_{i,t-1} + X'_{ti}\,\Theta + HS'_{i,t}\,\gamma + Year\ 2009_t + \nu_i + \varepsilon_{it} \quad (2)$$

where $PPVT_{it}$ represents the child's PPVT Z-scores for child *i* in period *t* (i.e., 2006/7 and 2009/10). $D_{i,t-1}$ captures whether the mother suffered from depression in *t-1* (i.e., 2002 and/or 2006/7). The parameter of interest, β, measures performance in the PPVT at *t* for children whose mothers were depressed in *t-1*. Because the identification strategy relies on changes in maternal depression between the first and second rounds of the survey, in this model β captures the effect of cases of depression that do not span both rounds of data collection, which are likely to represent more temporary or milder cases of depression.

Year 2009 is a binary variable that controls for the average changes in PPVT Z-scores between 2006/7 and 2009/10. X_{ti} is a vector of child, household, and community observable,

time-varying characteristics that can lead to differences in cognitive ability across children and influence their parents investments in them (wealth, exposure to shock, and urban/rural location). In addition, demographic controls include maternal age, an age quadratic term for the child and community controls, including access to social assistance programs and violent crimes in the community, both of which have been documented to affect children cognition (for a review, see Shonkoff and Phillips 2000).

I also include controls for children's health status (HS), including birth weight, height-for-age Z-scores and long-term health conditions and disability in the specification to address the possibility that these factors influence performance on the PPVT. ν_i represents unobserved child characteristics that are fixed over time that affect children's cognitive ability. ε_{t+1ij} is a random, idiosyncratic error term.

4.3 Data

The source of data for this chapter is the Young Lives Peru Survey (YL), the same as that for chapter 2, and is described in section 3.4 of that chapter. There are currently three waves of available data: the baseline round, conducted in 2002 when the index children were aged 6-20 months, the first follow-up conducted in 2006/7, when they were between 4 and 6 years old, and the last round in 2009/10, when they were between 7 and 8 years of age. This essay uses all three waves of data. Of the 2,000 index children in the baseline round, the analysis here focuses on the sample of 1113 present in all three waves for whom data on maternal mental health and PPVT scores are available.

4.3.1 Measurement Variables

I use Peabody Picture Vocabulary Test (PPVT) scores (Dunn et al.1986) as the measure of cognitive achievement, the outcome of interest for this study. Performance on the PPVT at early

ages has been shown to be predictive of schooling and labor market outcomes later in life (e.g., Cunha and Heckman 2007; Case and Paxson 2008; Schady 2011; Powell and Diamond 2012). The PPVT measures receptive vocabulary; children are shown slides, each of which has four pictures, and are asked to identify the picture that corresponds to objects or actions named by the test administrator (children do not need to name the objects or actions, or be able to read or write them). The test continues until the child has made six mistakes in the last eight slides. The number and the level of difficulty of questions differ according to children's age (see Cueto et al. 2009). I therefore construct age specific z-scores by subtracting the month-of-age-specific mean of the raw score and dividing by the month-of-age-specific standard deviation. PPVT scores are available in the second and third round of the YL survey, i.e., when children were 4-6 and 7-8 years.

The explanatory variable is constructed using information on maternal common mental disorders from the Self Reporting Questionnaire 20 items (SRQ20), a screening (case-finding) tool included in the YL survey. This tool is recommended by the World Health Organization and has acceptable levels of reliability and validity in developing countries. The SRQ20 consists of 20 yes/no questions with a reference period of the previous 30 days. It is not diagnostic and cannot separate out anxiety from depression, but to the extent that these conditions are closely related and both of them can undermine the quality of care mothers provide to their children, the information gathered from the questionnaire is very valuable (henceforth, depression is used to referred to both cases of depression and/or anxiety).

Using the responses to the questionnaire, I use available standards validated against clinical assessments in Peru to determine whether the mother experienced anxiety/depression using a cut

off score of 7/8 yes answers to the SRQ20 to separate probable cases from non-cases. I use information on maternal mental disorders from the first and second round of the YL survey.

4.3.2 Preliminary Descriptive Statistics

Table 4.1 reports summary statistics of the variables used in the analysis for the sample under analysis. Cognitive outcomes, as measured by PPVT Z-scores are practically unchanged between rounds, even if, as expected, the mean score increases as the children age, reflecting a larger vocabulary. The average child in the sample scored 0.07 standard deviations above the mean PPVT score of a reference child in both 2006/7 and 2009/10. Children's height-for-Age Z-scores on the other hand show an improving trend. Approximately 16 percent of mothers in the sample are of indigenous origin, 79 percent are literate, and 42 percent have not completed primary school. On average, households in the sample are wealthier and 30 percent more likely to reside in urban areas in 2009/10 than in 2006/7.

Table A.4.1 compares the sample under analysis with the observations excluded from the study. A number of maternal characteristics differ in these two sub-samples, and the difference remains statistically significant across rounds of data: mothers in the sample are less likely to have completed primary school and are less likely to be literate than mothers in the rest of the sample. A few children characteristics also differ: children in the sample under study have worse nutritional status and test scores than the rest of the sample. Finally, the 1113 households in this study are poorer and less likely to reside in urban areas than those in the rest of the sample.

To allay concerns of sample selection, I estimated the effect of depression on cognition using cross-section OLS regressions for each round of data, as well as by pooling the data (not reported) and in all cases reject the null hypothesis that the results are significantly different between the group under analysis in this essay and the total sample. These results suggest that

sample selection should not be of particular concern and results could be potentially extrapolated to the larger sample.

Table 4.2 compares depressed mothers and their children with non-depressed mothers and their children in the sample under study.[20] As it is evident from the table, these groups are quite different. On average, depressed mothers are older, less educated, and more likely to be of indigenous origin (all statistically significant). Their children are younger and more likely to have considerably worse nutritional and cognitive outcomes: at age 4-6, their average height-for-age z-score is 27 percent of a standard deviation lower and 14 percent more likely to be stunted than children of mothers who are not depressed, while average PPVT Z-scores are 34 percent of a standard deviation lower at age 4-6 and 40 percent of a standard deviation lower at age 7-8 (all statistically significant). In addition, depressed mothers and their children live in households that are less wealthy, less likely to be urban and more likely to have experienced a shock (all statistically significant).

Finally, Table A.4.2 depicts maternal mental health status during children's first stage of life. Of the 1113 observations, 325 are depressed in 2002 and 143 in 2006/7, while 81 mothers are depressed in both rounds.

4.4 Results

4.4.1 Main Results

Table 4.3 reports the main results of the essay. Column 1 presents results for pooled OLS estimations, while column 2 shows results for the baseline fixed effects model as described in

[20] Table 4.2 depicts descriptive statistics by maternal depression in 2006. I also estimated the same descriptive statistics by maternal depression in 2002 (available upon request), which are very similar to the ones in the table described above.

equation (2).[21] As expected, maternal depression has a negative, although small impact on child cognition. Z-scores of children whose mothers were depressed are 8 percent of a standard deviation lower than Z-scores of children whose mothers were not depressed, a result that is statistically significant at the 10 percent level. The FE coefficient is half of that in column 1 and is statistically insignificant. The smaller and insignificant FE coefficient may be the result of the fact that FE estimations excludes cases of depression that span more than one round of data collection (see section 4.3), while Pooled OLS captures the effect of the average effect of depression in the sample. It is also possible that time-invariant unobserved child and/or household characteristics that mediate the effect of maternal depression on child cognition, such as child resilience, lead Pooled OLS results to overestimate the effect of maternal depression. I further analyze the differences between Pooled OLS and FE estimations in section 4.4.3 below.

The effects on child cognition of demographic controls in the table are as expected, both for the Pooled OLS and FE estimations. Z-scores of children whose mothers are literate are 21 percent of a standard deviation higher (statistically significant at the 1 percent level) than those of children whose mothers are not literate. When comparing across households (Pooled OLS), children's nutritional status also affects performance in the PPVT, although having long-term health problems does not necessarily do so. The coefficients for wealth are positive, statistically significant, and among the highest in Table 4.3, which, together with the effect of maternal literacy, is in line with research that points to socio-economic status gradients of cognition as measured by vocabulary (Schady 2011; Paxson and Schady 2007; Fernald et al. 2011; Naudeau et al. 2011; Schady et al 2015).

[21] All OLS specifications in this section (Pooled, lagged, and cross-section) were estimated using the full sample of observations, as well as the restricted sample used in the FE model. There are no significant differences in the results obtained with the different samples and, for comparability, only those with the restricted sample are reported.

I explore heterogeneous effects by a number of household, child, and maternal characteristics that have been identified in the literature as moderators of the effect of depression, using both Pooled OLS and FE models. First, I run separate regressions including interactions of maternal depression with different levels of household wealth, which, it is generally agreed, influences the extent to which maternal depression affects children's outcomes (Lovejoy 2000; Shonkoff and Phillips 2000). For the sample under study, however, there appear to be no differences in effect across levels of wealth, whether it is comparing the bottom quintile, quartile, tercile, or half of the distribution to the rest of the sample, as none of the interaction terms is significant for either the Pooled OLS or FE estimations (not reported).

I also analyze whether gender plays a role in determining the effect of maternal depression on child cognition by conducting separate estimations for boys and girls, given previous research that suggests that boys' cognitive functioning may be more sensitive than girls' to the effect of mothers' mental illness (Sharp et al 1995; Hay et al 2001; Kurstjens and Wolke 2001). Results for the FE model in Table A.4.3 are in line with previous evidence: while, on average, maternal depression does not appear to affect the accumulation of vocabulary during childhood for girls (column 3), PPVT Z-scores of boys whose mothers were depressed are .137 standard deviations lower than boys whose mothers did not experience a depressive episode (column 4), a results that is statistically significant at the 1 percent level. The difference between point estimates for boys and girls is statistically significant (not shown). For the Pooled OLS model, however, results in columns 1 and 2 suggest no heterogeneous effects by gender.

Finally, I explore whether the effects of exposure to depression during a child's young age varies depending on maternal characteristics by calculating the specifications separately for households in which the mother has a partner, as well as by maternal schooling levels (I split the

sample by incomplete primary or less, and complete secondary or more), given that these factors may modulate the impact of depression (Shonkoff and Phillips 2000; Wachs 2005; Patel 2006).[22] There are no apparent differences in the effect of maternal depression for mothers who have a partner vis-à-vis those who do not (Panel A in Table A.4). However, there are sizable differences on child performance on the PPVT by mother schooling levels (Panel B in Table A.4). While the impact of depression appears to be negligible in size and statistically insignificant for children of mothers who have at least completed primary school (columns 2 and 4), Z-scores of children with less educated mothers who were depressed at *t-1* are, on average, .123 standard deviations lower than Z-scores of children of less educated mothers who were not depressed when estimated with the Pooled OLS model (column 1), and .183 in the FE model (column 3). These coefficients are statistically significant at the one and five percent level, respectively, and the differences between coefficients by mother schooling levels are also significant (not shown).[23]

The heterogeneous effects by maternal education level suggest that lower education and associated conditions that undermine child cognitive development (e.g., sub-optimal childcare practices/skills or restricted access to quality material inputs and opportunities) may exacerbate the negative effect of maternal depression on the accumulation of early vocabulary for the children of less educated mothers.

4.4.2 Further Exploring Fixed Effects Results

The negligible magnitude and statistical insignificance of the FE coefficient for maternal depression in Table 4.3 is somewhat surprising considering the importance of early mother-child

[22] The literature suggests that the presence of other members in the household that provide support to the mother can buffer the effect of depression on children. In addition to the effect of whether the mother has a partner, I also investigated whether the effect of maternal depression on child cognition varied depending on the number of adults or female adults in the household. The results of these estimations (available upon request) suggest that there are no heterogeneous effects by household composition.

[23] Throughout section 4.4, the equality of coefficients is tested with F-test using restricted and unrestricted specifications.

interactions for cognitive and language competence. Are the insignificant FE results a consequence of omitted child or household fixed attributes, such as child personality traits like resilience, or household characteristics, like violence, that may be leading pooled OLS to overestimate the impact of maternal mental health on child cognition? Or are there other factors driving the baseline FE results?

I explore whether sample restriction, selective non-response, or the standardization of the test scores may be influencing results. First, I conduct an alternative estimation that excludes children with mothers who report they speak a language other than Spanish to rule out the possibility of selective non-response on the PPVT.[24] In Peru, children were given the option of taking the test in Spanish or Quechua, an indigenous language spoken primarily in rural areas of the highlands (Cueto and Leon 2009). While twenty-two percent of children in rural areas chose to take the test in Quechua, only 0.1 percent of children in urban areas did. Because children in households that speak Quechua or another indigenous language may have more limited vocabularies in any given language (Schady et al 2015), I re-estimate the FE model for children of Spanish speaking mothers only. Results of estimations excluding children in households where a language other than Spanish is spoken (Table A.4.5) are not substantially different than those in Table 4.3.

I next explore whether missing test scores may be influencing the results. A fraction of children in both rounds of data collection available did not take the PPVT (7 percent in round 2 and 10 percent in round 3). Although the reasons why these children did not take the test are not available in the survey, earlier work in Ecuador by Paxson and Schady (2010) and Schady (2011) has shown that children who miss a given test do worse on other tests, or on the same test in

[24] In the sample, 56 percent of the rural population and 17 of the urban population speak a language other than Spanish.

different survey waves, than other children with comparable wealth and parental schooling levels who took the test. Following Schady et al (2015), who argue that children who miss tests are likely to be "low performers", I assign these children a test score of zero. Results when missing test scores are replaced with zero values (not shown) are virtually identical to those from the FE baseline specification, both in terms of magnitude and statistical significance (Table A.4.5).

Lastly, I investigate whether results in Table 4.3 hold restandardizing the PPVT Z-score for rural/urban area of residency. I re-estimate the baseline specification in equation (2) using alternative age-specific Z-scores separately by area of residence that standardize for possible differences across urban and rural samples, following Cunha and Heckman (2007), and run separate regressions by area of residency. Results, however, are not significantly different using this standardization and confirm the patterns summarized above (Table A.4.5).[25]

4.4.3 Dissecting Pooled OLS Results

I return to the coefficient for maternal depression in column 1 of Table 4.3, which, although small, is statistically significant, in contrast with the robustly insignificant FE results above. I explore here whether there are any particular factors that may be driving the difference between Pooled OLS and FE estimations, beyond the classic interpretation that pooled OLS captures variation between units of analysis, while FE estimations captures within effects. I disaggregate the effects in column 1 of Table 4.3, which synthetizes the impact of maternal depression in 2002 and/or 2006/7 on performance on tests taken in 2006/7 and 2009/10, run separate regressions by year of maternal depression and year in which the child took the exam.

Table 4.4 shows a stark contrast between results in row (a), which captures the effect of depression episodes that occurred 4 to 8 years before the taking of the exam, and those in row (b),

[25] I also conducted the estimations in this section using Pooled OLS and corroborated that results in Table 3 are robust to the exclusion of non-Spanish speakers from the sample, replacing missing test scores with zero, and the standardization of the PPVT by area of residency. These estimations are available upon request.

which reflects the effects of depression episodes that occurred at most 4 years prior to the exam.[26] Whereas results in row (a) are very small in magnitude and statistically insignificant, those in row (b), are non-trivial in size and statistically significant, with contemporaneous effects (column 1) being the strongest (-0.167 and statistically significant at 5 percent level), which is consistent with the literature on the topic (Lovejoy et al 2000). Results in Table 4.4 suggest that the effect of depression on cognitive outcomes diminishes as the amount of time elapsed between exposure to the shock and the taking of the exam increases. It is possible that the reason behind the apparent non-residual effects of maternal depression on child cognition after two years of exposure to the shock is that the intensity of the average depression case in the sample is not debilitating enough for its effect on child cognition to persist over time.

I re-estimate the OLS regressions by year of maternal depression and year in which the child took the exam for the sub-sample of children whose mothers were depressed both in 2002 and 2006/7 and those who were not depressed in either round of data collection. These estimations are likely to capture the effect of less temporary cases of depression, given that mothers who are classified as depressed in both the first and second round of data are likely to be more prone to mental health issues than the average mother in the sample, if not subject to recurrent or chronic depression. Coefficients for maternal depression in Table 4.5 are sizable and statistically significant regardless of the time elapsed between exposure to the shock and the taking of the exam, lending weight to the hypothesis that chronicity of maternal depression may be driving the effect of depression on cognitive development. Finally, I estimated the OLS regressions by year of maternal depression and year in which the child took the exam excluding

[26] Because 70 percent of households in the sample were interviewed in 2007 during the second round of data collection and all but four households were interviewed in 2009 during the third round of data collection, the gap of time between depressive episodes in 2002 and the second round is at least 5 years, while that between depressive episodes in 2006/7 and the third round is at most 2 years.

mothers depressed both in 2002 and 2006/7. The coefficients for depression in these estimations (not shown) are very similar to those in Table 4.4 and fade with time, further supporting the argument that only cases of maternal depression that persist in time in the YL survey may negatively influence children's cognitive development.

Results in this section suggest a compelling angle from which to interpret the differences between the Pooled OLS and FE estimations in Table 4.3. If in fact only the most severe and debilitating cases of maternal depression have an effect on performance on the PPVT, and these severe cases are not captured in the YL data by mothers who were only depressed in one round of data collection, then the FE estimations may be providing a lower bound of the real effect of depression on child cognition given that they do not capture the effect of depression that spans both rounds of data collection because the empirical identification strategy relies on changes in depression between rounds. Despite the fact that the Pooled OLS results do not fully address the potential endogeneity of the child cognitive development-maternal mental health relationship (although the rich set of controls included in the estimations reduces the possibility of omitted variable bias), they may be capturing a more accurate representation of the relationship between maternal mental health and the development of child cognition.

4.4.4 Discussion of Possible Mechanisms

I exploit available information in the first round of the Young Lives survey on maternal childcare and mother-child interactions when children were between 6 and 24 months of age (i.e., year 2002, the first round of data collection) to explore whether there is evidence of ways in which maternal depression leads to worse outcomes for the children in the sample, along the same lines as in the second chapter of the dissertation. First, I tabulate available indicators of general maternal behavior and maternal responses to children's persistent crying by maternal mental

health status (Table 4.6).[27] Results are identical to those in Table 3.6 in chapter 2 (see section 3.5.3 in that chapter for discussion).

To complement the analysis, I calculate separate regressions by year of maternal depression and year in which the child took the exam including controls for the maternal caregiving practices and behaviors in Table 4.6, a childcare index that adds every positive behavior in Table 4.6 (e.g., vaccinate child) and subtracts negative behaviors (e.g., shakes child), as well as interactions of all such variables with depression. I conduct these estimations for both the full sample under study, as well as by excluding the sub-sample of mothers who were depressed only in one round of data collection. The coefficients for most interactions terms are insignificant (not shown). The exceptions include a positive and significant effect for giving water or medicine to the child (for both the full and restricted sample) and breastfeeding (only for the full sample), behaviors that could be capturing maternal pro-activity or responsiveness when the child is very upset. These positive effects persist until children are 7-8 years old. In contrast, spanking children when they are between 6 and 24 months of age while they persistently cry has a negative and sizable effect on performance on the PPVT at age 4-6 for children whose mothers were depressed, although the effect becomes insignificant by the time children are 7-8.

Results in this section suggest that the effect of maternal depression on child cognition is nuanced. On the one hand, Table 4.6 suggests that depressed mothers are more likely to disengage or to simply go through the motions, which could result not only in more serious injuries, but also reduced stimulation and suboptimal feeding practices, both essential for sturdy cognitive development during early life. On the other hand, the differential effects of maternal depression for children of depressed mothers who attend their children when they cry

[27] I also tabulated the indicators for maternal behavior for the sub-sample of mothers who were depressed both in 2002 and 2006/7. Results (available upon request) are very similar to those in Table 4.6.

persistently as opposed to those who do not suggests that these behaviors may moderate the effect of maternal mental illness on child cognition.

4.4.5 Alternative Measures of Maternal Well-Being

Taking advantage of information on maternal life satisfaction and optimism available in the YL survey, I explore how these two alternative measures of maternal well-being fair vis-à-vis maternal depression in predicting performance on the PPVT for the children in the study. Just like depression, life satisfaction and optimism inform maternal preferences, which in turn influence maternal childcare practices and related behavior, and are thus likely to have an effect on children's development as well. Also, just as with depression, life satisfaction and optimism can influence investment decisions that in turn determine children's aptitudes and tastes. They may also affect children's developmental trajectories more directly by shaping children's own satisfaction and optimism levels, given how influential parent-child interactions are during early life.

The empirical literature on the association between maternal life satisfaction and child development is very limited. Maternal sensitivity and responsiveness are influenced by maternal psychological well-being (Belsky 1997). Consequently, maternal life satisfaction is likely to affect both of these qualities, which are crucial for the development of mother-child attachment and its quality, a paramount determinant for early childhood development (Glaser 2000, Shonkoff and Phillips 2000, Farah et al 2006; Walker et al 2007). Maternal life satisfaction has been found to have an effect on early childhood cognition and socio-emotional outcomes, with higher levels of maternal satisfaction associated with enhanced child verbal skills and reduced socio-emotional problems (Berger and Spiess 2009).

The measure of life satisfaction in the YL survey is the Cantril ladder question, which asks respondents to compare their lives to the best possible life they can imagine on a ladder where 1 represents the worst and 9 represents the best possible life. Whereas the depression indicator in the YL survey is an objective measure that captures a pathological form of low well-being, life satisfaction is a subjective measure which encapsulates a wider range of perception related to overall well-being. As expected, the correlation between depression and life satisfaction for mothers in the survey is negative and statistically significant, but low (-.078).

To capture maternal optimism, I use maternal reactions to the following statement: "If I try hard, I can improve my situation in life." The correlation between depression and optimism for mothers in the survey is negative and statistically significant, but very low (-.031). Maternal depression can be considered at the opposite end of the well-being spectrum from high maternal satisfaction and optimism. And although people with more positive attitudes about their futures tend to report higher levels of life satisfaction, these two measures encapsulate different dimensions of well-being: life satisfaction captures happiness or cheerfulness; optimism is associated with a positive attitude and intrinsic motivation.

Graham (2015) cites a host of studies that suggest people with perception of more limited future opportunities have higher discount rates, both because they have less capacity to set aside their limited means to make those investments, and because they have less confidence that those investments will pay off. Although higher discount rates associated with lower maternal optimism could be a catalyst for lower development trajectories for their offspring, to my knowledge there are no empirical studies to date that investigate whether and how maternal optimism affects their children's development.

I analyze the effects of maternal optimism and life satisfaction performance on the PPVT for the children in the study and how they compare to the effect of maternal depression. Unlike the indicator for maternal depression, which captures data from 2002 and 2006/7, information on maternal life satisfaction and optimism in the survey were collected in 2006/7 and 2009/10. This data limitation restricts the comparison across all three measures of maternal well-being to OLS estimates of the effect of the alternative indicators of maternal well-being in 2006/7 on performance on the PPVT both in 2006/7 and 2009/10, which I present in Table 4.7.

Panel A of the table captures the effect of current maternal well-being on PPVT performance, while Panel B presents the effect of lagged maternal well-being on PPVT performance. As expected, the coefficient for maternal depression (column 1) in both panels is negative (and statistically significant), while those for life satisfaction (column 2) and optimism (column 3) are positive, although very small. The coefficient for current life satisfaction is statistically significant at the 1 percent level, while those for lagged life satisfaction and both current and lagged optimism are not statistically significant.

In addition, I conduct separate FE regressions, similar to the baseline fixed-effects model in equation (2), but replacing lagged maternal depression with the contemporaneous measures of maternal optimism and life satisfaction in the survey. Table A.4.6 presents the results of the estimations using with maternal life satisfaction as the main explanatory variable in column 1 and optimism in column 2. The coefficient for the alternative measures of maternal well-being are in line with those in Table 4.7: negligible in size and only statistically significant for life satisfaction.

The negligible effects of maternal life satisfaction and optimism on child cognition are complimentary with the analysis in the previous sections, which concluded that for maternal

depression to undermine the accumulation of vocabulary at young ages, it had to be sustained over time. The results in Table 4.7 suggest that a dynamic may be taking place at the opposite end of the spectrum in which indicators of well-being that capture a positive outlook have no measurable effect on child cognitive development. Thus it appears that only a highly negative maternal mind-set has a demonstrable effect on children's outcomes.

4.5 Conclusion

In this essay, I explore the extent to which maternal mental health illness affects child cognition. The identification strategy I use exploits variation in the timing of maternal depression within households in Peru, controlling for child heterogeneity given the longitudinal nature of the data. The essay's main results indicate that, while the effect of the average case of maternal depression in the sample is negligible and statistically insignificant, the effect of more severe or chronic cases of depression sizable and statistically significant, and persists over time. Estimations using alternative measures of maternal well-being point to no discernible effects of life satisfaction or optimism on child cognition, a finding that complements the results on maternal depression and lends weight to the argument that only severely low maternal well-being affects child cognition.

In addition to the main results discussed above, this essay also estimates heterogeneous effects by gender and maternal education level. When the impact of maternal depression is analyzed separately by gender, there is evidence of worse effects for boys, which may be associated with evidence that boys are more vulnerable in early life than girls (Eriksson et al. 2010). Because cognitive development is cumulative, the effect of maternal mental illness on boys is an issue of concern, particularly if these boys grow up to be the main wage earners of a household. Their worse cognitive outcomes early on in childhood could potentially undermine these boys' future education and labor market outcomes in adulthood. In addition, my results

suggest that the cognitive development of children of less educated mothers, who are likely to live in poorer households and be vulnerable in other ways, also suffers disproportionately from exposure to maternal depression.

Results in this essay underscore the incredible resilience of children, while at the same time provide evidence that severe stress and associated maternal common mental disorders can undermine children's development. The heterogeneous findings by gender and maternal education, along with those of longer episodes of depression, combined with extensive evidence in the literature of the disproportionately high prevalence rates of anxiety and depression among households with low socio-economic status cited throughout the dissertation, suggests that stress in general, and associated maternal mental illness in particular, constitutes yet another pathway from poverty to substandard developmental trajectories and potentially worse outcomes later in life, and that maternal mental illness may contribute to the intergenerational transmission of poverty.

What are the implications of these findings for policymakers? To the extent that the maternal depression-child cognitive development relationship is causal, findings suggest that a two-pronged approach may be necessary for protecting children's cognitive development from maternal depression. First, given its disease burden and the associated deleterious effects, a strong case can be made for recognizing maternal mental health problems as disorders of public health significance and integrating them as such into maternal and infant health policies (Patel 2002). For this to occur, the public health commitment to mental health problems should increase, particularly in developing countries, where the current commitment is minimal (Patel et al 2006).

As I document in the first and second chapters of the dissertation, cost-effective interventions that treat mental health issues affecting women in poorer households have been

successfully implemented in developed and developing countries. Most relevant to this essay, evaluations of interventions that, in addition to addressing maternal depression also include children themselves, reported improved mother-infant interaction and better cognitive development (Rahman et al 2013). Taking into account cultural differences and local sensitivities, similar initiatives could prove effective and efficient in improving maternal mental health in developing countries such as Peru, in so doing improving the livelihoods of children whose early development is hindered by maternal depression.

In addition, the heterogeneous results in this essay suggest that the child cognition nexus is complex, determined not only by maternal illness, but also child, maternal and household characteristics that interact in ways that are not yet fully understood. Consequently, the most effective way to protect children's welfare may be to target children themselves and build support systems at the household, community, or institutional level that protect vulnerable children's outcomes. Programs and policies that promote poor children's cognitive development directly, such as by improving access to quality pre-school programs, or indirectly, by promoting cognitive stimulation at home and improving the quality of home environments, may help prevent and/or compensate for early deficits related to maternal depression. And given the hierarchical and interdependent nature of development, the earlier in life the intervention, the better. The magnitudes of the differentials in this essay are in line with the effects of a number of interventions in Latin America aimed at improving the cognitive development of poor young children, going from 0.10 standard deviations for cash transfers to very poor households in Nicaragua (Macours et al. 2012), to 0.23 standard deviations for a program that increases preschool availability in Argentina (Berlinski et al 2009); and 0.25 standard deviations for a program of home visits in Colombia (Attanasio et al. 2012).

High-quality childcare can also protect children from family-based risk, including exposure to maternal depression. In the United States, this has been a primary rationale for early intervention programs that provide high-quality center-based child care for children living in poverty and for children in the child welfare system, with evidence pointing to warmer and more responsive behavior for poor mothers of six-month-old babies, compared to similar poor mothers who were rearing their babies at home or were using full-time, lower quality care (NICHD Early Child Care Research Network 1997 in Shonkoff and Phillips 2000). Although it is not clear whether having access to high-quality childcare would positively influence maternal behavior for women who suffer from severe and/or chronic depression, it could alleviate some of the stressors in these women's lives. Regardless, childcare could partially shelter children from the toxic stress, neglect and/or maltreatment associated with maternal depression (Cohn et al 1986, 1991).

Finally, it is worth briefly discussing how the main results of this chapter relate to those in the second chapter of the dissertation. While the analysis in each essay was not guided by the objective to compare the effect of depression on each studied outcome, but rather to provide as in-depth of an analysis as possible in each case, the fact that, to my knowledge, this dissertation is the first study that analyzes both nutritional and cognitive outcomes using the same data, creates an opportunity to examine the findings jointly. To begin with, the comparison of these chapters' main results, that is, that controlling for time-fixed heterogeneity, average maternal depression does not have a significant effect on average child cognition, yet does affect average nutritional status, is thought provoking. Differences in methodological approaches and the sub-samples analyzed in the essays undoubtedly account for some of the differences in the essays' findings. Still, taken together, the results in these essays could suggest that nutritional status, as measured by height-for-age, is more susceptible to maternal stress, anxiety and depression during

early life than cognitive development (as measured by receptive vocabulary). Is it possible that these results suggest that, while on average, child's nutrition status is likely to be undermined by conditions that interfere with maternal behavior, cognitive development may be less vulnerable to such conditions, given that it can still benefit from other factors in the child's environment (e.g., family, school, community members)?

The comparison of results in these two essays highlights another relevant dimension in the maternal depression-ECD outcome relationship: the role of unobservables and chronic depression in determining the effect of maternal depression on ECD. As the essays point out, both unobservables and chronic depression play a role in explaining the differences in results between Pooled OLS and FE estimations. However, it would appear as though unobservables and the severity and/or chronicity of maternal mental health affect the studied nutritional and cognitive outcomes differently. The fact that the FE estimate of the effect of maternal depression on child nutrition in chapter 2 is larger than the Pooled OLS, even if FE estimations exclude the effect of longer and/or more severe episodes of maternal depression, would suggest that fixed, unobservable effects are specifically linked to nutritional determinants in such a way that they reduce the effect of depression (for instance, mothers' personality traits, such as sense of dutifulness, could be overriding the negative effects of chronic depression).

In this essay, the larger and significant Pooled OLS coefficient for maternal depression could suggest not only that the biases of the determination of cognitive ability from constant unobservables go in the opposite direction, but also that the severity or chronicity of maternal mental health plays a larger role on child cognition than do unobservables. In fact, the results of Pooled OLS estimations for mothers who were depressed in both rounds of data collection point

to a large and significant negative effect of depression on early child cognition, while estimates for mothers depressed in only one rounds suggest no effect.

Importantly, however, in this essay, FE estimations control for time-fixed characteristics at the child level, while FE in chapter 2 are at the mothers' level, which could suggest that child attributes that do not vary with time play a larger role in determining the effect of maternal depression than do maternal characteristics. Alternatively, it could be the case that by controlling for child fixed effects, characteristics that could help explain variation in child performance in the PPVT are not being considered in the analysis.

To be sure, there are myriad other factors that should be considered when trying to understand the differing results between these two essays, including the role of unobservable characteristics that are not fixed over time (see FEIV results in chapter 2), and the way all the factors just described interact. As much as my study of the effect of maternal depression on child nutrition and cognition taking advantage of the wealth of information in the YL survey constitutes a contribution to the field, it also raises as many questions as it addresses. The literature provides no point of reference to compare the effects of depression on nutrition and cognition among the same population. Consequently, the findings in this dissertation ultimately point to the need for research that provides the necessary context to achieve a more integrated understanding of how maternal depression affects the whole child, as well as the way that factors such as maternal characteristics and behavior, child attributes, and poverty interact with stress to influence child outcomes.

Table 4.1: Descriptive Statistics by Year

Variables	Year 2006			Year 2009		
	Mean	S.D	N	Mean	S.D	N
Maternal characteristics						
Age of the mother (in years)	31.46	0.2	1113	33.73	0.20	1113
Mother has partner (%)[a]	87.96	0.098	1113	--	--	--
Indigenous ethnic group (%)[a]	15.81	0.109	1113	--	--	--
Less than primary school (%)[a]	41.69	0.148	1113	--	--	--
Literate (%)[a]	79.16	0.122	1113	--	--	--
Household characteristics						
Wealth index	0.50	0.007	1113	0.56	0.01	1113
Live in Urban area (%)	58.40	0.148	1113	75.74	0.13	1113
Shock (%)	53.46	0.15	1113	53.28	0.15	1113
Child characteristics						
Age (in months)	63.51	0.141	1113	94.90	0.11	1113
Eldest (%)	16.44	0.111	1113	23.45	0.13	
Male (%)	49.87	0.15	1113	49.87	0.15	1113
Birth weight	3.21	0.015	1113	--	--	--
Height for age Z-score	-1.42	0.032	1113	-1.05	0.03	1113
Long-term health problems (%)	18.87	0.117	1113	8.90	0.085	1113
PPVT score	30.07	0.524	1113	47.63	0.39	1113
PPVT Z-score	0.07	0.029	1113	0.07	0.03	1113
Community characteristics						
Violent crime in community (%)	32.17	0.14	1113	35.58	0.14	1113
Social assistance (education) in community	95.33	0.063	1113	98.02	0.04	1113

Source: based on Peruvian Young Lives survey, first, second, and third round.
Note: Sample of children with available information on maternal depression in 2006 and PPVT scores in 2006 and 2009.
*Information available in the first round of data collection in 2002.

Table 4.2: Descriptive Statistics by Maternal Depression in 2006

Variables	Mother depressed	Mother not depressed	Difference	
Panel A - 2006/7				
Age of the child (in months)	62.23	63.70	-1.46	***
Child is eldest (%)	11.89	17.11	-5.23	*
Child is male (%)	44.76	50.62	-5.86	
Birth weight	3.18	3.21	-0.03	
Height for age z-score	-1.65	-1.38	-0.27	***
Long-term health problems (%)	13.29	8.25	5.04	*
Stunted (%)	37.76	26.80	10.96	**
PPVT score	23.08	31.10	-8.02	***
PPVT Z-score	-0.23	0.11	-0.34	***
Age of the mother (in years)	34.08	31.07	3.01	***
Mother has a partner (%)	89.51	87.73	1.78	
Mother indigenous (%)	24.48	14.54	9.94	***
Mother's edu: primary or less (%)	56.64	39.49	17.16	***
Mother is literate (%)	67.83	80.83	-12.99	***
Wealth index	0.42	0.51	-0.09	***
Urban (%)	38.46	61.34	-22.88	***
HH experienced shock (%)	71.33	50.83	20.50	***
Panel B - 2009/10				
Age of the child (in months)	94.71	94.93	-0.21	
Eldest (%)	15.39	24.64	-9.26	***
Height for age z-score	-1.36	-1.00	-0.36	***
PPVT score	42.78	48.35	-5.56	***
PPVT Z-score	-0.28	0.12	-0.40	***
Age of the mother (in years)	36.52	33.32	3.21	***
Wealth index	0.49	0.58	-0.08	***
Urban (%)	60.14	78.04	-17.90	***
HH experienced shock (%)	60.84	52.17	8.67	**
Number of observations	143	970		

Source: Own estimations using Peruvian Young Lives survey, 1st, 2nd and 3rd round.
Note: Differences in characteristics between both groups are statistically significant *** at 1%, ** at 5%, and * at 10%. Sample of observations with available information on maternal depression in 2002 and 2006 and PPVT scores in 2006 and 2009.

Table 4.3: Impact of Maternal Depression on Child PPVT scores

Dependent variable: PPVT Z-score	Pooled [1]	Fixed [2]
Maternal Depression	-0.0755*	-0.0395
	(0.0441)	(0.0524)
Mother's age	-0.000606	0.00424
	(0.00289)	(0.0257)
Mother indigenous (%)	0.0589	
	(0.0682)	
Mother is literate (%)	0.212***	
	(0.0598)	
Wealth index	1.840***	0.445**
	(0.113)	(0.211)
Live in urban area	0.361***	0.285***
	(0.0541)	(0.0730)
Year 2009/10	0.784***	0.485*
	(0.140)	(0.294)
Household experienced shock	0.0543	0.0716**
	(0.0339)	(0.0358)
Child age in months	-0.222***	-0.188***
	(0.0220)	(0.0194)
Child age in years squared	0.00120***	0.00117***
	(0.000140)	(0.000122)
Eldest (%)	-0.0252	
	(0.0455)	
Male (%)	0.0447	
	(0.0335)	
Birth weight	0.00956	
	(0.0340)	
Height-for-age	0.100***	-0.0301
	(0.0190)	(0.0362)
Long-term health problems	0.0132	0.0290
	(0.0469)	(0.0533)
Violent Crime in Community	-0.0459	-0.0477
	(0.0372)	(0.0546)
Social Assistance (Education) in Community	0.177*	0.197*
	(0.0988)	(0.112)
Constant	7.945***	13.09***
	(0.858)	(2.489)
Observations	2,225	2,225
R-squared	0.348	0.140

Source: Peruvian Young Lives survey 2002, 2006/7, and 2009/10.
Note: Robust standard errors in parenthesis * significant at 10%, ** significant at 5%, and *** significant at 1%.

Table 4.4: Dissecting Pooled OLS Results

Dependent variable:	PPVT Z-score in 2006/7 [1]	PPVT Z-score in 2009/10 [2]
(a) Mother depressed in 2002	-0.0481	0.0263
	(0.0571)	(0.0523)
(b) Mother depressed in 2006/7	-0.167**	-0.133*
	(0.0668)	(0.0698)
Observations	1,113	1,112

Source: Peruvian Young Lives survey 2002, 2006/7 and 2009/10.
Note: Robust standard errors in parenthesis. * significant at 10%, ** significant at 5%, and *** significant at 1%. All regressions include same demographic controls, as well as a control for Year 2009 as in Table 4.3.

Table 4.5: Chronic Depression vs. Non-Depression

Dependent variable:	PPVT Z-score in 2006/7 [1]	PPVT Z-score in 2009/10 [2]
(b) Mother depressed in 2002 & 06/07	-0.249***	-0.180*
	(0.0801)	(0.0926)
Observations	807	806

Source: Peruvian Young Lives survey 2002, 2006 and 2009.
Note: Robust standard errors in parenthesis. * significant at 10%, ** significant at 5%, and *** significant at 1%. All regressions include same demographic controls, as well as a control for Year 2009 as in Table 4.3.

Table 4.6: Maternal Behavior by Maternal Depression - Year 2002

Variables	Mother depressed	Mother not depressed	Difference	
Panel A - General Childcare Behaviors				
Attended antenatal control	93.50	92.52	0.98	
Child has received vaccines [a]	98.91	99.07	-0.16	
Duration of breastfeeding	70.26	68.39	1.86	
Mother is still breastfeeding	89.86	87.36	2.51	
Child regularly left alone with children under 5yrs	2.71	1.61	1.10	
Child has had serious injuries [b]	21.14	10.93	10.21	***
Panel B - Maternal response to persistent crying				
Carries child	25.75	25.89	-0.15	
Soothes/Sings to child	7.59	8.17	-0.58	
Rocks child	19.51	21.86	-2.35	
Gives child water	2.98	3.80	-0.82	
Smacks child	1.90	1.15	0.75	
Shakes child	0.00	0.12	-0.12	
Pinch/squeezes child tightly	0.00	0.12	-0.12	
Threatens Child	0.27	0.35	-0.07	
Takes child to doctor	8.40	5.29	3.11	
Gives child medicine	4.88	4.03	0.85	
Bottle/breastfeed child	49.32	45.34	3.98	
Puts child face down in crib	0.00	0.23	-0.23	
Swaddles child tightly	0.00	0.12	-0.12	
Lets child cry	2.44	1.96	0.48	
Other	45.80	49.14	-3.34	
Observations	325	788		

Source: Own estimations based on Peruvian Young Lives survey 2002.

Note: Differences in characteristics between both groups are statistically significant *** at 1%, ** at 5%, and * at 10%. Sample of observations with available information on maternal depression in 2002 and 2006 and PPVT scores in 2006 and 2009.

a. Vaccines include: polio, BCG, and measles

b. Injuries include: serious fall or burn, and broken bone.

Table 4.7: The Effect of Alternative Measures of Maternal Well-Being on Child Cognition

Measures of Maternal Well-Being	Depression	Maternal Satisfaction	Perception of Mobility
	[1]	[2]	[3]
Panel A - Dependent variable: PPVT Z-score in 2006/7			
Maternal Well Being in 2006 (current)	-0.167**	0.0367***	0.0675
	(0.0668)	(0.0128)	(0.103)
Observations	1113	1112	1113
R-squared	0.340	0.341	0.337
Panel B - Dependent variable: PPVT Z-score in 2009/10			
Maternal Well Being in 2006 (lagged)	-0.133*	0.00882	0.0119
	(0.0698)	(0.0131)	(0.117)
Observations	1112	1111	1112
R-squared	0.378	0.375	0.376

Source: Peruvian Young Lives survey 2002, 2006 and 2009.

Note: Robust standard errors in parenthesis. * significant at 10%, ** significant at 5%, and *** significant at 1%. All regressions include same demographic controls as in Table 4.3.

Appendix

Table A.4.1: Study's vs. Rest of Sample

Variables	Study's Sample	Rest of Sample	Difference	
Panel A – 2006/7				
Age of the child (in months)	63.42	63.51	0.09	
Eldest (%)	0.17	0.16	0	
Male (%)	0.51	0.5	-0.01	
Birth weight	3.2	3.21	0.01	
Height for age z-score	-1.62	-1.42	0.21	***
Long-term health problems (%)	0.08	0.09	0.01	
PPVT score	26.02	30.07	4.05	***
PPVT Z-score	-0.19	0.07	0.26	***
Age of the mother (in years)	31.1	31.46	0.36	
Mother has a partner (%)	0.84	0.88	0.04	*
Mother indigenous (%)	0.14	0.16	0.02	
Mother's edu: primary or less (%)	0.53	0.42	-0.11	***
Mother is literate (%)	0.74	0.79	0.05	**
Wealth index	0.43	0.49	0.06	***
Urban (%)	0.49	0.58	0.09	***
HH experienced shock (%)	0.56	0.53	-0.03	
Number of observations	1113	468		
Panel B – 2009/10				
Age of the child (in months)	95.02	94.9	-0.13	
Eldest (%)	0.27	0.23	-0.04	
Male (%)	0.49	0.5	0.01	
Height for age z-score	-1.22	-1.04	0.18	***
Long-term health problems (%)	0.09	0.09	0	
PPVT score	44.85	47.63	2.78	***
PPVT Z-score	-0.15	0.07	0.22	***
Age of the mother (in years)	33.43	33.73	0.3	
Mother has a partner (%)	0.87	0.88	0.01	
Mother indigenous (%)	0.15	0.16	0.01	
Mother's edu: primary or less (%)	0.52	0.42	-0.1	***
Mother is literate (%)	0.75	0.79	0.05	*
Wealth index	0.49	0.56	0.07	***
Urban (%)	0.64	0.76	0.12	***
HH experienced shock (%)	0.57	0.53	-0.04	
Number of observations	1113	468		

Source: Own estimations using Peruvian Young Lives survey, 1st, 2nd and 3rd round.

Table A.4.2: Maternal Depression in Stage 1 –
Comparison across YL rounds

	Round 2		
Round 1	No Depression	Depression	Total
No Depression	726	62	788
Depression	244	81	325
Total	970	143	1,113

Source: Peruvian Young Lives survey, 1st and 2nd rounds.
Note: Sample of observations with available information on maternal depression in 2002 and 2006 and PPVT scores in 2006 and 2009.

Table A.4.3: Impact of Maternal Depression on Child Cognition - Heterogeneous Effects by Gender

Dependent variable: PPVT Z-score	Pooled OLS		Fixed Effects	
	Girls	Boys	Girls	Boys
	[1]	[2]	[3]	[4]
Depressed mother	-0.0929	-0.0492	0.0600	-0.137*
	(0.0596)	(0.0661)	(0.0683)	(0.0801)
Demographic controls	Yes	Yes	Yes	Yes
Year 2009	Yes	Yes	Yes	Yes
Fixed effects	Yes	Yes	Yes	Yes
Observations	1,116	1,109	1,116	1,109
R-squared	0.390	0.312	0.162	0.138

Source: Peruvian Young Lives survey 2002, 2006 and 2009.

Note: Robust standard errors in parenthesis. * significant at 10%, ** significant at 5%, and *** significant at 1%. All regressions include same demographic controls, as well as a control for Year 2009 as in Table 4.3.

Table A.4.4: Impact of Maternal Depression on Child Cognition - Heterogeneous Effects by Maternal Characteristics

Dependent variable: PPVT Z-score	Pooled OLS		Fixed Effects	
	[1]	[2]	[3]	[4]
Panel A - Mother has a Partner	Yes	No	Yes	No
Depressed mother	-0.0437	-0.0877	-0.0537	-0.212
	(0.0557)	(0.149)	(0.0472)	(0.129)
Demographic controls	Yes	Yes	Yes	Yes
Year 2009	Yes	Yes	Yes	Yes
Fixed effects	Yes	Yes	Yes	Yes
Observations	1,958	267	1,958	267
R-squared	0.141	0.239	0.344	0.432
Panel B - Maternal education level	Primary or lower	Secondary or higher	Primary or lower	Secondary or higher
Depressed mother	-0.123*	-0.0123	-0.183**	0.0936
	-0.0622	-0.0615	(0.0821)	(0.0651)
Demographic controls	Yes	Yes	Yes	Yes
Year 2009	Yes	Yes	Yes	Yes
Fixed effects	Yes	Yes	Yes	Yes
Observations	927	1,298	927	1,298
R-squared	0.191	0.133	0.226	0.256

Source: Peruvian Young Lives survey 2002, 2006 and 2009.

Note: Robust standard errors in parenthesis. * significant at 10%, ** significant at 5%, and *** significant at 1%. All regressions include same demographic controls, as well as a control for Year 2009 as in Table 4.3.

Table A.4.5: Further Exploring Fixed Effects Results

Dependent variable: PPVT Z-score	Excluding non-Spanish speakers [1]	Replacing missing test scores with zero [2]	Standardizing test scores by urban/rural [3]
Depressed mother	-0.0395	-0.0442	-0.0178
	(0.0547)	(0.0583)	(0.0472)
Demographic controls	Yes	Yes	Yes
Year 2009	Yes	Yes	Yes
Mother fixed effects	No	No	Yes
Observations	1,922	2,223	2,949
R-square	0.129	0.146	0.084

Source: Peruvian Young Lives survey 2002, 2006 and 2009.
Note: Robust standard errors in parenthesis. * significant at 10%, ** significant at 5%, and *** significant at 1%. All regressions include same demographic controls, as well as a control for Year 2009 as in Table 4.3.

Table A.4.6: Impact of Maternal Well Being on Child PPVT scores

Dependent variable: PPVT Z-score	Maternal Satisfaction [1]	Perception of Mobility [2]
Maternal Well Being	0.0214*	0.111
	(0.0116)	(0.0746)
Mother's age	-0.00667	0.00681
	(0.0259)	(0.0254)
Wealth index	0.412**	0.450**
	(0.209)	(0.211)
Live in urban area	0.272***	0.278***
	(0.0736)	(0.0730)
Year 2009/10	0.455	0.499*
	(0.295)	(0.296)
Household experienced shock	0.0706**	0.0852**
	(0.0359)	(0.0353)
Child age in months	-0.187***	-0.183***
	(0.0193)	(0.0193)
Child age in years squared	0.00117***	0.00114***
	(0.000122)	(0.000121)
Height-for-age in 2006	-0.0278	-0.0302
	(0.0359)	(0.0364)
Long-term health problems in 2006	0.0266	0.0354
	(0.0528)	(0.0533)
Violent Crime in Community	-0.0497	-0.0512
	(0.0549)	(0.0546)
Social Assistance (Education) in Community	0.178	0.181
	(0.113)	(0.114)
Constant	12.86***	12.59***
	(2.492)	(2.457)
Observations	2,221	2,215
Children	1,113	1,113
R-squared	0.142	0.136

Source: Peruvian Young Lives survey 2002, 2006, and 2009.
Note: Robust standard errors in parenthesis * significant at 10%, ** significant at 5%, and *** significant at 1%.

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