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Fetal Origins and Parental Responses

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Abstract

We review the literature on how parental investments respond to health endowments at birth. Recent studies have combined insights from an earlier theoretical literature on how households allocate resources within the family, with a growing empirical literature that identifies early life health shocks using sharp research designs. We describe the econometric challenges in identifying the behavioral responses of parents and how recent studies have sought to address these challenges. We also discuss the emerging literature that has considered how there may be dynamic complementarities in parental investments due to the developmental nature of human capital production and how there may be multiple dimensions of skill. We find that thus far, the bulk of the empirical evidence is consistent with notion that parents reinforce initial endowments.

I. Introduction

The question of how parental investments respond to their children's endowments has emerged as fertile ground for theoretically-minded and research design-focused applied microeconomists alike. The burgeoning literature traces its origins to early work on intra-household resource allocation that was firmly grounded in theory, e.g. Becker and Tomes, 1976. The new phase of research on parental behaviorial responses has been infused with insights from the fetal origins literature which has emphasized sharp identification strategies. The literature has also been invigorated by contributions to our understanding of the developmental nature of human capital production during childhood, summarized and formalized by Heckman (2007).

Understanding this behavioral response is of broad and compelling interest – what parents do in response to endowment differences is something many of us can relate to as children or parents (or aliens observing humans interact with their children). At the same time for empirically minded economists, the literature maintains the virtues of the design-based literature because behavioral responses are potentially as well identified as "reduced form" damage to later-life outcomes. It is just as reasonable to use parental investment measures as dependent variables as it is to use long-term outcomes, so long as one is interested in tracing the effect of an exogenous early-life shock. Recent design-based papers have successfully exploited this opportunity to consider behavior.

Heckman famously bemoaned that: "In some quarters of our profession, the level of discussion has sunk to the level of a *New Yorker* article" (Angrist & Pischke, 2010). Perhaps part of this perceived degeneration is the narrowness of questions sometimes posed in individual design-based analyses. At this point, we think that economics literature on fetal origins has succeeded in demonstrating that a broad spectrum of environmental influences has causal effects on later-life effects – it's not a curiosity limited to famines or pandemics, but rather that the prenatal period is a key developmental window. More satisfying to some might be the central role for behavior and some formal modeling thereof, which at a

minimum can help distinguish economics from epidemiology.[1] As the review below indicates, sometimes behavior seems to respond to endowment shocks and sometimes it does not. Overall, we see relatively limited evidence for compensatory responses by parents, particularly when design-based studies are considered (Table 1). That, we are only beginning to understand whether the parental response is an important component to the later-life outcomes we care about most (e.g. health, cognitive ability, or productivity in adulthood). Thus, we sound a note of caution that while responsive behavior may be of natural interest to full-blooded economists, we should not be seduced by a surpassing interest in behavior *per se*. To maintain relevance outside of family economics, this interest should be scaled by behavior's importance to understanding developmental outcomes. For example, while it could be the case that parents reinforce in response to prenatal health shocks, it might turn out that such behavior tends to only play a small role compared to the purely biological mechanisms set in motion by the initial shock itself.

Distinguishing the role of behavior from biology in understanding "fetal origins" effects is a challenge. First, for mundane reasons: when one includes an endogenous variable as a regressor in attempt to "control for" parental responses, one can introduce bias (see Angrist and Pischke, 2009). For certain worthwhile questions, it may be better to simply ignore parental behavior and be agnostic about its role, as the early design-based literature usually did (often because investments went unobserved). Second, interpreting the role of behavior may be nuanced for reasons specific to the topic at hand, e.g. how substitutable we think investments are across stages of development (or dimensions of capacity). A goal for future work is to try and integrate endowment shocks, responsive behavior, and developmental outcomes into a coherent whole, a point previously made by Bleakley (2010).

This review article begins by defining and describing some of the key concepts and obstacles to estimation in section II. In section III we discuss a selection of recent empirical studies on parental responses that illustrates a range of methodological approaches. Here, we also highlight the recent

[1] The preeminence of identification strategies also distinguishes economic analyses from those in epidemiology. Interestingly, earlier analyses in epidemiology featured a more design-based approach to observational data, e.g. Heider 1934 and Stein et al. 1975, than more recent epidemiological work.

literature on "dynamic complementarities" in the production of human capital and how it has informed our understanding of parental investment behavior, noting that dynamic complementarities are particularly challenging to demonstrate in design-based analyses. In section IV we discuss some very recent empirical work by Heckman and co-authors that has begun to consider multiple dimensions to endowments and investments and the implications of such models on parental responsiveness. Section V concludes.

II. Background

Definitions and concepts

In this section, we briefly review some basic concepts in the fetal/developmental origins literature that are used in the remainder of the review. For a more comprehensive and formal treatment, please see Heckman (2007) and Almond and Currie (2011).

It is common to refer to the stock of capacities at birth as the birth endowment. For the most part, studies have treated the birth endowment as unidimensional. As we discuss later, many recent studies have used birth weight as a measure of this endowment and such studies often have "health" in mind as the key dimension. An exogenous component of the birth endowment can be isolated by considering prenatal shocks. If postnatal investments in human capital are positively correlated with the shock, they are said to be *reinforcing*. They are considered *compensating* if the correlation is negative. One issue which we will return to in section IV is whether it matters whether we think of there being multiple dimensions to human capital (e.g. health and cognitive ability) and whether the endowment shock and the investment responses refer to the same dimension.

It is tempting to think that whether parents reinforce or compensate within families in response to prenatal shocks would largely be driven by parental preferences, in particular, the degree to which parents have an aversion to inequality among their children. However, in an optimizing framework, one needs also to consider how responsive postnatal investments affect subsequent capacity stocks (e.g. health in

adulthood). If substitutability between prenatal shocks and postnatal investments is high, then compensation is more likely. If the elasticity of substitution is very poor (e.g. Leontieff in prenatal and postnatal investments), reinforcement is more likely. In general the more extreme the production technology, the less we learn about parental preference from the sign of the investment response (Almond & Currie, 2011). In this respect, basic formal modeling helps us interpret the design-based evidence. One interpretation of the results from design-based literature on fetal origins that finds large effects of prenatal shocks on long-term outcomes is that the elasticity of substitution between prenatal and postnatal periods is low.

The recent literature on the developmental nature of human capital production has introduced new concepts that are relevant to understanding parental responsiveness to initial endowments. One important idea in developmental models is that the effect of an investment flow in human capital in a particular period of childhood may depend on the level or "stock" of human capital in the preceding period. If the return to investment is larger when the stock is higher in the preceding period, this is referred to as a *dynamic complementarity* (Heckman, 2007). In the presence of dynamic complementarities early in childhood, one might expect parents to be more likely to make reinforcing investments. A related concept, *self-productivity* (Heckman, 2007), by contrast, is about levels rather than investment flows and simply refers to the extent to which the level of human capital in one period depends on the level of human capital in the preceding period. More interestingly perhaps, this can include effects across dimensions of capacity (e.g. cognitive ability helps you form health).

Empirical Challenges

Practical obstacles to estimating a causal effect of a prenatal shock to endowments on parental investment responses provides some context for the various empirical strategies that have been utilized. First, ideally one wants to use a measure of the endowment at birth that is a meaningful indicator of health

or human capital at birth that is easily observable to the parent.[2] Early studies in the literature on household allocation often did not have good measures of endowments and had to use a variety of indirect strategies to infer such endowments. Second, for credible identification one would like the variation in the measure of endowments to reflect exogenous differences. For example, some of the differences in endowments at birth (e.g. birth weight) are driven by prenatal investments such as behaviors during pregnancy (e.g. nutrition, smoking, drinking, health examinations). A positive correlation between the endowment at birth and a postnatal investment could simply reflect the correlation between unobserved prenatal and postnatal investments rather than a behavioral response to the endowment.

Third, ideally one wants a measure of the parental post-natal investment that inherently reflects a behavioral response on the parent. Suppose it were the case that for institutional reasons (e.g. living in a particular state) some parents were required to immunize their children against certain diseases. In that case one might worry that the use of vaccinations as a measure of postnatal investments could be a poor measure of parental response.[3] More worrying might be a parental "response" that is actually a mechanical effect of the initial treatment (more on this below).

Many early studies in the literature used completed years of education as a measure of parental investment. This can be problematic for various reasons, including that children may play an important role in deciding how much schooling they will actually complete. If one uses a measure of child endowments (e.g. test scores) that in part captures aspects of the child's personality (e.g. perseverance) then this can also induce a spurious correlation between endowments and subsequent investments.

In recent years the empirical literature on parental responses to child endowments has made important advances in at least two areas. First, recent studies have utilized better data to construct more direct measures of both children's health endowment and parental investments. For example, the more

[2] It could be that the researcher observes something (e.g. birth weight) that is correlated with a better metric that parents observe and the researcher does not, but one would then like a sense of the relationship between the variables.

[3] This could be particularly problematic if those same parents were also required to undertake similar prenatal investments.

widespread use of natality data has provided researchers access to data such as birth weight and breast-feeding (at hospital discharge) and the Demographic and Health Surveys (DHS) to measure parental investments. Second, and perhaps more importantly, the literature has employed a variety of methodological approaches to deal with the challenge of how to credibly identify parental investment responses that are causally linked to the stock of human capital at birth. Stalwarts who take stock from design-based studies alone may find little evidence for compensatory response patterns.

III. Review of recent empirical studies

We organize studies by the basic types of methodological approaches used. As the Table 1 summary indicates, a variety of methodological approaches has been used and a variety of responses ranging from reinforcing, through zero, to compensatory have been found. Overall, we interpret the current state of the literature to suggest investments are frequently not compensatory and often reinforcing. This is consistent with a strongly developmental production function, as the design-based fetal origins literature suggests.

Family Fixed Effects

The fetal origns literature has spurred a resurgence of interest in investment allocations across children within the household. Datar et al (2010) is among the first studies to directly measure both child endowments and parental investments. Specifically they use the Children of the National Longitudinal Survey of Youth (CNLSY) data and use the birth weight of children as a proxy for endowment at birth. They use measures of breast-feeding initiation, well-baby visits, immunization and pre-school attendance in order to capture postnatal investments by parents. Their main estimates rely on a family fixed effects estimator that relates the difference in parental investments among siblings to differences in birth weight. They find that children who are normal birth weight ($\geq$ 2500 grams) are 5 to 11 percent more likely to receive parental investments compared to their low birth weight siblings. These results suggest that parents reinforce endowment differences rather than compensate for them.

As part of their analysis they also find that an increase in the number of low birth weight siblings that a child has leads to greater parental investments in that index child. One concern with their approach is that parental investments such as well care visits may increase when one has a low birth weight sibling simply because of the greater ease of access to care generated by the heightened attention given to the low birth weight sibling. Such an effect would imply a different mechanism for parental response than a deliberate decision on the part of parents to invest in the relatively advantaged child.[4]

A more general concern with studies that use family fixed effects models is that they rely on the assumption that there are no sibling-specific unobserved differences that could account for both their birth weight differences and their subsequent post-natal investments. Datar et al (2010) attempt to address this concern by including a variety of sibling specific measures (e.g. family income, mother's age, mother's education, first month of prenatal care, smoking or alcohol use during pregnancy) that could account for a common pattern in sibling differences. As a robustness check, they also use only siblings born up to two years apart and find similar effects. Nevertheless one may still be concerned that there may be unobserved sibling specific factors that are correlated with both lower birth weight and lower parental investments that confound a causal interpretation even for siblings born with two years.[5] We will return to this general issue below.

Hsin (2012) also uses a sibling model with fixed effects but uses data from the Child Development Supplement of the Panel Study of Income Dynamics (PSID-CDS). Like Datar et al (2010), Hsin measures child endowments directly using birth weight. An important innovation is to analyze two sibling-specific, time–based measures of parental investment among children aged 12 or under. The first

[4] The authors consider the possibility that the likelihood of breastfeeding may be reduced if a child is very low birth weight and is placed in a neonatal intensive care unit. They find similar effects when they drop very low birth weight children from the sample. However, the authors do not consider other health factors that could lead to a positive association between birth weight and breastfeeding. For example, children born prematurely may not be able to breastfeed initially.

[5] Del Bono et al (2012) use data from the U.S. National Survey of Family Growth to estimate how breast-feeding is affected by the birth weight of previously born siblings and find evidence of compensating parental investments. They first estimate mother fixed effects and child specific idiosyncratic birth endowments using a dynamic structural model where identification is based on using a fixed effects and instrumental variables (FE-IV) estimator. They then regress breastfeeding on the birth weight of previous siblings while including the mother fixed effects and child-specific idiosyncratic endowments from the FE-IV model as additional regressors.

is the total amount of time that the mother spends with the child and the second is a measure of time spent with the child on activities that are directly related to human capital development. The latter measure includes time spent reading, playing, doing hobbies and doing homework together. Hsin reports that the maternal time spent with children is identical in only about 23 percent of the sibling pairs and in some cases the differences in maternal time are large.

The use of time-based measures of parental investment during childhood potentially presents some advantages over other measures of "investment" during the immediate postnatal period (e.g. breast-feeding) that could be directly related to birth weight for reasons unrelated to parental decision-making. On the other hand, sibling differences in maternal time could be highly age dependent and adjusting maternal time for age (as Hsin does) may not perfectly address confounding influences.

The results suggest an important role for mother's education in determining whether parents compensate or reinforce. Specifically, in a model without maternal education, Hsin finds no statistically significant effect of log birth weight on maternal time investments. However, when she interacts log birth weight with maternal education she finds a statistically significant negative effect suggesting that more educated parents are more likely to compensate. Hsin plots a preferred set of estimates based on a model that uses piece wise linear splines in mother's education for a sample of siblings under the age of 6. These results imply that while low educated mothers (less than 12 years of schooling) reinforce birth weight differences, better educated mothers compensate. She argues that in the aggregate the compensatory effects dominate.

As Almond and Currie (2011) note, several explanations might account for this relationship. It could be that the elasticity of substitution between consumption and human capital investment could be higher for families of lower socioeconomic status leading them to be more likely to reinforce a negative shock to the birth endowment. Alternatively, it could be that families of lower socioeconomic status are credit constrained and may be forced to shift resources to the better endowed child due to limited resources.

Meanwhile, Datar et al. (2010) report finding no significant differences by maternal education in their CNLSY data (implemented by interacting birth weight variable with maternal education). However, Restrepo (2011) likewise uses the CNLSY but uses a different set of proxies for parental investment that are measured later in childhood, finds a very similar pattern of results as Hsin does with the PSID-CDS. This suggests the possibility that the timing when investments are measured may be important.

As mentioned above, a key assumption is that there are no unobserved sibling-specific effects that are correlated with both birth weight and the measure of parental investments. In light of Hsin's results, however, an alternative explanation based on unobservables would have to explain a negative correlation between birth weight and maternal time investments (during childhood) among highly educated mothers and a positive correlation between birth weight and maternal time investments among less educated mothers. This "non-monotonicity" would likely require a more complicated confounding story in order to account for Hsin's findings.

Aizer and Cunha (2012) also use a family fixed effects framework and provide some notable advances in measuring parental investments. The Collaborative Perinatal Project (NCPP) collected very detailed data on the characteristics of parents and children based on nearly 60,000 births in 11 cities that occurred between 1959 and 1965. To assess parental investments they use information derived from a psychologist's ratings of a mother's parenting behavior when her child is 8 months old along many dimensions (e.g. expressions of affection, handling of the child, management of the child, responsiveness to the needs of the child). Aizer and Cunha are motivated by research on "attachment theory" which suggests that when children develop strong bonds with parents it improves their neurological development, leads to a greater capacity to learn, and has been associated with improvements in measures of cognitive ability.

To measure endowments they use a rich set of measures taken at birth including birth weight, gestation length, body size, and head circumference. Following Rosenzweig and Wolpin (1988) they use a fixed effects model that includes a variety of covariates capturing several key aspects of prenatal

parental investments: smoking during pregnancy, nutrition, whether the mother was trying to conceive. They then construct a residual component that can be thought of as an endowment measure that is net of these key prenatal investments, using factor analysis on the residuals of the different endowment measures. With this approach they argue that they address measurement error and endogeneity. Using this method, they find that parenting behavior is positively associated with their measures of endowments suggesting that parents use post-natal investments to reinforce differences.

One possible concern with Aizer and Cunha's approach is that the measures of parenting behavior that they use could potentially simply reflect the personality that their children are born with and that these innate personality differences could in turn, shape the quality of interactions parents have with children (see, e.g., Harris (1998)). The estimated effects could then reflect the correlation between the residual component of health endowments and personality. (We discuss Aizer and Cunha 2012 further in the subsection below on dynamic complementarity).

Twins

An alternative approach that is similar to the family fixed effects model but which narrows the potential scope for confounding influences is to compare twins. Simply put, it is virtually impossible for parents to deliberately treat their twins differently during the *in utero* period. As part of a larger analysis,[6] Royer (2009) uses the Early Childhood Longitudinal Study, Birth Cohort (ECLS-B) which contains a sample of nearly 1500 twins, to study differential investment responses to twin differences in birth weight. Specifically Royer examines whether neonatal intensive care use or the number of days in a hospital (which can be viewed as investment decisions made by health professionals) is related to birth weight and finds weak evidence of compensatory responses. She also reports finding no effects of birth weight differences on breast feeding.

[6] The main analysis in Royer (2009) uses California natality files to study the short and long-term effects of birth weight differences among twins.

Building upon (i.e. borrowing) Royer's idea, Almond and Currie (2011) use the same ECLS-B sample to examine a host of measures that reflect parental investment responses slightly later in childhood. They find few cases of differential parental behavior that are significant. They find that parents are more concerned about whether a low birth weight twin is ready for school. In some samples they also find differences in the timing of the introduction of solid food. They find no differences, however, in whether parents reprimand, praise, caress or otherwise behave differently among their twin children.

While the use of data on twins rather than siblings helps address the concern about sibling-specific unobserved factors, it is not a panacea. On the one hand, even twin endowment differences may come bundled across dimensions (see Section IV below on Multi-dimensional capacity). On the other hand, postnatal allocation decisions for twins may not generalize well. In particular, one might be concerned that it is simply very costly to implement favoritism among twin children and it therefore may be much more difficult to identify instances of reinforcing or compensating behavior.

Research Designs in Observational Data

As mentioned in the introduction, the design-based literature in fetal origins has until fairly recently ignored parental responses (often for data reasons). A number of recent empirical papers have made use of the insight that econometrically we are still on *terra firma* so long as behavior is the dependent variable together with some richer datasets. This has yielded some new and credible estimates on parental behavior.

Kelly (2011) uses the geographic variation in the spread of the 1957 influenza epidemic across the U.K. to identify the effects of prenatal exposure to influenza on birth weight and on children's test scores. The study uses the National Child Development Survey (NCDS) which follows a large sample of children who were born in one week in March of 1958 and who were potentially exposed to the Asian flu pandemic *in utero*. The epidemic struck England between September and November of 1957. Kelly

finds that only mothers with certain characteristics (those who smoked during pregnancy or were of short stature) had lower birth weight children as a result of flu exposure. In contrast lower childhood test scores were found for those with exposure to the virus irrespective of maternal characteristics. The study explicitly acknowledges the possibility that responsive behavior on the part of parents could constitute part of the reduced form effect that is identified. To address this, Kelly uses two approaches. First, Kelly use parental investment measures as a dependent variable. Second the parental investment measures are interacted with the exposure measure. Kelly reports that in neither case is there evidence that postnatal parental investments responded to the epidemic. [7]

Bharadwaj et al. (2012A) take a different methodological approach, using administrative data from Chile and Norway to implement a regression discontinuity (R-D) design. Infants who weigh under 1500 grams are classified as very low birth weight (VLBW), and are often provided access to special medical treatments (e.g. surfecant) after birth. Bharadwaj et al. (2012A) show that infants who are just below the cutoff not only received greater access to medical care after birth but also experienced improved test scores and higher grades in childhood compared to those whose birth weight is just above the VLBW cutoff. Like Kelly, Bharadwaj et al. (2012A) explicitly consider the extent to which these effects may be driven by parental responses by using a variety of measures of parental investment as dependent variables. They find no evidence of differences around the VLBW cutoff in the quality of schools attended, the time spent by parents reading to children, whether the child was enrolled in child care by age 5, or whether the mother returned to work after child birth. While these results appear internally valid, it remains an open question how they may generalize to other aspects of neonatal care or higher birth weight infants.

Akresh et al. (2012) use data from a sample of households in rural Burkina Faso to estimate how parental investments in education and time spent by children on household labor responds to differences

[7] While the measures of investment and the detail results are not reported in Kelly (2011), in private communication Kelly reports using measures such as time spent reading to children, time spent on outings with a child and teacher assessments of parental interest in the child's education. Kelly also suggests that while her effects were statistically insignificant, and of mixed sign, her data may not have had sufficient power to detect effects.

in cognitive ability between siblings. A key difference between their analysis and the sibling models described earlier is that they use rain fall shocks experienced early in life as an instrument for cognitive skills. Using this approach they find that higher ability children have higher rates of school enrollment and are less involved in child labor activities suggesting that parents reinforce endowment differences.

At first blush the study appears to offer some advantages relative to studies that only rely on family fixed effects for identification by introducing the rainfall shock which is plausibly exogenous. On the other hand the prolonged lag in this study between when the endowment shock occurs (mainly *in-utero*) and when the endowment and parental investment is measured (between the ages of 5 and 14) introduces some issues about interpreting the effects, particularly when combined with the broad nature of the shock. For example, it is likely that there were many household investment decisions (e.g. from rain-induced income changes) in the post-natal period that could have contributed to both cognitive ability and the likelihood of school enrollment measured later in childhood. In contrast the papers that rely on family models but measure endowments at birth (e.g. birth weight) and immediate investment responses (e.g. breastfeeding) may be conceptually more relevant.

Tropical disease has also been used by a number of studies to demonstrate long-term effects of health impairments early in life (e.g., Bleakley, 2007 and Barreca 2010). In a recent study set in Mexico, Venkataramani (2012) links malaria eradication in one's year of birth to a number of outcomes including improved cognitive test scores measured in adulthood. Venkataramani addresses the potential for parental investment responses to mediate these effects by examining the timing of schooling investments. He argues that given a positive endowment shock, a standard human capital model would predict that children would likely start school at an earlier age on average. This is because parents who would have otherwise delayed school entry because the marginal returns to schooling did not yet outweigh the marginal costs to schooling, may now find that with the improved learning capacity of their children due to malaria eradication, it would make sense to have children start school at an earlier age. Given that

there are few outside opportunities to schooling in the labor market for young children (that could also benefit from a positive shock), this is a relatively unambiguous prediction.

On the other hand, Venkataramani argues that it is ambiguous whether an endowment shock would affect the age at which children *leave* school when they are older. This is because at later ages it is more likely that improved cognitive abilities could confer advantages both for learning and outside options in the labor market. At later ages the endowment shock could lower the marginal costs by more than it improves the marginal benefits of education. Indeed, Venkataramani finds that malaria eradication appears to both lower the age at which children start school and the age at which they finish school. Since the age of school entry is likely a decision made by parents, this provides evidence that parents reinforce endowment shocks. However, since this is the only measure of parental investment. Further as was the case with Akresh et al (2012) one might prefer to see more immediate investment responses.

Adhvaryu and Nyshadham (2012) present perhaps the most compelling and interesting evidence thus far on parental responses. They build on previous work by Field et al. (2009) who showed that a large scale iodine supplementation program for women of child-bearing age in Tanzania led to increases in educational attainment among children who were exposed to the program *in utero*. Medical studies have shown that iodine deficiency early in pregnancy can inhibit normal neurological development. Adhvaryu and Nyshadham follow up on this prior work by examining how parental investments responded to the plausibly exogenous improvement in the cognitive endowment of children. Specifically they use data from the 1999 round of the Tanzania Demographic and Health Surveys (DHS) containing a rich set of measures of post-natal parental investments including the duration of breastfeeding, and vaccinations among children under the age of 5.

They find that children are more likely to be breastfed and are more likely to be immunized if they were exposed to the iodine supplementation program. Further, they find that there are spillover effects on siblings. Controlling for one's own exposure, parental investments are larger if one has

siblings that were exposed to the iodine supplementation program. One threat to the research design is if there were other aspects of the iodine supplementation program (e.g. health information) that might have direct effects on the likelihood of women undertaking investments. Adhvaryu and Nyshadham cite prior evidence in the literature suggesting that no such other aspects of the program existed. They further show that the program did not appear to directly affect measures of neonatal investment or measures of the health endowment at birth as measured by birth weight or perceived size at birth.

The results suggest that while parents invest more in a child with higher cognitive endowments (i.e. reinforce), they may also invest more in his or her siblings. This implies that studies that rely on family models to identify sibling differences may be missing an important aspect of household allocation decisions and under-estimating the total effect on parental investments.[8] Nevertheless, and appealing feature of this study is that it arguably considers a specific treatment that is known to affect cognitive ability but is not strongly associated with health more generally. This stands in contrast to studies that have relied on birth weight –which may not serve as a useful indicator for whether there has been an impairment to cognitive function.[9] To the extent that the real question of interest relates to how parental investments specifically relate to cognitive endowments, this may advantageous. In addition, they use key measures of post-natal parental investments that should occur fairly quickly after birth. Finally, they are also able to take account of other observable measures of the health endowment that likely reflect prenatal investments as well as measures of neonatal investments.

Random Assignment

Thus far, we have not encountered any studies that use randomized control trials (RCT) to identify parental responses to birth endowments. We expect this to change. For example, Li et al (2009)

[8] This parallels the criticism that Gluckman and Hanson (2005, p101) has made of twin studies in the fetal origins literature that have relied on birth weight differences to measure fetal injury and which have not found differences in hypertension later in life because these studies failed to understand that in some cases both fetuses are affected by the fetal environment even if this is not reflected in birth weight differences.

[9] Almond and Mazumder (2011) and Kelly (2011) also argue that birth weight may not capture biological adaptive responses that affect latent health or cognition.

analyze the effects of a double blind RCT that provided multi-micronutrient supplementation to several thousand pregnant women in rural China on measures of offspring mental and psychomotor development at up to age 1. Similarly Vaidya et al (2008) implemented an RCT in Nepal to identify the effects of iron or folic acid supplementation during the prenatal period on various measures of childhood size, illness and blood pressure. At some cost, both studies could follow-up with both the treatment and control groups to assess parental responsive behaviors. As in development economics, it may be useful for researchers interested in fetal origins to become more engaged in RCTs of the kind that have been traditionally used by the scientific community in order to better understand how parental behaviors are affected by random treatments during the prenatal period.

Fetal origins aside, Gelber and Isen (2011) used randomized access to Head Start programs among 3 and 4 year-olds to evaluate the effects of Program access on parental investments. Their study uses a very large set of extremely detailed measures of parental involvement in many learning activities such as how often they read to their child, how often they have children read stories, whether they helped their child practice spelling, rhyme words or write letters, words or numbers. In total they have 87 measures of parental involvement retrospectively reported by parents. They find that across many but not all measures that there are statistically significant positive effects of the program on parental involvement.[10] They find that the effects persist even after the program has ended. They even find large effects among time spent with fathers after the program has ended among children who do not live with their fathers.

A key issue in interpreting Gelber and Isen's results is understanding the underlying mechanism behind their findings. They argue that their results are consistent with the possibility that parents are more involved with their children because they find such investments are complementary with the improvement in cognitive or non-cognitive skills induced by Head Start. However, they cannot conclusively rule out whether the greater involvement by parents is simply due to the fact that parental

[10] The paper uses a generalized Hausman test and finds that the effects are jointly significantly different from zero.

involvement itself is a key feature of the Head Start program,[11] i.e. a mechanical effect as mentioned in Section II.

Indirect evidence

Finally, a number of papers have produced indirect evidence on whether parents reinforce or compensate for prenatal endowments. One approach is to compare simple OLS estimates which rely on cross-sectional variation to family fixed effects model which only use within family variation. If family differences among siblings are reinforced (compensated) then under some assumptions the fixed effects estimates would be larger (smaller) than the OLS estimates. Almond, Edlund, and Palme (2009) study the effects of exposure to radioactive fallout from the Chernobyl episode on the educational outcomes of Swedish students who were exposed *in utero*. They find that their estimates are somewhat larger when they use family fixed effects than when they use OLS. This leads them to conclude that "to the extent that parents responded to the cognitive endowment, such responses may have been reinforcing." Bharadwaj et al. (2012B) examine the effects of prenatal exposure to warm temperatures on childhood test scores.[12] In their case they find very little difference between OLS and fixed effects leading them to conclude "that parents do not appear to respond to health or other detriments caused by temperature."

Black et al. (2010) consider in whether an increase in family size leads to a reduction in IQ scores. One approach they use is to estimate the effect of the birth of twins, which may constitute an unexpected increase in family size, on the IQ scores of *existing* children. Using this strategy they do find negative effects of an increase in family size. Rosenweig and Zhang (2006) have made the criticism that studies such as these may understate the true effect because parents may reallocate resources in favor of the heavier first born child rather than to the lower birth weight twins (i.e. reinforce). They advocate that

[11] In one exercise they use a detailed list of 58 characteristics of Head Start centers (e.g. whether parents volunteer in the centers) that presumably would be the main mechanisms by which Head Start could conceivably *directly* affect the level of involvement of parents and find that these characteristics are not highly correlated with the effect of Head Start centers on parent investments. The authors acknowledge that while these results are suggestive that the effects are not driven by direct effects of Head Start on parent involvement, that they are not dispositive.

[12] Bharadwaj et al. (2012B) use a different set of assumptions than described here to infer what the difference between OLS and fixed effects estimates imply about whether parents compensate or reinforce.

researchers address this by controlling for the birth weight of the twin birth. If parents reinforce, then presumably controlling for birth weight should yield even larger negative effects on the IQ scores of existing children. Black et al., however, find that when they control for birth weight that the effects on the IQ scores of the first child older disappear. They conclude that their finding is "consistent with compensatory investment behavior by parents."

Dynamic Complementarities

Dynamic complementarities are defined in the unidimensional case as cases where changes in the return to developmental investments are increasing with changes in the baseline stock. In a multidimensional world, it could be that that postnatal cognitive investments have a higher return when the iodine-induced cognitive endowment is higher. Dynamic complementarities are one theoretical channel by which subsequent investments might optimally reinforce previous stocks (and previous shocks to those stocks). There is clearly much interest in this channel in the emerging literature.

That said, we think making a water tight empirical case for dynamic complementarities is more challenging than simply demonstrating that investments respond to shocks: familiar identification strategies in fetal origins literature are sufficient for the latter but not the former. Causal inference on dynamic complementarities requires: a) exogenous variation in the baseline stock, and; b) exogenous variation in subsequent investment. One can then trace the effects of the interaction on the return. In an observational setting, this may be asking for "lightning to strike" twice: two identification strategies affecting the same cohort but at adjacent developmental stages. Clearly this is a tall order.

In general, empirical studies (struggle to) feature at most one identification strategy. Even if that individual identification strategy is valid, familiar issues like omitted variables bias creep back in to undermine inference on the existence of dynamic complementarities. As a case in point, Aizer and Cunha (2011) claim to use an "exogenous increase in preschool availability to identify…complementarities with early stocks of human capital" which they conclude provides "strong evidence of complementarity

between investments and early human capital." This inference is drawn from the finding that those with higher Bailey test scores at month 8 benefit more from (arguably exogenous) variation in subsequent investments. However, missing is a reason why *only* the Bailey test score is different at month 8 and not other characteristics of the child. This omission is particularly vexing in a multidimensional conception of capacity. Absent a second source of exogenous variation, 8 month bailey test score may be correlated with a variety of other characteristics that may also influence the return to the subsequent investment (e.g motivation, health, patience), which are difficult to observe. Therefore, the return to investments may be different due to complementarities between cognitive investments and accumulation of non-cognitive skills, akin to equation 1 in the Section IV (from equation 19 in Conti et al). A host of alternative factors are not held constant and could affect the return to subsequent Head Start investment. Nor does inclusion family fixed effects provide a solution – raw sibling differences also come bundled, and inclusion of additional controls can even exacerbate omitted variables bias (Clarke, 2005).

That said, we don't view dynamic complementarities as a "fundamentally unidentified question" (Angrist and Pischke, 2009). One could imagine a controlled intervention with two distinct treatment arms targeting adjacent developmental ages for the same cohort. Clearly, such an intervention would require longitudinal data on an especially large sample (i.e. not the easiest place to start). Absent researcher manipulation, it seems those analyzing observational data will need to get especially lucky. A recent attempt in this spirit is by Bhalotra and Venkataramani (2012), who overlay the diffusion of sulfa drugs among children with racial segregation to consider long-term effects on schooling, income, and disability.

Overall, the evidence for dynamic complementarities is mainly descriptive at present. A few studies (Chay et al., 2009; Heckman et al., 2010; Kelly, 2011) have found larger treatment effects at higher capacity levels using quantile estimators, which is consistent with the existence of dynamic complementarities. Again, there are other channels besides dynamic complementarities that could explain these patterns and these three studies are commendably circumspect in invoking the dynamic

complementarity story – it's not their *raison d'etre*. The descriptive evidence that exists is an invitation to sharpen empirical tests, much as early descriptive evidence on fetal origins provoked stronger (generally corroborative) analyses. Eventually we might understand whether dynamic complementarities are an important motivating factor behind responsive parental investments and fetal origins effects more generally.

Summary of Evidence

Table 1 summarizes evidence from the recent empirical literature.[13] We roughly categorize studies into one of four categories: 1) those that find either no effects or small effects on parental responses; 2) those that only find evidence of compensating behavior; 3) those that find only evidence of reinforcing behavior; and 4) those that find mixed evidence.

We have placed a total of five studies in the first category that finds no effects. Of these, two are based on twins comparisons and one is based on a regression discontinuity involving comparisons of very small infants. For reasons mentioned above, their interpretation might be qualified. We have found only two recent studies that only find evidence consistent with compensatory investments, our second category. In one of the studies, that by Black et al. (2010), the issue of parental responsiveness was not really a focal point; the evidence is more indirect and was simply presented as a robustness check. For our third category, those that find only evidence of reinforcement we have placed a total of nine studies, three of which use family fixed effects. Finally, an additional five studies find evidence of both compensating and reinforcing behavior.

Overall, the balance of the evidence seems to be tipped towards finding that parental investments are reinforcing. To the extent that compensating behavior occurs, the evidence suggests it takes place more for higher SES families. There is evidence from two developing countries (China and Ethiopia) of

[13] We chose to limit the evidence to studies from the past 3 or 4 years that have generally used better measures of both endowments and investments. The one exception is Ayalew (2005) which though published in 2005 is unique in that it considers multiple dimensions of investments.

compensating behavior along the health dimension but reinforcing behavior along the cognitive dimension. Although many studies seem to find unambiguous evidence of reinforcement, given the nascent stage of the literature, we do not wish to push this conclusion too far. If biology is doing the "heavy lifting" in terms of outcomes, these investments may still not be of first order importance.

IV. Multi-dimensional capacity

The early modeling of human capacity formation featured a multi-dimensional conception of capacity. For example, capacity could include dimensions of health, cognitive skills, and non-cognitive skills. In general, recent empirical work on parental investments response to endowment shocks has glossed over this potential multi-dimensionality in investments and capacity.

An important exception distinguishes between health *H* and other skills *C* (Conti, Heckman, Yi, Zhang, 2011). The formation of health at a given developmental stage may be intertwined with the accumulated stock of other skills, and vice versa. Thus, we could have a health production technology like:

$$\theta_{t,2}^{H} = (\theta_{t,1}^{C})^{\gamma}[\beta_{\theta}\theta_{t,1}^{H} + \beta_{I}I_{t,1}^{H}]^{1-\gamma}$$

Thus, higher stocks of cognitive skills at the end of period 1 aid in the formation of health through health investments *I*.[14] These production technologies are nested within a convention intra-household resource allocation framework. An empirical prediction of their model is that when a shock to early childhood health occurs to one child, it may be optimal for parents to compensate (help offset) the shock to that child's health but reinforce (exacerbate) the shock in terms of subsequent cognitive investments. Conti et al. find support for this model in an analysis of data on Chinese twins, where direct parental investment measures are observed. The intertwining of cognitive and health dimensions in the production of subsequent capacities means, essentially, that optimal parental responses may be

[14] While the production function above assumes a Cobb-Douglas relationship across health and cognitive dimensions, Conti et al. 2011 show that a more general CES production function yields similar predictions.

heterogeneous and somewhat nuanced. For this reason, it becomes difficult to interpret estimates of "fetal origins" effects from the "reduced form" literature as providing a lower or upper bound on biological effects (effects absent responsive behavior).

Conti et al. sound an articulate and worthwhile note of caution on the interpretation of empirical studies related to the multi-dimensionality of capacity and its formation. This multi-dimensionality may help explain why the literature has "yet to achieve a consensus" (Conti et al.) on whether parental investments tend to be reinforcing or compensating – it may depend on the dimension considered. Indeed, in the context of their theoretical framework, the ambiguity may be greater still. Conti et al assume the birthweight difference within twin pairs has an immediate effect on the early health endowment but not on the cognitive endowment (their equations 1 and 2). Leaving aside the merit of this assumption, consider an alternative shock that had a purely cognitive initial effect, such as that characterized by Almond, Edlund, and Palme (2009) or Advaryu and Nyshadham (2012). Since the Conti et al. model is symmetric with respect to cognitive and health dimensions, we could use it to interpret a purely cognitive shock but swapping the dimension labels of "cognitive" versus "health". In this "photo negative" framework, we would now expect compensation along the educational dimension and reinforcement along the health dimension – i.e. the opposite of the Conti et al. empirical finding.

Indeed, it may be even easier to generate theoretical ambiguity in whether to compensate versus reinforce along different dimensions of capacity. We can simplify the Conti et al. 2011 framework by assuming just one child and taking health out of the production function for cognitive capacity and vice versa. Now, the level of cognitive capacity does not affect the productivity of investments in health in producing next period's health (and vice versa). Instead, we can allow for differing "own" production technologies by which health investments generate health and cognitive investments generate cognitive ability. Arbitrarily, we could assume a relatively developmental production technology for cognitive ability:

$$\theta_C = B[\gamma_C(\bar{I}_{1C} + (1-\beta)\mu)^{\phi} + (1-\gamma_C)I_{2C}^{\phi}]^{1/\phi}$$

And a non-developmental production technology for health:

$$\theta_H = \gamma_1(\bar{I}_{1H} + \beta\mu) + \gamma_2 I_{2H}$$

When ß=1, we have a pure health shock. Using a cobb-douglas child quality function like Conti et al.'s equation 18, we should compensate the health shock. If the health shock is positive, it's optimal to use that bounty to invest in the cognitive dimension, thereby reinforcing the positive health shock in the child with additional cognitive investments. The difference in the elasticities of substitution across the two production functions drives the asymmetric investment response. As we do not yet have a good sense of what these elasticities of substitution are for differing dimensions of capacity, reinforcement versus compensating strategies may be an artifact of these differences rather than a capacity intertwining like that depicted in Conti et al. equation 19.

As in the Conti et al. framework, the situation above is reversed when ß=0 and we have a purely cognitive shock. It is now optimal to increase health investments in response to an increase in the (cognitive) endowment, and reduce cognitive investments. Moreover, it is difficult to know at what value of ß our investment strategy flips. Even in this simple model, the intermediate "no investment response" value of ß is a non-obvious function of the production technology parameters. Even in the design-based literature, the early-life shocks often come bundled (affecting multiple dimensions at the same time), so it may be inappropriate to assume a uni-dimensional shock and trace the multidimensional investment response: it may instead be the multidimensionality of the initial shocks that drives the multidimensional response.

To summarize, allowing for different dimensions of capacity makes the exercise of interpreting empirical evidence more nuanced. Pursuant to the discussion above, future empirical work should consider along what dimensions an initial shock strikes (e.g. what's ß?) and the potential for

multidimensional impacts later in life and the correspondence between these dimensions over time. More challenging from a data perspective is to also consider the response of different dimensions of parental response. At this early stage, it is difficult to know whether the multidimensional nature of human capacity formation is mainly of conceptual interest. Future work in the design-based tradition can help shed light on this question. In the meantime, the basic point of Conti et al. goes through: we should exercise caution in interpreting fetal origins effects as upper versus lower bounds, particularly when within-family estimates are considered.

Does the Bumble Bee Fly?

Bleakley (2010) sounds a sobering note on the interpretation of analyses of parental investments and their optimized response to early-life shocks. His focus is on parental investments in education, but speaks more generally to inputs in the production adult capacity, income, etc. One can decompose the response of capacity due to a health shock into that attributable to the direct effect of health on capacity/income, and that operating through investments. At the optimal level of investments, the marginal return should be zero (i.e., the envelope theorem). While this need not imply that the change in investments due to an early-life shock is zero, Bleakley argues their effect on "what matters" may be zero at their optimal level.

Bleakley's point underscores the need for new studies that can assess not just the response of investments but their effect on later-life capacity. Bleakley also highlights the point that the inframarginal return on investment may change with an endowment shock: the quality of given level of investments improves even if the effect of the last unit of investment is zero. Again, a "lightening strikes twice" design would be a good starting point for testing this hypothesis. For the moment, we are left to explain why investments to the extent they do respond empirically to endowment shocks, more often than not seem to go in the reinforcing direction.[15] Bleakley also discusses potential endogenous response in the

[15] In Bleakley's model $b_{ee}<0$: the marginal benefit of (schooling) investment falls with more investment.

child's opportunity cost of schooling, whereas the childhood investments we have in mind typically occur before such options become important.)

Even with the envelope theorem in mind, investments may still have first order effects on things we care about. To the extent there are externalities to childhood investments (as is often invoked with education), then parental decision makers are not investing the optimal amount insofar as society at large is concerned and the optimized marginal investment consequential. Uncertainty in the returns to childhood investments or a divergence in whose utility is being optimized through investments (parents of kids?) could likewise lead to sub-optimal investment levels and thereby magnify the effect of parental investment decisions. Nevertheless, it is worth reiterating Bleakley's overarching point that investments are a means to an end: we should seek to integrate consideration of investment response with the response of later-life outcomes that enter directly into utility.

V. Conclusion

How parents respond to endowment shocks is a subject of inherent interest made more so by the confluence of researchers and researcher styles working on it. It is refreshing to have joint work between Anna Aizer (Brown) and Flavio Cunha (University of Pennsylvania), Hoyt Bleakley talking about the envelope theorem, and James Heckman talking about when reduced form estimates might or might not be interpreted as biological effects. We expect this area to be focus of continued research attention because the nature of the behavioral response and its importance to long-term effects is still being debated. The current scorecard seems to tilt against compensatory investments. Given the lens it provides on behavior and parent-child interactions, those well outside the "fetal origins" camp can follow developments and any regularities uncovered with interest. Finally, learning more about this area may help inform appropriate individual and policy responses to fetal origins: how to harness the critical developmental window to make more cost-effective investments.

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Table 1: Summary of Empirical Studies on Parental Responses to Endowments

Study	*Country*	*Methodology*
1. No Effects or small effects		
Bharadwaj, Loken and Neilson (2012)	Chile, Norway births	Regression Discontinuity at 1500 Grams
Bharadwaj, Graff Zivin and Neilson (2012)	Chile	Indirect, Temperature in Utero, OLS vs F.E.
Royer (2009)	US	Twins
Almond and Currie (2011)	US	Twins
Kelly (2011)	UK	Flu Exposure in Utero
2. Only Compensating Responses		
Black et al. (2010)	Norway	Indirect, Family Size Effects
Del Bono et al. (2012)	US	Structural Model with fmaily fixed effects
3. Only Reinforcing Responses		
Aizer and Cunha (2012)	US	Family Fixed Effects
Adhvaryu and Nyshadham (2012)	Tanzania	Iodine Supplementation in Utero
Venkataramani (2012)	Mexico	Malaria Eradication
Akresh et al. (2012)	Burkina Faso	Rainfall in Utero
Bhalotra and Venkataramani (2012)	US	Access to Sulfa Drugs in Infancy
Datar et al (2010)	US	Family Fixed Effects
Gelber and Isen (2011)	US	Random Assignment of Head Start
Almond, Edlund and Palme (2009)	Sweden	Exposure to Radiation in Utero
Rosenzweig and Zhang (2009)	China	Family Fixed Effects
4. Evidence of Both Compensating and Reinforcing Responses		
Conti, Heckman, Yi and Zhang (2011)	China	Twins, Multiple Dimensions
Restrepo (2012)	US	Family Fixed Effects
Parman (2012)	US	Flu Exposure in Utero
Hsin (2012)	US	Family Fixed Effects
Ayalew (2005)	Ethiopia	Family Fixed Effects

Appendix to Section IV on Multi-dimensional Capacity, Almond & Mazumder

Capacity has two dimensions, health H and cognitive ability C.

$$\theta_H = \gamma_1(\bar{I}_{1H} + \beta\mu) + \gamma_2 I_{2H}$$

$$\theta_C = B[\gamma_C(\bar{I}_{1C} + (1-\beta)\mu)^\phi + (1-\gamma_C)I_{2C}^\phi]^{1/(\phi)}$$

where $\phi \leq 1$, and $0 \leq \beta \leq 1$.
Parents value a summary measure of child quality:

$$U = U(\theta_H, \theta_C) = \theta_H^{\alpha_H}\theta_C^{\alpha_C}$$

and exhaust budget: $\bar{I}_{1H} + \bar{I}_{1c} + I_{2h} + I_{2C} = y$ or

$$I_{2h} + I_{2C} = y'.$$

Optimizing:

$$\frac{\delta U}{\delta \theta_H}\frac{\delta \theta_H}{\delta I_{H*}} = \frac{\delta U}{\delta \theta_C}\frac{\delta \theta_C}{\delta I_{C*}}$$

I_{H*} and I_{C*} are the optimized levels of period 2 investments, I_{2H}, I_{2C}.

$$\alpha_H\theta_H^{\alpha_H-1}\theta_C^{\alpha_C}\gamma_2 = \alpha_C\theta_H^{\alpha_H}\theta_C^{\alpha_C-1}\frac{1}{\phi}B[C]^{\frac{1}{\phi}-1}(\phi)(1-\gamma_c)I_{C*}^{\phi-1} \tag{1}$$

$$\frac{\alpha_H}{\alpha_C}\gamma_2 = \frac{\theta_H}{\theta_C}B[C]^{\frac{1}{\phi}-1}(1-\gamma_c)(y'-I_{H*})^{\phi-1} \tag{2}$$

$$\frac{\alpha_H}{\alpha_C}\gamma_2 = \frac{\theta_H}{[C]}(1-\gamma_c)(y'-I_{H*})^{\phi-1} \tag{3}$$

$$\frac{\alpha_H}{\alpha_C}\gamma_2[C] = \theta_H(1-\gamma_c)(y'-I_{H*})^{\phi-1} \tag{4}$$

$$G \equiv \frac{\alpha_H}{\alpha_C}\gamma_2[C] - \theta_H(1-\gamma_c)(y'-I_{H*})^{\phi-1} \tag{5}$$

$$G = \frac{\alpha_H}{\alpha_C}\gamma_2[\gamma_C(\bar{I}_{1C}+(1-\beta)\mu)^\phi+(1-\gamma_C)(y'-I_{H*})^\phi]-[\gamma_1(\bar{I}_{1H}+\beta\mu)+\gamma_2 I_{H*})](1-\gamma_c)(y'-I_{H*})^{\phi-1} \tag{6}$$

$$\frac{\delta I_{H*}}{\delta \mu} = -\frac{\frac{\delta G}{\delta \mu}}{\frac{\delta G}{\delta I_{H*}}} \tag{7}$$

$$\frac{\delta G}{\delta I_{H*}} = \frac{\alpha_H}{\alpha_C}\gamma_2\phi(1-\gamma_C)(y'-I_{H*})^{\phi-1}(-1)$$
$$-\theta_H(1-\gamma_C)(\phi-1)(y'-I_{H*})^{\phi-2}(-1) - \gamma_2(1-\gamma_C)(y'-I_{H*})^{\phi-1} < 0 \tag{8}$$

$$\frac{\delta G}{\delta \mu} = \frac{\alpha_H}{\alpha_C}\gamma_2\gamma_C\phi(\bar{I}_{1C}+(1-\beta)\mu)^{\phi-1}(1-\beta)$$
$$-\gamma_1\beta(1-\gamma_C)(y'-I_{H*})^{\phi-1} \tag{9}$$

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