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Parental Income and Children's Life Course: Lessons from the Panel Study of Income Dynamics

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Abstract

This article reviews how the Panel Study of Income Dynamics (PSID) has contributed to our understanding of the links between childhood economic conditions— in particular, the household incomes with very young children—and the economic attainment and health of those children when they reach adulthood. From its beginning, the PSID has provided data useful for addressing intergenerational questions. In the mid-1990s, PSID data supported a series of studies that link early childhood income to early adult attainments, particularly to completed schooling. At the same time, discoveries in neurobiology and epidemiology were beginning to provide details on the processes producing the observed correlations. These discoveries led to a more recent set of PSID-based studies that focus not only on labor market and behavioral outcomes, but also on links between income in the earliest stages of life (including the prenatal period) and adult health. Links between economic disadvantage in childhood and adult health, and the developmental neuroscience underlying those links, are promising areas for future research.

Keywords

PSID; intergenerational mobility; poverty; early life conditions

Our review of how the Panel Study of Income Dynamics (PSID) has contributed to our understanding of links between childhood economic conditions—in particular income very early in life—and adult well-being is designed to complement the article by Hofferth et al. (this volume), which discusses the contributions of the PSID's Child Development Supplements (CDS) begun in the late 1990s. The CDS collects data on children when they are children, and allows analysts to link child outcomes to the rich recent economic and demographic data gathered in the PSID's core data collection. We focus on the longer-run PSID-based opportunities to follow children from birth into adulthood, and to relate

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NOTE: Portions of this article are drawn from Duncan (2002).

demographic and economic conditions across childhood and adolescence to attainment, behavioral, and health outcomes of children when they are in middle adulthood.

Although the PSID was not conceived as an intergenerational study, design decisions taken prior to its initial wave of data collection have provided analysts with data that are useful for addressing intergenerational questions. By its 25th birthday (in the mid-1990s), the PSID had followed its sample long enough to begin supporting a series of studies linking childhood income and early adult attainments. As the scope of its data on children has expanded, evidence showing that poverty early in life was associated with worse adult health has focused on complementary discoveries in epidemiology and, most recently, developmental neuroscience, which are beginning to shed light on the stress- and immune-function processes that might be producing the observed correlations.

A Little History

The PSID was not conceived as an intergenerational study, nor even as a long-term study. It emerged from Lyndon Johnson's War on Poverty, after the Office of economic Opportunity (OEO) directed the U.S. Bureau of the Census to conduct a nationwide assessment of the extent to which the War on Poverty was affecting people's economic well-being. This Census Bureau study, called the Survey of Economic Opportunity (SEO), completed interviews with about 30,000 households, first in 1966 and again in 1967.

Interest in continuing this survey of economic "trajectories," but also in avoiding the bureaucratic requirements of the Census Bureau, led James D. Smith and his OEO colleagues to approach James Morgan at the Survey Research Center (SRC) about interviewing a nationally representative subsample of approximately 2,000 low-income SEO households over a period of five years. Morgan was initially reluctant to take on this task because the seriously flawed OEO design called for following only low-income households. He argued for the virtues of complete population representation, pointing out, for example, that understanding why nonpoor households fell into poverty was at least as interesting as knowing why poor households climbed out. Morgan won the argument, talking OEO into funding a design in which 2,000 randomly chosen, initially poor OEO households were combined with a fresh cross-section of about 3,000 households from the SRC national sampling frame. When weighted, the combined sample was representative of the entire population of the United States, including nonpoor as well as poor households. But the disproportionately large number of low-income households produces large analysis samples of black and other disadvantaged groups.

The year 1972 proved to be momentous for the PSID. As its original five years were coming to an end, then-President Nixon took the dramatic step of abolishing the OEO virtually overnight. Responsibility for the PSID was transferred to the Assistant Secretary for Planning and Evaluation (ASPE) at the Department of Health, Education, and Welfare (now Health and Human Services). A delightful, but perhaps apocryphal, story has it that the PSID's dossier contained the note "This is a 10-year study," although there had been no such official determination. At any rate, visionary ASPE officials like Larry Orr saw the value of continuing to support the PSID, and an additional five years of ASPE funding kept the PSID

alive until primary responsibility for its funding was taken over by the National Science Foundation (NSF). the NSF's Daniel Newlon was indispensable in simultaneously garnering support for the PSID within NSF and insisting that the study be as responsive as possible to the broader research community. Underscoring his and the study's success, on the occasion of NSF's 50th anniversary in 2000, the PSID was named one of the "Nifty Fifty"—the 50 most important projects in NSF's history.

How the PSID Became an Intergenerational Study

The PSID's value for intergenerational studies of childhood income rests on key design decisions made as the project began. Virtually all longitudinal studies focus on cohorts of individuals—those ages 14–22 in 1979 for the National Longitudinal Survey of Youth (NLSY79); students enrolled in grades 7–12 in the National Longitudinal Study of Adolescent to Adult Health (Add Health); 12th graders in Wisconsin schools in the Wisconsin Longitudinal Study; children born in a single week in 1970 in England, Scotland, and Wales in the 1970 British Cohort Study; and, reaching back in time, "normal" Berkeley children born in late 1928 and early 1929 in the Berkeley Growth Study.[1]

The PSID was conceived as a study of family income dynamics and began in 1968 with a national sample of roughly 5,000 U.S. households. However, it defined its "sample members" as all 18,000 individuals present in those 1968 households plus, as time passed, all individuals born to the original 18,000 sample members during the study period, and then all individuals born to the individuals born to the original 18,000 sample members, and so on. In other words, the PSID has attempted to trace all of the individuals living in or descending from its original households, whether or not they have continued to reside in the same dwelling or with the same people. So when an original sample family separates through, say, divorce, both the adults and, if present, their children, are followed. Most important for intergenerational studies, when children present in the original set of families leave the parental nest, they too are followed into their new households. And, crucially, when children born into PSID families eventually leave home, they too are followed. Whereas other longitudinal studies follow a given sample of persons, the PSID follows a bloodline; everyone born to or added to a household of someone with the original PSID "gene," defined by residence in an interviewed household in 1968, is included.

These rules have many advantages. First, the samples of families and individuals are continuously representative of the U.S. population in terms of all demographic changes other than immigration (Duncan and Hill 1989). Most important for intergenerational studies, they provide a representative sample of children who are observed throughout much or all of their childhood as well as during many of their adult years. Another advantage of following all children living with or born to original PSID sample members is that it provides data on complete sets of siblings, which is particularly useful for the estimation of family–fixed effects models.

1."Normal" was the study's term for children born to white, English-speaking parents who could be regarded as permanent residents of Berkeley and were willing to take their children to the Institute for Child Welfare for the required series of examinations (Jones and Bayley 1941).

Intergenerational studies that rely on certain elements of childhood socioeconomic status—in particular retrospectively recalled parental educational and occupational attainment, as well as family structure—do not need the longitudinal scope of a study like the PSID or Children of the NLSY79. But if the focus is on a child's family income, and the relevant measurement period for family background spans most of childhood and adolescence, then a long-term household panel study is necessary because respondents are typically unable to recall income for more than a couple of years (Bound et al. 1994).[2] And if conditions in utero and in early childhood are to be linked to health and attainments in midlife, cohorts must be followed for more than three decades.

Linking Family Income to Children's Attainments: Early Theory

From the inception of the work relating childhood poverty to child outcomes, economists, sociologists, and psychologists had offered field-specific theoretical frameworks and empirical tests of those frameworks. However, the explosion of research in developmental neuroscience, combined with that field's strong interest in poverty and stress, has opened up new opportunities in developmental theory and for empirical work.

Already in place by the mid-1990s, the two main theoretical frameworks describing these processes were resources and investment, on one hand, and family and environmental stress on the other. Pioneered by Becker (1991), the resources and investment perspective holds that children from poor families lag behind their economically advantaged peers, in part because their parents have less time and money to invest in them. In schematic form:

$$\textit{Economic adversity} -> \textit{less parental investment in children} -> \textit{worse cognitive and noncognitive outcomes} -> \textit{less schooling} -> \textit{lower wage rates}$$

Since this perspective is perhaps the best known, we will not review it here. Instead, we concentrate on stress and other child systems that appear to be affected by early environments.

Family and environmental stress

Economically disadvantaged families experience higher levels of stress in their everyday environments than more affluent families, and these disparities may affect children's development. The family stress model was first developed by Glen Elder to document the influence of economic loss during the Great Depression (Elder 1974). According to this perspective, poor families face significant economic pressure as they struggle to pay bills and purchase important goods and services, and these economic pressures, coupled with other stressful life events that are more prevalent in the lives of poor families, create high levels of psychological distress, including depressive and hostile feelings, in poor parents (Kessler and Cleary 1980; McLeod and Kessler 1990).

[2]If economic resources are to be captured by exogeneous program and policy information (as in the Hoynes et al. 2016 study of food stamps), accurate yearly information on children's residential history is needed.

Psychological distress spills over into marital and coparenting relationships. As couples struggle to make ends meet, their interactions tend to become more hostile and conflicted, and this leads them to withdraw from each other (Brody et al. 1994; Conger and Elder 1994). Parents' psychological distress and conflict, in turn, are linked with parenting practices that are on average more punitive, harsh, inconsistent, and detached, as well as less nurturing, stimulating, and responsive to children's needs (McLoyd 1990):

$$\textit{Economic adversity} -> \textit{less sensitive parenting} -> \textit{worse youth mental health, behavior, and achievement} -> \textit{worse adult outcomes}$$

Methodologically strong studies have not found much evidence for income effects on these processes, however. For example, Morris et al. (2001) examined data from welfare-to-work experiments and found very few impacts on parenting behavior or parent-child relationships. Family stress caused by economic adversity may still matter for longer-run development, but parenting may not be the pathway by which that impact is generated.

A related body of work focuses on the variety of adverse childhood experiences (ACEs) that are linked to low family income during childhood (Centers for Disease Control 2014). Such experiences might include family disruption, parental ill health and substance use, childhood exposure to crime and abuse or neglect. Because these factors are for the most part endogenous to family income they are generally not included in the empirical models of the studies to which we turn next.

Linking Family Income to Children's Attainments: Empirical Research from 1995–2010

Armed with these two models of how economic adversity affects children's development, we turn now to the task of placing PSID research into the large empirical literature that arose in the 1990s. Several early review articles (e.g., Corcoran 1995; Haveman and Wolfe 1995) and books (e.g., Mayer 1997) summarized the voluminous correlational literature linking family income and developmental outcomes in adolescence and early adulthood. Their consensus was that: (1) correlations with parental income varied from one outcome to another; (2) for achievement-related outcomes such as completed schooling and early adult labor market success, the estimated associations with parental income were usually statistically significant (although there was little consensus regarding the size of these associations); and (3) if the confounding effects of unmeasured parental and neighborhood characteristics were not taken into account, even the mostly modest estimates of the effects of parental income might well be upwardly biased. None of the reviewed work had been able to take a whole-childhood look at family income.

Also in the mid-1990s, Duncan and Brooks-Gunn (1997) organized a coordinated analysis by twelve groups of researchers working with ten different developmental datasets, most of which offered at least short-run longitudinal measurement of parental family income as well as some measures of the achievement, behavior, and/or health of individuals at various points in childhood or early adulthood. A common element across the conference papers was a required "replication" analysis in which the same measures—family income, maternal

schooling, family structure—were included in a regression model predicting child and adult outcomes. The results suggested that family income at times had large but somewhat selective associations with children's attainments (Figure 1).

The most important finding was that the childhood stage over which income was measured made a difference. Specifically, family economic conditions in early and middle childhood appeared to be more consistently important for predicting achievement test scores a few years later than were economic conditions during adolescence for predicting test scores taken in adolescence. Experimental evidence from the Negative Income Tax (NIT) experiments of the 1970s and 1980s, and from the welfare reform experiments in the 1990s produced broadly similar short-run patterns (Duncan, Magnuson, and Votruba-Drzal 2014). In particular, none of the achievement studies using exclusively adolescence-based income measures found large effects. In contrast, all the studies in which income was measured during early childhood found substantial associations with early measures of achievement. Left unanswered in these and all other analyses is the importance for adolescent and early-adult outcomes of family economic conditions in the earliest stages of childhood.

Extending a preliminary analysis in the summary chapter of the Duncan and Brooks-Gunn book, Duncan et al. (1998) provided the first analysis to include family income throughout childhood. To explore the stage-specific patterns suggested by the Duncan and Brooks-Gunn (1997) studies, they broke up the PSID's 15-year time series of childhood income into three equal stages and included all three, plus demographic controls, in their predictor list. Although correlational, estimates of, say, effects of early childhood income controlling for income in middle-childhood and adolescence likely suffer from less omitted variable bias than if the regression controls were limited to demographic measures other than income.

More specifically, Duncan et al. (1998) drew a sample consisting of 1,323 children born between 1967 and 1973 who were observed in PSID families for the entire period between birth and ages 20–25. Outcomes included years of children's completed schooling, high school completion and the timing of a possible nonmarital birth (for females only). They estimated both linear and nonlinear functions for income, with the most interesting results coming from the models using a piecewise linear (spline) function split at $20,000 (in 1993 dollars) and dummy variables that imposed no functional form on the income relationship.

The results showed more consistent income associations with school attainment than with nonmarital childbearing, as well as consistently stronger associations for increments to low as opposed to high income levels. But the regressions also revealed that the timing of economic deprivation mattered, with income early in life having the most consistent associations with the attainment outcomes (Figure 2). For example, after controlling for income in other stages and other family conditions, children in families with birth-to-age-five annual incomes between $15,000 and $25,000 averaged two-thirds of a year more schooling—about one-third of a standard deviation—than children in families with an income of less than $15,000. In contrast, increments to income in middle childhood and adolescence were not significant predictors of the schooling outcomes. The potentially unique importance of early childhood economic conditions was clearly the most intriguing result of the article.

Duncan, Ziol-Guest, and Kalil (2010) extended this stage-specific work by focusing on a broader range of outcomes, including, for the first time, health conditions in adulthood, and applying an updated definition of the “early childhood years” that included the prenatal period. Their study was based on 1,589 children born between 1968 and 1975, and used information on their adult outcomes between ages 25 and 37. Health conditions were assessed in 2006, when these individuals were between the ages of 30 and 37.

As in Duncan et al. (1998), Duncan, Ziol-Guest, and Kalil (2010) measured income across several distinct periods of childhood, distinguishing income early in life (prenatal through 5th year) from income in middle childhood and adolescence. Regressions related these outcomes to stage-specific childhood income plus a host of demographic control variables. To allow for nonlinear income effects, spline functions split income at $25,000 (in 2010 dollars). Concentrating on the estimated slopes for low-income segments, increases in early childhood income were associated with higher adult earnings and work hours as well as reductions in food stamp receipt (Figure 3). Income increases later in childhood did not show significant associations with either earnings or work hours, although income increases in both childhood periods were associated with lower food stamp receipt. As in Duncan et al. (1998), income in early childhood failed to correlate significantly with either arrests or incarceration for males, or with nonmarital births for females. These findings reinforced the existing evidence on the possible role of early life economic circumstances for adult attainments but not behaviors.

A closer look at the patterns of attainment-related results in Duncan, Ziol-Guest, and Kalil (2010) produced some surprises. First, given the links between early life income and completed schooling found in earlier studies, it was odd that early life income was not associated with completed schooling assessed when respondents were in their late twenties or early thirties. Duncan, Ziol-Guest, and Kalil (2010) investigated this issue and found that income very early in life was indeed associated with “on-time” schooling, defined in various ways to reflect schooling completed in very early adulthood. But a considerable amount of post-secondary schooling for low-income adults is completed when they are in their twenties, and these increments to completed schooling were essentially independent of patterns of childhood income. Taking these increments into account weakened considerably the associations between childhood income and eventual educational attainment.

Extending the Conceptual Models

Recent work with the PSID and other developmental data has led to major reconsiderations of the investment and family stress models. In particular, the family stress model’s narrow focus on environmental conditions and parental mental health and parenting has been broadened by neurobiological evidence on the importance of maintaining tolerable levels of stress for both parents and children, and by a cognitive psychological perspective on links among stress, information processing, and decision-making (Mullainathan and Shafir 2013). Increasingly sophisticated empirical studies suggest linkages between income support and maternal stress (W. Evans and Garthwaite 2014).

Complementary studies in psychology and social epidemiology show that both in utero environments and early childhood experiences can have long-run impacts on adult physical and mental health (Sapolsky 2004; Strauss 1997). The influential “fetal origins hypothesis” posits a programming process whereby stimulants and insults during the prenatal period have long-lasting implications for physiology and disease risk (Barker et al. 2002). Later research, including in economics, has linked stressors and environmental toxins experienced during pregnancy, including naturally occurring phenomena such as famines, flu epidemics, and pollution, to metabolic disorders and heart disease in later life (Almond and Currie 2011).

Chronic stress from growing up poor could also play a role in neurocognitive or biological dysregulation (Conger et al. 2002; Conger and Dogan 2007). In a correlational analysis, G. Evans and Schamberg (2009) showed that childhood poverty is associated with chronically elevated physiological stress (measured in this study by allostatic load, a summary index of six risk factors for physiological wear and tear on the body) in early adolescence, which is in turn correlated with worse performance of working memory (a key determinant of cognitive function) in late adolescence. A more telling analysis of possible links between early economic disadvantage and stress-related adult outcomes is the Hoynes, Schanzenbach, and Almond (2016) PSID-based study of food stamps and adult levels of a metabolic index thought to be strongly correlated with allostatic load.

Theory also suggests that early childhood income and adult health may be linked through immune-related processes (Ziol-Guest et al. 2012). First, as noted previously, the “fetal origins hypothesis” posits a biological programming process whereby exposures and insults during the prenatal period have long-lasting implications for physiology and disease risk (Strauss 1997). Second, it is now well documented that psychological stress has an impact on cellular and humoral immune processes (Mathews and Janusek 2011; Segerstrom and Miller 2004), and chronic stress from growing up poor may also play a role in dysregulation across multiple physiological systems, with the effects persisting (or possibly compounding) into adulthood (G. Evans and Schamberg 2009). Thus, childhood poverty may actually calibrate immune system responsivity, dysregulating inflammatory processes and resulting in a shift toward proinflammatory states (Hänsel et al. 2010).

Ziol-Guest et al. (2012) used the PSID to investigate whether low income during early childhood, relative to other stages of childhood, is associated with immune-mediated, chronic health conditions in adulthood, as well as the extent to which these adult health conditions might explain associations between early childhood income and adult productivity. The outcomes examined in that analysis (hypertension, arthritis) are typically considered diseases of old age, but the PSID data show that adults 30–41 years of age who were poor in early childhood reported hypertension about 19 percent of the time across three adult interviews, and arthritis about 11 percent of the time (Figure 3). These rates are approximately twice those reported by adults who were not poor in early childhood. Moreover, these relationships—particularly early onset arthritis—partially account for the associations between early childhood poverty and adult productivity, as measured by adult work hours and earnings.

The specificity of the associations matters a great deal. First, they are much stronger for income in the first years of life (between the prenatal year and age two) than later in childhood. This is consistent with the hypothesis that these early years represent a sensitive period during which social processes become embedded in biology, and that epigenetic modifications may be responsible for these associations (Hertzman and Boyce 2010). Second, the associations are much stronger at lower, rather than higher, income levels. This is in keeping with the findings of a number of recent studies showing that income plays a more potent role in later life outcomes for those at the bottom end of the income distribution (Duncan, Ziol-Guest, and Kalil 2010). The Ziol-Guest et al. (2012) results support the hypothesis that inflammatory changes constitute a core biological process that initiates and mediates associations between early childhood poverty and adult disease. Of course, the PSID can only suggest that these processes might be at work. Direct measurements of immune parameters early in life are clearly needed for a more definitive judgment.

A second, and related, finding is that while early childhood income was associated with annual earnings, it was not associated with hourly earnings (wage rates). Earnings are, of course, the product of work hours and the hourly wage rate, so this combination of results indicates that the "action" for the early childhood income/adult earning linkage may lie in an ability to sustain part- or full-time employment during prime adult years rather than in earning more for every hour worked. Since neither mid-adult schooling nor wage rates are related to childhood income, the standard Beckerian story of family human capital investment presented earlier may be relatively unimportant in explaining the lifelong impacts of early economic adversity.

But if neither the human capital (more schooling and higher wage rates) nor the behavioral (less crime and nonmarital births) outcomes account for links between early life income and adult earnings, what does? Consistent with the "early origins" work in social epidemiology and neuroscience, it appears that early life income has long-term effects on work-limiting health conditions. The operational pathway, in other words, may be:

$$\textit{Early economic adversity} -> \textit{dysregulation of child stress and immune function} -> \textit{worse adult human capital and health} -> \textit{worse adult labor market outcomes}$$

Emerging adult health problems (in the mid- to late-thirties) were explored briefly in Duncan, Ziol-Guest, and Kalil (2010) and in greater depth in Ziol-Guest et al. (2012). Although increments to early childhood income did not appear to correlate with self-rated overall adult health or diabetes, Ziol-Guest et al. (2012) found that increments to low income early in life were associated with reductions in the likelihood of hypertension, early onset arthritis, and reports of health-related work limitations; as well as reductions in the likelihood of obesity (Ziol-Guest, Duncan, and Kalil 2009). As explained below, corroborating evidence comes from the Hoynes, Schanzenbach, and Almond (2016) analysis of the impacts on cardiovascular health of early life exposure to the food stamp program. To better understand the early childhood income/adult health relationships estimated with the PSID, it is important to place emerging ideas from the field of developmental neuroscience in the broader context of developmental models of family effects.

Does Correlation Approximate Causation?

Given the nonexperimental nature of the estimates of income “effects” that we have reviewed, it is useful to compare them with evidence from experimental and quasi-experimental studies. Because we focus on links between childhood income and outcomes in early adulthood, the number of causal estimates is low. Two studies explore the causal impact of income during adolescence and completed schooling, whereas one looks at income across childhood and adult health.

As a PSID-based reference point for completed schooling outcomes, Duncan, Kalil, and Ziol-Guest (2017) relate completed schooling by age 25 to childhood income measured between ages 10 and 15. They find that a one-unit (approximately three-fold) increase in log family income is associated with a .63 year increase in completed schooling. The 95 percent confidence interval around that point estimate ranges from .52 to .74. Drawing data from the New Jersey Negative Income experiment, Mallar (1977) estimated that the 50-percent increase in family income caused by the NIT treatment was associated with between one-third and one-half more years of schooling. Assuming a 50 percent increase in income, the Duncan, Kalil, and Ziol-Guest (2017) .63 coefficient and its standard error would have predicted education increases of between .21 and .31 years, which is somewhat below Mallar’s results.

Akee et al. (2010) used revenue for tribal members generated by the opening of a casino to estimate that a $3,900 annual income increase (on an average base income of $20,919) caused statistically insignificant .38- and .12-year increases in educational attainment for the two cohorts studied. Our .629 coefficient would have predicted that the income increase would have led to an attainment increase ranging between .07 to .58 years. However, for families poor at least once prior to the casino transfers, the increased schooling was between .45 and 1.13 years, depending on the cohort, with the latter being much higher than the Duncan, Kalil, and Ziol-Guest (2017) coefficient would have predicted. For the never-poor in the Akee et al. (2010) study, the change was a statistically insignificant .17-year decrease, which is smaller than we would have predicted. these back-of-the-envelope calculations suggest that the PSID adolescent income “effects” on completed schooling are, if anything, conservative.

Turning to health outcomes, Hoynes, Schanzenbach, and Almond (2016) investigated whether access to food stamps in childhood reduces the incidence of metabolic syndrome decades later. They find a remarkable graded relationship between the timing of the introduction of food stamps relative to a child’s age, with children conceived in or born into counties in which food stamps were available scoring about .40 standard deviations lower on the metabolic syndrome index in their late 20s and 30s than children conceived in or born in counties without food stamps.

Extending the analysis in Ziol-Guest et al. (2012), we estimated a model in which a metabolic index score measured between ages 30 and 39 is related to income averaged over three periods: the prenatal year to age 2, ages 3–5, and ages 6–15. As in Hoynes et al., we find an age-graded relationship for increments to low income, with much larger coefficients

for the early life segments than for ages 6–15. The corresponding coefficient in the Hoynes et al. article is between 1.5 and 3.0 times larger than the coefficient estimated in the Ziol-Guest et al. (2012) extension.[3] Overall, this look at the schooling and health analyses of income effects reveals no evidence of worrisome upward biases in estimates based on nonexperimental analyses that include extensive controls.

Conclusion

The newest evidence emerging from the PSID points to an important role for child stress and possibly immune function in linking early childhood economic conditions to outcomes (both economic and health) in adulthood. Adverse childhood economic conditions may affect children's stress and immune functioning directly, through a biological programming process. These new results are consistent with the hypothesis that the early years represent a sensitive period during which social processes become embedded in biology, and that epigenetic modifications may be responsible for some of these associations. This evidence suggests a need to reconsider the theoretical model that has predominated in the literature, which focuses on investment and parenting behavior.

The long-run effects of these processes are only now being identified in the PSID respondents. As the sample ages, however, the increasing prevalence and diversity of poor health conditions will allow for greater exploration of this hypothesis. Direct measurement of early childhood stress and immune parameters is another research priority, although not one well suited to a population study like the PSID.

Biography

Greg Duncan is a distinguished professor in the School of Education at the University of California, Irvine. Duncan spent the first two decades of his career at the University of Michigan working on, and ultimately directing, the Panel Study of Income Dynamics (PSID) data collection project.

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References

Akee Randall K., Copeland William E., Keeler Gordon, Angold Adrian, and Jane Costello E. 2010. Parents' incomes an children's outcomes: A quasi-experiment using transfer payments from casino profits. American Economic Journal: Applied Economics 2 (1): 86–115. [PubMed: 20582231]

Almond Douglas, and Currie Janet. 2011. Killing me softly: The fetal origins hypothesis. Journal of Economic Perspectives 25 (3): 153–72. [PubMed: 25152565]

[3]Details are available upon request.

Barker David J. P., Eriksson Johan G., Tom Forsén, and Clive Osmond. 2002. Fetal origins of adult disease: Strength of effects and biological basis. International Journal of Epidemiology 31 (6): 1235–1239. [PubMed: 12540728]

Becker Gary S. 1991. A treatise on the family. Cambridge, MA: Harvard University Press.

Bound John, Brown Charles, Duncan Greg J., and Rodgers Willard L.. 1994. Evidence on the validity of cross-sectional and longitudinal labor market data. Journal of Labor Economics 12 (3): 345–68.

Brody Gene H., Stoneman Zolinda, Flor Douglas, Chris McCrary, Lorraine Hastings, and Conyers Olive. 1994. Financial resources, parent psychological functioning, parent co-caregiving, and early adolescent competence in rural two-parent African-American families. Child Development 65 (2): 590–605. [PubMed: 8013241]

Centers for Disease Control. 2014. The Adverse Childhood Experiences (ACE) Study. Washington, DC: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control, Division of Violence Prevention.

Conger Rand D., and Dogan Shannon J.. 2007. Social class and socialization in families. In Handbook of socialization theory and research, eds. Grusec JE and Hastings PD, 433–60. New York, NY: Guilford Press.

Conger Rand D., and Elder Glen H. Jr. 1994. Families in troubled times: Adapting to change in rural America. New York, NY: Aldine de Gruyter.

Conger Rand D., Lora Ebert Wallace, Yumei Sun, Simons Ronald L., McLoyd Vonnie C., and Brody Gene H.. 2002. Economic pressure in African American families: A replication and extension of the family stress model. Developmental Psychology 38:179–93. [PubMed: 11881755]

Corcoran Mary. 1995. Rags to rags: Poverty and Mobility in the United States. Annual Review of Sociology 21 (1): 237–67

Duncan Greg J. 2002. The PSID and me. In Landmark studies of the 20th century in the US, eds. Erin Phelps, Furstenberg Frank F. Jr., and Anne Colby, 133–63. New York, NY: Russell Sage Foundation.

Duncan Greg J., and Brooks-Gunn Jeanne, eds. 1997. Consequences of growing up poor. New York, NY: Russell Sage Foundation.

Duncan Greg J., Brooks-Gunn Jeanne, Wei-Jun. Yeung, and Judith Smith. 1998. How much does childhood poverty affect the life changes of children? American Sociological Review 63:406–23.

Duncan Greg J., and Hill Daniel H.. 1989. Assessing the quality of household panel data: The case of the Panel Study of Income Dynamics. Journal of Business and Economic Statistics 7 (4): 441–52.

Duncan Greg J., Kalil Ariel, and Ziol-Guest Kathleen M.. 2017. Increasing inequality in parent incomes and children's schooling. Demography 54 (5): 1603–1626. [PubMed: 28766113]

Duncan Greg J., Magnuson Katherine, and Votruba-Drzal Elizabeth. 2014. Boosting family income to promote child development. The Future of Children 24 (1): 99–120. [PubMed: 25518705]

Duncan Greg J., Magnuson Katherine, and Votruba-Drzal Elizabeth. 2017. Moving beyond correlations in assessing the consequences of poverty. Annual Review of Psychology 68:413–34.

Duncan Greg J., Ziol-Guest Kathleen M., and Ariel Kalil. 2010. Early-childhood poverty and adult attainment, behavior, and health. Child Development 81 (1): 306–25. [PubMed: 20331669]

Elder Glen H. 1974. Children of the Great Depression. Chicago, IL: University of Chicago Press.

Evans William N., and Garthwaite Craig L.. 2014. Giving mom a break: the impact of higher EITC payments on maternal health. American Economic Journal: Economic Policy, American Economic Association 6 (2): 258–90.

Evans Gary W., and Schamberg Michelle A.. 2009. Childhood poverty, chronic stress, and adult working memory. Proceedings of the National Academy of Sciences 106:6545–6549.

Hänsel Alexander, Hong Suzi, Camara Rafael JA, and Von Kaenel Roland. 2010. Inflammation as a psychophysiological biomarker in chronic psychosocial stress. Neuroscience and Biobehavioral Reviews 35:115–21. [PubMed: 20026349]

Haveman Robert, and Wolfe Bobbi. 1995. The determinants of children's attainments: A review of methods and findings. Journal of Economic Literature 33 (4): 1829–1878.

Clyde Hertzman, and Boyce W. Thomas. 2010. How experience gets under the skin to create gradients in developmental health. Annual Review of Public Health 31:329–47.

Hoynes Hilary, Schanzenbach Diane W., and Almond Douglass. 2016. Long-run impacts of childhood access to the safety net. American Economic Review 106 (4): 903–34.

Jones Harold E., and Bayley Nancy. 1941. The Berkeley Growth Study. Child Development 12 (2): 167–73.

Kessler Ronald C., and Cleary Paul D.. 1980. Social class and psychological distress. American Sociological Review 45 (3): 463–78. [PubMed: 7406359]

Mallar Charles D. 1977. The educational and labour-supply responses of young adults in experimental families. In The New Jersey Income-Maintenance Experiment, volume II: Labour-supply responses, eds. Watts Harold W. and Albert Rees, 163–84. New York, NY: Academic Press.

Mathews Herbert L., and Linda Witek Janusek. 2011. Epigenetics and psychoneuroimmunology: Mechanisms and models. Brain, Behavior, and Immunity 25:25–39.

Mayer Susan E. 1997. What money can't buy: Family income and children's life chances. Cambridge, MA: Harvard University Press.

McLeod Jane D., and Kessler Ronald C.. 1990. Socioeconomic status differences in vulnerability to undesirable life events. Journal of Health and Social Behavior 31 (2): 162–72. [PubMed: 2102495]

McLoyd Vonnie C. 1990. The impact of economic hardship on black families and children: Psychological distress, parenting, and socioemotional development. Child Development 61 (2): 311–46. [PubMed: 2188806]

Morris Pamela, Huston Aletha, Duncan Greg, Crosby Danielle, and Bos Johannes. 2001. How welfare and work policies affect children: A synthesis of research. New York, NY: Manpower Research Demonstration Corporation.

Mullainathan Sendhil, and Shafir Eldar. 2013. Scarcity: Why having too little means so much. New York, NY: Times Books, Henry Holt and Company.

Phelps Erin, Furstenberg Frank F., and Ann Colby, eds. 2002. Landmark studies of the 20th century in the US. New York, NY: Russell Sage Foundation.

Sapolsky Robert 2004. Social status and health in humans and other animals. Annual Review of Anthropology 33:393–418.

Segerstrom Suzanne C., and Miller Gregory E.. 2004. Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. Psychological Bulletin 130:601–30. [PubMed: 15250815]

Strauss Richard S. 1997. Effects of the intrauterine environment on childhood growth. British Medical Bulletin 53:81–95. [PubMed: 9158286]

Ziol-Guest Kathleen M., Duncan Greg J., and Kalil Ariel. 2009. Early childhood poverty and adult body mass index. American Journal of Public Health 99 (3): 527–32. [PubMed: 19106427]

Ziol-Guest Kathleen M., Duncan Greg J., Kalil Ariel, and Boyce W. Thomas. 2012. Early childhood poverty, immune-mediated disease processes, and adult productivity. Proceedings of the National Academy of Sciences 109 (Supplement 2): 17289–17293.

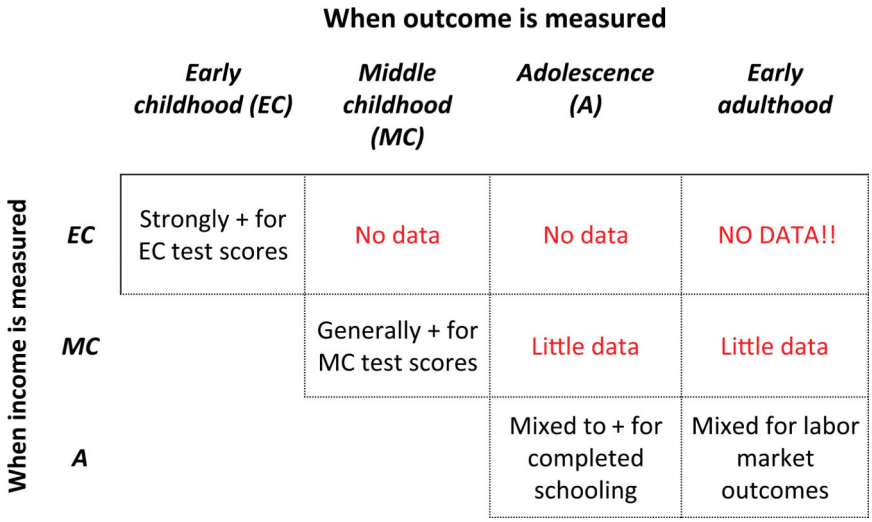

FIGURE 1. General Coefficient Patterns in Twelve Studies
SOURCE: Duncan and Brooks-Gunn (1997), Table 18.1.

When income is measured	Schooling years and HS degree	Hazard of non-marital birth
Age 0-5	Always +	*Null*
Age 6-10	*Null*	*Null*
Age 11-15	Usually +	Usually -

FIGURE 2. Coefficient Patterns for Early Adult Outcomes

SOURCE: Duncan et al. (1998).

NOTE: Regressions include control for race, gender, number of siblings, mother's completed schooling and age at the time of the child's birth, region, and stage-specific measures of family structure, maternal employment, and residential mobility across childhood.

Adult outcome	Age when income is measured	
	Prenatal to age 2 or 0-5	Age 6-15 or 10-15
"On time" education	+	often +
Education age 30+	ns	ns
Earnings, age 25-37	+	ns
Work hours, age 25-37	+	ns
Wage rate, age 25-37	ns	ns
Food stamp receipt	-	-
Work limitations, early 30s	-	ns
Arthritis, early 30s	-	+
Hypertension, early 30s	-	ns
Arrests, jail and OWB	ns	ns

FIGURE 3. Associations Between Income Increases and Adult Outcomes, by Childhood Stage, Slope for Low Income

SOURCE: Duncan, Ziol-Guest, and Kalil (2010); Ziol-Guest et al. (2012).