

Relationships Between Poverty and Psychopathology

A Natural Experiment

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THE ASSOCIATION BETWEEN POVERTY and mental illness has been described throughout the world and throughout history.¹⁻⁹ Clinicians and researchers have noted the difficulty of untangling the effects of "social causation, . . . adversity and stress associated with low social statuses" from those of "social selection, [which] posits that genetically predisposed persons drift down to or fail to rise out of" poverty.¹⁰

Recent research has emphasized the role played by genetics in an individual's vulnerability to a wide range of psychiatric disorders. Social selection is an example of a theory consistent with gene-environment correlation, in that affected individuals, and often their family members with them, drift down into poverty (and thus into environments that in themselves increase risk for mental illness), while social causation theories reflect a gene-environment interaction in which genetic risk remains latent unless individuals are exposed to the stress of poverty, often by situations beyond their control. The distinction can be important in suggesting different strategies for prevention or treatment.¹¹

For editorial comment see p 2063.

Context Social causation (adversity and stress) vs social selection (downward mobility from familial liability to mental illness) are competing theories about the origins of mental illness.

Objective To test the role of social selection vs social causation of childhood psychopathology using a natural experiment.

Design Quasi-experimental, longitudinal study.

Population and Setting A representative population sample of 1420 rural children aged 9 to 13 years at intake were given annual psychiatric assessments for 8 years (1993-2000). One quarter of the sample were American Indian, and the remaining were predominantly white. Halfway through the study, a casino opening on the Indian reservation gave every American Indian an income supplement that increased annually. This increase moved 14% of study families out of poverty, while 53% remained poor, and 32% were never poor. Incomes of non-Indian families were unaffected.

Main Outcome Measures Levels of *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*, psychiatric symptoms in the never-poor, persistently poor, and ex-poor children were compared for the 4 years before and after the casino opened.

Results Before the casino opened, the persistently poor and ex-poor children had more psychiatric symptoms (4.38 and 4.28, respectively) than the never-poor children (2.75), but after the opening levels among the ex-poor fell to those of the never-poor children, while levels among those who were persistently poor remained high (odds ratio, 1.50; 95% confidence interval, 1.08-2.09; and odds ratio, 0.91; 95% confidence interval, 0.77-1.07, respectively). The effect was specific to symptoms of conduct and oppositional defiant disorders. Anxiety and depression symptoms were unaffected. Similar results were found in non-Indian children whose families moved out of poverty during the same period.

Conclusions An income intervention that moved families out of poverty for reasons that cannot be ascribed to family characteristics had a major effect on some types of children's psychiatric disorders, but not on others. Results support a social causation explanation for conduct and oppositional disorder, but not for anxiety or depression.

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Disentangling the effects of social causation and social selection ideally requires an experimental design that manipulates poverty levels and studies the effects on mental illness.¹¹ Income ex-

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EXHIBIT H	MentalHealth	Document consists of 10 pages
<input checked="" type="checkbox"/>	Entire document provided.	
<input type="checkbox"/>	Due to size limitations, pages ____ through ____ provided.	
A copy of the complete document is available through the Research Library (775-684-6827 or e-mail library@icb.state.nv.us).		
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periments, such as this, have occasionally been done; for example, the New Jersey Negative Income Tax experiment of the 1960s,^{12,13} and its replications.¹⁴⁻¹⁶ However, none of these studies investigated the effects of relief from poverty on mental health. Researchers have had to take advantage of retrospective recall,¹⁷ nonexperimental prospective studies,¹⁸ or at best quasi-experimental situations such as the depression of the 1930s,¹⁹ the farm crisis of the 1980s,²⁰⁻²² or the immigration of European and North African Jews to Israel.¹⁰ The extent to which the association between poverty and mental illness reflects social causation or social selection is difficult to test using such natural experiments, because of the difficulty of disentangling movement into or out of poverty from other characteristics that might have caused the change in income. Problems of interpretation are compounded if the quasi-experimental manipulation was unexpected, and if measures before and after change are not available. A truly experimental manipulation of income that showed an improvement in children's behavioral symptoms is the Minnesota Family Investment Program,²³ but this study was restricted to single-parent families who were long-term recipients of welfare.

In the middle of an 8-year, community-based study of the development of mental illness in children, we were confronted with a natural experiment in which income levels in an entire community were raised. We used this conjunction of longitudinal evaluation and natural experiment to test the effect of social causation on the trajectory of child and adolescent psychopathology. We examined the mental health of children whose families moved out of poverty, compared with children whose families remained poor despite the intervention and with those who were never poor. If family poverty caused specific emotional and behavioral problems in children,^{1,4,24} then after poverty was removed these psychiatric symptoms should improve or disappear.

METHODS

Setting and Population

The Great Smoky Mountains Study is a longitudinal study of the development of psychiatric disorder and need for mental health services in rural and urban youth.^{25,26} A representative sample of 1420 children aged 9, 11, and 13 years at intake was recruited from 11 counties in western North Carolina. Potential participants were selected from the population of some 20000 children, using a household equal probability, accelerated cohort design.²⁷ Over several years of data collection, each age cohort reaches a given age in a different year, thus controlling for cohort effects.²⁸

American Indian children were oversampled, to make up 25% of the final sample. The Eastern Band of Cherokee Indians live on a federal reservation that extends into 2 of the 11 counties. The tribe has an enrolled membership of approximately 8000. The final sample consisted of 350 Indian children (81% of those recruited) and 1070 non-Indian children (80% of those recruited); 92.5% of the latter were white and 7.5% African American. In the analyses, each individual's contribution was weighted proportionately to his/her probability of selection into the study, so that the results are representative of the whole population of children of this age. During the 8 years (1993-2000) of this ongoing study, 3 children have died, and 6% have completed only 1 interview. The mean response rate was 83%. Attrition and nonresponse were found equally in all the ethnic and income groups.

Intervention

Beginning in 1996, tribal members began to receive income from a gambling casino that opened on the reservation. Under the terms of the agreement with the casino operators, every man, woman, and child receives a percentage of the profits, paid every 6 months. Children's earnings are paid into a trust fund until the age of 18 years. The payment has increased each year, reaching around \$6000 by 2001. The opening of the casino also increased the number of jobs

available, in the casino itself or in surrounding motels and restaurants. These jobs are available to both Indians and non-Indians, but Indians receive preference in hiring at the casino itself.

Procedures

Families were interviewed, usually at home, once a year from 1993 through 2000. Parent and child signed informed consent forms. The study and consent forms were approved by Duke University's institutional review board. Individuals then were interviewed in separate rooms by different interviewers. All interviewers were residents of the study area; some were American Indian. Interviewers had bachelor's degrees but were not clinically licensed. They received 1 month of training and were under constant quality control, which was maintained by postinterview reviews of interview schedules, notes, and tape recordings by supervisors and study faculty.

Measures

Child and Adolescent Psychiatric Assessment. The Child and Adolescent Psychiatric Assessment²⁹ is a structured interview for use with both children and parents or guardians that enables interviewers to determine whether symptoms, as defined in an extensive glossary, are present or absent, and to code their frequency, duration, and onset. The Child and Adolescent Psychiatric Assessment scoring algorithms can be used to generate either diagnoses made using the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)*,³⁰ or scale scores that count the number of DSM-IV psychiatric symptoms relating to any of 29 separate diagnoses or groups of diagnoses. For these analyses, in addition to DSM-IV diagnoses, scale scores were created to cover 2 broad categories of symptoms: those occurring in an emotional disorder (depression or anxiety) and those consistent with a behavioral disorder (conduct disorder or oppositional defiant disorder).

To obtain relatively stable estimates of symptom scores for each child over

time, we calculated 3 mean 4-year symptom scores for the period before the casino opened (1993-1996): 1 for all symptoms, and 1 each for behavioral and emotional symptoms separately. Another 3 symptom scores were calculated for the 4-year period after the casino opened (1997-2000). These 6 mean symptom scores served as the primary outcome measure for all analyses. Children also were classified as having 1 or more emotional disorders or behavioral disorders in the period before and after the casino opening. Both types of disorder were entered together into the models to control for comorbidity.³¹

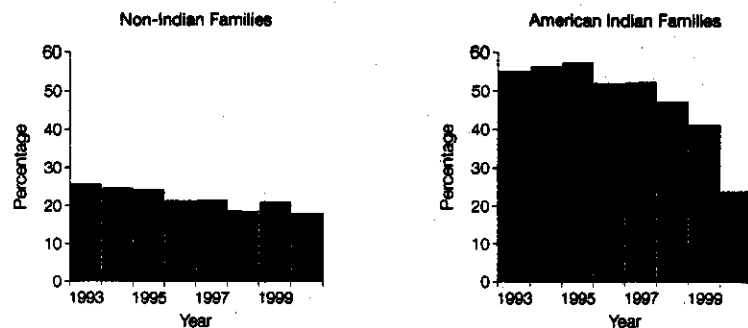
Classification Variable. The adult respondent (usually the mother) provided information about total family income and sources of income (from earnings, welfare, etc) and rank ordered the sources from the largest to the smallest percentage of total family income. The mean family income for the 4 years before and the 4 years after the casino opened was calculated separately. Families were defined as *poor* if the mean income for the 4-year period, adjusted for family size and missing data, was below the federal poverty line for that year, using the Department of Health and Human Services guidelines (available at <http://www.census.gov/hhes/poverty/threshld.html>). Results of repeated analyses using the median were very similar.

Families then were classified into 3 groups: (1) persistently poor, those families below the federal poverty line before and after the casino opened; (2) ex-poor, those families who moved out of poverty after the casino opened; and (3) never poor, those families above the poverty line before and after the casino opened. The fourth possible group, the newly poor (those families who were not poor before the casino opened but became poor later), were excluded from all analyses because of the small number of them ($n=8$) among the American Indian families.

Analyses

We applied a marginal model approach (generalized estimating equa-

Figure. Annual Percentage of Non-Indian and Indian Families Below the Poverty Line



tions) to the analysis of these longitudinal data. Generalized estimating equations is a method developed for dealing with complex longitudinal, repeated, or clustered data, in which the observations within each cluster are correlated with each other.^{32,33} Generalized estimating equations model the effects of predictors (ie, covariates) on the marginal expectations (ie, means), while also accounting for the associations (correlations) among observations from each individual. The parameter estimates were obtained by minimizing a score function that is a generalization of the weighted least squares approach. The SAS program PROC GENMOD, version 8.02 (SAS Institute, Inc, Cary, NC) with a Poisson link function was used in the analyses. In this representative population sample, the number of children with diagnoses was not large, so symptom scale scores were used for several of the analyses presented herein. Statistical significance was set at .05.

RESULTS

Association Between Poverty and Psychiatric Disorder

Across the 8 years of the study, a significant negative correlation between family income and child psychiatric diagnoses ($r=-0.13$, $P<.001$) and number of symptoms ($r=-0.15$, $P<.001$) was observed. The correlation was similar in both non-Indian ($r=-0.19$, $P<.001$) and Indian children ($r=-0.17$, $P<.001$). Children living in poverty were more likely than nonpoor children to have a psychi-

atric disorder (22% vs 15%, odds ratio [OR], 1.6; 95% confidence interval [CI], 1.4-1.8; $P<.001$). Thus, the data are consistent with a relationship between poverty and childhood mental illness.

Effects of the Casino on Family Income

The FIGURE shows that whereas in non-Indian households poverty decreased linearly over the 8 years of the study at a mean rate of 1%, the percentage of Indian families in poverty increased between 1993 and 1995, and then decreased 5% between 1997 and 1998, 6% between 1998 and 1999, and 18% between 1999 and 2000. These results show that the income generated by the casino had an effect on Indian family poverty but not on non-Indian family poverty from 1996 to 2000.

For American Indian and non-Indian children, the proportion of families were 53.2% and 20.2% for persistently poor, 14.4% and 10.3% for ex-poor, and 32.4% and 69.5% for never poor, respectively. The casino income did not succeed in lifting all Indian families out of poverty; more than half (54%) remained poor throughout the study (compared with only 20% of white children). However, a higher proportion of Indian than of white families moved out of poverty after the casino opened (OR, 1.3; 95% CI, 1.1-1.7; $P=.02$).

Family Income and Child Mental Illness

We first used the Indian sample to test whether a change in family resources, in-

dexed by moving above the federal poverty line, had an effect on the likelihood of psychiatric symptoms. Analyses then were repeated in non-Indian families who moved out of poverty around the same time. We predicted that we should see similar results, but the hypothesis that these symptoms were caused by the increase in income could not be as strong because of possible confounders.

TABLE 1 shows the mean number of psychiatric symptoms of any kind in American Indian children, before and after the casino opened, by poverty group. The cells labeled "contrast" present the results of 9 between-group pair-

wise contrasts of interest. For example, exponentiating the contrast estimate for the comparison between persistently poor vs never-poor groups during the 4-year period before the casino opened (eg, $e^{0.463}$) gives a difference in log odds of approximately 1.59, suggesting that the odds of having a psychiatric symptom for the persistently poor group are 59% higher than the odds for the never-poor group. Column contrasts compare symptom scores across time within a group.

Children of American Indian never-poor and persistently poor families maintained a steady mean (SD) total

number of psychiatric symptoms, low and high respectively, before and after the casino opened. Children whose families moved out of poverty, however, showed a significant decrease in the mean number of psychiatric symptoms after the casino opened ($P = .02$).

Between-group comparisons by poverty group revealed that over the 4 years before the casino opened, children whose families were to move out of poverty had the same number of symptoms as those who were to remain poor, and both groups had significantly more symptoms than did children whose families were never poor. The situation changed dramatically after the casino opened. Between-group comparisons showed that children of ex-poor families had the same number of psychiatric symptoms as the never poor, and significantly fewer symptoms than the persistently poor. Thus far, the data supported a social causation hypothesis, demonstrating a decrease in symptoms in children whose families moved out of poverty.

We next tested whether the effect of moving out of poverty applied equally to behavioral and emotional symptoms. Across the whole sample, the mean (SD) number of behavioral symptoms was almost the same before (2.0 [2.7]) and after (2.1 [3.2]) the casino opened. TABLE 2 shows that this increase was restricted to children from persistently poor families, whose mean symptom level rose by 21%. Ex-poor children showed a 40% decrease in behavioral symptoms. Children from never-poor families maintained a steady, low level of behavioral symptoms before and after the opening of the casino. Before the casino opened, children from families destined to move out of poverty had almost as many behavioral symptoms as the persistently poor. After the casino opened, the mean level of behavioral symptoms in children from ex-poor families was almost identical to that of never-poor children, and significantly lower than the mean for persistently poor children (Table 2).

The pattern for emotional symptoms was much less marked (TABLE 3). Results from the overall Poisson re-

Table 1. Mean Annual Frequency Scores of Total Psychiatric Symptoms of American Indian Children Averaged Separately Over the 4-Year Period Before and After the Casino Opened

	Before Casino	After Casino	Contrast Before vs After Casino, OR (95% CI)*
Persistently poor, mean (SD)	4.38 (3.79)	4.71 (4.79)	0.91 (0.77-1.07), $P = .24$
Ex-poor, mean (SD)	4.28 (3.54)	2.90 (2.48)	1.5 (1.08-2.09), $P = .02$
Never poor, mean (SD)	2.75 (2.55)	2.78 (3.76)	1.0 (0.72-1.4), $P = .98$
Contrast persistently vs ex-poor OR (95% CI)*	1.02 (0.76-1.39)	1.69 (1.19-2.41)	
P value	.88	.003	
Contrast persistently vs never poor* OR (95% CI)	1.59 (1.24-2.04)	1.76 (1.22-2.54)	
P value	<.001	.003	
Contrast ex- vs never-poor* OR (95% CI)	1.55 (1.11-2.17)	1.04 (0.67-1.61)	
P value	.009	0.88	

Abbreviations: CI, confidence interval; OR, odds ratio.

*The cells labeled "Contrast" present the results of between-group pairwise contrasts of interest. The parameter for each contrast estimate was converted into an adjusted OR. An OR greater than 1 indicates that the odds of an increase in the mean number of psychiatric symptoms are higher in row 2 (or column 2) than in row 1 (or column 1). Values less than 1 indicate the odds of an increase in the mean number of psychiatric symptoms higher in row 1 (or column 1) than in row 2 (or column 2). The strength of association increases with the deviation from 1.

Table 2. Mean Annual Frequency Scores of Behavioral Psychiatric Symptoms of American Indian Children Averaged Separately Over the 4-Year Period Before and After the Casino Opened

	Before Casino	After Casino	Contrast Before vs After Casino, OR (95% CI)*
Persistently poor, mean (SD)	2.41 (2.69)	2.91 (3.80)	0.80 (0.64-1.01), $P = .06$
Ex-poor, mean (SD)	2.25 (2.65)	1.34 (2.07)	1.66 (0.97-2.83), $P = .07$
Never poor, mean (SD)	1.30 (2.11)	1.37 (1.93)	0.95 (0.62-1.44), $P = .80$
Contrast persistently vs ex-poor* OR (95% CI)	1.07 (0.70-1.64)	2.21 (1.24-3.95)	
P value	.75	.007	
Contrast persistently vs never poor* OR (95% CI)	1.86 (1.25-2.78)	2.19 (1.47-3.28)	
P value	.002	<.001	
Contrast ex- vs never poor* OR (95% CI)	1.73 (1.03-2.91)	0.99 (0.53-1.86)	
P value	.04	.98	

Abbreviations: CI, confidence interval; OR, odds ratio.

*See Table 1 for explanation.

gression showed no significant interaction between poverty group and time.

Replication in Non-Indian Sample

The social causation theory was subjected to a powerful test in the Indian community, because the income from the casino came to every family. No such powerful test of the effect of relieving poverty was available for the non-Indian families. However, some non-Indian families did move out of poverty around the same time, after they had been in the study for 4 years, while others remained poor or were never poor. We repeated the analyses with non-Indian children, testing the same hypotheses. Results were similar to those seen in Indian children (TABLE 4).

How Does Relief of Poverty Affect Children's Psychopathology?

We ran a series of test for mediators of the link between poverty and psychopathology; that is, factors causally related to the symptoms that could be affected by relief from poverty. A strict mediational model³⁴ requires that the significant effect of changing poverty status on the children's symptoms should become less significant once the putative mediator is entered into the model; the mediator itself must be significantly associated with poverty status.

Potential mediators examined were traumatic life events (eg, parent separation or divorce, sexual or other physical abuse, unplanned pregnancy), neglect, harsh or inconsistent parenting, overprotective or intrusive parenting, lax supervision, and maternal depression. Only 1 stressor met the requirements specified by Baron and Kenny³⁴ as a full mediator: failure of parents to provide adequate supervision. This stressor was coded from parents' answers to a set of questions such as, "How often is [the subject] out without your knowing where s/he is?" Lax supervision is defined as inability to exercise effective control once a week or more often. As required for a mediational model, the 3 main effects analyses all were significant in a series of Poisson regressions: the effect of chang-

ing poverty level on the moderator, level of parental supervision ($\beta = -0.59$; $SE = 0.14$; $\chi^2 = 17.2$; $P < .001$), effect of changing poverty level on psychiatric symptoms ($\beta = 0.18$; $SE = 0.062$; $\chi^2 = 8.2$; $P = .004$), and the effect of supervision on psychiatric symptoms ($\beta = 1.07$; $SE = 0.13$; $\chi^2 = 71.2$; $P < .001$). When the mediational model was run, including both supervision and changing poverty status, the effect of changing poverty level on psychiatric symptoms became nonsignificant ($\beta = 0.04$; $SE = 0.05$; $\chi^2 = 0.59$; $P = .44$). The mediating effect of parental supervision accounted for approximately 77% of the effect of changing poverty level on the number of psychiatric symptoms during the 4 years after the opening of the casino.

The model produced the same results for both girls and boys.

In a set of exploratory analyses, we examined differences of 26 variables between the 3 groups before and after the casino opened that might explain why parents who were ex-poor were able to maintain better supervision of their children; factors included single-parent or step-parent household, parental mental illness, drug abuse or crime, traumatic life events, and lack of time to spend with child because of other demands (eg, large family or working 2 jobs). Full details can be obtained from the author.

Three of the 26 variables were distinguished among the groups, all having to do with time constraints in the family. In the ex-poor households, the

Table 3. Mean Annual Frequency Scores of Emotional Psychiatric Symptoms of American Indian Children Averaged Separately Over the 4-Year Period Before and After the Casino Opened

	Before Casino	After Casino	Contrast Before vs After Casino, OR (95% CI)*
Persistently poor, mean (SD)	1.56 (1.51)	1.55 (1.71)	1.0 (0.83-1.2), $P > .99$
Ex-poor, mean (SD)	1.56 (1.13)	1.14 (0.95)	1.43 (1.02-2.03), $P = .04$
Never poor, mean (SD)	1.10 (0.87)	1.07 (1.82)	1.04 (0.70-1.56), $P = .85$
Contrast persistently vs ex-poor*			
OR (95% CI)	1.01 (0.75-1.34)	1.44 (1.0-2.09)	
P value	.97	.05	
Contrast persistently vs never poor*			
OR (95% CI)	1.42 (1.12-1.80)	1.48 (0.94-2.33)	
P value	.004	.09	
Contrast ex- vs never poor*			
OR (95% CI)	1.42 (1.06-1.90)	1.03 (0.62-1.71)	
P value	.02	.92	

Abbreviations: CI, confidence interval; OR, odds ratio.

*See Table 1 for explanation.

Table 4. Mean Annual Frequency Scores of Total Psychiatric Symptoms of Non-Indian Children Averaged Separately Over the 4-Year Period Before and After the Casino Opened

	Before Casino	After Casino	Contrast Before vs After Casino, OR (95% CI)*
Persistently poor, mean (SD)	6.36 (4.97)	6.51 (5.96)	1.01 (0.86-1.18), $P = .92$
Ex-poor, mean (SD)	6.65 (4.80)	4.48 (3.59)	1.52 (1.19-1.95), $P < .001$
Never poor, mean (SD)	4.66 (4.30)	4.03 (4.65)	1.15 (1.04-1.27), $P = .007$
Contrast persistently vs ex-poor*			
OR (95% CI)	0.96 (0.77-1.19)	1.47 (1.12-1.93)	
P value	.69	.006	
Contrast persistently vs never poor*			
OR (95% CI)	1.36 (1.16-1.60)	1.58 (1.30-1.93)	
P value	<.001	<.001	
Contrast ex- vs never poor*			
OR (95% CI)	1.43 (1.18-1.73)	1.08 (0.84-1.38)	
P value	<.001	.55	

Abbreviations: CI, confidence interval; OR, odds ratio.

*See Table 1 for explanation.

number of single-parent households decreased ($\chi^2=4.22$, $P=.04$), the number of households with 2 working parents increased ($\chi^2=6.04$, $P=.01$), and a measure of time demands placed on the index parent decreased ($\chi^2=6.74$, $P=.03$). For each of these measures, the ex-poor families were significantly different from the never-poor families before the casino opened and were significantly different from the persistently poor household after it opened.

COMMENT

As Dohrenwend et al¹⁰ pointed out a decade ago,¹⁰ "Social causation and social selection theories both predict an inverse relation between socioeconomic status and various types of psychopathology. Our problem, therefore, has been to identify circumstances in which the 2 theories make different predictions."

The present study took a longitudinal approach to the problem, arguing that if the reason for the well-established association between poverty and child psychopathology^{1,4,5,24,35} was the social selection of mentally ill families into poverty, then relieving poverty would leave the association intact. If, on the other hand, poverty had a causal role in the symptoms, then alleviating it would reduce the level of symptoms. An event that substantially increased the income of every man, woman, and child in a community provided a natural experiment that we could use to test these competing models. We found the following: (1) Moving out of poverty was associated with a decrease in frequency of psychiatric symptoms over the ensuing 4 years; by the fourth year the symptom level was the same in children who moved out of poverty as in children who were never poor. (2) Adding to the income of never-poor families had no effect on frequency of psychiatric symptoms. (3) The effect of poverty was strongest for behavioral symptoms (those included in the DSM-IV diagnoses of conduct and oppositional disorder). Little effect of moving out of poverty on emotional symptoms (DSM-IV anxiety and depression) was observed. (4) The effect of relieving poverty was mediated by 1 stressor: level

of parental supervision. (5) The same models run using the non-Indian participants showed similar results.

These findings thus support a social causation for behavioral problems. Anxiety and depression symptoms were more common in poor children, but moving out of poverty was not followed by a reduction in these symptoms. There are several possible explanations for this. Anxiety and depression in children and adolescents may be caused by some characteristics of poor families not directly related to poverty; for example, they may carry a higher genetic loading for these conditions, as a social selection hypothesis (gene-environment correlation) would suggest.³⁶ Alternatively, the remarkable speed of the change in behavioral symptoms after poverty was lifted may be specific to those symptoms; it might take longer for the reduction in poverty-induced family stress to be reflected in children's mood and anxiety levels. In fact, surprisingly little evidence is available linking childhood anxiety or depression directly to poverty,^{4,24,37-39} and it may be that poverty seriously increases risk for anxiety and depression only in adulthood.^{40,41} Effects on attention-deficit/hyperactivity disorder are not reported here because the prevalence of attention-deficit/hyperactivity disorder decreased to 0 in all groups as the children moved through adolescence, so there was a confound between age and the pre- and post-study design that made the findings uninterpretable.

Theory would predict similar findings in the general population, in this study represented by the non-Indian children, and indeed the findings were similar; when families moved out of poverty we saw a reduction in behavioral symptoms. The problem is with interpretation: did stronger families work their way out of poverty, did moving out of poverty improve the risk environment for children, or both? Only an experimental or quasi-experimental design can dis-aggregate these 2 possibilities.⁴²

Among the wide range of potential mediators of the effect of poverty on behavioral symptoms, only parental

supervision emerged as a mediator. As the study participants moved into adolescence, the number of parents who believed that they provided adequate supervision decreased across the sample, but it decreased less in the ex-poor than in the persistently poor group. With only 50 children in the ex-poor group, the study lacked power to explore the reasons for this decrease. However, exploratory analyses showed that the 3 family characteristics on which the ex-poor families resembled persistently poor families before the casino opening and never-poor families after the casino opening all had to do with the amount of time that the index parent had to pay attention to the child. Like all the other families in the study, the number of households with 2 parents working full time increased over time, but ex-poor families reported a reduction in time demands and in the number of single-parent households (both of which increased in never-poor and persistently poor households). This finding raises the possibility that children's symptoms, particularly those of oppositional and deviant behavior, are affected by economic constraints on parents' ability to devote scarce time resources to supervision. The fact that 3 significant associations are conceptually linked through their relationship to family time constraints suggest that our findings are valid.

The Great Smoky Mountains Study has some advantages for addressing the relationship between poverty and psychopathology. First, the outcomes were measured in children who played little role in generating the family's social status, and therefore offered a fairly clean test of competing theories. Second, the study provided a within-subjects design, following the same children over an 8-year period; a stronger test than is provided by a between-subjects design.⁴³ Third, this was a representative population sample. Fourth, the study included a direct manipulation of a key explanatory variable: an increase in family income in the American Indian families that was not caused by family characteristics that also could affect children's behavioral symptoms.

The study also has important limitations. The sample was not large, and only a small proportion of children (14.4% of Indians, 10.3% of non-Indians) moved out of poverty. The study lacked power for within-group analyses at the level of diagnoses, which required us to test for differences at the level of symptom scales rather than at diagnostic categories. An ethnic difference correlated 100% with the study intervention. We replicated the findings in non-Indian families, as a social causation theory would predict, but this group's income change could not be disentangled from characteristics that might be causally linked to both moving out of poverty and improved child mental health. While the rural setting of the study is in some ways an advantage, in enabling us to disentangle poverty and urban residence, replication in an urban sample would increase the generalizability of the findings. Most importantly, the study examined psychiatric symptoms only at the level of parent- and child-reported phenomena; we could not explore the psychophysiological processes that changed to bring about a reduction in behavioral symptoms when the stress of poverty was relieved.

Selection and causation are both compatible with a genetic basis to psychopathology. Social selection implies a correlation between genes and environment such that individuals with a genetic liability have difficulty climbing out of poverty, while social causation implies an interaction: genetic liability to a disease is expressed under the stress of poverty. Questions about which genes and about the developmental processes that lead to their expression in the form of behavioral symptoms are still unanswered.

Author Contributions: Dr Costello, as primary investigator, had full access to all of the data in this study and takes responsibility for the integrity of the data, and the accuracy of the data analysis.
Study concept and design: Costello, Compton, Angold.
Acquisition of data: Costello, Keeler, Angold.
Analysis and interpretation of data: Costello, Compton, Angold.
Drafting of the manuscript: Costello, Compton, Keeler.
Critical revision of the manuscript for important intellectual content: Costello, Angold.
Statistical expertise: Compton, Keeler.
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Unique Lipoprotein Phenotype and Genotype Associated With Exceptional Longevity

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INDIVIDUALS WITH EXCEPTIONAL LONGEVITY have been generally spared from major age-related diseases that are responsible for most deaths in elderly persons, such as cardiovascular disease (CVD), diabetes mellitus, Alzheimer disease, and cancer.¹ Various studies suggest that genetic determinants of exceptional longevity are highly heritable.^{2,3} Siblings of centenarians have an 8- to 17-fold higher probability of living past the age of 100 years, accounting for only approximately 1 of 10000 individuals in the general population.^{2,3} The offspring of long-lived parents have an approximately 50% lower prevalence of hypertension, diabetes mellitus, myocardial infarction, and stroke/transient ischemic attacks compared with age-matched control groups.³ Furthermore, at least 1 study linked a locus on chromosome 4 to exceptional longevity.⁴ Identification of biological markers and genes that are conducive to exceptional longevity may provide insights into mechanisms that protect from a host of common diseases and/or slow the biological processes of aging.

Longevity genes have been demonstrated in other species but the relevance to humans is controversial.¹ In contrast, rodent models of aging and ag-

Context Individuals with exceptional longevity have a lower incidence and/or significant delay in the onset of age-related disease, and their family members may inherit biological factors that modulate aging processes and disease susceptibility.

Objective To identify specific biological and genetic factors that are associated with or reliably define a human longevity phenotype.

Design, Setting, and Participants In a case-control design, 213 Ashkenazi Jewish probands with exceptional longevity (mean [SD] age, 98.2 [5.3] years) and their offspring (n=216; mean [SD] age, 68.3 [6.7] years) were recruited from 1998 to 2002, while an age-matched control group of Ashkenazi Jews (n=258) and participants from the Framingham Offspring Study (n=589) were accepted as control groups.

Main Outcome Measures Detailed questionnaires, physical examination, and blood samples were taken, including assessment of lipids and lipoprotein subclass levels and particle sizes by proton nuclear magnetic resonance. Samples were also genotyped for the codon 405 isoleucine to valine (I405V) variation in the cholesteryl ester transfer protein (CETP) gene, which is involved in regulation of lipoprotein and its particle sizes.

Results High-density lipoprotein (HDL) and low-density lipoprotein (LDL) particle sizes were significantly higher in probands compared with both control groups ($P=.001$ for both), independent of plasma levels of HDL and LDL cholesterol and apolipoprotein A1 and B. This phenotype was also typical of the proband's offspring but not of the age-matched controls. The HDL and LDL particle sizes were significantly larger in offspring and controls without hypertension or cardiovascular disease, ($P=.001$ and $P=.008$, respectively). Furthermore, lipoprotein particle sizes, but not plasma LDL levels, were significantly higher in offspring and controls without the metabolic syndrome ($P<.001$). Probands and offspring had a 2.9- and 3.6-fold (in men) and 2.7- and 1.5-fold (in women) increased frequency, respectively, of homozygosity for the 405 valine allele of CETP (VV genotype), respectively, compared with controls ($P<.001$ for both). Those probands with the VV genotype had increased lipoprotein sizes and lower serum CETP concentrations.

Conclusions Individuals with exceptional longevity and their offspring have significantly larger HDL and LDL particle sizes. This phenotype is associated with a lower prevalence of hypertension, cardiovascular disease, the metabolic syndrome, and increased homozygosity for the I405V variant in CETP. These findings suggest that lipoprotein particle sizes are heritable and promote a healthy aging phenotype.

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Poverty and Child Mental Health

Natural Experiments and Social Causation

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MOST SOCIAL SCIENTISTS HAVE TENDED TO ASSUME that poverty predisposes to mental disorder, on the grounds that numerous studies have shown statistical associations between the two.^{1,2} However, as Costello et al³ point out in this issue of THE JOURNAL, causal inference is problematic in the absence of experimental evidence because of the crucial need to differentiate between social selection and social causation.^{4,5} In other words, does the statistical association reflect the tendency for individuals with mental disorder to drift into or remain in poverty, or does the experience of poverty itself predispose to mental disorder?

The problem arises when both poverty and the mental disorder apply to the same individuals, as would be the case with studies of adults, but it is especially a concern when poverty derives from the parents, with the effects being considered with respect to mental disorder in the children. Sometimes social selection vs social causation is viewed as a choice between genetic mediation (leading to social selection) and environmental mediation (leading to social causation). However, the issues are much broader than that, particularly when dealing with effects on children. Families in poverty are far from a random selection of the population, and parents who are poor may be so because of either genetic predisposition or environmental adversities or, more usually, a mixture of the two.

It is rare that opportunities arise to put the social selection vs social causation dichotomy under rigorous test. Costello and colleagues³ seized such an opportunity that occurred when a casino was established on an American Indian reservation during the course of their own longitudinal epidemiological study of child psychopathology. The natural experiment derives from the agreement that every adult and child tribe mem-

ber would receive income from the proceeds of the casino and that this would happen without regard for what the families did. Compared with the 4 years before the casino was opened, the 4 years after the opening were accompanied by a reduction in the number of Indian families with incomes below the federal poverty line, whereas there was no effect on income levels in non-Indian families. The findings from the study showed that the Indian children whose family income was no longer below the poverty line showed a significant reduction in behavioral symptoms of oppositional/defiant and conduct disorder, although there was no effect on anxiety and depression symptoms. The study allows a reasonably strong inference that the effect represented social causation.

It was also important to determine why the relief of poverty had this beneficial effect on behavioral symptoms in the children. Further analysis revealed that improved parental supervision of the children was associated with a reduction in the proportion of single-parent households, an increase in the number of households in which both parents worked, but a decrease in the time demands that were placed on the key parent. Improved parental supervision accounted for about 77% of the effect of changing poverty level on the number of psychiatric symptoms among the children.³ The inference, therefore, is that relief of poverty had benefits for the children when it led to improved parental supervision. In other words, the benefits deriving from relief of poverty were mediated by changes in parenting.

The finding that the effects of poverty, and the benefits associated with its relief, are mediated by the impact on the quality of parenting, and especially on parental supervision, is broadly in keeping with the findings from most nonexperi-

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mental studies⁶⁻⁸ as well as with those from an experimental welfare program in Minnesota.⁹ The implication is that poverty constitutes a distal risk factor (because it makes good parenting more difficult) but that the more direct proximal risk mechanism concerns aspects of parenting. Some caution is needed before concluding that it is parental supervision, as such, that matters. Although that may be the case, Kerr and Stattin¹⁰ suggested that much of the apparent beneficial effect of good parental supervision actually derived from the children letting parents know where they were and what they were doing. Doubtless there are 2-way effects, but that cannot be the whole explanation in the study by Costello et al because the benefits of relief of poverty were associated with a reduction in single-parent households, an increase in both parents working, and gains in the time available to the parents. These findings, however, do suggest that part of the benefit of the casino derived from the increase in the jobs available as well as from the income provided as a direct supplement.

Two further issues arise from the authors' findings. First, it is not clear why the income supplement and the greater availability of jobs enabled only 14% of the Indian families to rise above the federal poverty line. Was this because those who failed to rise out of poverty had greater financial needs (perhaps because of having a greater number of children), because they wasted the money gained, or because they failed to take the job opportunities or were not able to use the increased resources to improve their parenting? The policy implication is that because the benefits of relief of poverty are indirect, it cannot be expected that more money will, in itself, provide the hoped-for benefits. Rather, steps need to be taken to make it more likely that economic gains will be translated into benefits in family functioning.

Second, it is necessary to consider the effects on mental disorders in childhood of the increase in affluence, and the reduction in poverty, that has taken place over the last half century in most, but not all, developed countries.¹¹ If the relief of poverty truly has behavioral benefits for children, it might be expected that the economic gains that have taken place over time would have brought about reductions in children's behavioral disturbance. It is striking that the evidence shows that, to the contrary, the rates of antisocial behavior have increased over the same period that income levels have increased. This contrasts with the evident benefits of higher income levels on improved physical health.¹¹

Of course, time trend data have important limitations. The aggregated data do not allow an accurate determination of the effects of changed economic resources on individual children. It is commonly assumed that inequalities in economic resources, rather than absolute levels, are the key factor with respect to physical health. Nevertheless, this distinction does not seem to help with regard to the lack of evident benefits on behavioral disturbance in young people from economic gains in the population as a whole. Time trend data do not necessarily contradict the findings of Costello et al because the benefits of increased income were indirect and were me-

diated by changes in family function. The apparent lack of benefits for child mental health from improvements in a population's overall economic situation may mean either that, for some reason, the economic benefits did not translate into improved family functioning or that such benefits were offset by other negative changes in the society as a whole. Moreover, time trend data largely concern levels of crime as reflected in official statistics, rather than clinically significant mental disorder. Conversely, the findings of Costello et al involve self-reported information from parents and children and lack external verification by reports from teachers or police.

Clearly, many questions remain about the ways in which changes in economic level affect family functioning and the ways in which such effects do or do not have benefits for children. In the meantime, it is important to recognize the great value of this carefully assessed natural experiment regarding the behavioral benefits of relief of poverty in American Indian children living on a reservation. The findings go a long way in demonstrating the reality of social causation, and the findings on mediation draw attention to the features of family functioning that particularly merit further attention. Societies need to recognize that economic levels do have important implications for both family functioning and child mental health^{12,13} but, equally, policy makers need to ensure that economic benefits actually have the intended psychological benefits. Much remains to be learned about what makes the difference in translating economic gains into psychological improvements for parents and children.

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