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The Developmental Approach to Child and Adult Health

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ABSTRACT

The Developmental Approach to Child and Adult Health[1]

Pediatricians should consider the costs and benefits of preventing rather than treating childhood diseases. We present an integrated developmental approach to child and adult health that considers the costs and benefits of interventions over the life cycle. We suggest policies to promote child health which are currently outside the boundaries of conventional pediatrics. We discuss current challenges to the field and suggest avenues for future research.

JEL Classification: I12, I18

Keywords: health, prevention, remediation, capabilities, technology of capability formation

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1 Introduction

Modern medicine focuses on treatment rather than prevention. Great strides have been made in finding effective cures for many pediatric illnesses. Novel drugs and innovative new treatments have been developed. Child health insurance coverage has been expanded, and vaccination programs have rendered certain diseases extremely rare. While these are important advances of which pediatricians are justly proud, the field can do even better.

By many indicators of child health, the United States has not kept up with progress in other industrialized countries. One leading example is infant mortality (Heisler, 2012). For this and other child conditions, many causes are preventable. Yet a growing body of knowledge about the origins of child disease has not made its way into mainstream pediatric practice. Strategies currently well outside the boundaries of medicine can be very effective in preventing illness and promoting health.

Recent evidence from both the biological and social sciences points to the importance of the early years in building the foundations for lifelong health (Knudsen et al, 2006). It is now recognized that human development is a dynamic process that starts in the womb (Barker, 1998; Gluckman and Hanson, 2006), and that early life conditions affect the emergence and evolution of human traits (Conti and Heckman, 2012b) which affect a variety of adult outcomes, including health (Almlund et al., 2011; Almond and Currie, 2011; Conti and Heckman, 2010). Later life environmental influences on development also matter, as does resilience in response to adversity. For policy purposes, it is not enough to know that early-life conditions matter. It is important to know the costs and benefits of remediating early life deficits at different stages of the lifecycle.

A developmental focus suggests new channels of policy influence. Early childhood interventions that enrich the environments of disadvantaged children can be effective policy tools to prevent disease and promote health. An integrated developmental approach to health, starting before conception, is needed to analyze synergies in producing health, cognition, and other mental and behavioral traits, and to model the economic, social and biological mechanisms that produce health over the life course and transmit it across generations.

2 The Longlasting Effects of Early Life Experiences

The contribution of the social and economic circumstances of early childhood to health throughout the lifecourse is now well documented (Anda et al., 1999; Danese et al., 2008). Some of the most compelling evidence on the consequences of early maternal and social deprivation comes from children raised in the adverse settings of Romanian orphanages of the 1980s and 1990s. The most recent research shows a high degree of persistence until 15 years of age of cognitive impairments, sub-optimal physical development and behavioral problems (Rutter, 2010). Detrimental long-term effects of early life adverse rearing conditions on physical health and behavioral development have also been recently shown in experimental studies on nonhuman primates (Feng et al., 2011; Conti et al., 2012). Environmental enrichment later in life can partially remediate the consequences arising in part from adverse early environments, both in animals (Francis et al, 2002; Champagne et al., 2003; Rutter, 2010) and in humans, even following severe deprivation (Sheridan et al., 2012). In every case, the timing of the intervention is a crucial factor. The earlier the intervention, the higher the probability of remediating early disadvantage.

In recent years we have begun to gain a much more sophisticated understanding of how the circumstances in which children are born and raised 'get under the skin', and affect the biological development of the brain and of the rest of the body. Studies of stress response pathways, allostatic load, neuronal development, and more recently, epigenetic mechanisms, have shown that the environment can become biologically embedded in the body, in ways that can affect – also through latent pathways – health across the lifecourse. However, the exact mechanisms through which the environment operates and the nature of the biological embedding are just beginning to be understood. The current state of knowledge suggests that adverse conditions early in life induce changes in brain structure and functions, and that these environmental stressors can affect epigenetic programming of long-term changes in neural development and behaviors (Murgatroyd et al., 2009). The temporal nature of brain development implies that environmental exposures at different ages will affect different areas of the brain. However, the way epigenetic marks translate from a transient state to lasting cellular memory is still unknown. Experimental evidence for rhesus monkeys suggests that early adversity gets under the skin and establishes stable marks very early and independently of cumulative exposures (Cole et al., 2012). Comparable evidence for humans finds persistent epigenetic differences associated with early adversity (Heijmans et al., 2008). However, the quantitative importance and the causal nature of these biological changes still needs to be rigorously established.

On the other hand, the family environments in which children are being raised have been worsening over time (McLanahan, 2004). Divorce rates have been on the rise, and less-educated women tend to be single parents. They also tend to work in lower wage jobs and invest less in their children than more educated women (Heckman, 2008). The economic recession and the

financial stress coming with it has been a primary contributor to family conflict. And, for the first time in more than thirty years, mental health problems have displaced physical conditions as the leading causes of disabilities[2] in U.S. children (Slomski, 2012). It is hard to believe that the rise of developmental disorders such as ADHD, which are caused by multiple and complex genetic and environmental factors, might be due entirely to better diagnostic tools or thresholds. Nonetheless, this change in the epidemiology of child illness finds the pediatric system unprepared and there is the risk of incurring huge costs in the future if they are not properly faced. Child mental health problems affect a wider variety of adult outcomes than physical conditions, ranging from reduced educational attainment to increase in the probability of engaging in unhealthy behaviors (Goodman et al., 2011; Conti et al., 2012).

This evidence suggests that inequalities in endowments and environments present at birth can affect the biology of the body, propagate throughout childhood, and persist into adulthood. It also reveals promising avenues for interventions, by enriching the nurturing environments of children born in disadvantaged families and allow them to develop their full potential. However, despite a large body of evidence on the beneficial effects of such policies (Almond and Currie, 2011; Cunha et al., 2006), early childhood programs still are not considered an option in pediatric practice. Before reviewing evidence from interventions, we now present a lifecycle developmental framework to conceptualize and interpret it.

[2] The leading causes of limitation in usual activities due to chronic conditions in U.S. children are: speech problems; learning disability; ADHD; other emotional, mental, and behavioral problems; and asthma or breathing problems, see Halfon et al. (2012).

3 The Biology and Economics of Health and Human Development

Heckman (2007) and Cunha and Heckman (2007) develop a framework for analyzing the expression and evolution of capabilities, since conception through adulthood, which links early-life conditions to late-life outcomes by accounting for intervening mechanisms and a variety of exposures at different levels. This framework recognizes the multiple nature of capabilities, the synergies across them, and the need to consider the child in her entirety, by developing her cognitive potential, together with her physical and mental health. Hence, the different aspects of the well-being of the child can be written in terms of the capability vector θ_t, which can have different components, such as cognition ($\theta_{C,t}$), mental ($\theta_{MH,t}$) and physical health ($\theta_{PH,t}$). Capabilities are the capacities to function effectively in economic and social life. There are many different capabilities. These capabilities of the child can have different weights in affecting adult outcomes, so that a shortfall in one dimension can be compensated by a greater strength in another. Other capabilities, instead, can have a great degree of specificity. Some of these traits might operate as determinants of choices, while others operate through purely biological mechanisms.[3] These capabilities are not solely genetically determined. They are produced early in the life of the child, and their evolution over time can be represented by the following dynamics of capability formation (Heckman, 2007):

$$\theta_{t+1} = f_t(\underbrace{\theta_t}_{\text{Self-Productivity}}, \underbrace{I_t}_{\text{Investments}}, \underbrace{h_t}_{\text{Environments}}, \underbrace{\theta_t^P}_{\text{Parental Traits}}).$$

This equation captures the notion that the development of child health and other capabilities in subsequent periods depends on the stock already present, and on parental traits, environments,

[3] For example, an obese child is likely to become an obese adult, since the metabolic rate is set early in life.

and investments,[4] starting with the initial endowments determined at conception (θ_0), which are function of maternal investments in pregnancy (I_{-1}). It also embeds the idea that capabilities at one age enhance capabilities at later ages.

For example, a healthy child who is able to pay attention in class learns more and increases her cognitive ability. This can, in turn, have positive effects on her mental and physical health. This framework also recognizes that the productivity of investments depends on the age at which they are made, so that it is easier to develop certain capabilities at certain ages. This depends on the plasticity of the organs which govern the functions underlying these capabilities. If investment effects are especially strong in one period, that is called a sensitive period. If investments are productive in only one period, that is called a critical period. An important feature of this framework is the complementarity of capabilities with investments, i.e. the fact that investments are more productive in children with higher stocks of capabilities. This not only implies that providing early nurturing environments to children affects their health and development, but also that early life interventions have to be followed-up with quality schooling and health care, in order for them to be effective in the long-term.

[4] The latter include investments made by parents, teachers and doctors in the health and development of the child.

Figure 1: A Life Cycle Framework for the Development of Capabilities

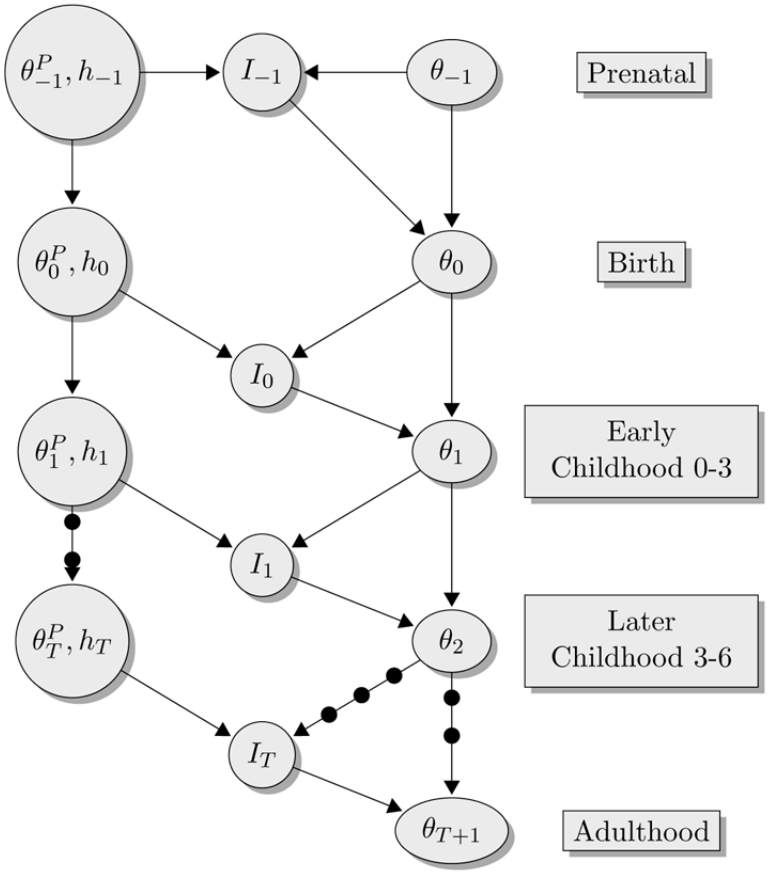


Figure 1 summarizes the framework graphically. Progress has been made in estimating some of the linkages displayed in this figure but most remain unknown.[5] The figure suggests both opportunities and dangers. Investments and environments shape capabilities. There are many stages and strategies for intervention. Understanding at what stages investments are most effective for shaping which capabilities will inform health policy. To make wise policy choices, it is necessary to have a deeper understanding of the mechanisms – both social and biological – displayed in Figure 1. Knowing that early-life conditions causally affect adult health does not tell us the channels through which they operate or the mechanisms through which effective remediation might occur. Early-life conditions might trigger a series of later-life events that

[5] Estimates reveal sensitive periods in early life (before age 10) for cognitive capabilities and sensitive periods for behavioral traits through adolescence (Cunha et al., 2010).

shape the capabilities that produce adult outcomes. Perhaps later-life interventions are highly effective and later life remediation is effective. Alternatively, early-life conditions might affect biology in an irreversible way. In that case, early life interventions are essential. The current literature offers only hints at answers to these questions. When the causal links of Figure 1 are fleshed out, analysts will be better able to suggest effective, age-graded policies for promoting child health and development. Surely our ability to design effective policies will increase as evidence from the biological sciences on windows of plasticity for specific organs sharpens. For the time being, we review in the following promising interventions on the basis of the best available evidence.

4 Policies to Promote Child Health and Development

In this section we review recent evidence on the effectiveness of interventions at different stages of childhood, which compensate, in part, for the risks arising from growing up in disadvantaged environments.

4.1 Early Interventions

We first consider research focusing on the earlier channels of influence displayed in Figure 1. The most reliable evidence on the effectiveness of early interventions comes from experiments that substantially enriched the early environments of children born in disadvantaged families. We consider two iconic interventions, the Perry Preschool Project and the Abecedarian Project, since they have been evaluated by the method of random assignment, they have long-term follow-ups, and their health effects have recently been investigated.

The High/Scope Perry Preschool Project (PPP) is a social experiment designed to evaluate the impact of the novel Perry curriculum on highly disadvantaged African-American children of 3-5 years of age. It was administered to five cohorts of children during the early- to mid-1960s in the district of the Perry Elementary School in Ypsilanti, Michigan. The intervention consisted of 2.5 hours classes for five days per week during the regular school year, and included weekly home visits lasting 1/1.5 hours. The curriculum was based on the concept of *active learning*, which is centered around play, based on problem-solving, and placed within a structured daily routine. The final sample consisted of 123 children over five entry cohorts.

The Carolina Abecedarian Project (ABC) is a social experiment designed to test if an intellectually stimulating environment could prevent the development of mild mental retardation for disadvantaged children. The intervention was much more intense than the Perry. It was year-round and full-day. It consisted of a two-stage treatment: a preschool treatment targeting early childhood education (from 0.4 months until 5 years), and a subsequent school-age treatment targeting initial schooling (from 5 to 8 years). It used a systematic curriculum specially developed by Sparling and Lewis (Sparling and Lewis, 1979, 1984) that consisted of a series of 'learning games', but also included a nutritional and health care component.[6] It was administered to four cohorts of children born between 1972 and 1977 and living in or near Chapel Hill, North Carolina (NC). Eligibility was based on a High-Risk Index computed from 13 socioeconomic factors capturing disadvantage. The final sample consisted of 111 children recruited over a 5-year period, resulting in four cohorts. Unlike Perry, ABC provided access to full health services to participants as well as adequate nutrition.

[6] The treated children had breakfast, lunch and an afternoon snack at the child care center, and were also given pediatric care.

Both the Perry and the Abecedarian interventions show consistent patterns of successful outcomes for treatment group members as compared to control group members for both boys and girls (Heckman et al., 2010; Campbell et al., 2008). While among Perry participants, an initial increase in IQ gradually faded out in the four years following the intervention, still at the oldest age studied (age 40), treated individuals had attained higher levels of education, earned higher wages, and were less likely to be on welfare and to commit crime than the controls.

Heckman et al. (2012) show that the effects of the intervention on life outcomes operate primarily through the program's reduction in child's externalizing behavior. Previous attempts at analyzing the effects of these programs on health and risk behaviors (Muennig et al., 2009, 2011) have not accounted for the variety of statistical challenges that these small sample RCTs pose, have not investigated gender differences in the treatment effects, and have not considered health effects across the whole lifecourse of the subjects. Conti et al. (2012) overcome all these limitations and carry out an extensive analysis by taking advantage of all the health information available in the Abecedarian and Perry samples, since early childhood, and including newly collected unique biomedical data. They find statistically significant and economically important program effects for both males and females which were not uncovered in the pooled gender analysis, and which survive when simultaneously tackling all the statistical challenges. Both the Abecedarian and the Perry participants had significantly fewer behavioral risk factors (such as smoking, drinking, drug use, adhering to safe traffic practices), and higher health care coverage. The Abecedarian participants had also a leaner physical constitution in childhood;[7] additionally, the analysis of the biomeasures recently collected for this sample reveals that they were in better physical health also by the time they reached their mid-thirties. See Table 1 for an overview of

[7] Childhood health measures are not available for the Perry sample.

the results. Other studies – such as the Nurse-Family Partnership (Kitzman et al., 1997), which visits pregnant girls and teaches them prenatal health practices and parenting – also provide evidence of a variety of positive health effects from early environmental enrichment (Heckman et al., 2012). Prenatal home visiting programs such as the NFP or the Doula[8] are also particularly appealing, both because they reach at-risk families as early as possible, and because they intervene at the same time on children and adolescent mothers, by affecting those traits still amenable to change during adolescence (Brown and Hurlock, 1977).

[8] Doulas are trained mentors from the community who help young expectant mothers by encouraging healthy prenatal practices, offering support during labor and delivery, and fostering bonds between babies and their parents (Klaus et al., 1993).

Table 1: Health Effects from the Perry and the Abecedarian Interventions—Selected Results

Perry Preschool Project (PPP)					Abecedarian Project (ABC)				
Variable Description	**Control Mean**	**Diff. Means**	**Asym. *p*-value**	**IPW *p*-value**	**Variable Description**	**Control Mean**	**Diff. Means**	**Asym. *p*-value**	**IPW *p*-value**
Behavioral Risk Factors									
Never drunk without permission by age 15 (F)	0.682	0.152	0.140	**0.040**	Started smoking by age 15 (parent report) (M)	0.190	-0.114	0.127	**0.064**
Never smoked marijuana by age 27 (F)	0.364	0.156	0.146	**0.089**	First tried marijuana before age 17 (F)	0.393	-0.233	**0.031**	**0.053**
Drinks alcohol never or once in a while at age 27 (F)	0.773	0.107	0.169	**0.013**	First drink before age 17 (F)	0.571	-0.291	**0.016**	**0.047**
Non-smoker at age 27 (M)	0.462	0.119	0.164	**0.080**	Started smoking regularly before age 17 (M)	0.304	-0.189	**0.052**	**0.030**
Always wears a seat belt at age 40 (M)	0.618	0.182	**0.057**	**0.080**	Always wears a seat belt at age 21 (F)	0.500	0.220	**0.052**	**0.028**
Non-smoker at age 40 (M)	0.472	0.161	**0.098**	**0.020**	Has drank and driven in past month at age 21 (F)	0.222	-0.102	0.170	**0.042**
Any change in diet in past 15y at age 40	0.229	0.151	**0.097**	**0.018**	Physical activity in past week at age 21 (F)	0.071	0.249	**0.010**	**0.012**
Health Care Coverage									
Covered by health insurance in past 15y at age 40 (F)	0.682	0.068	0.308	**0.044**	Covered by health insurance at age 30 (M)	0.476	0.228	**0.057**	**0.088**
Health									
Never classified as mentally impaired by age 19 (F)	0.636	0.280	**0.010**	**0.036**	BSI Depression score at age 21 (F)	59.643	-5.601	**0.026**	**0.002**
					BMI z-score at 60m (M)	0.601	-0.695	**0.009**	**0.026**

Notes: This table is made of two panels, the left panel for the Perry Project, the right panel for the Abecedarian Project. Each panel is made of five columns: (1) The first column describes the variable of interest, the age at which the information was collected, and the gender (M=male, F=female). PPP: `Any change in diet' is in relation to health reasons. ABC: `Wearing a seat belt' refers to when riding in a car driven by someone else. `Physical activity' is defined as exercise or participation in sports activities for at least 20 minutes on 4 or more of the past 7 days at age 21. The BMI z-score is computed using the 2000 CDC growth charts. BSI=Brief Symptom Inventory. (2) The second column shows the mean of the control group, i.e. the group who was not administered the intervention. (3) The third column shows the unconditional difference in means between the treatment and the control group. (4) The fourth column presents the asymptotic *p*-value of the one-sided single hypothesis based on the *t*-statistic associated with the unconditional difference in means in (3). (5) The fifth column presents the one-sided single hypothesis constrained permutation *p*-value based on the IPW (Inverse Probability Weighting) *t*-statistic associated with the difference in means between treatment and control groups. We implement conditional inference to account for compromisation of the randomization protocol. We use permutation-based inference to account for the small size of the samples. By constrained permutation we mean that permutations are done within strata defined by the pre-program variables used in the randomization protocol. We use IPW to account for non-random attrition. Note all these treatment effects survive correction for multiple hypothesis testing. See Conti et al. (2012) for details.

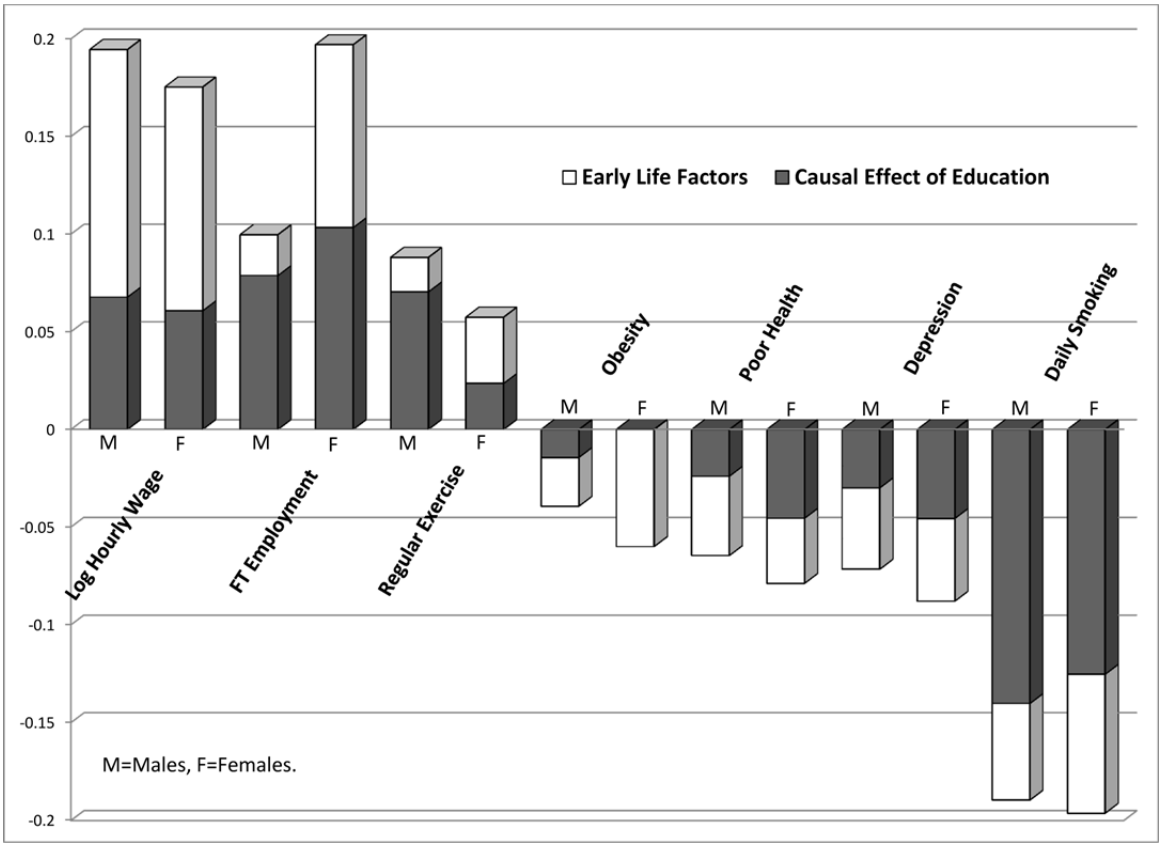
4.2 Later Life Interventions

While the economic benefits to early interventions are substantial (Heckman et al., 2010), at the same time we cannot abandon the children who have had no access to this foundational opportunity and the adults, who did not have such opportunities as children. We now consider the effectiveness of later channels of influence displayed in Figure 1. The key to successful remediation is to invest in carefully designed programs which address those capabilities amenable to change. On the one hand, while the reversibility of structural and functional changes in the brain following early life adverse conditions has not been systematically investigated, recent research in humans is beginning to document the effects of specific interventions in adults to reduce stress and promote mental well-being (Davidson and McEwen, 2012). On the other hand, while environmental enrichment in puberty has shown positive effects both in animals (Imanaka et al., 2008) and in humans (Fisher et al., 2007), the optimal timing and duration, and the most effective components of these interventions are not well understood yet. Nonetheless, knowledge is accumulating rapidly, and designing and implementing biologically-based interventions is the key to promoting the health of the future generation, while not abandoning the current one.

Here we consider in particular one later intervention: education. Is education policy a promising avenue for promoting child health? Specifically what is the effect of education in the adolescent years on health and healthy behaviors? Enhanced capabilities promote schooling, and also promote health and healthy behaviors, both beyond their direct effects on education and through the effects of education on health (Conti et al., 2010). While gaps in cognitive ability emerge early and persist strongly, those in other mental traits are less persistent. The fluidity of personality traits over the adolescent years is associated with the slowly emerging prefrontal

cortex (Steinberg, 2008). At current levels of practice, adolescent remediation for cognitive deficits has largely proved ineffective. Adolescent interventions in personality are more promising instead, although there is less evidence to date on them (Almlund, Duckworth, Heckman, and Kautz, 2011; Heckman and Kautz, 2012; Heckman, Humphries, and Kautz, 2012; Borghans, Diris, Heckman, Kautz, and ter Weel, 2012). Traits beyond cognition have been shown to play a fundamental role in understanding disparities in health and health behaviors by education (Conti and Heckman, 2010; Conti and Hansman, 2012).

Figure 2: Mean Differences in Outcomes Due to Early Life Factors versus the Causal Effect of Education



But what is the relative importance of education as compared to factors formed before the adolescent years? Figure 2, based on British data analyzed by Conti et al. (2010), shows the effect (by gender) of attendance beyond compulsory schooling on health and healthy behaviors measured at age 30. The height of the bars (including both the light and dark portions) displays mean differences in a variety of outcomes between those who stop their education at the minimum compulsory schooling level and those who stay-on. It is clear that more educated individuals are better off under a variety of dimensions. However, the crucial question is how much of the difference between more and less educated individuals is *caused* by education, so that increasing the educational level of the population would be an effective health policy. If the more educated individuals are in better health simply because they have better capabilities developed during the early years, which are associated both with increased education and better health outcomes, then early intervention is a more effective strategy to promote health not only in childhood, but throughout the lifecourse. In order to answer this question, Figure 2 decomposes the drivers of a variety of outcomes by gender. The dark portion of each bar in the graph is the causal contribution of education, and the light portion quantifies the contribution of early capabilities (cognition, mental and physical health traits) shaped by family investments and environments. It shows that these early life factors account for at least a half of the adult disparities in poor health, depression, and obesity. In addition, these early life traits promote education, which has independent effects on outcomes, in particular on healthy behaviors.

Finally, capabilities developed in early childhood can also affect health in the next generation, both directly, and by affecting education and the choices that the mothers make while pregnant. Conti, Heckman, Pinger, and Zanolini (2012) study the determinants of newborn health outcomes as a crucial link in the intergenerational transmission of disadvantage, using

British data on a cohort of women born in 1958.[9] They analyze the role of maternal endowments and investments (education and smoking in pregnancy) on the probability of having a baby who is small for gestational age (SGA). They estimate the total impact of maternal endowments on birth outcomes, and analyze the mechanisms through which they operate. They find that cognition affects the health of the newborn primarily through education, that personality traits mainly operate by changing smoking behavior, and that the physical fitness of the mother has a direct, "biological" effect on SGA. Additionally, they estimate the causal effects of maternal choices, and find significant variability in the effects of education and smoking in pregnancy along the distribution of maternal physical traits, which suggests that women with a less healthy physical constitution should be the primary targets of prenatal interventions.

Such evidence shows that the capabilities developed in early childhood may have longlasting benefits, not only in adulthood but also into the next generation. It also underscores the importance of going beyond a mere collection of treatment effects or correlations between early life conditions and later life outcomes, in order to understand the mechanisms through which these interventions operate and their benefits emerge.

5 Lessons for Health Policy

Three important lessons emerge from this research that should shape future policies to improve the health of the children.

Lesson 1: Develop the Whole Child

To promote the health of the children, pediatricians and policymakers should consider their overall well-being, by viewing the child in her entirety as a human being *in fieri*. Currently,

[9] The National Child Development Study (NCDS).

health policy in the United States focuses primarily on extending health insurance coverage. While universal health insurance is an important ingredient in promoting the health of children, it is not the only one. We must also improve the conditions in which children live, and promote the diverse capabilities – not only health, but also cognition and character traits – which will allow them to become highly adaptive, productive, and healthy adults.

In their quest for accountability in public investments, policymakers must hold themselves accountable for developing the whole child and evaluating progress based on measurements that reflect the full range of capabilities that are essential for success in life and that are highly valued in the labor market. Policy makers have to go beyond tests of cognition as the indicator of child development. They should develop more inclusive measures. A neglected avenue of investigation to promote health is interventions that form these capabilities by exploiting the synergisms among diverse policies. A good health policy may be a good family policy. This argument goes beyond just considering education or nutrition but is consistent with both and integrates these more specialized emphases into a general framework.

Lesson 2: Start Early in Life

We live in an era of substantial and growing social and economic inequality. A large body of research confirms that the accident of birth is a primary source of inequality. Families play a powerful role in creating adult outcomes, by shaping the capabilities of children. Health inequalities between the advantaged and the disadvantaged open up early in the lives of children, well before they enter school. They are already visible at birth. In fact, they can be detected even before birth. Parental choices and family environments are major causal factors.

Family environments in the United States have deteriorated over the past 40 years. A greater fraction of children is being born into disadvantaged families with fewer parenting resources. At

the same time, parents in the top-earning families invest far more in their children than ever before. Due to growing inequality in parental resources and child-rearing environments, the disparity of resources between the haves and the have-nots has increased substantially. If this trend is not reversed, it will create greater economic and social polarization and health disparities in the next generation. Supplementing at-risk families with quality early childhood development resources can help stem this inequality and promote health. (Heckman, 2008)

Lesson 3: Prevention, Not Remediation

Early intervention is far more effective than later remediation. The capabilities that matter can be created. Child health is not solely determined by family income, but also by the parenting resources – the attachment, the guidance and the supervision accorded to children, as well as the quality of the schools, of the neighborhoods and of the hospitals surrounding them. Investments in early childhood development can improve cognitive and character traits and the health of disadvantaged children. Such early efforts promote schooling, reduce crime, foster workforce productivity, reduce teenage pregnancy and foster healthy behaviors. The rates of return on these investments are higher than stock market returns, even in normal times.

The substantial benefit from early investments arises because life cycle skill formation is dynamic in nature. Capabilities cross-foster each other. Early health is critical to this developmental process. A healthy child is ready to engage, will learn more, and is more likely to be a healthy and productive adult. The longer society waits to intervene in the life of a disadvantaged child, the more costly it is to remediate disadvantage in the form of public job training, convict rehabilitation programs, adult literacy programs, or treatment for chronic health conditions. And there is no equity-efficiency trade-off for early interventions for the disadvantaged.

Acting on this knowledge requires a paradigm shift. The pediatric health system, developed more than fifty years ago to primarily cure acute infectious diseases, has to interface and to interact with families, schools, and local communities. A shift to primary prevention will require pediatricians to go beyond the current "siloed" policies which provide short-term fixes using separated and limited budgets, towards an integrated approach to child and adult health and development.

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