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AN EXPLORATION OF THE DYNAMIC RELATIONSHIP BETWEEN HEALTH AND COGNITIVE DEVELOPMENT IN ADOLESCENCE

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ABSTRACT

This paper is an empirical exploration of the dynamic relationship between health and cognitive development in a longitudinal data set compiled from two nationally representative cross-sections of children. Our results indicate that there is feedback both from health to cognitive development and from cognitive development to health, but the latter of these relationships is stronger. They also indicate that estimates of family background effects taken from the dynamic model - which can be assumed to be less influenced by genetic factors are smaller than their cross-sectional counterparts, but some still remain statistically significant.

The first finding calls attention to the existence of a continuing interaction between health and cognitive development over the life cycle. The second finding suggests that nurture "matters" in cognitive development and health outcomes.

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An Exploration of the Dynamic Relationship Between Health and Cognitive Development in Adolescence

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Recent studies of children have documented the existence of a relationship between health and cognitive development, reporting typically that good health is associated with higher levels of cognitive development (Edwards and Grossman 1979 and the references cited therein). This association may arise from causality running in one or both directions. Poor health may impede cognitive development in diverse ways. Children who had excessively low birth weights may experience defective brain functioning and abnormally low IQ's throughout their lives. Children who are frequently sick or who are undernourished may be less well able to benefit from school instruction because they either are absent from school or are lethargic and passive when present at school. A similar comment can be made about children with vision or hearing problems. Causality runs in the other direction when more intelligent children and adolescents are better able to manage or avoid health problems. Such children can better understand and follow instructions, and they might be more conscientious about taking prescribed medicine or following a specified treatment. In addition, they may better appreciate the importance of eating a nutritious diet and act appropriately.

While existing studies of childhood document this association between health and cognitive development, they do not provide much evidence concerning the direction of causality. This is because they rely almost exclusively on cross-sectional data. The use of cross-sectional data does not necessarily preclude the investigation of causality, of course, but in the present context the underlying theory does not yield enough prior restrictions to allow one to address this issue. Another stumbling block that arises when one tries to unravel the complicated health-cognitive development relationship with cross-sectional

data is the impossibility of holding constant unmeasurable genetic factors which may be correlated with both health and cognitive development.

A partial remedy for these problems lies with the use of longitudinal data. With such data it is possible to directly model and estimate the dynamic relationship between health and cognitive development. Causality is probed by examining which attribute of children is statistically prior to the other. For example, if it is found that early health status influences later IQ but that early IQ does not influence later health status, it is concluded that health affects IQ but not vice-versa. (This notion of causality is akin to that of Granger 1969). The problem of separating out the impact of unmeasured genetic factors is not so readily dealt with, but it may have less damaging consequences when longitudinal as opposed to cross-sectional data are used.

In this paper we investigate the relationship between health and cognitive development using a longitudinal data set compiled from two nationally representative cross-sections of children: Cycles II and III of the Health Examination Survey (HES). Cycle II samples 7,119 noninstitutionalized children aged 6 to 11 years in the 1963-65 period; and Cycle III samples 6,768 noninstitutionalized youths aged 12 to 17 years in the 1966-70 period. There are 2,177 children common to both cycles, and they were examined in both periods. These 2,177 children constitute the sample on which our longitudinal analysis is based. For these 2,177 children we have measures of health and cognitive development in both periods (childhood and adolescence) and an array of family background variables taken from the first period.

Two multivariate equations are estimated with these data. The first relates adolescent health to childhood health, childhood cognitive development, and family background; and the second relates adolescent cognitive development to childhood cognitive development, childhood health, and family background.

Thus, the resulting estimates will enable us to compare the effect of prior health on current cognitive development with the effect of prior cognitive development on current health. As a byproduct, these equations provide sharper estimates of the environmental as opposed to genetically-related impacts of selected family background variables on children's health and cognitive development.

I. Some Theoretical Considerations

The general type of model estimated here can be represented by the following equation

$$(1) \quad y_{i,t} = A\, y_{i,t-1} + B\, x_{i',t-1} + \varepsilon_{i,t}$$

where $y_{i,t}$ represents a vector of health and cognitive development measures in period t for individual i, $x_{i,t}$ is a vector of economic and background variables for that individual in period t, and A and B are matrices of coefficients.[1] The variables in $x_{i,t}$ are those that determine the quantity and productivity of the various inputs in the health and cognitive development production functions: family income, parents' educational attainment, family size, and the prices of medical care, schooling, and nutrition.[2] Some of these variables vary through time and some are assumed to be constant in all periods. In the special case where $y_{i,t}$ is a dichotomous measure (when it denotes the presence or absence of a particular illness, for example), equation (1) can be directly interpreted as a transition probability function: it gives the probability that individual i has a given health status in time t conditional on his health status in time t-1 and on the values of the other predetermined variables in t-1.

Estimation of this type of model improves on existing cross-sectional analysis of causality because it explicitly treats the time sequence of changes in health and cognitive development. Briefly, this approach, suggested by Granger (1969), relies on a temporal ordering of events: a variable x is said to cause y if predictions of y conditional on lagged values of y and x both are statistically superior to predictions conditional on lagged values of y alone. In this setting, causality between cognitive development and health can be discovered by examining the coefficients of childhood health in the adolescent cognitive IQ development equation and the coefficients of childhood cognitive development in the adolescent health equation.

The problem raised by omitted genetic factors is less tractable. Nevertheless, if such factors can be assumed to operate once and for all by determining the "endowed" levels of health or cognitive development [$y_{i,0}$], past values of these variables will fully embody and control for all genetic effects. Under this assumption, the fact that one cannot directly measure genetic factors does not mar the above analysis of causality. Even as restrictive an assumption as this, however, cannot rescue cross-sectional work because cross-sectional data do not typically include past values of the dependent variable.[3]

An additional implication of this assumption is that the estimated impacts of the various family background measures and of early health or IQ represent true environmental (as opposed to genetic) effects. That is, they represent effects that operate through the parents' demand for health or cognitive development inputs or through the degree of productive efficiency. This is in contrast to estimates generated from cross-sectional data. In the latter case, the relationship between parents' educational attainment and children's IQ, for example, reflects both an environmental effect (more highly educated mothers do a better job of educating their children) and a genetic effect (more highly educated mothers have on average greater native intelligence,

which is passed genetically to their children). When it is assumed that early health or cognitive development fully embodies the genetic contribution, family background variables will reflect only environmental influences.

Admittedly, this assumption concerning genetic impacts is very restrictive. With data like ours, however, which covers only two points in time, it is impossible to partition the effect of the unobservable genetic factors from other time-invariant factors without making some fairly restrictive assumptions. We choose to make this particular assumption for the balance of this paper because it has the advantage of permitting us to use single equation estimation techniques, a not insignificant consideration with a data set as large as this one.[4]

To better illustrate the exact nature of this assumption and its necessity, we present the following simplified two-period formulation, of which our model is a special case (the i's are suppressed for simplicity):

$$(2a) \quad H_1 = a_1 GH + b_1 E + \varepsilon_1$$

$$(2b) \quad H_2 = a_2 GH + b_2 E + c_2 H_1 + d_2 Q_1 + \varepsilon_2$$

$$(3a) \quad Q_1 = \alpha_1 GQ + \beta_1 E + \varepsilon'_1$$

$$(3b) \quad Q_2 = \alpha_2 GQ + \beta_2 E + \gamma_2 Q_1 + \delta_2 H_1 + \varepsilon'_2.$$

In this two-period model H_t represents health, Q_t represents cognitive development, GH represents the time-invariant genetic health endowment, GQ represents the time-invariant cognitive endowment, and E represents a time-invariant background variable. Since GH and GQ are unobserved, we write H_2 and Q_2 in terms of the predetermined values of H and Q [assuming a_1 and α_1 do not equal zero]:

$$(2c) \quad H_2 = \left[\frac{a_2}{a_1} + c_2\right] H_1 + b_2 \left[1 - \frac{a_2}{a_1}\frac{b_1}{b_2}\right] E + d_2 Q_1 + \left[\varepsilon_2 - \frac{a_2}{a_1}\varepsilon_1\right]$$

$$(3c) \quad Q_2 = \left[\frac{\alpha_2}{\alpha_1} + \gamma_2\right] Q_1 + \beta_2 \left[1 - \frac{\alpha_2}{\alpha_1}\frac{\beta_1}{\beta_2}\right] E + \delta_2 H_1 + \left[\varepsilon_2' - \frac{\alpha_2}{\alpha_1}\varepsilon_1'\right].$$

In the context of this model the assumption of no direct genetic effects after the first period is equivalent to fixing a_2 and α_2 at zero. When these are not zero, one cannot determine directions of causality because the error terms in the equations are correlated with the explanatory variables and this correlation leads to biased estimates of both d_2 and δ_2. Nor can one obtain unbiased estimates of pure environmental effects because the reduced form coefficients of the background variable (E) and of the lagged dependent variable (Q_1 or H_1) embody both genetic (a_1 and a_2 or α_1 and α_2) and environmental (b_2 and c_2 or β_2 and γ_2) impacts.[5]

II. Empirical Implementation

A. The Data

Equations (2b) and (3b) are estimated (under the assumptions that $a_2 = 0$ and $\alpha_2 = 0$) using the longitudinal sample compiled from Cycles II and III of the HES. Both cycles are described in detail in NCHS (1967a) and (1969), respectively. Ninety-nine percent of the youths in the longitudinal sample are between the ages of 12 and 15 years at the time of Cycle III, and the remaining one percent are 16 years old.

The HES data include medical histories of each youth provided by the parent, information on family socioeconomic characteristics, birth certificate information, and a school report with data on school performance and classroom behavior provided by teachers or other school officials. Most important, there are objective measures of health from detailed physical examinations and scores on psychological (including IQ and achievement) tests. The physical examinations were given to the children and youths by pediatricians and dentists, and the IQ and achievement tests were administered by psychologists, all of whom were employed by the Public Health Service at the time of each cycle of the HES.

This paper uses only those data for white adolescents who at the time of the Cycle II exam lived with either both of their parents or with their mothers only. Black adolescents are excluded from the empirical analysis because Edwards and Grossman (1979, 1980, forthcoming) have found significant race differences in slope coefficients in cross-sectional research using Cycles II and III. Separate estimates for black adolescents are not presented because the black sample is too small to allow for reliable coefficient estimates. Our working sample also excludes observations for which data are missing.[6] The final sample size is 1,434.

The health and cognitive development measures are described below. In labeling these measures, we denote those that refer to childhood (from Cycle II) by the number 1 at the end of the variable name, and those that refer to adolescence (from Cycle III) by the number 2.

B. Measurement of Cognitive Development

Two measures of cognitive development are used: an IQ measure derived from two subtests of the Wechsler Intelligence Scale for Children (WISC1, WISC2), and a school achievement measure derived from the reading and

arithmetic subtests of the Wide Range Achievement Test (WRAT1, WRAT2). Both measures are scaled to have means of 100 and standard deviations of 15 for each age-group (four-month cohorts are used for WISC and six-month cohorts are used for WRAT).[7] WISC is a common IQ test, similar to (and highly correlated with results from) the Stanford-Binet IQ test (NCHS 1972). The full test consists of twelve subtests, but only two of these--vocabulary and block design--were administered in the HES. IQ estimates based on these two subtests are highly correlated with those based on all twelve subtests (NCHS 1972). Similarly, a test score based on the reading and arithmetic subtests of Wide Range Achievement Test have been found to be highly correlated with the full test and with other conventional achievement tests (NCHS 1967b).

C. Measurement of Health

The measures of childhood and adolescent health are: the periodontal index (APERI1, APERI2); obesity (OBESE1, OBESE2); the presence of one or more significant abnormalities as reported by the examining physician (ABN1, ABN2); high diastolic blood pressure (HDBP1, HDBP2); the parent's assessment of the youth's overall health (PFGHEALTH1, PFGHEALTH2); and excessive school absence for health reasons during the past six months (SCHABS1, SCHABS2). These six measures are negative correlates of good health, and with the exception of the periodontal index, they are all dichotomous variables. Detailed definitions of these health measures (as well as the cognitive development measures) appear in Table 1. All but two of the measures--APERI and ABN--are adequately explained by the table. Additional discussion of APERI and ABN follows.

The periodonal index (APERI, APERI2) is a good overall indicator of oral health as well as a positive correlate of nutrition (Russell 1956). It is obtained from an examination of the gums surrounding each tooth and is scored in such a way that a higher value reflects poorer oral health.[8] Because the periodontal index has marked age and sex trends, our measure is computed as

TABLE 1

Definitions of Health and Cognitive Development Measures

Variable Name	Sample[a] Mean	Sample Standard Deviation	Definition	Source[b]
A. Cognitive Development Measures				
WISC1[c]	103.508	13.924	Youth's IQ as measured by vocabulary and block design subtests of the Wechsler Intelligence Scale for Children, standardized by the mean and standard deviation of four-month age cohorts, in Cycles II and III, respectively	4
WISC2[c]	104.513	13.998		
WRAT1[c]	103.568	12.017	Youth's school achievement as measured by the reading and arithmetic subtests of the Wide Range Achievement Test, standardized by the mean and standard deviation of six-month age cohorts, in Cycles II and III, respectively	4
WRAT2[c]	104.112	13.563		
B. Health Measures				
APERI1[d]	-.055	.792	Periodontal Index, standardized by the mean and standard deviation for one-year age-sex cohorts, in Cycles II and III, respectively	3
APERI2[d]	-.138	.852		
ABN1	.096	.294	Dummy variables that equal one if the physician finds a significant abnormality in examining the youth, in Cycles II and III, respectively	3
ABN2	.188	.391		
HDBP1	.054	.226	Dummy variables that equal one if youth's average diastolic blood pressure is greater than the 95th percentile for the youth's age and sex class, in Cycles II and III, respectively	3
HDBP2	.054	.227		

(continued on next page)

TABLE 1 (concluded)

Variable Name	Sample[a] Mean	Sample Standard Deviation	Definition	Source[b]
OBESE1 OBESE2	.110 .094	.312 .292	Dummy variables that equal one if youth's weight is greater than the 90th percentile for youth's age, sex, and height class, in Cycles II and III, respectively	3
PFGHEALTH1 PFGHEALTH2	.441 .272	.497 .445	Dummy variables that equal one if parental assessment of youth's health is poor, fair or good in Cycles II and III, respectively. Variable equals zero if assessment is very good in Cycle II and very good or excellent in Cycle III; there is no excellent category in Cycle II	1
SCHABS1 SCHABS2[e]	.033 .054	.178 .221	Dummy variables that equal one if youth has been excessively absent from school for health reasons during the past six months, in Cycles II and III, respectively	5
SCHABSUK1	.068	.252	Dummy variable that equals one if information about school absence in Cycle II is not available (see footnote 5)	5

Footnotes to TABLE 1

[a]The means and standard deviations are for the sample of 1,434 white youths described in the text.

[b]The sources are 1 = parents, 2 = birth certificate, 3 = physical examination, 4 = psychological examination, 5 = school form.

[c]The mean of this variable is not equal to 100 because standardization was done using the entire Cycle II or Cycle III sample rather than the subsample reported here. In particular the mean in excess of 100 reflects the better cognitive development of white youths compared to black youths.

[d]The mean of this variable is not zero because standardization was done using the entire Cycle II or Cycle III sample rather than the subsample reported here. In particular the negative mean reflects the better oral health of white youths compared to black youths.

[e]The mean and standard deviation are based on a subsample of 1,321 youths for whom the school form was available.

the difference between the adolescent's (or child's) actual index and the mean index for his or her age-sex group divided by the standard deviation for that age-sex group. Note that oral health is one of the few aspects of health for which a well-defined continuous index has been constructed.

Significant abnormalities (ABN1, ABN2) are defined to be heart disease; neurological, muscular, or joint conditions; other major diseases; and in Cycle III only, otitis media. This minor difference between the definitions of ABN1 and ABN2 will have little impact on our results because otitis media constitutes only a small percentage (about 1 percent) of all reported abnormalities in Cycle III.

In choosing these six particular health measures, our overriding consideration was diversity.[9] Indeed, it is the well-known multidimensional nature of health that led us to study a set of measures rather than a single composite index. Diversity is desired not only with respect to the systems of the body covered, but also with regard to the degree to which the health conditions can be affected by environmental influences. For example, both obesity and the periodental index are greatly affected by life style and preventive medical care. In the case of either of these measures, therefore, one would expect to observe a significant impact of family background variables. On the other hand, health problems like high blood pressure and significant abnormalities may not be responsive to family or medical intervention. Such measures may, however, have an impact on other aspects of health or on cognitive development. Subjective health measures like the parents' assessment of the child's health or school absenteeism have the advantage of reflecting people's perceptions about their health. But, at the same time, they may depend on the socioeconomic status of the family. For example, parents with low levels of income and schooling may be dissatisfied with many aspects of their lives

including the health of their offspring. (This type of reporting bias is largely controlled for in our analysis, however, because we hold constant both a group of socioeconomic variables and the lagged value of the subjective measure.) A secondary criterion used in choosing the health measures was prevalence. In particular, we avoided health problems like abnormal hearing that have a relatively low prevalence in this cohort.

D. Measurement of Other Variables

In addition to lagged (i.e. childhood) health and cognitive development, each equation includes the set of family and youth characteristics defined in Appendix Table 1. All family and youth characteristics are taken from Cycle II (except for the variable INTERVAL which measures the elapsed time between the child's two examinations). The child's age as of the Cycle II exam and/or his sex are also included when the dependent variable is not age and/or sex adjusted (that is, for ABN, PFGHEALTH, SCHABS, WISC, and WRAT).[10]

The rationale for including each of these youth and family characteristics variables has been discussed extensively elsewhere (Edwards and Grossman 1979, 1980, forthcoming) and will not be treated here. In the empirical section we discuss the effects of only the most important family background variables: mother's schooling (MEDUCAT), father's schooling (FEDUCAT), and family income (FINC). We view parents' schooling as representing the parents' efficiency in the production of their offspring's health and cognitive development, and family income as representing the family's command over resources.

III. Empirical Results

Ordinary least squares multiple regression equations for the dependent variables WISC2, WRAT2, APERI2, ABN2, HDBP2, PFGHEALTH2, OBESE2, and SCHABS2 are given in Tables A-2 through A-9 in the appendix. Since the six adolescent

health measures are negative correlates of good health, negative (positive) effects of family background and lagged cognitive development in the health equations reflect factors associated with better (poorer) health outcomes. Alternatively, positive coefficients of lagged health in the current health equations signify that poor health in childhood is associated with poor health in adolescence. Finally, negative coefficients of lagged health in the current cognitive development equations mean that poor health in childhood reduces cognitive development in adolescence.

Although five of the eight dependent variables are dichotomous, the method of estimation is ordinary least squares. Preliminary investigation revealed almost no differences between ordinary least squares estimates and dichotomous logit estimates. Given the size of our sample and the minimal improvement in the accuracy of the estimates, we decided to rely on OLS estimation. When the dependent variable is dichotomous, the estimated equation can be interpreted as a linear probabilty function.

A. Causal Priorness

In order to address the issue of the direction of causality between health and cognitive development, we present in Table 2 an 8 by 8 matrix of lagged coefficients from the 8 equations. The off-diagonal elements of the matrix provide information with regard to mutual feedback between health and cognitive development, mutual feedback between various health conditions, and mutual feedback between IQ (WISC) and achievement (WRAT). The elements on the main diagonal of the matrix are the own-lagged effects, or the regression coefficients of the lagged dependent variable.

We begin by looking at the own-lagged effects. The size of the own-lagged coefficients are an indication of the persistence of each health condition. For example, if the coefficient of the lagged dependent variable is

TABLE 2

Regression Coefficients of Lagged Health and Lagged Cognitive Development[a]

Current \ Lagged	WISC1	WRAT1	APERI1	ABN1	HDBP1	PFGHEALTH1	OBESE1	SCHABS1
WISC2	.603 (27.35)	.231 (9.47)	-.164 (-0.54)	-1.619 (-2.15)	-1.791 (-1.82)	.388 (0.84)	.946 (1.33)	-.650 (-0.53)
WRAT2	.192 (9.79)	.728 (33.52)	-.073 (-0.27)	-.204 (-0.30)	-.740 (-0.85)	-.341 (-0.82)	.421 (0.66)	-.699 (-0.64)
APERI2	-.004 (-2.25)	-.005 (-2.35)	.340 (12.16)	-.005 (-0.07)	-.195 (-2.15)	.039 (0.90)	.114 (1.73)	.146 (1.28)
ABN2	-.001 (-0.98)	-.002 (-2.00)	.020 (1.41)	.146 (4.16)	.042 (0.91)	.001 (0.03)	.049 (1.47)	.064 (1.11)
HDBP2	-.001 (-1.11)	-.0003 (-0.51)	-.003 (-0.41)	.030 (1.47)	.169 (6.38)	-.010 (-0.78)	.096 (4.97)	.033 (1.00)
PFGHEALTH2	-.001 (-0.92)	-.003 (-2.76)	.019 (1.22)	.139 (0.37)	.043 (0.86)	.243 (10.43)	.019 (0.52)	.096 (1.54)
OBESE2	.0001 (0.08)	-.001 (-1.45)	-.014 (-1.60)	-.020 (-0.91)	.013 (0.43)	-.016 (-1.15)	.512 (24.28)	.007 (0.18)
SCHABS2	.0002 (0.34)	-.001 (-1.78)	.004 (0.49)	.011 (0.49)	.009 (0.34)	.045 (3.45)	.020 (1.03)	.159 (4.60)

[a] t-ratios are in parentheses. The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test. Source: Appendix Tables A-2 through A-9.

close to one, this signifies that the health condition (or the stochastic process governing the occurrence of that condition) has a relatively low frequency and is slow to change. Coefficients close to zero indicate a higher frequency process. For slowly changing conditions one would expect to find that other explanatory variables (besides the lagged dependent variable) will not have as large effects as they would for conditions that are more readily altered. When the dependent variable is dichotomous, the own-lagged coefficient can be directly interpreted as the degree of persistence in the particular aspect of health in question: in this case the lagged coefficient is the difference between the expected conditional probability of an adolescent health condition given that the same condition was present in childhood and the conditional probability given that the condition was absent in childhood. Each of the eight own-lagged effects is positive and statistically significant at all conventional levels of confidence.[11] The coefficients range from a high of .73 in the case of WRAT to a low of .15 in the case of ABN.[12] Among the dichotomous variables, obesity is the most persistent: obese children have approximately 50 percentage point higher probabilities of being obese adolescents than do non-obese children.

The cross-lagged effects, however, appearing off the diagonal in Table 2, are the primary focus of this paper. From these coefficients, it appears that causality runs more strongly from cognitive development to health than vice versa. When the two cognitive development measures are the dependent variables, only two of the six health measures (ABN1 and HDBP1) have significant impacts on WISC2; and none have significant impacts on WRAT2 (the latter statement holds whether the statistical test is done on each health variable separately or on the set of six). In the two cases where there is a significant impact, the effect is as expected, with poorer health being associated with lower values of WISC2. When the health

measures are the dependent variables, one or both of the cognitive development measures have significant impacts for four of the six health measure: APERT2, ABN2, SCHABS2, and PFGHEALTH2 (these results hold whether the statistical test is done on WISC1 and WRAT1 separately or together). In all four cases, higher levels of WISC1 or WRAT1 are associated with better health. To conclude, while these off-diagonal elements affirm a two-way relationship between health and cognitive development, the link from cognitive development to health appears to be the stronger one.

Several other interesting relationships are evident in Table 2. There is evidence of mutual feedbacks between IQ and achievement: childhood achievement has a significant impact on adolescent IQ even when childhood IQ is held constant; and childhood IQ has a significant impact on adolescent achievement when childhood achievement is held constant. There are also dependencies between some of the health measures: obesity in childhood is related to poorer oral health and high blood pressure in adolescence,[13] and a parental rating of health in childhood as poor, fair, or good (as opposed to very good) is associated with excessive school absence due to illness in adolescence. Finally, there is one seemingly "perverse" and statistically significant relationship in the table: high blood pressure in childhood is associated with better oral health in adolescence.

B. Family Background Effects

A secondary objective of this paper is to obtain better estimates of the impacts of environmental factors on health and cognitive development. The three environmental measures we focus on are mother's schooling (MEDUCAT), father's schooling (FEDUCAT), and family income (FINC).

Coefficients of these three variables in the adolescent health and cognitive development functions are shown in Table 3. Two types of estimates are reported. Those in the first three columns, labeled cross-sectional coefficients,

TABLE 3

Regression Coefficients of Parent's Schooling and Family Income[a,b]

Dependent Variable \ Independent Variable	Cross-Section Coefficients			Dynamic Coefficients		
	MEDUCAT	FEDUCAT	FINC	MEDUCAT	FEDUCAT	FINC
WISC2	.986	.904	.288	.146	.207	.135
	(6.19)	(6.80)	(3.33)	(1.32)	(2.24)	(2.27)
WRAT2	.942	.805	.271	.177	.136	.103
	(6.03)	(6.18)	(3.20)	(1.79)	(1.65)	(1.94)
APERI2	-.039	-.019	0.005	-.023	-.006	.0001
	(-3.67)	(-2.17)	(-0.91)	(-2.25)	(-0.75)	(0.00)
ABN2	-.002	-.005	.004	.003	-.003	.005
	(-0.36)	(-1.26)	(1.60)	(0.51)	(-0.65)	(1.83)
HDBP2	-.005	.002	-.001	-.003	.003	-.001
	(-1.84)	(0.66)	(-0.33)	(-0.89)	(1.07)	(-0.53)
PFGHEALTH2	-.015	-.012	-.007	-.009	-.006	-.001
	(-2.71)	(-2.47)	(-2.21)	(-1.69)	(-1.23)	(-0.47)
OBESE2	-.012	.00002	.001	-.005	.001	.0004
	(-3.19)	(0.00)	(0.55)	(-1.42)	(0.53)	(0.21)
SCHABS2	-.010	.003	-.002	-.008	.004	-.001
	(-3.11)	(1.09)	(-1.32)	(-2.46)	(1.35)	(-0.66)

[a] t-ratios are in parentheses. The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test. The cross-sectional coefficients are taken from multiple regressions that contain all family and youth characteristics. The dynamic coefficients are taken from multiple regressions that contain all variables.

[b] Source: Appendix Tables A-2 through A-9 (dynamic estimates only).

are taken from multiple regressions that control for all of the family and youth characteristics listed in Appendix Table 1 but exclude all lagged (childhood) cognitive development and health measures. The estimates in the last three columns, labeled "dynamic" coefficients, are taken from multiple regressions that include all lagged cognitive development and health measures in addition to the family and youth characteristics. The first set of estimates shows background effects as typically computed in a cross-section. The second set shows background effects estimated in a dynamic context which controls for initial levels of cognitive development and health. As we argued in Section I, the "dynamic" estimates are free of genetic bias if genetic effects are fully embodied in the early health and cognitive development measures.[14] Under this assumption, then, the "dynamic" coefficients represent the pure contribution of the home environment to cognitive development and health outcomes in the interval between Cycles II and III.

Let us consider first the impacts of the three family background variables on cognitive development. In the cross-section estimates, all six family background coefficients are positive and statistically significant, and they tend to remain significant when the lagged variables are included. The magnitudes of the "dynamic" family background effects are, however, much smaller than the magnitudes of the cross-sectional effects. To be precise, the ratios of "dynamic" coefficients to the corresponding cross-sectional coefficients range from .15 in the case of mother's schooling in the WISC2 equation to .47 in the case of family income in the same equation.

In the case of adolescent health, the difference between cross-section and dynamic family background estimates is less dramatic. First, fewer of the cross-section estimates themselves show significant impacts: only mother's educational attainment is a consistently important variable (except when ABN2

is the dependent variable). Father's educational attainment has significant positive health impacts for the periodontal index and the subjective health rating, and family income is significant in determining only the subjective health rating. All of the statistically significant background effects are reduced in absolute value when childhood health and cognitive development are included in the equations. The ratios of the "dynamic" coefficients to the corresponding cross-sectional coefficients range from .14 in the case of family income in the PFGHEALTH2 equation to .80 in the case of mother's schooling in the SCHABS2 equation. Moreover, there are only three statistically significant dynamic coefficients: those belonging to mother's schooling in the APERI2, PFGHEALTH2, and SCHABS2 equations.

A clear message in Table 3 is that the "dynamic" estimates of family background effects on cognitive development and health are much smaller than the corresponding cross-sectional estimates. The important point here, however, is not that the "dynamic" estimates of background effects are smaller than the cross-sectional estimates. This decline was to be expected if our procedure does in fact remove much of the genetic effects otherwise embodied in the family background variables.[15] Rather, it is the fact that after removing the genetic component from the family background variables, family background, and especially mother's education, remains an important determinant of cognitive development and of some aspects of health. This finding is strong evidence that the family environment plays an important role in the overall development of adolescents.

An interesting sidelight to the discussion of family background effects is found in a comparison of the results for cognitive development versus health. First, regardless of which set of estimates are used, family background variables as a group are less likely to have significant impacts on adolescent health than on adolescent cognitive development. Second, according

to the "dynamic" estimates, either one year of additional educational attainment for either parent or one thousand additional dollars of family income are associated with roughly the same increase in WISC2 or WRAT2. For the health measures, however, the "dynamic" estimates show that mother's educational attainment tends to have a larger impact than the other variables, and it is frequently the only background variable to be statistically significant. Taken together, these points suggest that there is more "home production" of health than of cognitive development--at least in the period between childhood and adolescence.

IV. Summary and Implications

Our exploration of the dynamic relationship between health and cognitive development in adolescence has generated two important results. first, there is feedback both from health to cognitive development and from cognitive development to health, but the latter of these relationships is stronger. Second, estimates of family background effects taken from the dynamic model--which can be assumed to be less influenced by genetic factors--are smaller than their cross-sectional counterparts, but some still remain statistically significant.

The first finding calls attention to the existence of a continuing interaction between health and cognitive development over the life cycle. Since an individual's cognitive development (measured by IQ or achievement tests) is an important determinant of the number of years of formal schooling that he ultimately completes (see Grossman 1975), our findings may be viewed as the early forerunner of the positive impact of schooling on good health for adults in the United States reported by Grossman (1975), Shakotko (1977), and others.

The second finding suggests that nurture "matters" in cognitive development and health outcomes. All three background variables are important contributors to cognitive development, but mother's schooling is singled out as the crucial component of the home environment in adolescent health outcomes. This is an especially strong result because in the words of Keniston and the Carnegie Council on Children: "Doctors do not provide the bulk of health care for children; families do (1977, p. 179)." Since the mother spends more time in household production than the father, her characteristics should be the dominant factor in outcomes that are determined to a large extent in the home. The importance of mother's schooling in obesity and oral health is notable because these are outcomes that are neither irreversible or self-limiting. Instead, they can be modified by inputs of dental care, medical care, proper diet, and parents' time.

The two findings interact with each other. Cognitive development in childhood has a positive effect on health in adolescence, and cognitive development in childhood is positively related to parents' schooling and family income. Both findings imply that the health of adults is heavily dependent upon their home environment as youths. They also imply that public policies aimed at children's and adolescents' health must try to offset the problems encountered by offspring of mothers with low levels of schooling. In particular, they should try to improve the skills of uneducated mothers in their capacity as the main provider of health care for their offspring.

APPENDIX TABLE 1
Family and Youth Characteristics[a]

Variable Name	Sample[b] Mean	Sample Standard Deviation	Definition	Source[c]
FEDUCAT[a]	11.310	3.355	Years of formal schooling completed by father	1
MEDUCAT	11.216	2.704	Years of formal schooling completed by mother	1
FINC	8.060	4.607	Continuous family income (in thousands of dollars) computed by assigning mid-points to the following closed income intervals, $250 to the lowest interval, and $20,000 to the highest interval. The closed income classes are: $500 - $999 $1,000 - $1,999 $2,000 - $2,999 $3,000 - $3,999 $4,000 - $4,999 $5,000 - $6,999 $7,000 - $9,999 $10,000 - $14,999	1
LESS20	3.700	1.813	Number of persons in the household 20 years of age or less	1
MWORKFT MWORKPT	.149 .149	.356 .356	Dummy variables that equal one if the mother works full-time or part-time, respectively; omitted class is mother does not work	1
NEAST MWEST SOUTH	.265 .315 .203	.442 .465 .402	Dummy variables that equal one if youth lives in Northeast, Midwest, or South, respectively; omitted class is residence in West	1

(continued on next page)

APPENDIX TABLE 1 (continued)

Variable Name	Sample Mean[b]	Sample Standard Deviation	Definition	Source[c]
URB1 URB2 URB3 NURB	.189 .126 .200 .140	.392 .331 .400 .347	Dummy variables that equal one if youth lives in an urban area with a population of 3 million or more (URB1); in an urban area with a population between 1 million and 3 million (URB2); in an urban area with a population less than 1 million (URB3); or in a non-rural and non-urbanized area (NURB); omitted class is residence in a rural area	1
LIGHTA	.008	.091	Dummy variable that equals one if youth's birth weight was under 2,000 grams (under 4.4 pounds)	2
LIGHTB	.054	.227	Dummy variable that equals one if youth's birth weight was equal to or greater than 2,000 grams but under 2,500 grams (under 5.5 pounds)	2
BWUK	.138	.345	Dummy variable that equals one if youth's birth weight is unknown	2
FYPH	.068	.252	Dummy variable that equals one if parental assessment of child's health at one year was poor or fair and zero if it was good	1
BFED	.302	.459	Dummy variable that equals one if the child was breast fed	1
LMAG	.057	.231	Dummy variable that equals one if the mother was less than 20 years old at birth of youth	1
HMAG	.119	.324	Dummy variable that equals one if mother was more than 35 years old at birth of youth	1

(continued on next page)

APPENDIX TABLE 1 (concluded)

Variable Name	Sample[b] Mean	Sample Standard Deviation	Definition	Source[c]
NOFATH	.047	.213	Dummy variable that equals one if mother was more than 35 years old at birth of youth	1
FIRST	.292	.455	Dummy variable that equals one if youth is the first born family	1
TWIN	.028	.165	Dummy variable that equals one if youth is a twin	1
FLANG	.110	.312	Dummy variable that equals one if a foreign language is spoken in the home	1
MALE	.522	.500	Dummy variable that equals one if youth is a male	1
AGE	9.712	1.042	Age of youth	1
INTERVAL	42.327	6.404	Number of months between the physical examinations given for the Cycle II survey and the Cycle III survey	3

[a]All family and youth characteristics are from Cycle II unless otherwise stated.

[b]The means and standard deviations are for the sample of 1,434 white youths described in the text.

[c]The sources are 1 = parents, 2 = birth certificate, 3 = physical examination, 4 = psychological examination, 5 = school form.

APPENDIX TABLE 2

Ordinary Least Squares Regression of W1SC2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	.207	2.24	FIRST	.960	1.77
MEDUCAT	.146	1.32	TWIN	-2.177	-1.58
FINC	.135	2.27	FLANG	.643	0.85
LESS20	.138	0.97	MALE	2.674	5.95
MWORKPT	.965	1.53	AGE	-	-
MWORKFT	-.390	-0.61	INTERVAL	-.004	-0.09
NEAST	4.503	6.44	WISC1	.603	27.35
MWEST	2.297	3.64	WRAT1	.231	9.47
SOUTH	1.189	1.60	APERI1	-.164	-0.54
URB1	-1.428	-2.09	HDBP1	-1.791	-1.82
URB2	-.488	-0.65	PFGHEALTH1	.388	0.84
URB3	-.729	-1.15	OBESE1	.946	1.33
NURB	-.182	-0.25	SCHABS1	-.650	-0.53
LIGHTA	4.636	1.78	SCHABSUK1	.959	1.10
LIGHTB	.291	0.27	ABN1	-1.619	-2.15
BWUK	.235	0.34			
FYPH	-1.597	-1.81	CONSTANT	8.810	
BFED	1.174	2.36			
LMAG	.830	0.83	Adj. R^2	.658	
HMAG	.055	0.08			
NOFATH	1.908	1.81	F[b]	79.74	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

APPENDIX TABLE 3

Ordinary Least Squares Regression of WRAT2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	.136	1.65	FIRST	.125	0.26
MEDUCAT	.177	1.79	TWIN	-.950	-0.77
FINC	.103	1.94	FLANG	.285	0.42
LESS20	.067	0.53	MALE	-.739	-1.85
MWORKPT	.794	1.42	AGE	-	-
MWORKFT	.142	0.25	INTERVAL	-.058	-1.66
NEAST	4.089	6.57	WISC1	.192	9.79
MWEST	2.404	4.29	WRAT1	.728	33.52
SOUTH	1.526	2.30	APERI1	-.073	-0.27
URB1	-.874	-1.44	HDBP1	-.740	-0.85
URB2	.295	0.44	PFGHEALTH1	-.341	-0.82
URB3	1.799	3.19	OBESE1	.421	0.66
NURB	-.028	-0.04	SCHABS1	-.699	-0.64
LIGHTA	1.560	0.67	SCHABSUK1	.265	0.34
LIGHTB	.408	0.43	ABN1	-.204	-0.30
BWUK	-.829	-1.37			
FYPH	-.684	-0.87	CONSTANT	4.336	
BFED	1.081	2.45			
LMAG	.032	0.03	Adj. R^2	.712	
HMAG	1.334	2.16			
NOFATH	-.355	-0.38	F[b]	101.983	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

APPENDIX TABLE 4

Ordinary Least Squares Regression of APERI2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	-.006	-0.75	FIRST	.042	0.83
MEDUCAT	-.023	-2.25	TWIN	.176	1.39
FINC	.0001	0.00	FLANG	-.047	-0.67
LESS20	.023	1.77	MALE	-	-
MWORKPT	.001	0.00	AGE	-	-
MWORKFT	-.004	-0.07	INTERVAL	-.026	-7.03
NEAST	-.194	-3.02	WISC1	-.004	-2.25
MWEST	-.162	-2.79	WRAT1	-.005	-2.35
SOUTH	.012	0.17	APERI1	.340	12.16
URB1	.117	1.86	HDBP1	-.195	-2.15
URB2	.001	0.00	PFGHEALTH1	.039	0.90
URB3	-.023	-0.40	OBESE1	.114	1.73
NURB	.055	0.84	SCHABS1	.146	1.28
LIGHTA	-.371	-1.55	SCHABSUK1	.016	0.19
LIGHTB	-.078	-0.80	ABN1	-.005	-0.07
BWUK	.035	0.56			
FYPH	.067	0.82	CONSTANT	2.268	
BFED	-.072	-1.57			
LMAG	-.008	-0.09	Adj. R^2	.165	
HMAG	-.017	-0.26			
NOFATH	.106	1.39	F[b]	9.34	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

APPENDIX TABLE 5

Ordinary Least Squares Regression of ABN2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	-.003	-0.65	FIRST	-.013	-0.53
MEDUCAT	.003	0.51	TWIN	-.069	-1.08
FINC	.005	1.83	FLANG	-.029	-0.82
LESS20	-.010	-1.48	MALE	.006	0.27
MWORKPT	-.003	-0.12	AGE	.015	1.44
MWORKFT	.012	0.40	INTERVAL	-.003	-1.40
NEAST	-.057	-1.75	WISC1	-.001	-0.98
MWEST	.014	0.47	WRAT1	-.002	-2.00
SOUTH	.096	2.77	APERI1	.020	1.41
URB1	.077	2.42	HDBP1	.042	0.91
URB2	-.058	-1.65	PFGHEALTH1	.001	0.03
URB3	.052	1.77	OBESE1	.049	1.47
NURB	.006	0.17	SCHABS1	.064	1.11
LIGHTA	-.013	-0.10	SCHABSUK1	.006	0.15
LIGHTB	-.027	-0.54	ABN1	.146	4.16
BWUK	-.012	-0.38			
FYPH	.143	3.45	CONSTANT	.430	
BFED	.040	1.74			
LMAG	-.036	-0.76	Adj. R^2	.043	
HMAG	.030	0.92			
NOFATH	.007	0.14	F[b]	2.78	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

APPENDIX TABLE 6

Ordinary Least Squares Regression of HDBP2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	.003	1.07	FIRST	.013	0.87
MEDUCAT	-.003	-0.89	TWIN	.034	0.92
FINC	-.001	-0.53	FLANG	.035	1.73
LESS20	-.001	-0.33	MALE	-	-
MWORKPT	-.009	-0.50	AGE	-	-
MWORKFT	-.007	-0.41	INTERVAL	-.002	-1.62
NEAST	.014	0.77	WISC1	-.001	-1.11
MWEST	.021	1.22	WRAT1	-.0003	-0.51
SOUTH	.049	2.43	APERI1	-.003	-0.41
URB1	.041	2.22	HDBP1	.169	6.38
URB2	.030	1.49	PFGHEALTH1	-.010	-0.78
URB3	.032	1.84	OBESE1	.096	4.97
NURB	.011	0.56	SCHABS1	.033	1.00
LIGHTA	.012	0.17	SCHABSUK1	.018	0.75
LIGHTB	-.005	-0.17	ABN1	.030	1.47
BWUK	.026	1.39			
FYPH	.0004	0.00	CONSTANT	.168	
BFED	.003	0.24			
LMAG	-.019	-0.71	Adj. R^2	.051	
HMAG	.015	0.82			
NOFATH	.027	0.96	F[b]	3.28	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

APPENDIX TABLE 7

Ordinary Least Squares Regression of PFGHEALTH2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	-.006	-1.23	FIRST	.008	0.29
MEDUCAT	-.009	-1.69	TWIN	-.140	-2.02
FINC	-.001	-0.47	FLANG	-.029	-0.77
LESS20	.009	1.20	MALE	-.002	-0.10
MWORKPT	.031	0.97	AGE	.010	0.92
MWORKFT	-.015	-0.48	INTERVAL	.001	0.31
NEAST	-.024	-0.69	WISC1	-.001	-0.92
MWEST	-.046	-1.44	WRAT1	-.003	-2.76
SOUTH	-.025	-0.66	APERI1	.019	1.22
URB1	.022	0.65	HDBP1	.043	0.86
URB2	.007	0.19	PFGHEALTH1	.243	10.43
URB3	.039	1.23	OBESE1	.019	0.52
NURB	.048	1.34	SCHABS1	.096	1.54
LIGHTA	-.239	-1.82	SCHABSUK1	-.052	-1.20
LIGHTB	.103	1.91	ABN1	.139	0.37
BWUK	-.015	-0.45			
FYPH	.153	3.45	CONSTANT	.639	
BFED	-.020	-0.81			
LMAG	.047	0.94	Adj. R^2	.147	
HMAG	-.002	-0.04			
NOFATH	.062	1.16	F[b]	7.86	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

APPENDIX TABLE 8

Ordinary Least Squares Regression of OBESE2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	.001	0.53	FIRST	-.002	-0.15
MEDUCAT	-.005	-1.42	TWIN	-.013	-0.32
FINC	.0004	0.21	FLANG	-.001	-0.06
LESS20	-.007	-1.73	MALE	-	-
MWORKPT	.008	0.45	AGE	-	-
MWORKFT	.027	1.41	INTERVAL	-.001	-1.07
NEAST	-.009	-0.45	WISC1	.0001	0.08
MWEST	-.011	-0.59	WRAT1	-.001	-1.45
SOUTH	-.012	-0.56	APERI1	-.014	-1.60
URB1	-.021	-1.05	HDBP1	.013	0.43
URB2	-.029	-1.28	PFGHEALTH1	-.016	-1.15
URB3	-.011	-0.61	OBESE1	.512	24.28
NURB	-.007	-0.31	SCHABS1	.007	0.18
LIGHTA	-.035	-0.46	SCHABSUK1	-.034	-1.34
LIGHTB	.020	0.64	ABN1	-.020	-0.91
BWUK	-.005	-0.26			
FYPH	.024	0.91	CONSTANT	.284	
BFED	-.014	-0.99			
LMAG	-.016	-0.53	Adj. R^2	.314	
HMAG	.058	2.81			
NOFATH	-.022	-0.70	F[b]	20.30	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

APPENDIX TABLE 9

Ordinary Least Squares Regressions of SCHABS2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	.004	1.35	FIRST	-.005	-0.32
MEDUCAT	-.008	-2.46	TWIN	-.017	-0.44
FINC	-.001	-0.66	FLANG	-.004	-0.20
LESS20	-.005	-1.14	MALE	-.019	-1.52
MWORKPT	-.014	-0.79	AGE	.017	2.69
MWORKFT	.008	0.43	INTERVAL	.001	0.76
NEAST	-.005	-0.26	WISC1	.0002	0.34
MWEST	-.010	-0.56	WRAT1	-.001	-1.78
SOUTH	.004	0.20	APERI1	.004	0.49
URB1	-.009	-0.47	HDBP1	.009	0.34
URB2	-.020	-0.96	PFGHEALTH1	.045	3.45
URB3	-.001	-0.06	OBESE1	.020	1.03
NURB	-.057	-2.87	SCHABS1	.159	4.60
LIGHTA	-.078	-1.09	SCHABSUK1	-.015	-0.62
LIGHTB	-.005	-0.17	ABN1	.011	0.49
BWUK	-.015	-0.77			
FYPH	.018	0.73	CONSTANT	.045	
BFED	-.013	-0.91			
LMAG	-.038	-1.34	Adj. R^2	.043	
HMAG	-.026	-1.31			
NOFATH	.024	0.81	F[b]	2.63	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

FOOTNOTES

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[1]This is a reduced form equation derived by solving a system of equations that include a family utility function (with the health and cognitive development of each child in each period as arguments), a children's health production function, a production function for children's cognitive development, and a wealth constraint. Note that at any point in time, t, both $y_{i,t-1}$ and $x_{i,t-1}$ are predetermined variables.

[2]A detailed discussion of the types of variables included in $x_{i,t}$ can be found in Edwards and Grossman (1979 and 1980).

[3]One technique that has been used in cross-sectional analysis is to include indicators of the unobserved variable. These indicators, which are not themselves part of the original cross-section specification, are taken to be instruments for the unobserved variable. An example is the inclusion of test scores as a proxy for ability in earnings equations. Investigators generally acknowledge that this is a second-best procedure because it introduces an errors-in-variables bias which may be nearly as large as the original omitted-variables bias [Griliches (1974)].

[4]See Shakotko (1979) for an alternative model formulated in the spirit of the ability-bias problem as described, for example, by Griliches (1977).

While relaxing the restrictive assumption in the present paper regarding genetic embodiment, Shakotko requires an alternative set of restrictions in order to identify and estimate a factor structure.

[5]Since H_1 is correlated with the error term in equation (2c), the coefficient of Q_1 in this equation is biased unless the partial correlation between Q_1 and H_1 with E held constant is zero. This is extremely unlikely because GQ and GH are bound to be related, probably, in a positive manner. The same comment applies to the coefficient of H_1 in equation (3c). Note that if the partial correlation between E and H_1 or between E and Q_1 is non-zero, ordinary least squares of the reduced form environmental parameters, given by the coefficients of E in (2c) or (3c), are biased.

[6]We did not, however, exclude observations from the analysis if data were missing for the school absenteeism variables (SCHABS1, SCHABS2) and birth weight variables (LIGHTA, LIGHTB). (These variables are described in Section II-C below). Information on school absenteeism is taken from the school form completed by the child's school. This form is missing for roughly 7 percent of the sample. Since excessive absence due to illness is the only variable taken from this form, a dummy variable that identifies youths with missing Cycle II school forms (SCHABSUK1) is included in all regression equations as an independent variable. Youths without a Cycle III school form are eliminated from the empirical analysis only when SCHABS2 is the dependent variable. Birth weight is taken from the child's birth certificate, which is missing for 14 percent of the sample. Since birth weight is the only variable taken from the birth certificate, we do not delete these observations, but rather we include a dummy variable that identifies youths with missing birth certificates (BWUK) in the regression equations.

[7]Although these and other test scores have been widely criticized, they are used here and elsewhere because they are so readily obtainable and because they roughly comparable across diverse populations. WISC and WRAT are adjusted for sex as well as for age in some studies, but the variables used here are not sex-adjusted.

[8]Kelly and Sanchez (1972, pp. 1-2) describe the periodontal index as follows:

> Every tooth in the mouth ... is scored according to the presence or absence of manifest signs of periodontal disease. When a portion of the free gingiva is inflamed, a score of 1 is recorded. When completely circumscribed by inflammation, teeth are scored 2. Teeth with frank periodontal pockets are scored 6 when their masticatory function is unimpaired and 8 when it is impaired. The arithmetic average of all scores is the individual's [periodontal index], which ranges from a low of 0.0 (no inflammation or periodontal pockets] to a high of 8.0 (all teeth with pockets and impaired function).

[9]The choice of appropriate measures of health in childhood and adolescence is discussed in detail in Edwards and Grossman (1979, 1980, and forthcoming).

[10]The periodontal index and the two cognitive development measures are continuous variables. In these cases we have experimented with the raw score as the dependent variable in a multiple regression that includes in the set of explanatory variables age in Cycle II, the square of age, the time interval between the Cycle II and III examinations, the square of the interval, the product of age and the interval, and a dummy variable for male adolescents. The results obtained (not shown) with respect to family background, lagged health, and lagged cognitive development effects are similar to those reported in Section III.

[11]Statements concerning statistical significance in the text refer to the 5 percent level in a one-tailed test except when the direction of the effect is unclear on a priori grounds or when the estimated effect has the "wrong sign." In the latter cases two-tailed tests are used.

[12]If the dynamic processes that we study have the same structures over time and if cross-lagged effects are ignored, they all have stable long-run solutions. To be specific, if $H = aH_{t-1} + bE$, the long-run solution, obtained by setting $H_t = H_{t-1}$, is $H_t = (b/1-a)\ E$. This is a stable solution when a is positive and smaller than one.

[13]This finding is consistent with cross-sectional results reported by the 1977 National Heart, Lung, and Blood Institute's Task Force. The Task Force points out that obesity is a risk factor in the incidence of high blood pressure in adolescents.

[14]Some evidence supporting the validity of this assumption appears in Appendix Tables A2 through A9. In particular, the coefficients of birth weight, mother's age at the birth of the youth, and parental assessment of the youth's health in the first year of his life are almost never statistically significant. These variables are proxy measures of the genetic endowment. If they had had large significant impacts in the dynamic equations, this would have thrown into question the validity of our assumption.

[15]Even if the family background variables had no genetic components, we would still expect the "dynamic" coefficients to be smaller than the cross-sectional coefficients because the "dynamic" estimates represent short-run effects in the sense that they hold constant the lagged values of health and cognitive development. Since these lagged values themselves depend on family background, the cumulative or long-run impacts of family background

are likely to exceed the "dynamic" or short-run impacts. To be precise, if cross-lagged effects are ignored, a full representation of the dymamic health process that we study is (ignoring stochastic terms):

$$H_1 = \alpha GH + b_1 E, \text{ and}$$

$$H_t = a_t H_{t-1} + b_t E, \quad t=2,\ldots, n.$$

Solving recursively, one obtains

$$H_t = \left[\prod_{i=1}^{t} a_i\right] GH + \left[b_t + \sum_{i=1}^{t-1} b_i \prod_{j=t-1}^{i} a_j\right] E.$$

The parameter of E in the above equation is the cumulative environmental effect. If the b_i all have the same sign, the long-run parameter unambiguously exceeds b_t in absolute value. Of course, the long-run parameter estimate may be larger or smaller than the cross-sectional estimate if GH is omitted from the equation.

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