

# The Great Recession, genetic sensitivity, and maternal harsh parenting

Dohoon Lee<sup>a,1</sup>, Jeanne Brooks-Gunn<sup>b</sup>, Sara S. McLanahan<sup>c,1</sup>, Daniel Notterman<sup>d</sup>, and Irwin Garfinkel<sup>e,1</sup>

<sup>a</sup>Department of Sociology, New York University, New York, NY 10012; <sup>b</sup>Teachers College and the College of Physicians and Surgeons, Columbia University, New York, NY 10027; <sup>c</sup>Center for Research on Child Wellbeing and Office of Population Research, Princeton University, Princeton, NJ 08544; <sup>d</sup>College of Medicine, Pennsylvania State University, Hershey, PA 17033; and <sup>e</sup>School of Social Work, Columbia University, New York, NY 10027

Contributed by Sara S. McLanahan, June 30, 2013 (sent for review May 2, 2013)

**Using data from the Fragile Families and Child Wellbeing Study, this study examined the effects of the Great Recession on maternal harsh parenting. We found that changes in macroeconomic conditions, rather than current conditions, affected harsh parenting, that declines in macroeconomic conditions had a stronger impact on harsh parenting than improvements in conditions, and that mothers' responses to adverse economic conditions were moderated by the *DRD2* *Taq1A* genotype. We found no evidence of a moderating effect for two other, less well-studied SNPs from the *DRD4* and *DAT1* genes.**

The only thing we have to fear is fear itself—nameless, unreasoning, unjustified terror.

President Franklin Delano Roosevelt's opening remarks in his first inaugural address

The “Great Recession” (2007–2009) entailed the largest contraction in economic output in the United States since the Great Depression. In this article, we examine the effects of the Great Recession on harsh parenting, using data from the Fragile Families and Child Wellbeing Study (FFS). We extended prior work by examining the effects of changes in macroeconomic conditions as well as current economic conditions, by examining both declines and improvements in economic conditions, and by determining whether mothers' responses to economic conditions were moderated by the *DRD2* gene.

The family stress model, developed from studies of the Great Depression and the Iowa Farm Crisis, posits that economic hardship leads to stress, which, in turn, leads to deterioration of parental relationships, mental health, parenting quality, and, ultimately, child wellbeing (1, 2). Associations consistent with the family stress model have been found in many other small samples throughout the United States and Europe. Whereas most studies in this literature suffer from omitted variables bias—the effects of economic hardship are identified off differences between families who do and do not experience large economic losses—more recent studies have reported similar findings using more exogenous measures of economic conditions, e.g., plant closings (3, 4), changes in income transfer policy (5, 6), and unemployment rates (7, 8). We also used more exogenous measures and hypothesized that higher unemployment rates were associated with higher levels of harsh parenting.

Stress may result not only from the actual experience of adversity but also from uncertainty and the anticipation of adversity. Mother monkeys parent less well—and their offspring do less well—when foraging in poor environments compared with rich environments. However, both mothers and offspring do worst when poor and rich environments are varied randomly, suggesting that uncertainty or insecurity may be more stressful than the actual experience of adversity (9, 10). Among humans, anticipation of adverse events with high salience is thought to elicit stress or anxiety as well as changes in decision making, risk aversion, and aggression (11–14). Although the unemployment rate is a good indicator of the probability that an individual will

be unemployed, it may not fully capture the stress associated with the anticipation of economic adversity. Hedonic adaptation theory (15) suggests that the emotions elicited by any level of the unemployment rate depend upon the previous level. For example, an unemployment rate of 8% will elicit hope and confidence if the previous rate was 10%, but fear and anxiety if the previous level was 6%. Similarly, an 8% rate will elicit greater fear and anxiety if the previous rate was 4% rather than 7% because the size (or rate) of the change is much larger for the former than the latter. Finally, research in behavioral economics demonstrates that people's responses to losses are greater than their responses to gains of equal size (16, 17). Drawing on these ideas from the behavioral sciences, we hypothesized that harsh parenting is associated with both the direction and the rate of change in macroeconomic conditions and that declines in economic conditions have larger effects on parenting than improvements in conditions.

Researchers have long observed that individuals vary in their response to events such as job loss or worsening economic conditions. Early psychological studies attributed such differences to temperament or behavioral reactivity (18). More recently, the advent of molecular genetics has allowed researchers to examine genotypic variance to see whether individuals with certain genetic profiles reacted more negatively than others when exposed to adverse environments (19). The concept of genetic vulnerability has recently been complemented by the concept of genetic sensitivity, which posits that individuals with certain genetic profiles have worse outcomes in unfavorable social environments but better outcomes in favorable environments (20–24). A common criticism of this literature is that people may select their environments, meaning that gene-by-environment interactions ( $G \times E$ ) may be due to gene-by-environment correlations ( $rGE$ ) or gene-by-gene interactions ( $G \times G$ ) (25). The Great Recession and changes in macroeconomic conditions more generally are beyond the control of a particular individual and thereby provide a more exogenous measure of the environment.

The dopaminergic system helps regulate emotional and behavioral responses to environmental threats and rewards. Low efficiency in this neural system has been shown to result in aggression, impulsivity, and attention deficit hyperactivity disorder (26–29). Of particular interest is the *Taq1A* single nucleotide polymorphism (SNP) (rs1800497), which is located in the 3' UTR of the *DRD2* gene. The T allele of this SNP eliminates the *Taq1* site by replacing a cytosine base with a thymine base. Although far from conclusive, research to date suggests that individuals possessing at least one T allele have fewer D2 dopamine

Author contributions: D.L., J.B.-G., S.S.M., D.N., and I.G. designed research; D.L., J.B.-G., S.S.M., D.N., and I.G. performed research; D.L. and D.N. analyzed data; and D.L., J.B.-G., S.S.M., D.N., and I.G. wrote the paper.

The authors declare no conflict of interest.

Freely available online through the PNAS open access option.

<sup>1</sup>To whom correspondence may be addressed. E-mail: mclanaha@princeton.edu, dohoon.lee@nyu.edu, or ig3@columbia.edu.

This article contains supporting information online at [www.pnas.org/lookup/suppl/doi:10.1073/pnas.1312398110/-DCSupplemental](http://www.pnas.org/lookup/suppl/doi:10.1073/pnas.1312398110/-DCSupplemental).

receptors in their brains and are more susceptible to reactive aggression (26, 30) than individuals with the CC alleles. Based on these findings, we hypothesized that mothers' harsh parenting behavior as a response to adverse macroeconomic conditions is moderated by their *DRD2 Taq1A* genotype.

The FFS is a population-based, longitudinal birth cohort study of 4,898 children born in 20 large American cities between 1998 and 2000 (31). By design, three fourths of the children in the study were born to unmarried mothers and one fourth to married mothers. Mothers were interviewed at birth and reinterviewed when the child was ~1, 3, 5, and 9 y old. Data on harsh parenting were measured when the child was 3, 5, and 9 y old. In year 9, saliva DNA samples were collected from mothers using the Oragene-DNA sample collection kit (DNA Genotek) (details are available in *SI Materials and Methods*). The analytic sample with valid observations on study variables contained 2,612 mothers (Table S1).

Maternal harsh parenting was measured using 10 items from the Conflict Tactics Scale (32). Five items measured psychological harsh parenting, and five measured corporal punishment. Subscales were combined to construct a composite measure of maternal harsh parenting (Cronbach's alpha = 0.75). Mothers' risk of exposure to unemployment was measured by the city-level unemployment rates (UR). Anticipation of unemployment or economic adversity was measured directly by the national consumer sentiment index (CSI) and indirectly by the rate and direction of change in UR and CSI. The national CSI is salient for predicting stress because of the ubiquity of television and other national media. Further, because consumer sentiment is a leading indicator and unemployment rate is a lagging indicator of changes in the business cycle, we included both indicators in our model. Levels of UR and CSI were measured at the time of interview and merged with the FFS data. The rate and direction of change in UR and CSI were measured by computing the difference between the value at the time of interview and the 3-mo lagged value divided by the lagged value, and multiplied by 100.

Ordinary least squares regression was used to estimate the association of maternal harsh parenting with macroeconomic conditions, *DRD2* genotype, and their interactions. To test the hypothesis that declines in macroeconomic conditions have a stronger impact on harsh parenting than improvements, we used a spline function, which allowed positive and negative changes in each macroeconomic indicator to have their own slopes and converge at zero. We exploited multiyear variations in UR and CSI by pooling the FFS data from years 3, 5, and 9 ( $n = 6,492$  person-years). SEs were adjusted for within-individual correlation. All models controlled for age, race/ethnicity, immigration status, educational attainment, poverty status, family structure,

child sex, and child age (in months) at the time of interview. We also controlled for interview wave, quarter of interview month, and city of residence (analysis details are provided in *Materials and Methods*).

## Results

Table 1 presents our main results. Column 1 shows the main effects of levels of and changes in macroeconomic conditions (Table S2). Deteriorations in macroeconomic conditions, indicated by positive changes in UR and negative changes in CSI, were associated with increases in maternal harsh parenting. A ten percent increase in UR led to a 1.6-unit increase in the number of harsh parenting behaviors ( $b = 0.16$ , 95% CI = [0.05, 0.27],  $P = 0.005$ ). Although not statistically significant, a 10% decrease in the CSI also led to a 1.3-unit increase in harsh parenting ( $b = 0.13$ , 95% CI = [-0.10, 0.35],  $P = 0.27$ ). By contrast, improvements in macroeconomic conditions, indicated by negative changes in UR and positive changes in CSI, were associated with much smaller, statistically insignificant changes in harsh parenting ( $P = 0.83$  and  $P = 0.43$ , respectively). Finally, the levels of UR and CSI affected harsh parenting, but not in the expected direction ( $b = -0.70$ , 95% CI = [-1.34, -0.06],  $P = 0.03$  and  $b = 0.18$ , 95% CI = [0.00, 0.36],  $P = 0.046$ , respectively). As discussed below, however, the level effects were not robust to model specification. In sum, harsh parenting was not positively associated with high levels of unemployment but rather with increases in the unemployment rate and declines in consumer sentiment, suggesting that the anticipation of adversity was a more important determinant of harsh parenting than actual exposure.

Columns 2 and 3 of Table 1 present the full  $G \times E$  interaction model to test whether the association between macroeconomic indicators and harsh parenting varied by mothers' *DRD2 Taq1A* genotype. The effect of deteriorating macroeconomic conditions was more pronounced for mothers carrying a T allele than for mothers carrying the CC allele. For the T allele carriers, increases in the UR and declines in the CSI strongly increased harsh parenting. A 10% increase in UR and decrease in CSI led to a 2.3- and 4-unit increase in harsh parenting ( $b = 0.23$ , 95% CI = [0.07, 0.38],  $P = 0.005$  and  $b = 0.40$ , 95% CI = [0.09, 0.72],  $P = 0.01$ , respectively). For the CC allele carriers, however, these changes had inconsistent and insignificant effects. A 10% increase in UR and decrease in CSI were associated with a 0.8 increase and 1.8 decrease in harsh parenting ( $b = 0.08$ , 95% CI = [-0.07, 0.24],  $P = 0.30$  and  $b = -0.18$ , 95% CI = [-0.50, 0.14],  $P = 0.27$ , respectively). As a result, the effect of deteriorating macroeconomic conditions differed significantly between the T and CC allele carriers ( $F(2, 2611) = 3.36$ ,  $P = 0.03$ ).

**Table 1. The association among macroeconomic conditions, the *DRD2 Taq1A* polymorphism, and harsh parenting**

Variable	All ( $b$ /95% CI)	T ( $b$ /95% CI)	CC ( $b$ /95% CI)
% change in local UR			
Improving	0.03/[-0.20, 0.25]	-0.21/[-0.51, 0.09]	0.25/[-0.09, 0.60]
Deteriorating	0.16**/[0.05, 0.27]	0.23**/[0.07, 0.38]	0.08/[-0.07, 0.24]
% change in CSI			
Deteriorating	0.13/[-0.10, 0.35]	0.40*/[0.09, 0.72]	-0.18/[-0.50, 0.14]
Improving	-0.05/[-0.19, 0.08]	-0.05/[-0.24, 0.13]	-0.05/[-0.24, 0.15]
Level of local UR	-0.70*/[-1.34, -0.06]	-0.57/[-1.43, 0.28]	-0.75/[-1.71, 0.21]
Level of CSI	0.18*/[0.00, 0.36]	0.31*/[0.07, 0.55]	0.02/[-0.24, 0.28]
Wave dummies?	Yes	Yes	Yes
Season dummies?	Yes	Yes	Yes
City dummies?	Yes	Yes	Yes
Covariates?	Yes	Yes	Yes
<i>N</i>	6,492	3,498	2,994

\*\* $P < 0.01$ , \* $P < 0.05$  (two-tail tests).

By contrast, the effect of improving macroeconomic conditions on harsh parenting was less pronounced. For the T allele carriers, improvements in UR and CSI reduced harsh parenting, but the coefficients were smaller and not statistically significant ( $b = -0.21$ , 95% CI =  $[-0.51, 0.09]$ ,  $P = 0.17$  and  $b = -0.05$ , 95% CI =  $[-0.24, 0.13]$ ,  $P = 0.57$ , respectively). For the CC allele carriers, improvements in UR and CSI were associated with either increases or decreases in harsh parenting, which were inconsistent and insignificant ( $P = 0.15$  and  $P = 0.63$ , respectively). Overall, these  $G \times E$  results lend support to our hypothesis. The fear of economic adversity, as indicated by deteriorating macroeconomic conditions, played a more critical role in parenting behavior for mothers with a T allele of *DRD2 Taq1A* polymorphism than for mothers with the CC allele. We found no evidence of a moderating effect for two other, less well-studied SNPs from the *DRD4* and *DAT1* genes.

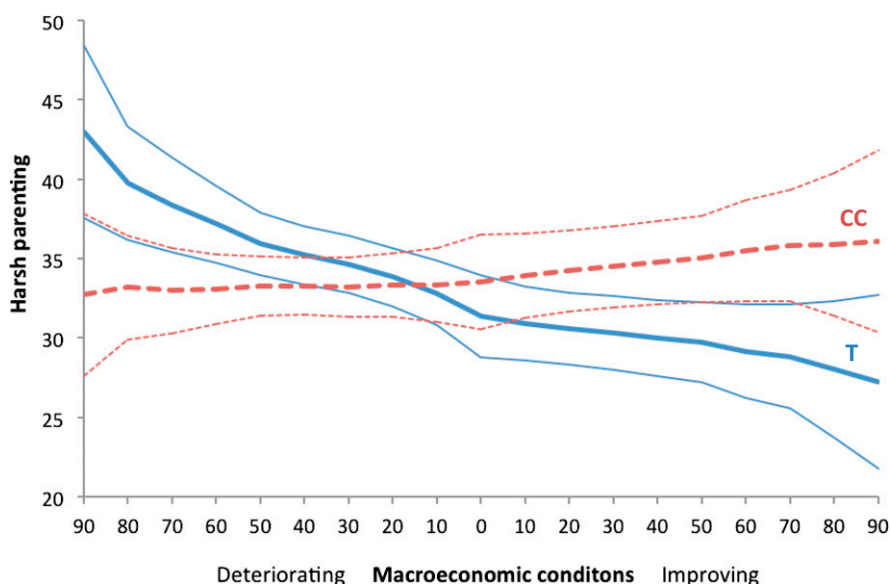
Because the results above were obtained by estimating the effects of changes in UR and CSI in the same model, it is difficult to interpret the model without considering all of the coefficients at once. In some situations, changes in UR and CSI could give conflicting signals about changes in the economy. To further examine the implications of our  $G \times E$  model for genetic sensitivity, we used the coefficients in columns 2 and 3 of Table 1 to simulate the responses of mothers with a T allele and mothers with the CC allele to unambiguous deteriorations and unambiguous improvements in macroeconomic conditions. In Fig. 1, predicted harsh parenting is shown on the vertical axis and changes in macroeconomic conditions are shown on the horizontal axis. Deteriorating and improving macroeconomic conditions were measured (in percentile terms) as the distance from zero change (in the middle). Inspection of 95% confidence bands indicated that, for mothers with a T allele, harsh parenting clearly increased as macroeconomic conditions worsened and decreased as macroeconomic conditions improved. By contrast, for mothers with the CC allele, harsh parenting changed little if at all in response to changes in macroeconomic conditions. These results suggest that the parenting behaviors of the T allele carriers were more sensitive than those of the CC allele carriers to changes in macroeconomic conditions.

We extended our analysis in nine ways. First, to further explore heterogeneous effects by genotype, we reestimated our

$G \times E$  model using two less well-studied SNPs (rs1800955 and rs40814) from the *DRD4* and *DAT1* genes (details are given in *SI Materials and Methods*). The effects of macroeconomic conditions on harsh parenting did not vary by these two dopamine markers (Table S3 and Figs. S1 and S2).

Second, to the extent that the risk of experiencing unemployment is heterogeneous within the population and that individuals are aware of their differential risks, we should observe heterogeneity of response not only by genotype but also by differences in the risk or salience of adverse economic conditions across the population (33). Thus, we explored heterogeneity of response by education, race/ethnicity, family structure, and child sex. For the first three characteristics, we found some evidence for heterogeneous responses, with responses being more negative for those most likely to be adversely affected (Table S4).

Third, the absence of a positive association between the level of the local UR and harsh parenting was surprising, and thus we extensively examined plausible alternative specifications to see whether there was any evidence to support a positive and significant UR level effect (Table S5). With one minor exception, we found no supporting evidence. Fourth, we investigated whether the level effects of UR and CSI, which were in the unexpected direction, were driven by their functional forms (Table S6). The level coefficients were indeed sensitive to alternative functional forms, suggesting that this effect should not be given much weight. Fifth, we examined whether the change coefficients were robust to the exclusion of the UR and CSI level variables (Table S7). Excluding these variables did not alter our findings. Sixth, we examined whether family income loss at the individual level was associated with increases in harsh parenting and whether controlling for actual income loss affected our main results (Table S8). Although income loss at the individual level was associated with increases in harsh parenting, especially for the T allele carriers, the association was puzzling, as larger losses did not lead to harsher parenting. Seventh, we examined whether our results might be strengthened if we used employment-population ratios rather than unemployment rates (Table S9). The results were consistent with, but a little weaker in significance than, those in Table 1. Eighth, we examined whether our results were sensitive to our treatment of mothers who moved to a different city during the study (Table S10). Accounting for mothers' cross-city



**Fig. 1.** Differential harsh parenting responses to deteriorating and improving macroeconomic conditions (measured as percentile changes) by the *DRD2 Taq1A* polymorphism.

moves did not change the results. Ninth and lastly, we examined psychological harsh parenting and corporal punishment separately (Table S11). Both sets of results were consistent with those using the composite measure of maternal harsh parenting.

## Discussion

This paper extends prior work by examining the relationship between adverse macroeconomic conditions and maternal harsh parenting. Because the Great Recession and other more moderate changes in macroeconomic conditions are exogenous to the individual, our approach allowed us to address shortcomings in the literatures on both the family stress model and the genetic sensitivity model. We found that the rate and direction of change in macroeconomic conditions rather than actual conditions affected harsh parenting, and that declines in conditions had a stronger impact on parenting than improvements in conditions. We also found that mothers' responses to changes in economic conditions were moderated by their genetic profiles, such that mothers with the "sensitive" genotype did worse than their counterparts in a deteriorating economy and better in an improving economy.

Although we found no evidence that the level of the local UR was positively associated with harsh parenting, caution is warranted in interpreting this finding. Actual reductions in income between interviews did result in greater harsh parenting. The strong effects for the *Taq1A* polymorphism of the *DRD2* gene should also be interpreted with caution insofar as we found little effect for other dopamine genes that were plausible candidates. More research is needed to disentangle the complex pathways through which a variety of genetic markers in the dopaminergic system operate together. Finally, although our findings are consistent with the argument that uncertainty and anticipation of adverse conditions are mechanism through which a deteriorating economy leads to aggressive parenting, these constructs were not directly measured in this study.

Nonetheless, we provide strong evidence that changes in macroeconomic conditions, rather than current conditions, affect harsh parenting, that declines in macroeconomic conditions have a stronger impact on harsh parenting than improvements in conditions, and that mothers' responses to adverse economic conditions are moderated by the *DRD2 Taq1A* genotype. These findings demonstrate the importance of attending to the non-economic costs of macroeconomic changes, population heterogeneity in response to macroeconomic shocks, and exogenous measurement of environmental stressors in  $G \times E$  research.

## Materials and Methods

The FFS is a national longitudinal birth cohort study that follows 4,898 children, 1,186 of whom were born to married parents and 3,712 were born to unmarried parents (31). They represent children born in 20 large US cities with population greater than 200,000 between 1998 and 2000. Baseline interviews were conducted shortly after the birth, with mothers interviewed in the hospital and fathers interviewed either in the hospital or wherever they could be located. Follow-up surveys were conducted when the focal child was 1, 3, 5, and 9 y of age. In year 9, saliva DNA samples were collected from mothers using the Oragene• DNA sample collection kit (DNA Genotek) and retained at room temperature until DNA extraction (laboratory of D.N., Princeton University, Princeton, NJ) according to the protocol supplied by

the manufacturer. The analytic sample consisted of 2,612 mothers who (i) were interviewed at least once during the years 3, 5, and 9 surveys, (ii) provided usable saliva during the year 9 survey, and (iii) had valid observations on study variables (details on variable construction and dopamine genetic variation are provided in *SI Materials and Methods*). For access to the FFS data, visit [www.fragilefamilies.princeton.edu/index.asp](http://www.fragilefamilies.princeton.edu/index.asp). The study protocol was approved by the institutional review board of Princeton University and informed consent was obtained in line with the study protocol.

We conducted our analysis by estimating two models. The first model examined the effect of macroeconomic conditions on harsh parenting. To test the hypothesis that declines in macroeconomic conditions have a stronger impact than improvements, we applied a spline function to the measures of changes in the local UR and the CSI (34). It allowed deteriorating and improving changes in each macroeconomic indicator to have their own slopes and at the same time their effects to converge at zero. To exploit multiyear variations in UR and CSI, we pooled the FFS data from years 3, 5, and 9 with SEs adjusted for within-individual correlation. The equation for the model is given by:

$$MHP_{it} = a + b_1CE_{it} + b_2LE_{it} + cX_{it} + dWave_{it} + eSeason_{it} + fCity_{it} + \varepsilon_{it}, \quad [1]$$

where  $MHP_{it}$  is mother  $i$ 's harsh parenting score in year  $t$ ,  $CE$  denotes deteriorating and improving changes in UR and CSI,  $LE$  denotes levels of UR and CSI, and  $X$  is a vector of the control variables described above. We included in the model interview wave-, season-, and city-fixed effects to rule out the possibility that periodic, seasonal, and geographical variations could induce a spurious association between macroeconomic conditions and harsh parenting.

The second model examined the  $G \times E$  effect on harsh parenting of macroeconomic conditions and *DRD2 Taq1A* genotype. We used the full interaction  $G \times E$  model such that Eq. 1 was estimated separately for mothers carrying a T allele and for mothers carrying the CC allele. We found that the effect of deteriorating macroeconomic conditions differed significantly between mothers with a T allele and those with the CC allele ( $F(2, 2611) = 3.36, P = 0.03$ ). In addition, among mothers carrying a T allele, the effect of deteriorating macroeconomic conditions, indicated by positive changes in UR and negative changes in CSI, differed significantly from that of improving macroeconomic conditions, indicated by negative changes in UR and positive changes in CSI ( $F(1, 1396) = 10.75, P = 0.001$ ). However, among mothers carrying the CC allele, the effect of deteriorating macroeconomic conditions did not differ significantly from that of improving macroeconomic conditions ( $F(1, 1214) = 1.38, P = 0.24$ ).

The simulation results in Fig. 1 were derived as follows. To calculate the predicted values of harsh parenting from our  $G \times E$  model in Table 1, levels of UR and CSI were set to their mean values and all covariates to their observed values. To plot the predicted values when macroeconomic conditions declined, deteriorating changes in UR and CSI were assigned to their percentile values whereas improving changes in UR and CSI were assigned to zero. Conversely, to plot the predicted values when macroeconomic conditions improved, improving changes in UR and CSI were assigned to their percentile values whereas deteriorating changes in UR and CSI were assigned to zero. We plotted these predicted values on harsh parenting against changes in macroeconomic conditions with 95% confidence bands.

**ACKNOWLEDGMENTS.** We would like to thank Charles Manski and Stephen Suomi for helpful comments. We also benefited from the feedback provided by Dalton Conley, Jennifer Jennings, Douglas Miller, Patrick Sharkey, Florencia Torche, Jane Waldfogel, Lawrence Wu, and Yang Yang. Major funding for the Fragile Families and Child Wellbeing Study came from the Eunice Kennedy Shriver National Institute of Child Health and Human Development through Grants R01HD36916 and R01HD066054, as well as a consortium of private foundations.

- Elder G (1974) *Children of the Great Depression* (Westview, Boulder, CO).
- Elder G, Conger R (2000) *Children of the Land* (Univ of Chicago Press, Chicago).
- Page M, Stevens A, Lindo J (2009) Parental income shocks and outcomes of disadvantaged youth in the United States. *An Economic Perspective on the Problems of Disadvantaged Youth*, ed Gruber J (Univ of Chicago Press, Chicago), pp 213–235.
- Oreopoulos P, Page M, Stevens A (2008) The intergenerational effect of worker displacement. *J Labor Econ* 26:455–483.
- Milligan K, Stabile M (2011) Do child tax benefits affect the wellbeing of children? Evidence from Canadian child benefit expansions. *Am Econ J* 3:175–205.
- Dahl G, Lochner L (2012) The impact of family income on child achievement: Evidence from the Earned Income Tax Credit. *Am Econ Rev* 102:1927–1956.
- Berger RP, et al. (2011) Abusive head trauma during a time of increased unemployment: A multicenter analysis. *Pediatrics* 128(4):637–643.
- Wood JN, et al. (2012) Local macroeconomic trends and hospital admissions for child abuse, 2000–2009. *Pediatrics* 130(2):e358–e364.
- Rosenblum LA, Pautly GS (1984) The effects of varying environmental demands on maternal and infant behavior. *Child Dev* 55(1):305–314.
- Coplan JD, et al. (1998) Cerebrospinal fluid concentrations of somatostatin and biogenic amines in grown primates reared by mothers exposed to manipulated foraging conditions. *Arch Gen Psychiatry* 55(5):473–477.
- Loewenstein GF, Weber EU, Hsee CK, Welch N (2001) Risk as feelings. *Psychol Bull* 127(2):267–286.
- Berkowitz L (1990) On the formation and regulation of anger and aggression: A cognitive-neoassociationistic analysis. *Am Psychol* 45(4):494–503.
- Baumeister RF, Vohs KD, DeWall CN, Zhang L (2007) How emotion shapes behavior: Feedback, anticipation, and reflection, rather than direct causation. *Pers Soc Psychol Rev* 11(2):167–203.

14. Wilson T, Gilbert D (2003) *Affective Forecasting*, Advances in Experimental Social Psychology, ed Zanna M, (Academic, San Diego), Vol 35, pp 345–411.
15. Frederick S, Loewenstein G (1999) Hedonic adaptation. *Well-Being: The Foundations of Hedonic Psychology*, eds Kahneman D, Diener E, Schwarz N (Cambridge Univ Press, New York), pp 302–329.
16. Tversky A, Kahneman D (1974) Judgment under uncertainty: Heuristics and biases. *Science* 185(4157):1124–1131.
17. Kahneman D, Slovic P, Tversky A (1982) *Judgment under Uncertainty: Heuristics and Biases* (Cambridge Univ Press, New York).
18. Kagan J, Reznick JS, Snidman N (1988) Biological bases of childhood shyness. *Science* 240(4849):167–171.
19. Caspi A, et al. (2002) Role of genotype in the cycle of violence in maltreated children. *Science* 297(5582):851–854.
20. Ellis BJ, Essex MJ, Boyce WT (2005) Biological sensitivity to context. II. Empirical explorations of an evolutionary-developmental theory. *Dev Psychopathol* 17(2):303–328.
21. Belsky J, Bakermans-Kranenburg M, van Ijzendoorn M (2007) For better and for worse: Differential susceptibility to environmental influences. *Curr Dir Psychol Sci* 16:300–304.
22. Ellis BJ, Boyce WT, Belsky J, Bakermans-Kranenburg MJ, van Ijzendoorn MH (2011) Differential susceptibility to the environment: An evolutionary–neurodevelopmental theory. *Dev Psychopathol* 23(1):7–28.
23. Bakermans-Kranenburg MJ, van Ijzendoorn MH (2011) Differential susceptibility to rearing environment depending on dopamine-related genes: New evidence and a meta-analysis. *Dev Psychopathol* 23(1):39–52.
24. Caspi A, Hariri AR, Holmes A, Uher R, Moffitt TE (2010) Genetic sensitivity to the environment: The case of the serotonin transporter gene and its implications for studying complex diseases and traits. *Am J Psychiatry* 167(5):509–527.
25. Conley D (2009) The promise and challenges of incorporating genetic data into longitudinal social science surveys and research. *Biodemography Soc Biol* 55(2):238–251.
26. Noble EP, Blum K, Ritchie T, Montgomery A, Sheridan PJ (1991) Allelic association of the D2 dopamine receptor gene with receptor-binding characteristics in alcoholism. *Arch Gen Psychiatry* 48(7):648–654.
27. Bannon M, Sacchetti P, Granneman J (1995) The dopamine transporter: Potential involvement in neuropsychiatric disorders. *Psychopharmacology: The Fourth Generation of Progress*, eds Borroni E, Kupfer D (Raven, New York), pp 179–188.
28. Gizer IR, Ficks C, Waldman ID (2009) Candidate gene studies of ADHD: A meta-analytic review. *Hum Genet* 126(1):51–90.
29. Pavlov KA, Chistiakov DA, Chekhonin VP (2012) Genetic determinants of aggression and impulsivity in humans. *J Appl Genet* 53(1):61–82.
30. Blum K, et al. (1995) Dopamine D2 receptor gene variants: Association and linkage studies in impulsive-addictive-compulsive behaviors. *Pharmacogenetics* 5:121–141.
31. Reichman N, Teitler J, Garfinkel I, McLanahan S (2001) Fragile families: Sample and design. *Child Youth Serv* 23:303–326.
32. Straus MA, Hamby SL, Finkelhor D, Moore DW, Runyan D (1998) Identification of child maltreatment with the Parent-Child Conflict Tactics Scales: Development and psychometric data for a national sample of American parents. *Child Abuse Negl* 22(4):249–270.
33. Manski C (2004) Measuring expectations. *Econometrica* 72:1329–1376.
34. Marsh L, Cormier D (2001) *Spline Regression Models* (Sage, Thousand Oaks, CA).