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ADOLESCENT HEALTH, FAMILY BACKGROUND, AND PREVENTIVE MEDICAL CARE

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ABSTRACT

This paper investigates the health of white adolescents, focusing particularly on the roles of family background and preventive medical care. This emphasis is motivated in part by our desire to study adolescent health in the context of the nature-nurture controversy. The findings indicate first, that family characteristics (especially mother's schooling) do have a significant impact on adolescent health and second, that preventive care is an important vehicle for this impact in the case of dental health but not in the case of physical health measures. Similarly, the greater availability of dentists has a positive impact on dental health, but greater availability of pediatricians does not alter the physical health measures. On the basis of these results we predict that goverment efforts to improve the dental health of adolescents with policies to lower the cost of dental care or increase the availability of dentists are much more likely to be successful than similar policies directed at improving their physical health.

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Linda N. Edwards and Michael Grossman*

This paper investigates the health of white adolescents, focusing particularly on the roles of family background and preventive medical care. This emphasis is motivated in part by our desire to study adolescent health in the context of the nature-nurture controversy. Despite the existence of a massive literature on the relative importance of heredity (nature) and the home and school environment (nurture) in the determination of cognitive development,[1] the corresponding issue has not been directly addressed by researchers in child and adolescent health. This is partly because much of the health research is limited either to poverty or to minority populations (Hu 1973; Kessner 1974; Inman 1976; Dutton 1978; Dutton and Silber 1979), and partly because researchers who use representative samples do not adopt the multivariate context necessary for distinguishing between genetic and environmental influences (Douglas 1951; Douglas and Bloomfield 1958; Kellmer-Pringle, Butler, and Davie 1966; Haggerty, Roghmann, and Pless 1975; Zimmer 1978). Our research uses multivariate statistical techniques to provide some evidence of the degree to which nurture--that is, the family and local environment--acts in determining the health levels of a representative sample of white adolescents.

One aspect of the adolescent's environment, medical care, has been recognized as the logical vehicle for public policy aimed at improving adolescent health. For example, Newberger, Newberger, and Richmond (1976), Keniston and the Carngeie Council on Children (1977), and Marmor (1977) all have proposed that national health insurance should provide coverage of

prenatal care, pediatric care, and dental care. Bills with this aim have been introduced in Congress by Senator Jacob K. Javits and Congressman James H. Scheuer, both of New York. To cite another illustration, recently-enacted Federal legislation has attempted to increase the availability of pediatricians and dentists in medically underserved areas to expand the use of preventive care in such areas. The Emergency Health Personnel Act of 1970 (PL 91-623) created the National Health Service Corps., whose members are assigned to health manpower shortage areas. The Health Professions Assistance Act of 1976 (PL 94-484) encourages new graduates of medical and dental schools to locate in urban ghettos and rural regions by forgiving their medical education loan obligations. Further, the Health Maintenance Organization Act of 1974 (PL 93-222) gives priority for developmental funding of HMOs in medically deprived areas. One objective of our research is to provide estimates of the potential payoffs to national health insurance and medical manpower policies directed at improving youths' health.

The specific health indicators we study are oral health, obesity, anemia, and corrected distance vision. These four are chosen not only because they represent health problems that create discomfort for the teenager, but more importantly, because they may be good predictors of subsequent adult health. Indeed, they all partly reflect poor health habits that are likely to persist into adulthood. With the growing evidence that adults' choice of life styles and health behaviors can have important impacts on their health (Breslow and Klein 1971; Fuchs 1974a, 1974b; Grossman 1975; Manheim 1975), it is natural to look into adolescence to understand the formation of these habits. A second motivation

for choosing these indicators is that they represent health problems that are capable of being affected by family decisions concerning diet and other forms of at-home health care, as well as by pediatric and dental care. This is in contrast to many adolescent health problems that are either self-limiting, such as morbidity from acute conditions, or irreversible, such as congenital abnormalities of the neurological system.

To analyze these health problems we use data from Cycle III of the U.S. Health Examination Survey (HES), an exceptional source of information about a national sample of 6,768 noninstitutionalized youths aged 12 to 17 years in the 1966-70 period.[2] The data comprise complete medical histories of each youth provided by the parent, information on family socioeconomic characteristics, and birth certificate information. Most important, there are objective measures of health from detailed physical examinations given to the youths by pediatricians and dentists employed by the Public Health Service. These data are supplemented by two medical resource inputs specific to the youth's county of residence (the number of pediatricians per capita and the number of dentists per capita) and information on the presence of controlled or natural fluorides in the water supply system that services the youth's community. The last piece of information enables us to evaluate the impact of a collective, as opposed to an individual, preventive dental practice.

These data are used to estimate two types of relations: a health production function and a derived demand function for preventive care. The resulting estimates permit us to answer the following four questions. What is the size of the home environmental effect on adolescent oral and physical (obesity, anemia, corrected distance vision) health outcomes?

How important is the home environment as a determinant of the demand for preventive dental and pediatric care? How large are the effects of dentists, preventive dental care, and fluoridation on oral health outcomes? How large are the effects of pediatricians and preventive pediatric care on physical health outcomes? In addressing the last two questions, we recognize explicitly the common-sense proposition that an increase in a community's physician or dental manpower will not increase health outcomes unless it encourages more utilization of medical care services. Previous empirical work on the impact of physicians or dentists on health has not taken account of this restriction (for example, Newhouse and Friedlander 1977).

Our findings indicate first, that family characteristics do have a significant impact on adolescent health and second, that preventive care is an important vehicle for this impact in the case of dental health but not in the case of the three physical health measures. Similarly, the greater availability of dentists has a positive impact on dental health, but greater availability of pediatricians does not alter the physical health measures. On the basis of these results we predict that government efforts to improve the dental health of adolescents with policies to lower the cost of dental care or increase the availability of dentists are much more likely to be successful than similar policies directed at improving their physical health.

I. Analytical Framework

In a previous paper (Edwards and Grossman 1980), we have argued that offsprings' health can be examined fruitfully within the context of the economic models of fertility developed by Becker and Lewis (1973), Willis

(1973), and Ben Porath and Welch (1976). In these models the parents' utility function depends on their own consumption, their family size, and the "quality" of each child. Child "quality" refers to those characteristics of the child that generate utility for the parents: his health, sex, wealth, social adjustment, intellectual development, etc. Therefore, when parents choose their optimal family composition, they choose not only how many children they will have but also what portion of the family's resources will be devoted to each child. This choice is made in the usual way: parents choose the number and quality of children, as well as of other consumption goods, so as to maximize their utility subject to the constraints imposed by their wealth (their potential earned and nonearned income) and the various prices they face. In the case of children, there is a further constraint in the form of children's genetic endowments which in part determine their quality. Genetic endowments act as a constraint because they are largely outside of the family's control.

The prices of children and of the various components of their quality are determined by a fundamental insight embedded in the household production function approach to consumer behavior: consumers produce their basic objects of choice with inputs of goods and services purchased in the market and their own time (Becker 1965). This insight is of particular relevance in dealing with children and their health because parents obviously do not buy these objects of choice directly in the market; both a child's home environment and his genetic endowment are important determinants of his ultimate health level. Therefore, the price of health depends on the cost of the parents' or other caretakers' time, and the prices of medical care, nutrition, and any other purchased inputs used to

improve children's health. It also depends on the number of children in the family because the more children there are in the family, the more costly it is to raise their average health level. In addition, to the extent that there are systematic differences in the ability of families to produce children's health with given inputs, these differences in efficiency are also relevant. For example, more educated parents are more likely to be able to follow doctors' instructions, to have general information about nutrition, and to be willing and able to acquire medical information from published materials. Consequently, one would expect more educated parents to be more efficient in producing healthy children.

Given these considerations, the following factors are expected to influence children's health levels: the child's exogenous (genetic) health endowment, family wealth, parents' wage rates, family size, parents' educational attainment and other measures of their efficiency in household production, and the direct and indirect costs of medical care and other market health inputs (vitamins, sanitation, etc.).[3] (The indirect costs of medical care are generated by the time spent in traveling, waiting, and obtaining information about this care.[4]) The relationship between the child's ultimate health and this set of factors may be termed a demand function for the output of health. In this demand function a positive association between children's health and family wealth is predicted (assuming that child health is a normal good). Similarly, a positive association is expected between both parents' education and children's endowed health status and children's ultimate health status. Negative associations would be anticipated between all of the prices of health inputs and children's health, and between family size and children's health. Parents' wage rates may have negative or positive effects

on children's health levels depending on whether the household production of children's health is more or less time intensive than the production of other aspects of child quality and/or other types of parents' consumption commodities. In this framework a child's health is treated as a single datum--his permanent health measured, say, at the beginning of adulthood or as an average over his childhood and adolescence. This type of model is not formulated to explain variations in health over childhood or to examine the child's contribution to his own health.[5]

The above model provides a useful setting within which to view adolescent health, but empirical estimation of the resultant "demand for health" function would not yield answers to the questions posed in the introduction. Such estimates would only yield information about the total impact of family characteristics or medical input prices on children's health. To determine the effect of preventive care on health we need estimates of the health production function. Similarly, to determine whether families with specific characteristics are more efficient at producing healthy children also requires estimates of this production function. Alternatively, to assess the role of family characteristics in determining the amount of preventive care received by adolescents, an estimate of a derived demand function for medical care is needed. Finally, a computation of the impact of health manpower availability on adolescent health requires not only the above functions but also a set of market demand and supply for health manpower functions. In the latter case, we employ a simplified approach which yields rough estimates of these manpower availability effects on health.

A. The Health Production Function

A simple, linear health production function[6] is represented by

$$(1) \qquad H = \beta_0 + \beta_1 E + \beta_2 G + \beta_3 M + \beta_4 X + \beta_5 R + u_1 \quad .$$

Here H is a health measure, E is a vector of family efficiency characteristics, G is a vector of the adolescent's endowed health characteristics, M is a medical or dental care input, X is a vector of other family inputs (nutrition, parents' time, etc.), R is a vector of relevant regional characteristics (city size, region of the country, and whether or not the water supply is fluoridated), and u_1 is a random error term with the usual properties.

The health production function actually estimated in Section III does not correspond exactly to equation (1) because of inadequate data. First, data on the amount of "other" inputs (X) are not available. Therefore, we include the following proxy measures for X: family income, family size, and the mother's labor force status. Family income is positively related and family size is negatively related to nutrition and other unmeasured market health inputs. Family size and mother's labor force status are proxies for the amount of time the mother spends with each of her offspring. Women who work full-time or part-time in the labor market and women with many offspring have less time to spend with each one. In addition, our data do not include good information about curative care. Consequently, M represents only preventive care. This is not a serious deficiency because we have chosen health measures for which the impact of preventive care (with the associated remedial treatment) is

relatively large. (By focusing on health problems for which the medical input is primarily preventive, we also avoid the necessity of modeling the simultaneous determination of health levels and curative care utilization.)

B. The Derived Demand for Preventive Care

The derived demand function for medical care depends on the same set of variables as the demand function for health:

$$M = \gamma_0 + \gamma_1 F + \gamma_2 G + \gamma_3 P + \gamma_4 R + u_2 \quad . \tag{2}$$

F represents family income, education, family size, and other family characteristics affecting either the demand for health or the family's efficiency in producing healthy children; G and R are the same as in equation (1); P represents a vector of relevant direct and indirect input prices (wage rates, the cost of a doctor or dental visit, etc.); and u_2 is the usual random error term.

We cannot estimate this derived demand curve exactly as stated because data on P are not available. Inclusion of variables representing the mother's labor force status helps control for variations in the mother's wate rate. Other input prices are partially controlled for by the region and city-size variables in R. Finally, physician or dentist availability measures are included to represent differences in the direct and indirect costs of medical or dental care.[7] Thus, rather than equation (2), we estimate the following:

$$M = \alpha_0 + \alpha_1 F + \alpha_2 G + \alpha_3 D + \alpha_4 R + u_3 \quad . \tag{3}$$

where the vector F now includes the mother's labor force status and D

represents the number of pediatricians or dentists per capita in the adolescent's county of residence.

C. The Role of Health Manpower Availability

It is the inclusion of manpower availability measures in the derived demand for preventive care functions that permits us to obtain a rough assessment of the impact of health manpower on the demand for preventive care, and consequently, on adolescent health. Only a rough assessment is possible because to get precise estimates it is necessary to have, first, data on the direct and indirect costs of medical care and second, measures of the price elasticity of supply of physicians or dentists. Good estimates of the supply elasticities do not exist, and it is almost impossible to measure all of the indirect costs of medical care. Although data on direct costs do exist, they are not usually found in conjunction with the detailed health and family background data used here. Thus, our estimate of the impact of health manpower on health is the best that can be obtained given the limitations of existing data sets. The coefficients of the health manpower variables in the derived demand equations embody both the relationship between health manpower availability and direct and indirect medical care prices, and the relationship between medical care prices and the demand for preventive care.

Implicit in the above discussion is the assumption that an increase in a community's health manpower will not improve the health of adolescents unless it encourages a greater utilization of preventive care services. This assumption is explicitly incorporated in equations (1) and

(3): D is assumed to have no direct effect on health in equation (1) but alters health only via its impact on M in equation (3). Substituting equation (3) into equation (1) yields estimates of the total impact of doctor or dentist availability on health:

$$(4) \qquad H = \beta_0 + \beta_1 E + \beta_3 \alpha_1 F + (\beta_2 + \beta_3 \alpha_2) G + \beta_3 \alpha_3 D$$

$$\beta_4 X + (\beta_5 + \beta_3 \alpha_4) R + u_1 + \beta_3 u_3 \quad .$$

The total impact of pediatrician or dentist availability on health is given by $\beta_3 \alpha_3$. Note that an estimate of the total impact computed from individual estimates of α_3 and β_3 differs from that obtained from direct estimation of equation (4) because the latter does not incorporate the restriction that D does not appear in equation (1).[8]

D. The Role of Family Background Variables

To the extent that there are family background variables common to both the set E and the set F (parents educational attainment is a good example of one), the substitution in equation (4) provides an additional insight. Parents' education is clearly seen to have two effects on adolescent health: a direct or "efficiency" effect given by β_1 and an indirect or "allocative" effect given by $\beta_3 \alpha_1$. The latter refers to the ability of parents with greater schooling levels to select a better input mix in the production function.[9]

II. Empirical Implementation

Equations (1) and (3) are estimated using Cycle III data for white adolescents who live with either both of their parents or with their mothers only. Black adolescents are excluded from the empirical analysis. Preliminary results revealed significant race differences in slope coefficients so that pooling blacks and whites for estimation was inappropriate. Separate estimates for black adolescents are not presented because the black sample is too small to allow for reliable coefficient estimates. Observations are also deleted if there are missing data. The final sample size is 4,121. Table 1 contains definitions, means, and standard deviations of all of the dependent and independent variables. It also contains a notation concerning the source of each variable.

A. Measurement of Adolescent Health

In the introduction to this paper, we expressed an intention to study physiological measures of adolescent health that (1) reflect detrimental health behaviors or life styles that may persist and create more serious problems in adulthood and (2) relate to problems that can be modified by endogenous inputs in the health production function such as proper diet, parents' time, and especially preventive medical care.[10] Based on these criteria, we focus on two correlates of poor oral health: the periodontal index and the number of decayed permanent teeth; and on three correlates of poor physical health: obesity, abnormal corrected distance vision, and anemia as reflected by low hematocrit levels. All five measures clearly relate to conditions that can carry on into adulthood, and all can be modified by appropriate care. Dental care provided

TABLE 1

Definitions of Variables

Variable Name	Sample[a] Mean	Sample Standard Deviation	Definition	Source[b]
A. Health Measures				
APERI[c]	-.114	.857	Periodontal index, standardized by the mean and standard deviation of one-year age-sex cohorts	2
IDECAY[c]	-.146	.839	Number of decayed permanent teeth, standardized by the mean and standard deviation of one-year age-sex cohorts	2
OBESE	.103	.305	Dummy variable that equals one if the physician rates the youth as obese or very obese	2
PVIS	.042	.201	Dummy variable that equals one if youth wears glasses and his corrected binocular distance vision is 20/40 or worse or if youth does not wear glasses and his uncorrected binocular distance vision is 20/40 or worse	2
ANEMIA	.023	.149	Dummy variable that equals one if youth is a female whose hematocrit level is more than two standard deviations below the mean for females 12 to 17 years of age or if youth is a male whose hematocrit level is more than two standard deviations below the mean for his stage of sexual maturity	2
B. Preventive Medical Care Measures				
DTPREV	.697	.460	Dummy variable that equals one if youth saw a dentist for a check-up within the past year	1

TABLE 1 (continued)

Variable Name	Sample[a] Mean	Sample Standard Deviation	Definition	Source[b]
DRPREV	.588	.492	Dummy variable that equals one if youth saw a doctor for a check-up within the past year	1
FLUOR	.584	.493	Dummy variable that equals one if the community in which the youth lives uses naturally fluoridated or controlled fluoridated water	See text
C. Other Variables				
FINC	9.614	5.112	Continuous family income (in thousands of dollars) computed by assigning midpoints 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
FEDUCAT[d]	11.327	3.227	Years of formal schooling completed by father	1
MEDUCAT	11.142	2.843	Years of formal schooling completed by mother	1
NOFATH	.099	.297	Dummy variable that equals one if youth lives with mother only	1
FLANG	.139	.346	Dummy variable that equals one if a foreign language is spoken in the home	1
LESS20	3.360	1.853	Number of persons in the household 20 years of age or less	1

TABLE 1 (continued)

Variable Name	Sample[a] Mean	Sample Standard Deviation	Definition	Source[b]
MWORKFT MWORKPT	.268 .154	.443 .361	Dummy variable that equals one if the mother works full-time or part-time, respectively; omitted class is mother does not work	1
DENT	.584	.216	Number of dentists per thousand population in community of residence of youth	See text
PED	.051	.027	Number of pediatricians per thousand population in community of residence of youth	See text
NEAST MWEST SOUTH	.253 .291 .203	.435 .454 .402	Dummy variables that equal one if youth lives in Northeast, Midwest, or South, respectively; omitted class is residence in West	1
URB1 URB2 URB3 NURB	.193 .132 .194 .146	.395 .339 .396 .353	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
LMAG	.077	.267	Dummy variable that equals one if the mother was less than 20 years-old at birth of youth	1

TABLE 1 (continued)

Variable Name	Sample[a] Mean	Sample Standard Deviation	Definition	Source[b]
HMAG	.096	.294	Dummy variable that equals one if mother was more than 35 years-old at birth of youth	1
LIGHT1	.010	.098	Dummy variable that equals one if youth's birth weight was under 2,000 grams (under 4.4 pounds)	3
LIGHT2	.032	.177	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)	3
BWUK	.245	.430	Dummy variable that equals one if youth's birth weight is unknown	3
FYPH	.117	.321	Dummy variable that equals one if there was a medical difficulty with youth before the age of one year	1
ABN	.200	.400	Dummy variable that equals one if the diagonistic impression of the physician was that the youth had a significant abnormality	2
TWIN	.023	.150	Dummy variable that equals one if youth is a twin	1
FIRST	.497	.500	Dummy variable that equals one if youth is the first born in the family	1
AGE	14.335	1.661	Age of youth	1
MALE	.528	.499	Dummy variable that equals one if youth is a male	1

Footnotes to TABLE 1

[a]The means and standard deviations are computed for the sample of 4,121 white youths described in the text.

[b]The sources are 1 = parents, 2 = examination, 3 = birth certificate. See text for sources of FLUOR, PED and DENT.

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

[d]For youths who were not currently living with their father, father's education was coded at the mean of the sample for which father's education was reported.

by dentists has a direct impact on tooth decay and periodontal disease. The prescription of eyeglasses by an ophthalmologist or an optometrist can remedy abnormal distance vision. Pediatricians also play an important role in eye care because they often are responsible for examining a youth's eyes initially and referring his parents to an eye specialist if necessary. Finally, all of the health measures excluding vision reflect basic nutritional factors that can be modified by the appropriate diet. These measures are described in detail below.

The periodontal index (APERI) is a good overall indicator of oral health as well as a positive correlate of nutrition (Russell 1956). 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).

It is clear from this description that higher values of the periodontal index correspond to poorer dental health. Our measure, APERI, is scaled somewhat differently from that described above in order to remove the well-known age and sex trends in the periodontal index. APERI is computed as the difference between the adolescent's actual periodontal index and the mean index for his or her age-sex group divided by the standard deviation for that age-sex group.[11] A similar method of age and sex

standardization is used for our other measure of oral health, the number of decayed permanent teeth (IDECAY). We employ two measures of dental health because it is one of the few health problems for which well-defined continuous health measures have been developed.

Obesity is represented by a dichotomous variable that equals one if the physician rates the youth as obese or very obese (OBESE). The physician presumably takes account of the youth's height, age, and sex in making his evaluation.

Anemia is represented by a dichotomous variable that equals one if the youth's hematocrit level is "excessively" low (ANEMIA).[12] The hematocrit level of a female youth is considered to be excessively low if it is more than two standard deviations below the mean for all females 12 to 17 years of age. The hematocrit level of a male youth is considered to be excessively low if it is more than two standard deviations below the mean for all males in his stage of sexual maturity. This procedure is based on Daniel's (1973) findings that (1) hematocrit values differ by sex; (2) these values depend on sexual maturity rather than age for male adolescents; and (3) hematocrit levels are independent of age and sexual maturity for female adolescents.[13]

Abnormal corrected distance vision is denoted by a dichotomous variable that equals one if a youth wears glasses and his corrected binocular distance vision is 20/40 or worse or if a youth does not wear glasses and his uncorrected binocular distance vision is 20/40 or worse (PVIS). This standard of abnormal distance vision is the one used by National Center for Health Statistics (1972).

It is instructive to consider measures of adolescent health that are excluded by our selection criteria. Abnormal hearing is subject to

medical intervention, but the prevalence rate of this condition is less than 1 percent in the HES. Hence, it is far too rare to pose a threat to the future lifetime well-being of a significant percentage of adolescents. High blood pressure is not studied because there is a lack of consensus among pediatricians concerning the importance of this condition in adolescence and the appropriate treatment (National Heart, Lung, and Blood Institute's Task Force 1977). Moreover, the measures of high blood pressure in Cycle III are somewhat suspect (National Center for Health Statistics 1977). Congenital abnormalities are a source of current and future difficulties, but we do not study them because to a large extent they are irreversible. Parental ratings of adolescent health and other subjective indicators are avoided because of the possibility that responses depend on the parents' socioeconomic status. Parents with low levels of income and schooling are likely to be dissatisfied with many aspects of their life including the health of their offspring. Finally, we do not include measures relating to the "new morbidity" such as "learning difficulties and school problems, behavioral disturbances, ... and the problems of adolescents in coping and adjusting ..." [see Haggarty, Roghmann and Pless (1975), p. 316]. While such measures may well reflect life styles that have serious health consequences, they are unlikely to be revealed in a physical exam. Nor are they likely to be easily altered by preventive medical care. Although examination of these and other excluded health measures would be necessary to paint a complete picture of the health of this adolescent cohort, it is not relevant to the objectives of this paper.

B. Measurement of Preventive Dental and Medical Care

Preventive dental care is measured by a dichotomous variable that equals one if the youth saw a dentist for a check-up within the past year (DTPREV). Similarly, preventive pediatric care is measured by a dichotomous variable that equals one if the youth saw a doctor for a check-up within the past year (DRPREV). These variables distinguish between two groups of adolescents: (1) those who received preventive care; and (2) those who received no care at all or only curative care. These two measures of preventive care are preferred to alternatives like the number of dental or physician visits or the receipt of curative care alone because our measures are less likely to reflect reverse causality from poor health to more medical care. Of course, our measures reflect the possibility that adolescents received treatment as well as an examination, but the appropriate treatment of problems revealed by an annual check-up is an integral component of preventive care.

Fluoridation is indicated by a dichotomous variable that is equal to one if the community in which the youth resides uses naturally fluoridated or controlled fluoridated water (FLUOR). Naturally fluroridated communities are serviced by a water supply system that contains a natural fluoride content of 0.7 parts per million or higher. They are identified by the Division of Dental Health of the National Institutes of Health (1969). Controlled fluoridated communities are those that have adjusted the fluoride content of their water supply systems to the optimum level. They are identified by the Division of Dental Health of the National Institutes of Health (1970). For youths who reside in controlled communities, the fluoridation variable equals one only if the

date on which that youth was examined in the HES succeeds the date on which the community adjusted the fluoride content of its water supply system. This insures that youths in controlled communities actually were exposed to fluoridated water.[14]

C. The Pediatrician and Dentist Availability Measures

The youths in Cycle III were selected from 38 distinct primary sampling units. The primary sampling unit is a county or a group of several contiguous counties, some of which form a standard metropolitan statistical area. We obtained data on the number of dentists per capita (DENT) in each youth's primary sampling unit (hereafter termed his county or community of residence) for the year 1968 (the mid-year of the Cycle III survey) from publications of the American Dental Association. The number of pediatricians is not available for the years during which the HES was conducted (1966-70). Therefore, we use the number of pediatricians per capita in the county of residence (PED) for the year 1964 from the American Medical Association (Theodore and Sutter 1965).[15] We believe that the number of pediatricians in 1964 is a good proxy for the number in 1968. Although youths receive medical care from other types of physicians--general practitioners, internists, and ophthalmologists--these physicians also service adults while pediatricians do not. Therefore, we focus on pediatricians as the most important suppliers of physicians' services to youths.[16]

D. Measurement of Other Explanatory Variables

Many of the remaining explanatory variables called for in Section II require no further elaboration. Parents' educational attainment and family

income, for example, are adequately described in Table 1. Some of the other variables listed in Table 1, however, do require additional explanation.

Family size is represented by the number of people in the family who are under 20 years of age at the time of the Cycle III interview (LESS20). Consequently, it may overstate or understate actual completed family size.

Three measures of the family's efficiency in producing healthy children are included in addition to the parents' educational attainment. These are dichotomous variables that identify youths whose mothers were under age 20 when the youths were born (LMAG), youths from homes in which a foreign language is spoken (FLANG), and youths who live with their mothers only (NOFATH). Young mothers are notoriously less efficient at contracepting and may be similarly less efficient in producing healthy offspring. Foreign born families are likely to exhibit differences in household productive efficiency. The absence of her spouse from the household is likely to hinder the mother's allocative efficiency in selecting the input mix with which to produce health. The absence of a father also impinges upon the amount of time that a mother can spend with her children.[17]

The youth's endowed health status is represented by four variables relating to his early health. The first two (LIGHT1, LIGHT2) are dummy variables identifying youths of low birth weight. Low birth weight is a typical indicator of a less healthy birth outcome (for example, Birch and Gussow 1970). Birth weight was obtained from the youth's birth certificate. Since birth certificates are missing for approximately 25 percent of the sample and since we do not focus on the effects of birth weight, we do not delete these observations. Instead, we include a

dummy variable that identifies youths with missing birth certificates (BWUK) in the regression estimates. The third endowment measure is a dummy variable identifying youths whose mothers were over 35 years old at the youth's birth (HMAG). The rationale for including this variable is that older mothers are more likely to have offspring with health defects. The last of these measures is a dummy variable which identifies youths whose parents reported a medical difficulty with the youth before the age of one year (FYPH). Although parents' reports of youths' medical problems before the age of one year are subject to recall error, the first year of a child's life is likely to stand out in his parents' minds relative to other stages in his life cycle. Therefore, we believe that the measurement error in this variable is small.

Our current health indicator is used as a proxy for the child's unmeasured genetic health endowment and his health history beyond age one. This indicator is the presence of at least one significant abnormality as reported by the HES physician who examined the youth (ABN). Abnormalities include heart disease, neurological, muscular, or joint conditions; other major diseases; and otitis media. Except for the last condition, which constitutes a relatively small percentage of all reported abnormalities, these health problems are to a large extent congenital and irreversible.

We also control for several other characteristics of the youth which are not necessarily health related but may cause him to receive better or worse treatment within the family. They are his birth-order (FIRST) and whether or not he is a twin (TWIN). First born youths (or non-twins) will have greater access to individual parental attention because they

arrived in the family first (or they arrived alone). In addition, the youth's age (AGE) and sex (MALE) are included in regressions in which the dependent variable is not adjusted for age and sex (i.e. when the dependent variable is either obesity, abnormal corrected distance vision, preventive dental care, or preventive pediatric care).

Finally, three region variables (NEAST, MWEST, SOUTH) and four sizes of place of residence variables (URB1, URB2, URB3, NURB) are included to control for regional differences that are not otherwise taken into account. We are agnostic about the nature of these differences, but want to avoid the possibility that the health manpower and fluoridation effects are biased by an omission of unmeasured regional characteristics.

III. Empirical Results

In this section we present estimate equations (1) and (3) and compute the total impact of family characteristics and health manpower availability on adolescent health as given in equation (4). Equations (1) and (3) form a recursive system which can be estimated using single equation techniques as long as $E(u_1\ u_3) = 0$. We make this assumption here. Although all the dependent variables except the two oral health measures 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 obtained by the method of maximum likelihood. When the dependent variable is dichotomous the fitted equation can be interpreted as a linear probability function in which the regression coefficient of a given independent variable represents the change in the conditional probability

of poor physical health or receipt of preventive care for a one-unit change in the independent variable. The resultant estimates also embody the assumption that several variables that may be considered endogenous (mother's labor force status and family size, for example) are exogenous to adolescent health.[18] Finally, our estimates cannot be unambiguously interpreted as production functions or derived demand equations because insufficient data forced us to use proxy measures for some of the explanatory variables.

Estimates of the dental health production functions and the preventive dental care demand function are discussed in the first part of this section. The physical health production functions and the preventive pediatric care demand function are discussed in the second part. Both discussions are centered on answering the questions posed in the introduction concerning the roles of the family, preventive care, and health manpower availability in determining adolescent health. In examining the results, it is important to remember that the five health measures (APERI, IDECAY, OBESE, PVIS, ANEMIA) are negative correlates of good health, so that negative effects of independent variables in the production functions reflect factors associated with <u>better</u> health outcomes. The two preventive medical care measures (DTPREV, DRPREV), on the other hand, are positive correlates of care; thus positive effects of independent variables in the demand functions reflect factors associated with higher propensities to obtain preventive care.

A. <u>Oral Health</u>

Estimates of the oral health production functions and the preventive care demand function are in Tables 2 and 3, respectively. When the

TABLE 2
Ordinary Least Squares Estimates of Oral Health Production Functions[a]

Independent Variable	APERI		IDECAY		IDECAY (with APERI)	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
FEDUCAT	-.016	-2.95	-.011	-2.10	-.007	-1.41
MEDUCAT	-.030	-4.80	-.028	-4.67	-.021	-3.57
DTPREV	-.255	-8.47	-0.274	-9.42	-.212	-7.48
FLUOR	-.082	-2.96	-.159	-5.96	-.139	-5.39
FINC	-.007	-2.36	-.015	-4.85	-.013	-4.40
LESS20	.036	4.41	.023	2.90	.014	1.86
MWORKFT	.046	1.47	.088	2.91	.077	2.63
MWORKPT	-.007	-0.20	.055	1.54	.057	1.64
NEAST	.038	1.01	.264	7.26	.255	7.24
MWEST	-.072	-1.99	.183	5.24	.200	5.92
SOUTH	-.057	-1.42	.131	3.38	.145	3.85
URB1	.003	0.10	-.071	-1.85	-.072	-1.93
URB2	.027	0.63	-.028	-0.68	-.035	-0.87
URB3	-.088	-2.36	-.153	-4.25	-.132	-3.78
NURB	-.001	-0.00	-.077	-1.96	-.077	-2.02
LMAG	-.009	-0.17	-.032	-0.67	-.030	-0.64
HMAG	.023	0.47	-.048	-1.10	-.053	-1.26
LIGHT1	.187	1.40	-.127	-0.98	-.172	-1.38

TABLE 2 (concluded)

Independent Variable	APERI		IDECAY		IDECAY (with APERI)	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
LIGHT2	.081	1.10	.123	1.73	.103	1.50
BWUK	.014	0.45	.062	2.07	.058	2.02
ABN	.173	5.39	.032	1.04	-.009	-0.32
FYPH	.023	0.57	-.102	-2.64	-.107	-2.87
NOFATH	.016	0.33	.148	3.29	.145	3.31
FLANG	-.051	-1.28	-.172	-4.45	-.159	-4.27
TWIN	-.023	-0.26	.107	1.26	.112	1.37
FIRST	-.013	-0.47	-.024	-0.88	-.021	-0.79
APERI	-	-	-	-	.240	16.47
CONSTANT	.566		.532		.396	
Adj. R^2	.086		.114		.169	
F	15.85[b]		21.37[b]		31.99[b]	

[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.

TABLE 3
Ordinary Least Squares Estimate of Preventive Dental Care Demand Function[a]

Independent Variable	Regression Coefficient	t-Ratio	Independent Variable	Regression Coefficient	t-Ratio
FEDUCAT	.009	3.22	LIGHT2	.070	1.84
MEDUCAT	.023	7.27	BWUK	.005	0.32
FLUOR	.003	0.20	ABN	-.002	-0.14
DENT	.170	4.05	FYPH	.018	0.88
FINC	.011	6.99	NOFATH	-.025	-1.04
LESS20	-.033	-7.95	FLANG	-.037	-1.79
MWORKFT	-.034	-2.07	TWIN	-.002	-0.00
MWORKPT	.044	2.30	FIRST	.007	0.49
NEAST	.046	2.24	AGE	-.006	-1.53
MWEST	.041	2.17	MALE	-.022	-1.67
SOUTH	-.017	-0.83	CONSTANT	.331	
URB1	-.008	-0.37			
URB2	-.016	-0.64	Adj. R^2	.150	
URB3	.003	0.14	F[b]	27.02	
NURB	-.029	-1.37			
LMAG	-.065	-2.53			
HMAG	-.018	-0.75			
LIGHT1	.054	0.79			

[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.

number of decayed permanent teeth (IDECAY) is the dependent variable, two production functions are estimated. The first contains the same set of independent variables as the periodontal index (APERI) regression, while the second includes APERI as an additional independent variable. It has been suggested by Russell (1956) that variations in APERI result largely from genetic factors. If these genetic factors are correlated with the home environment and imperfectly measured by the health endowment variables, the second regression will give a more accurate estimate of the effects of the home environment on IDECAY than the first. Of course, APERI has an environmental component as well as a genetic component (as is evident from our estimate in Table 2). Therefore, the two IDECAY regressions contain upper and lower bound estimates of the impact of the environment on IDECAY.[19]

Most notable among the results are the large significant impacts of a preventive dental visit on both the periodontal index and the decay index.[20] The coefficient estimates imply that adolescents who did not have a preventive check-up within the past year have periodontal indices and decay scores that are each about .3 of a standard deviation worse than adolescents who received a check-up. When APERI is included in the decay equation, the decay differential between the two groups of adolescents declines to .2 but remains statistically significant. To gauge the magnitudes of these effects, recall that APERI and IDECAY have means of approximately zero and standard deviations of approximately one. Therefore, the oral health differentials associated with absence of preventive care are relatively large; they range from 20 to 30 percent of the standard deviations in the scores. Moreover, the differentials apply to a

substantial proportion of the sample: 30 percent of the youths in the HES did not have a check-up in the past year. These findings underscore the efficacy of preventive dental care.

The results pertaining to a publicly provided form of preventive care--water fluoridation--are also strong. Youths exposed to fluoridated water (FLUOR) have significantly better oral health than other youths at all conventional levels of confidence.[21] The fluoridation differentials are smaller, however, than the corresponding preventive dental care differentials in oral health. For example, the fluoridation coefficient in the periodontal index equation is one-third as large as the preventive dental care coefficient. In the decay equations, the ratio of the two coefficients ranges from three-fifths to two-thirds. Nevertheless, given that the per-child cost of fluoridation is substantially below the cost of a preventive dental visit, this remains a cost-effective method of improving dental health.[22]

Let us turn now to the role of the family in determining adolescent dental health levels. The four characteristics of the family environment we focus on are parents education (MEDUCAT, FEDUCAT), family income (FINC), family size (LESS20), and mother's labor force status (MWORKFT, MWORKPT). An overview of the production function estimates in Table 2 reveals that all six variables have statistically significant effects in the expected directions (with the exception of mother's labor force status in the periodontal index equation). Children of more educated parents have better oral health, as do children from families with higher income; while children whose mothers' are employed full-time or who come from larger families have poorer health. The impacts of these

variables on IDECAY are reduced in absolute value when APERI is held constant, but the pattern of statistical significance is not dramatically altered (only the coefficient of father's schooling becomes insignificant). It is clear, then, that these family characteristics have an important impact on adolescent dental health.

We interpret these findings as evidence that the home environment plays an important role in determining children's health. It can be argued, however, that our results do not really constitute strong evidence in favor of "nurture" because of the likelihood of positive correlations first between these family characteristics and the parents' health and second between the genetically determined components of parents' and childrens' health. Put differently, this argument states that family characteristics such as income or parental education largely reflect genetic health factors. For example, parents who are themselves healthy are more likely to be in the labor force and will have higher earnings. Or, parents who have had a healthy childhood and adolescence are more likely to have attained a higher level of education. Two of our findings, however, cast doubt on the applicability of this argument in our case. First, when we include APERI in the decay equation in an effort to more fully control for genetic factors, we still find that these family environment variables have significant impacts on IDECAY. This is noteworthy because the inclusion of APERI is likely to bias the coefficients of the family environment variables toward zero (see note 19).

A second and stronger reason revolves around the coefficients of the educational attainment of the two parents. If the education effect is primarily genetic, we would expect the coefficients of both mother's and father's education to be equal because both parents make an equal genetic contribution to the child. On the other hand, if the education

effect is primarily environmental, we would expect the impact of the mother's education to be larger because she is the family member most concerned with the children's health care. In Table 2 we observe that in every case the coefficient of mother's education exceeds that of father's education. In addition, despite a high correlation between the two education variables ($r = .61$), the difference in coefficients is always statistically significant at the 10 percent level.[23] Thus, our results clearly indicate that the family environment, and in particular, the mother's education, plays an important role in producing healthy children.

Besides having an important impact on the production of health, family characteristics work to improve adolescent health by increasing the probability that an adolescent receives preventive care. In Table 3 we see that all six of the family variables have significant impacts on the probability that an adolescent received preventive care. Children from families with higher annual income, more educated parents, and in which the mother works part-time, are more likely to receive preventive care, while children from larger families or families where the mother works full-time are less likely to receive preventive care. As an example of the magnitude of these effects, the probability that a child received preventive care in the previous year increases by about two percentage points for each additional year of education received by the mother and declines by about three percentage points for each additional child in the family. Once again we believe that these results reflect environmental rather than genetic influences: the mother's education coefficient is more than twice as large as the father's

education coefficient and the difference between them is statistically significant ($t = 2.82$).

To determine the total effect of family characteristics on health--both the direct effect embodied in the production function estimates and the indirect effect that operates through the family's proclivity to obtain preventive care--we compute the total impact of these family characteristics in Table 4. The reported coefficients are analogous to the sum $(\beta_1 + \beta_3 \alpha_1)$ in equation (4).[24] Comparison of the coefficients in Tables 3 and 4 indicates that the total impact is from 10 to 100 percent greater than the "direct" effect alone. We also observe, as before, a large and statistically significant (at the 5 percent level) difference between the impacts of fathers' and mother's education, again lending support to our conclusion that "nurture" matters.[25]

With regard to the role of health manpower, we see that it has a large significant effect on the family's propensity to obtain preventive care for its children (Table 3). An increase of one dentist per thousand population increases the probability that adolescents visited the dentist for preventive care in the previous year by 17 percentage points. This estimate is identical to one obtained by Manning and Phelps (1978) and is insensitive to the exclusion of region and size of place of residence from the equation.[26] The implied effect on adolescent health (assuming that dentist availability has no direct impact on adolescent health but operates only by increasing the family's propensity to obtain preventive care) is given in Table 4 and ranges from -.036 to -.047 of a standard deviation in the dental health measures.[27] Thus, an increase in the number of dentists in an area by one per thousand population is

TABLE 4

Total Impacts (Direct and Indirect) of Selected Variables on Oral Health

Variable \ Oral Health Measure	APERI	IDECAY	IDECAY (with APERI)
FEDUCAT	-.018	-.013	-.009
MEDUCAT	-.036	-.034	-.026
DENT	-.043	-.047	-.036
FINC	-.010	-.018	-.015
LESS20	.044	.032	.021
MWORKFT	.055	.097	.084
MWORKPT	-.018	.043	.048

equivalent in its effect on dental health to an increase in the level of the mother's education by one and one-third years.

It should be noted that the positive impact of dentists on the propensity to obtain a check-up is unlikely to reflect demand manipulation by dentists. The concept of demand manipulation refers to the ability of health personnel to shift the demand curve for their services, when all direct and indirect costs of these services are held constant. In his extensive treatment of this phenomenon, Pauly (forthcoming) shows that the demand manipulation effect should be larger in a sample of consumers with positive utilization than in a sample of all consumers. Moreover, his model gives no basis for expecting a demand manipulation effect in an equation that explains the probability of a check-up. Based on these considerations, we view the dental manpower variable as reflecting the importance of information, entry, travel, waiting, and direct costs in the parents' decision to obtain preventive dental care for their offspring.

Most of the other results in Table 3 are consistent with our expectations and will not be discussed.[28] We do wish to point out, however, that although fluoridation does have a significant impact on dental health, it is not significantly related to the probability of obtaining preventive dental care. This is not surprising since from a theoretical point of view either a positive or negative relationship could be predicted. If fluoridation is regarded as an increase in the child's health endowment, the quantity of care demanded should fall. On the other hand, if the increased endowment also increases the marginal product of preventive care, or if it lowers the psychic costs

of obtaining care by reducing the severity of the tooth decay uncovered by a preventive check-up, a positive effect on the quantity of care demanded would be predicted.[29] Both types of results have been reported in other studies. Manning and Phelps (1978) report mixed effects of duration of exposure to fluoridation on the propensity to obtain preventive dental check-ups for white children below the age of 15 in a 1970 health survey conducted by the National Opinion Research Center. Upton and Silverman (1972) use 1966 data for 15 midwestern towns, half of which used fluoridated water, and report fewer restorations of children's permanent teeth in the fluoridated towns.

We conclude this subsection by using our results to estimate the impacts of three government programs to improve the oral health of youths. First, consider a $1,000 income transfer to low-income families. As shown by the reduced-form coefficients of FINC in Table 4, the transfer would lower the periodontal index of youths from these families by .01 points and would lower their decay index by .02 points. (Such a program would naturally also have other beneficial effects on children and their families.) Next consider a program to reduce or eliminate regional differences in the number of dentists per thousand population. Dentists are more numerous in urban areas than in rural areas. To take two sites in the HES, there were 1.1 dentists per thousand population in San Francisco, California, while there were .2 dentists per thousand population in San Benito, Texas. Suppose that this difference were eliminated by raising the number of dentists in San Benito by one per thousand population. Then the periodontal index of youths in San Benito would fall by .04 points, and their decay index would fall by .05 points.[30] Finally,

consider an 80 percent reduction in the price of a dental check-up as a result of the enactment of a national health insurance plan for dental care with a 20 percent co-insurance rate. Based on research by Manning and Phelps on the impact of price on the propensity to obtain preventive dental care for children and youths, we estimate that such a policy would raise the probability of obtaining care by 16 percentage points. This would improve both the periodontal and the decay scores by .04 points.[31]

We view the above computations as illustrative rather than definitive. To choose among the three programs, information on the cost of each program and on the number of youths affected clearly is required. Moreover, as indicated in Section I, definitive computations of impact effects should take account of the supply elasticity of dental care and the exact nature of the relationship between dental manpower and the indirect costs of obtaining dental care.

B. Physical Health

Estimates of the physical health production functions and the preventive care demand function appear in Tables 5 and 6, respectively. Looking first at the production function estimates, we are struck by the fact that these physical health measures are much less amenable to statistical explanation than are the dental health measures. Of course, lower R^2's would be expected for the three physical health measures because they are dichotomous rather than continuous. But many fewer explanatory variables are statistically significant in the physical health case. Clearly unmeasured genetic or "luck" factors play a much larger role in the case of these health measures.

TABLE 5
Ordinary Least Squares Estimates of Physical Health Production Functions[a]

Independent Variable	OBESE		PVIS		ANEMIA	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
FEDUCAT	.001	0.46	-.002	-1.51	-.001	-0.65
MEDUCAT	-.005	-2.01	.001	0.71	-.002	-2.07
DRPREV	-.005	-0.50	-.005	-0.75	.002	0.47
FINC	.0002	0.20	-.001	-1.04	-.001	-1.39
LESS20	-.007	-2.41	.006	3.31	.004	2.60
MWORKFT	.005	0.40	.001	0.10	.002	0.37
MWORKPT	-.007	-0.54	-.00003	-0.00	.0003	0.00
NEAST	.040	2.94	-.003	-0.36	-.013	-1.93
MWEST	.036	2.80	.012	1.45	-.010	-1.63
SOUTH	-.020	-1.36	.009	0.93	.002	0.24
URB1	-.017	-1.15	-.0002	-0.00	.007	0.96
URB2	.023	1.47	.010	0.91	.010	1.33
URB3	-.011	-0.79	-.009	-1.01	.015	2.27
NURB	.016	1.09	-.008	-0.85	.014	1.94
LMAG	.023	1.27	-.011	-0.92	.005	0.54
HMAG	.017	1.01	.002	0.20	.003	0.40
LIGHT1	-.001	-0.00	.003	0.10	-.020	-0.84

TABLE 5 (concluded)

Independent Variable	OBESE		PVIS		ANEMIA	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
LIGHT2	-.010	-0.36	-.003	-0.17	-.010	-0.73
BWUK	.0004	0.00	-.012	-1.59	-.013	-2.39
ABN	.122	10.38	.009	1.17	-.008	-1.40
FYPH	-.006	-0.40	.006	0.59	.001	0.10
NOFATH	.021	1.22	.009	0.81	.006	0.71
FLANG	.015	1.06	.021	2.13	.001	0.20
TWIN	.004	0.10	.017	0.79	-.020	-1.25
FIRST	.021	1.95	.008	1.20	.003	0.66
AGE	-.004	-1.23	-.008	-4.28	-	-
MALE	-.053	-5.65	-.024	-3.87	-	-
CONSTANT	.190		.164		.049	
Adj. R^2	.039		.013		.008	
F	7.21[b]		2.97[b]		2.35[b]	

[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.

TABLE 6
Ordinary Least Squares Estimate of Preventive Pediatric Care Demand Function[a]

Independent Variable	Regression Coefficient	t-Ratio	Independent Variable	Regression Coefficient	t-Ratio
FEDUCAT	.013	4.28	LIGHT1	-.031	-0.40
MEDUCAT	.007	1.82	LIGHT2	.063	1.47
PED	.675	1.97	BWUK	.001	0.10
FINC	.005	2.59	ABN	.094	5.00
LESS20	-.017	-3.60	FYPH	.042	1.81
MWORKFT	.015	0.80	NOFATH	-.071	-2.61
MWORKPT	.005	0.24	FLANG	.017	0.72
NEAST	.062	2.77	TWIN	-.025	-0.49
MWEST	-.031	-1.50	FIRST	.058	3.47
SOUTH	-.021	-0.91	AGE	.004	0.95
URB1	.021	0.87	MALE	.064	4.30
URB2	.036	1.34			
URB3	-.014	-0.61	CONSTANT	.192	
NURB	-.037	-1.56	Adj. R^2	.067	
LMAG	-.032	-1.09			
HMAG	-.055	-2.09	F	11.99[b]	

[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.

In contrast to the results for preventive dental care, there is little evidence that preventive medical care is efficacious. Youths who saw a doctor for a check-up within the past year (DRPREV) have one-half percentage point smaller probabilities of being obese or of having abnormal corrected distance vision than other youths, and a one-fifth percentage point higher probability of having anemia. None of these three differentials is statistically significant. One possible explanation for these findings is that there are fairly long lags between the receipt of preventive care and an improvement in physical health. Alternatively, one might argue that physicians play a minor role in the outcomes studied here relative to unmeasured endogenous inputs such as proper diet. The non-significant impact of preventive care also means that family characteristics operate on health only through the production function. There are no indirect effects of the various family characteristics on physical health, only direct effects. Consequently, we do not present a table of "total" effects (comparable to Table 4) in the case of the physical health measures.

The relationship between family characteristics and health is also much weaker in the case of physical health. Most of the six family characteristics variables studied are not even statistically significant in the production function; only the mother's education and family size variables have significant impacts. Children of more educated mothers are less likely to be obese or anemic, and they are more likely to have poor vision (the latter relationship is not significant). Children from larger families are more likely to have poor corrected vision or be anemic, but they are less likely to be

obese. To get an impression of whether these effects can be viewed as environmental as opposed to genetic, we again look at the difference between the coefficients of the two parent's education variables. For both OBESE and ANEMIA, the mother's education coefficient is larger than the corresponding father's education coefficient, but for PVIS the opposite is true. Only in the case of obesity is the difference significant at the 10 percent level. Thus, in this case the evidence regarding a nature versus nurture interpretation of the family effects is not conclusive, but it does suggest that with respect to obesity at least one component of the family environment--mother's education--has an important impact.

We noted that the family size variable has a perverse sign in the obesity equation: children from larger families are in better rather than worse health in that they are less likely to be obese. The positive relationship between family size and the incidence of the other health problems is easy to rationalize (it may reflect a substitution away from higher "quality" children as the shadow price of quality rises),[32] but a justification for the negative relation reported for obesity is less obvious. One possible explanation for this negative family size effect (as well as for the positive income effect) is the existence of joint production among various aspects of quality. For example, families with fewer children or higher income may consume more rich and caloric foods. This consumption raises some aspects of quality but at the same time makes obesity more likely.

The finding of non-significant effects of family income in physical health outcomes has important implications. First, it suggests that

policies to improve the well-being of adolescents via income transfers would have little impact on our physical health measures. Second, this finding coupled with the significance of the mother's schooling variable underscores the key role in health production of nonmarket productivity as opposed to market goods and services as measured by family income and preventive care. This result echoes our earlier findings for a group of younger children (Edwards and Grossman 1980). In the case of obesity, we believe that the impact of mother's schooling reflects the information that highly educated mothers have acquired as part of the schooling process about the dangers of obesity and about what constitutes an appropriate diet.

Family effects in the derived demand for preventive pediatric care (Table 6) tend to be much stronger than they are in the production functions (although the R^2 in the preventive pediatric care equation is still substantially lower than in the preventive dental care equation). Among the six family variables, only the mother's labor force status variables do not have a significant impact on the family's probability of obtaining preventive care. Families with higher parental education and more income are more likely to get preventive care for their children while larger families are less likely to. In addition, father's education has a larger impact than mother's education. It is not clear how to interpret these results, however, since we have no evidence that preventive pediatric care is efficacious.

The last result to be discussed concerns the role of pediatrician availability. Similar to the corresponding findings for preventive dental care, the number of pediatricians per thousand population in the

county of residence (PED) has a positive and statistically significant regression coefficient in the demand curve for pediatric care. This finding complements those reported by Kleinman and Wilson (1977) and Colle and Grossman (1978). However, the implied effects of an increase of one pediatrician per thousand population are small (-.003, -.003, and .001 for OBESE, PVIS, and ANEMIA, respectively), primarily because the health impact of preventive care is small and not significant. Thus our findings indicate that a policy to increase pediatric manpower in medically underserved areas would not improve the physical health of adolescents--at least as represented by our three measures. Such a policy should be given a much lower priority than a analogous policy to expand dental manpower in areas characterized by shortages.[33]

IV. Summary and Implications

The purpose of this study has been to examine the determinants of the oral and physical health of white adolescents with special emphasis on the roles of family background and the use of preventive medical care. The main results of the study are (1) nurture plays an important role in determining oral health but less so for the other health problems studied; (2) preventive care is efficacious in the case of oral health but not for the other health problems studied; and (3) the three physical health measures are largely unexplained by the family and preventive care variables used here. Only mother's education and family size have significant impacts.

With respect to the first result,mother's schooling is singled out as a crucial component of the home environment. Although mother's schooling, father's schooling, family income, and family size all make

significant contributions to oral health, mother's schooling dominates father's schooling. Moreover, mother's schooling tends to dominate both income and father's schooling in the physical health equations, especially in the case of obesity. The finding that the impact of mother's schooling almost always exceeds that of father's schooling is especially important because equal effects would be expected if the schooling variables were simply proxies for unmeasured genetic endowments.

Two additional pieces of evidence underline the robustness of the finding that nurture "matters." First, the relative magnitude of the effect of the various family background variables on the index of tooth decay is not greatly altered when the periodontal index, a proxy for genetic oral health endowment, is held constant. Second, the identification of a plausible mechanism by which family characteristics influence adolescent health--preventive care--increases our confidence that these variables reflect a behavioral effect as opposed to a genetic effect or a statistical artifact.

With regard to the role of preventive dental care, youths who received a preventive dental check-up within the past year and youths exposed to fluoridated water have much better oral health than other youths. Moreover, the probability of a preventive examination is positively related to the number of dentists per capita in a youth's county of residence. This implies that a program to increase the availability of dentists in medically deprived areas would improve the oral health of youths in these areas. Indeed, we estimate that the payoffs to increasing dental manpower by one per thousand population are about the same as the payoffs to the coverage of preventive dental care under national health insurance.

The probability of obtaining a preventive check-up by a doctor is also positively related to family income and to the number of pediatricians per capita in the county of residence. But we have little evidence that preventive care delivered to youths by physicians is efficacious in terms of their physical health. Therefore, the payoffs to national health insurance for physicians' services delivered to youths or programs to increase the availability of doctors who treat youths are very small.

Our results for the physical health measures are weak, but one pair of findings does stand out. Adolescents are less likely to be obese if their mothers are highly educated, and they are more likely to be obese if they come from small families. The latter relation provides a partial explanation of the dramatic increase in obesity during recent decades since over the same period we have seen a startling decline in family size. The former relation, on the other hand, suggests a strategy for slowing down the trend in the incidence of this health problem. What is needed is a public information program--similar to that mounted in the case of childhood immunization--directed at alerting less educated parents, and especially mothers, to the dangers associated with childhood obesity.

Overall, what our results suggest is that selective rather than general programs would be most effective in improving the health of the population under 18 years of age. For instance, instead of providing complete coverage for physicians services delivered to persons from birth to age 18 under national health insurance, the government should direct its attention at prenatal care and physicians services

during the first year of life. It is known that appropriate prenatal and infant care can make a difference in terms of health outcomes (for example, Lewit 1977). Conversely, our results for oral health in this paper and in our previous research (Edwards and Grossman 1980) suggest that the payoffs to the coverage of dental care from the age it is first received until age 18 or beyond would be substantial.

FOOTNOTES

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[1]For a partial survey of this literature, see Grossman (1975) and Edwards and Grossman (1979).

[2]A full description of the sample, the sampling technique, and the data collection is presented in National Center for Health Statistics (1969).

[3]Children's health also depends on the prices of inputs used to produce other aspects of their quality and the prices of other forms of parents' consumption. The effects of these variables will not be studied here.

[4]For discussions of the indirect costs of obtaining pediatric care, see Colle and Grossman (1978) and Goldman and Grossman (1978).

[5]One possible objection to using this type of framework to analyze the health of adolescents is that the goals of parents and youths are likely to differ. For instance, cigarette smoking by a youth might increase his utility but reduce his parents' utility because it is detrimental to his current or future health. This type of conflict between parents and youths has been analyzed by Becker (1974) in the context of an economic model of social and family interactions. He shows that such conflicts are important when the parents' utility function depends on particular "merit" commodities consumed by the youth rather than on his consumption of all commodities. In such a case parents have an incentive to allocate resources not only to their children's consumption, but also to policing their offsprings' consumption patterns. An explicit melding of our model with Becker's would be a difficult task, and although it would alter the interpretations of the effects of various family characteristics, it would not add to or delete from the list of relevant explanatory variables.

[6]Given the essentially arbitrary scaling of all of our adolescent health measures and the general ignorance concerning the exact specification of a health production function, we believe that it is inappropriate to experiment with more sophisticated functional forms.

[7]The Bureau of Labor Statistics does collect measures of the prices of various goods and services, including physician and dental office visits, for 40 cities and four nonmetropolitan areas. We do not take price variables from this source because they are based on small samples.

and the sites in the HES survey are not identical to the sites in the BLS survey. On the other hand, the number of dentists and pediatricians are based on complete enumerations in all counties by the American Dental Association and the American Medical Association and can be matched easily to the HES sites. Thus the two manpower variables have little measurement error, while the price estimates from the BLS would contain a great deal of measurement error.

[8]This is in contrast to the work of Newhouse and Friedlander (1977) who fit an equation similar to our equation (4).

[9]The term "allocative effect" and the decomposition of the schooling parameter into direct and allocative components is due to Welch (1970). He uses this framework to study the impact of schooling on market production. Technically, schooling is a relevant determinant of the demand for medical care even if it has no allocative efficiency effect. In simple models of schooling as an efficiency variable in household production (Grossman 1972; Michael 1972), schooling raises the amount of health output obtained from a given vector of inputs. In such models schooling can lower the quantity of medical care demanded at the same time as it raises the quantity of health demanded. In particular, medical care would rise only if the income and price elasticities of demand for offspring's health exceeded unity. We stress a model that incorporates an allocative efficiency effect because schooling should increase the parents' knowledge about what constitutes an appropriate diet, when to take their children to the doctor or the dentist for a preventive check-up, how to follow the doctor's advice, and how to

foster appropriate oral hygiene behavior by their children. The ability of parents with extra schooling to select a better input mix, as well as to obtain a larger health output from given inputs, is likely to encourage them to demand larger quantities of preventive care even if the income and price elasticities of health are less than one. In part the effect may reflect a substitution toward preventive care and away from curative care.

[10]In adopting these two criteria for the selection of health measures, we are guided in part by Kessner's (1974) tracer methodology for studying the health of children and adolescents.

[11]If the actual periodontal index of each age-sex group is normally distributed, APERI could be translated directly into the youth's periodontal index percentile. We have experimented with the actual value of the periodontal index as the dependent variable in a multiple regression that includes age, the square of age, and a dummy variable for male adolescents in addition to the remaining independent variables. The results obtained (not shown) are similar to those reported in Section III.

[12]Dutton (1978) advocates the use of a continuous, rather than a discrete, measure of anemia. She conducts a multiple regression analysis of actual hematocrit levels of black children between the ages of 6 months and 4 years. The only statistically significant variables in this regression (at the 5 percent level) are age and sex. Therefore, it is not at all clear what we would gain by adopting her measure.

[13]Similar patterns are present in the Cycle III data. Tanner (1962) stresses the importance of sexual maturity in the determination of the health and cognitive development of adolescents. Preliminary analysis revealed, however, that sexual maturity does not have an effect on our health measures except in the case of hematocrit levels of females.

[14]Clinical evidence suggests that exposure to fluoridated water is particularly important if it occurs during the ages at which the permanent teeth are being formed (McClure 1962). These teeth do not appear until a child is approximately 6 years-old but start to be formed a few months after birth. Therefore, it is useful to identify youths who had been exposed to fluoridated water before they reached the age of 6 years. Unfortunately, we cannot do this because the youth's current residence alone is reported in the HES. We did create a fluoridation variable that identifies youths exposed before age 6 under the assumption of no migration, but it had no effect on oral health in regressions that included the fluoridation variable described in the text.

[15]Our measure of the number of dentists excludes those in the Federal dental service. The number of pediatricians pertains to those in private practice.

[16]Since pediatricians treat only children and youths, the number of pediatricians per person under a certain age (say age 18) might appear to be a more relevant measure than the number of pediatricians per capita. We did not employ such a variable for several reasons. First, the appropriate age cutoff is not obvious. Second, even if pediatricians do not treat youths beyond the age of 17, since mothers

typically are responsible for taking youths to the physician, the indirect costs of obtaining pediatric care might be more related to the number of pediatricians per woman with children below the age of 18 than to the number of pediatricians per person below the age of 18. Third and most important, there is little variation in persons under age 18 as a percentage of the population or in women with children under age 18 as a percentage of the population among the 38 sites in the HES.

[17]The educational attainment of absent fathers is not known. For children with absent fathers, we code FEDUCAT at the mean level of father's education in the subsample of youths who live with both parents. This coding scheme is consistent with the assumption that father's education has the same relationship with adolescent health whether or not the father is actually present. An alternative assumption is that father's education has no affect on adolescent health if he is absent. Under this assumption, the education of absent fathers would be coded at zero. Use of the alternative coding scheme would alter the regression coefficient of NOFATH but would not alter the coefficient of FEDUCAT or the coefficients of other independent variables in the regression.

[18]The health endowment variables are also endogenously determined because they are affected by family choices regarding prenatal care, timing of childbearing, and resources allocated to children since birth. Despite the endogeneity of the health endowment measures, mother's labor force status, and family size, preliminary computations revealed that the estimated coefficients of the other family background measures and

of preventive care are only slightly altered by the exclusion of these variables from the equations.

[19]Suppose that the periodontal and decay functions are

$$\text{APERI} = a_1 G + a_2 E + u_1 \qquad (1)$$

$$\text{IDECAY} = b_1 G + b_2 E + u_2 \quad , \qquad (2)$$

where G is genetic oral health endowment, E is the home environment, u_1 and u_2 are disturbance terms, and intercepts and other independent variables are ignored. Note that a_1, a_2, and b_2 are negative since a more favorable endowment or environment improves oral health. Solve equation (1) for G and substitute into equation (2) to obtain

$$\text{IDECAY} = b_1 a_1^{-1} \text{APERI} + (b_2 - a_2 b_1 a_1^{-1}) E + u_2 - b_1 a_1^{-1} u_1 \, . \qquad (3)$$

Clearly, the absolute value of the parameter of E in equation (3) is smaller than the absolute value of the corresponding parameter in equation (2). Note that APERI is negatively correlated with the composite disturbance term $(u_2 - b_1 a_1^{-1} u_1)$ in equation (3). Therefore, if the equation is estimated by ordinary least squares, the regression coefficient of APERI is biased toward zero and that of E away from zero provided E and APERI are negatively related. In the text we make the plausible assumption that this upward bias in the absolute value of the regression coefficient of E is offset by the fundamental difference between the structural parameters of E in equations (2) and (3). That is

we assume that the expected value of the regression coefficient of E understates $|b_2|$ even though it overstates $|b_2 - a_2 b_1 a_1^{-1}|$.

[20]Statements concerning statistical significance in the text are based on one-tailed tests 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.

[21]The estimated effects of fluoridation on oral health are not sensitive to the omission of the three region and four size of place of residence variables from the regressions. This indicates that the fluoridation variable is not simply a proxy for location.

[22]Consumer Reports (1978) cites a report in the New England Journal of Medicine which estimates the per capita cost of fluoridation to be about 10 to 40 cents per year (p. 393).

[23]The relevant "t" statistics for the three equations in Table 2 are 1.41, 1.79, and 1.48. Note that probable biases in the estimates of the two parents' education coefficients are likely to work towards a finding of no significant difference. The estimate of the direct efficiency effect of father's schooling may be biased away from zero; and the estimate of the direct efficiency effect of mother's schooling may be biased toward zero. The former bias is introduced if father's education serves as a proxy for permanent income (if there is measurement error in current family income). The latter bias is introduced if more educated mothers allocate less time to the production of adolescent oral health because they have a higher opportunity cost of time, and if

the opportunity cost of time effect is not fully reflected by the two measures of mother's labor force status. Along similar lines, the estimated father's education effect may be biased upward in the demand curve for preventive care. The mother's education effect is biased downward if oral health is "time-intensive" and if substitution in consumption outweighs substitution in production.

[24]These could be thought of as solved "reduced form" coefficients of the exogenous variables.

[25]As is expected on the basis of the education coefficients in Tables 2 and 3, the difference in "total" effects is larger than the difference in direct effects. The test of the significance of the difference between the "total" effect of mother's schooling and the "total" effect of father's schooling is based on the estimated reduced form--the ordinary least squares regression APERI or IDECAY on all the exogenous variables. This procedure is employed because standard errors of solved reduced-form coefficients and standard errors of difference between such coefficients are very difficult to compute. In every case, the estimated reduced-form difference between the schooling coefficient is exactly the same as the solved reduced-form difference. Therefore, the bias introduced by our test is minimal. The test statistics are 1.81, 2.23, and 2.82 for APERI, IDECAY, and IDECAY with APERI, respectively.

[26]Manning and Phelps estimate a discriminant function of the probability of obtaining a check-up. They point out that the coefficients in this equation approximate logit coefficients. Since they do not

indicate the mean probability of a check-up in their sample, we converted their logit coefficient of the number of dentists into a marginal effect at the mean check-up probability in the HES sample of .7. If m is the marginal effect of a given independent variable, b is its logit coefficient, and p is the probability of a check-up, the conversion formula is

$$m = bp(1 - p) \quad .$$

[27]The finding that the periodontal index is inversely related to the number of dentists differs from that of Newhouse and Friedlander (1977). Using adults in Cycle I of the HES, they report an insignificant positive effect of dentists per capita in the county of residence on the periodontal index. Their result is based on an ordinary least squares regression of the periodontal index on the number of dentists and other variables and does not embody the restrictions discussed in Part C of Section I.

[28] There are two "perverse" results that are statistically significant: youths from families in which a foreign language is spoken in the home (FLANG) have better oral health than other youths; and youths whose parents reported a medical difficulty with the youth before the age of one year (FYPH) have less decay than other youths. The first of these may be caused by genetic differences in oral health between native Americans and immigrants or the native-born offspring of immigrants. We offer no explanation for the latter finding.

[29]For a general discussion of endowment effects in models such as the one employed in this paper, see Tomes (1978). A detailed treatment of the role of fluoridation in dental care demand functions appears in Upton and Silverman (1972).

[30]The reduction in the decay score is taken from the reduced-form coefficient of DENT obtained from the decay function that excludes APERI.

[31]In their discriminant estimate of the decision for white children and youths to receive a dental exam, Manning and Phelps specify a price effect that varies with family income. Our extrapolation of their results assume (1) that family income equals $10,000 (the mean value in the HES), (2) that the uninsured price of check-up is $15, and (3) that the uninsured probability of a check-up is .7 (the mean in the HES). The reduction in the decay score is obtained from the decay function that excludes APERI.

[32]Alternatively, these effects may be attributed to a reduction in per capita income as family size rises with family income held constant. Indeed, the sign of the family size effect is opposite that of the family income effect in all three regressions. Yet something more than a mechanical relationship between family size and per capita income is required to account fully for the contribution of family size to health outcomes. For example, unlike the family size coefficients, the family income coefficients are not always statistically significant. In addition, computations reveal that the impact on physical health of a 1 percent

increase in family size is larger in absolute value than that of a 1 percent increase in family income.

[33]Some readers may object to the constraint in our recursive model that the direct effect of health manpower on health is zero. For the benefit of these readers, the estimated reduced-form coefficients of the number of pediatricians on obesity, abnormal vision, and anemia are -.316 (t = -1.47), -.010 (t = -0.07), and -.168 (t = -1.57), respectively The estimated reduced-form coefficients of the number of dentists on the periodontal index and decay are .128 (t = 1.55) and .085 (t = 1.08), respectively. These coefficients give a very different and, in our view, inappropriate picture of the payoff of a program to expand pediatric manpower compared to a program to expand dental manpower.

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