DOES HEALTH INSURANCE COVERAGE MITIGATE OR EXACERBATE SOCIOECONOMIC INEQUALITIES IN HEALTH IN THE U.S.? *

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Abstract:

This paper examines the institutional impact of health insurance coverage on the pathways leading from status attainment to adult health. Using data from the 1979 National Longitudinal Survey of Youth, the analyses consist of structural equation models of sibling resemblance (Hauser 1988).

Results suggest that the cumulative effects of income are partially mediated by the effects of health insurance. More specifically, these analyses indicate that health insurance and the source of coverage contribute to social inequalities in health through very different pathways: first, the number of years privately insured was found to compound the positive sibling-specific effects of status attainment on health when contrasted with the lack of insurance; second, public insurance was not found to differ in its effects on health from private insurance; and third, public insurance may have the potential to reduce socioeconomic inequalities from the family of origin when lack of insurance is the alternative.

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The assessment of the contribution of health insurance to social inequalities in health requires an understanding first of its relationship with socioeconomic status (SES), and secondly, of its relationship with health. Concerning the relationship of SES with health insurance, it is well-known that health insurance coverage, and the source of that coverage, are both strongly related to income in the United States, with more fortunate-individuals being more likely to have private than public, or no insurance (Kalleberg, Reskin and Hudson 2000; Mills and Bhandari 2003).

In turn, regarding the impact of health insurance on health, many studies show quite consistently that, relative to the lack of insurance, private insurance has a positive effect on health, even net of baseline socioeconomic status and health (Baker et al. 2001; Franks, Clancy and Gold 1993a, 1993b; Hahn and Flood 1995; Kasper, Giovanninni and Hoffman 2000; Short and Lair 1994). Yet, a number of previous studies also indicated that public health insurance has no such clear effects, as it appears in certain cases to be more detrimental to health than even being uninsured (Hahn and Flood 1995; Rogers et al. 2000; Ross and Mirowsky 2000; Short Hahn and Flood 1995; Rogers et al. 2000; Ross and Mirowsky 2000; Short and Lair 1994). In fact, Quesnel-Vallée (2004) recently argued that this counterintuitive negative relationship of public health insurance is unlikely to be causal, but rather may be due to the strong selection bias emanating from its stringent eligibility requirements. She then went on to show that this negative relationship becomes nonsignificant when greater controls for health and SES from the family of origin.

Conceptually, the effects of public insurance on health should not be different from those of private insurance; however, studies of access to care allow us to speculate that public insurance could have a slight negative effect compared with private insurance, due to the lack of continuous coverage and greater obstacles to obtaining care (Ayanian et al. 2000; Baker et al. 2002; Hadley 2003; IOM 2002a; Short, Monheit and Beauregard 1989; Sudano and Baker 2003). This would also imply that public insurance should have a positive effect on health relative to the lack of insurance only if it provides more continuous coverage and greater access to care.

Thus, compared with both the lack of insurance and public insurance, private insurance may contribute to socioeconomic differentials in health by compounding the positive effects of income on health. In contrast, being publicly insured rather than uninsured should reduce socioeconomic differentials in health. Indeed, if income has both a direct positive effect on health and a negative relationship to the likelihood of public insurance, which in turn has a positive relationship to health relative to the lack of insurance, then public health insurance will reduce the total effect of income on health.

Given the substantial challenges in analytic adjustments that were underscored by Quesnel-Vallée (2004) for studies of the impact of health insurance on general health and mortality in the United States, it is not overly surprising that only one prior study has explicitly set out to evaluate the contribution of health insurance to socioeconomic inequalities in health. Using longitudinal data from the 1995 survey of Aging, Status and the Sense of Control (ASOC), Ross and Mirowsky (2000) looked at the impact of being publicly or privately insured rather than uninsured at baseline on multiple health outcomes, access to care and economic hardship three years later. Controlling for the hazard of attrition, demographic characteristics, education, employment, household income and health measures at baseline and for significant changes

between time 1 and time 2 in these independent variables, they found no benefits of private insurance relative to being uninsured, while public insurance had a marginally significant negative effect on self-rated health.

Moreover, health insurance did not mediate the effects of SES on any of the health outcomes (similar analyses were not shown for access to care and financial hardship outcomes). In discussing these findings, the authors argue that, because it is not associated with better health, private insurance cannot therefore contribute to socioeconomic differentials. And while they recognize that the negative effect of public health insurance could have explained why lower SES individuals have worse health, they find no evidence of such mediating effects and thus conclude that health insurance does not contribute to the effects of SES on health.

Ross and Mirowsky's (2000) study has several strengths, such as longitudinal data, the same baseline measures of health as the outcomes, and explicit measures of economic hardship. They also went to great lengths to document the process whereby health insurance might result in socioeconomic inequalities by taking into account outcomes of access to care and of financial hardship. However, a number of limitations may explain why their results are at odds with previous studies' findings of the positive effects of private insurance (Baker et al. 2001; Franks et al. 1993a, 1993b; Hahn and Flood 1995; Kasper et al. 2000; Short and Lair 1994).

A first limitation of these analyses is that health insurance was measured at only one point in time, and outcomes three years later. Yet, this should not prove to be a crucial problem insofar as other prior studies suffered from similar limitations and still found positive effects of private insurance relative to being uninsured (Baker et al. 2001; Sorlie et al. 1994). A second and more substantial problem with having only two data points is that SES and baseline health were measured concurrently, not prior to health insurance, which impedes the logical and empirical demonstration that health insurance mediates the impact of SES on health.

Finally, more preoccupying still was the inclusion in the analyses of individuals over 65 (respondents in their sample ranged in age from 18 to 95 at baseline). Assuming that age and baseline health status would be sufficient to control for the substantial heterogeneities in health of such a broad population, the mere inclusion of elderly individuals eligible for Medicare in their analyses in effect prevents these findings from being comparable to any prior study of the effects of health insurance on general health and mortality, which all were limited to the adult nonelderly population. Moreover, this sample composition led to an atypical typology of health insurance, as elderly individuals with Medicare and supplemental insurance were classified as having private insurance, which introduces significant heterogeneity in the meaning of private insurance and in the population it covers. The pervasive lack of statistical significance of health insurance effects, and by extension of mediating effects of the relationship between SES and health, is more easily understood in light of these limitations.

In sum, I would argue that, as those of most previous studies, Ross and Mirowsky's (2000) results are affected by selection biases that confound the true effect of health insurance, and particularly of public insurance. As such, these findings cannot be considered definitive but rather call for extensions of this model controlling for more of the spurious effects of prior health and SES.

Conceptually, it may be more fruitful to model the potential contribution of health insurance to socioeconomic inequalities in health in the United States[1] more explicitly as a pathway effect,

[1] Of course, it should be made clear here that this study is not assessing the contribution of health insurance to socioeconomic inequalities in health per se. Indeed, socioeconomic inequalities or the social gradient in health are

whereby socioeconomic status in adulthood has an impact on the likelihood of coverage, which in turn has an impact on health that may either contribute to or reduce the total effects of socioeconomic status on health. Of course, the effects of socioeconomic status in adulthood should be modeled here to integrate the cumulative hypothesis of the life course effects of SES on health. Moreover, if socioeconomic status in adulthood is thought to result from a process of status attainment, this pathway model should be augmented to include the upstream processes that issue from the family of origin, as well as education, which also have an effect on health that may be mediated by health insurance.

However, this poses an estimation problem, as Quesnel-Vallée (2004) suggested that effects of health insurance must be measured net of those of family background. While she highlighted the contribution of fixed effects models to the study of the impact of health insurance on health, these models may not be best suited for the study of socioeconomic differentials in health in a status attainment framework. Indeed, this framework emphasizes the estimation of effects from the family of origin on health, which are simply cancelled out from siblings fixed effects models.

In fact, the solution may be found in Hauser's (1988) sibling resemblance models, which use structural equation modeling (SEM) to both control for unobserved effects from the family of origin and estimate their impact on other endogenous variables. Thus, this model should also allow to control for latent effects of socioeconomic status on adult health[2], to the extent that exposures were shared by siblings[3]. Thus, it appears that the sibling resemblance model offers great promise for understanding the processes by which health insurance contributes to a pathway model of the impact of status attainment on health.

HYPOTHESES

Previous studies have suggested the existence of pathway effects between childhood conditions, education, adult socioeconomic status and adult health and mortality. However, only one study has considered the contribution of health insurance to the relationship between adult socioeconomic status and adult health, which could be of considerable importance in the United States. Yet, while Ross and Mirowsky (2000) found no support for this pathway, substantial limitations to their analyses nevertheless warrant a reexamination of this hypothesis with more appropriate data and analytic methods. More specifically, given the substantial SES and health bias in health insurance coverage, influences from the family of origin on health insurance and health will be controlled for and estimated.

Pathway effects of status attainment and health insurance

Starting with the early adulthood factors, and going successively through the variables in temporal order, I expect to find the following pathways:

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population characteristics, and while they may emerge from individual differences in health along the socioeconomic structure, looking at individual differences in socioeconomic status and health only allows us to formulate hypotheses about how these population patterns eventually arise in the aggregate.

[2] I am referring here specifically to one of the three hypotheses suggested by Kuh et al. (2003) for the impact of socioeconomic status over the life course on adult health, and not more generally to latent variables in SEM models.

[3] As they would be for instance if the social status of the family did not change from one pregnancy to the next.

Early health: I hypothesize that early health will have both a direct and an indirect impact on adult health through financial resources and health insurance in mid-adulthood.
Education: Research on status attainment leads me to expect that the effects of family of origin on adult achievement (here financial resources) should work through education. In addition, education has been shown to have a significant effect on health net of adult socioeconomic status. Thus, I expect education to have both a direct and an indirect effect on health going through adult financial resources.
Financial resources: Here, cumulative financial resources are hypothesized to have both a direct and an indirect impact on health working through health insurance.
Health insurance: As hypothesized above, lacking health insurance should have negative cumulative effects on health when contrasted with both private and public health insurance. In turn, the cumulative effects of public insurance should not differ from those of private insurance.

These pathways entail that, net of family background effects, I expect private insurance to contribute to socioeconomic inequalities in health by adding to the total effect of financial resources on adult health. In turn, I also hypothesize that, relative to the lack of insurance, public insurance should reduce those inequalities.

METHODS

SIBLING RESEMBLANCE MODELS (BETWEEN- AND WITHIN-FAMILY ESTIMATORS)

Structural equation models (SEM) will be used here. As underscored above, the hypothesis of pathways effects has the potential to offer a lot to the understanding of the mechanisms linking socioeconomic status and adult health. While they have seldom been tested as such up until now with regards to health, pathway effects are conceptually recursive models. Thus, methods such as SEM are ideal for this model, as they allow to simultaneously test sets of linear structural equations where cause and effect relationships are hypothesized by the researcher.

However, use of SEM models in the current context raises certain methodological issues related first to the ordinal nature of the dependent variable, and second to the need to control for unobserved effects of the family of origin, while still estimating the impact of these effects on status attainment, health insurance and health. These issues will be discussed in turn, and a description of the model used here will conclude the section.

Using an ordinal indicator in SEM

One of the primary assumptions of structural equation modeling analysis is that the indicator variables must be continuous. While Bollen (1989) notes that this assumption is likely to always be violated due to the limits of our measurements[4], he also argues that the use of more coarsely categorized measures such as Likert scales demands particular attention.

Take for instance the ordinal measure of self-rated health (*srh*). As shown above, we assume, but do not observe, that this variable conceptually measures a latent continuous function of individuals' health, or wellness *srh**. Following Bollen's (1989) discussion on this topic, this underlying continuous variable can be used in SEM, but it is not so clear that its ordinal indicator

[4] For instance, age is conceptually a continuous measure, but we generally measure it ordinally, as the age at last birthday.

can, for three reasons. First, the measurement model for the latent continuous indicator *srh** may not hold with an ordinal indicator *srh*, as *srh**≠*srh* for at least some rows. Secondly, the distribution of the ordinal variables may differ from that of the latent continuous variables. Thus, even when *srh** and the other latent variables in the structural model are multinormal, the ordinal variable *srh* may be nonnormal. Finally, the covariance structure hypothesis that the population covariance structure of the observed variables ($\Sigma(\theta)$) equals the population covariance structure of the latent variables (Σ) may not hold with ordinal variables. Bollen deems this last consequence to be the most serious.

No measurement model is used in the current analyses. Thus, the first problem with using ordinal variables does not apply here. However, both the question of normality and of the equality of $\Sigma(\theta)$ and Σ are relevant and will be discussed sequentially.

Univariate, bivariate and multivariate normality assumptions. The second consequence underscored by Bollen (1989) hinges on the lack of normality of the ordinal variable's distribution. Univariate normality is of concern because it is related to the assumption of multivariate normality. Before assessing whether this assumption is violated, I will briefly review the main consequences of the lack of normality of indicator variables in SEM. The most commonly noted problem that has been noted with maximum likelihood (ML) estimation in the presence of non-normality is the inflation of Chi-square values and of standard errors, which would respectively bias the model towards type I error, or rejecting a model that should not be rejected (Bollen 1989; Curran et al. 1996). Still, simulations studies also suggest that ML parameter estimates show no evidence of bias when the model is properly specified (Curran et al. 1996).

In these analyses, the measure of self-rated health has five categories. Graphically, we can see from Figure 1 that this variable roughly follows the normal curve in terms of its kurtosis, but that it exhibits some left skewness, as there are fewer people in poor health than in excellent health. However, the Shapiro-Francia test of normality (Thode 2002) on this variable does not allow us to reject the null hypothesis of normal distribution (W'=0.99285; V'=1.243; Z=0.466; p=0.321). Thus, univariate normality does not appear to be an issue here, even with the dependent ordinal variable.

However, as it is possible for variables to have normal marginal distributions but no multivariate normality, we must also test for the latter. I rely here on two tests provided by PRELIS 2.5. First, bivariate tests of normality between the ordinal variables and the continuous variables and secondly the omnibus Mardia test for the joint hypothesis of no multivariate skew or kurtosis (see Bollen 1989 p. 423 for a more detailed description). The PRELIS tests of bivariate normality for each pair of variables use polychoric correlations and indicate that about one fifth of the 47 pairs tested here did not meet this assumption. However, these departures from normality were apparently not substantial, as the Mardia test for the null hypothesis of no excessive multivariate skew or kurtosis was found to be non-significant ($\chi^2_{df=2} = 1.652$; $p = 0.438$). Thus, the second consequence of using ordinal variables in SEM – the violation of normality assumptions – does not appear to be a significant problem in these analyses.

Equality of the population covariance matrix to the observed covariance. Yet, even in the presence of normality, the last potential consequence of using ordinal variables, namely that $\Sigma \neq \Sigma(\theta)$, or that the population covariance matrix not be equal to the observed covariance

matrix, remains an issue. Thus, Bollen (1989) argues that using a simple covariance matrix in this case would be erroneous, as it does not recognize the categorical nature of the data.

Therefore, a widespread strategy used to circumvent this issue has been to rely on a polychoric correlation matrix, which assumes that continuous variables underlie the categorical observed variables (see Bollen 1989, pp. 439-446 for a more involved derivation of these issues). A common procedure to estimate this matrix (and the one used in PRELIS) is to estimate the thresholds for each ordinal variable from univariate marginals and estimate the polychoric correlations on these thresholds.

Yet, while the polychoric *correlation* matrix has been most commonly used in the literature, Jöreskog (2004) developed a method that is more appropriate when response alternatives are the same for several variables in the model, which uses a polychoric *covariance* matrix that fixes the first and second thresholds of the latent variable to 0 and 1 respectively, and estimates the mean and variance of the underlying latent variables. The resulting covariance matrix (as well as the asymptotic covariance matrix) is simply a scaling of the polychoric correlation matrix using the standard deviations of the ordinal variables with fixed thresholds as a scale factor (see Jöreskog 2004 for the derivation).

In addition, Hipp and Bollen (2003) count three main estimation strategies currently used today to analyze these polychoric correlation/covariance matrices: weighted least squares (WLS), diagonally weighted least squares (DWLS) and maximum likelihood (ML).

Weighted least squares relies on the asymptotic covariance matrix to weigh the data to correct for non-normality, and was originally the main solution recommended by Bollen (1989). However, recent simulation studies have indicated that WLS estimators are not robust for models with many variables or all but very large samples[5] (Curran et al. 1996).

Diagonally weighted least squares employs only the diagonal of the asymptotic covariance matrix as a weight, which results in a more easily invertable matrix (Jöreskog and Sörbom 1984). However, information is lost in DWLS, since the off-diagonal elements are ignored, and this estimation method does not allow a straightforward comparison of nested models (Hipp and Bollen 2003).

Finally, Jöreskog (2004) has very recently developed a new estimation technique using maximum likelihood estimation but correcting chi-squares and standard errors for non-normality using the asymptotic covariance matrix. Because this method does not require that the asymptotic covariance matrix be inverted, it is more stable with samples as small as 200 observations. In LISREL 8.54, this method also yields the Satorra-Bentler chi-square, which was found to outperform other chi-square tests in robustness to non-normality in a variety of simulations (Curran et al. 1996).

Given the various advantages and disadvantages of these methods, I will first estimate a polychoric covariance matrix using PRELIS 2.5, because, as the next section will show, the models I estimate include two distinct ordinal variables measured on the same scale. In addition, I will follow Jöreskog's (2004) recommendation to use the third estimation method in LISREL 8.54, namely maximum likelihood estimation with asymptotic covariances correction of standard errors, as it appears to be the most stable with small samples.

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[5] Minimal sample sizes of 2,000 cases and even as high as 5,000 have been suggested.

Sibling resemblance models

While fixed effects models have obvious advantages for the study of the impact of health insurance on adult health (Quesnel-Vallée 2004), their contribution is much more limited as far as status attainment models are concerned. Indeed, the strength of fixed effects models lies in the differencing out of mean family effects, which essentially precludes the estimation of between-family effects on endogenous variables (Hauser, Sheridan and Warren 1999). In other words, fixed effects models would not permit the estimation of the role of family background in the relationship between status attainment, health insurance and adult health.

Building on Blau and Duncan (1967) and Jencks and his colleagues (Jencks et al. 1972, 1979), Hauser and Mossel (1985, 1987) have used SEM to model the statistical decomposition of variances and covariances into between-family and within-family components. They named this model the "sibling resemblance model" in recognition of its use of the similarity of siblings in estimating the effects of background on achievement. While the sibling resemblance model has generated a substantial amount of research on status attainment and occupational achievement (see Hauser et al. 1999 and Warren et al. 2002 for the most recent developments), it has never been used to study adult health up until now.

Error! Reference source not found. represents the typical structure of a sibling resemblance model, estimating here the impact of early health on adult health. Using this example, I will successively follow Hauser and Mossel (1987) and Hauser's (1988) demonstration to describe sibling resemblance models in general, and in the next subsection, I will expose the specific model tested here.

Using LISREL notation, and assuming that the variables are deviated from their means, the sibling resemblance model presented in Figure 2 can be expressed as a function of four observed variables: X_1 and X_2 are measures of early health respectively for the first and the second sibling (there was no particular order in the selection of siblings), and Y_1 and Y_2 are measures of adult health, again respectively for the first and the second sibling. Both X_1 and X_2 are indicators of the common family factor of early health ξ_3, and are also respectively indicators of the within-family (sibling-specific) components of early health ξ_1 and ξ_2, as is expressed in equations 1 and 2

$$X_1 = \xi_3 + \xi_1 \quad \textbf{(1)}$$

$$X_1 = \lambda^x_{23}\xi_3 + \xi_2 \quad \textbf{(2)}$$

Similar equations define the within and between dimensions of adult health:

$$Y_1 = \eta_3 + \eta_1 \quad \textbf{(3)}$$

$$Y_2 = \lambda^y_{23}\eta_3 + \eta_2 \quad \textbf{(4)}$$

In the previous four equations, $\text{cov}[\xi_i, \xi_j] = \Phi_{ij} = 0$ for $i \neq j$, which indicates that the within-family disturbances are not correlated to one another, nor to the between-family factor. Finally, the structural model in equations 5 to 7 specifies that the impact of early health on adult health is the following, respectively for sibling 1, families and sibling 2:

$$\eta_1 = \gamma_{11}\xi_1 + \zeta_1 \quad \textbf{(5)}$$

$$\eta_3 = \gamma_{33}\xi_3 + \zeta_3 \quad \textbf{(6)}$$

$$\eta_2 = \gamma_{22}\xi_2 + \zeta_2 \quad \textbf{(7)}$$

The loadings of 1 on the paths between the latent and observed variables indicate that these latent variables are in the metric of the observed variables. Finally, the 0 error term on this observed variable points to the assumption of perfect measurement (this is a necessary assumption, without which the model would not be identified).

As it is presented in Figure 2, the model is not identified, as there are more paths to estimate than elements in the covariance matrix. With four variables, we only have 10 sample moments (4 variances and 6 covariances), while the model to be estimated counts 11 parameters: 3 variances of ξ's, 3 variances of disturbances in η's, 3 structural regressions (γ's) and 2 scale factors (λ's). Thus, in order to identify the model, two other restrictions need to be imposed on the parameters, namely $\lambda_{23}^{x} = \lambda_{23}^{y} = 1$. I follow Hauser and Mossel (1987) in selecting these restrictions, as they allow the two pairs of sibling-specific latent variables to be in the same metric as the common family factor, which permits the comparison of the slopes among the three regressions γ_{11}, γ_{22}, and γ_{33}. With these additional restrictions, the model can be identified with 1 degree of freedom.

This brings us to the estimation of the effect of early health on adult health. In this model, the common family factor for adult health is a function of the common family factor for early health, and an error term ζ_2, just as each sibling's latent adult health is a function of their latent early health and an error term. In consequence, the effect of early health on adult health is expressed in these analyses by three coefficients, γ_{11}, γ_{22}, and γ_{33}, respectively for sibling 1's specific effects, the common family effects, and sibling 2's specific effects. Therefore, for instance, γ_{11} represents the effect of those latent factors that uniquely affect sibling 1's own early health on those factors that uniquely affect sibling 1's own adult health.

Therefore, comparing γ_{11}, γ_{22}, and γ_{33} also allows us to determine whether family background biases the relationship between early health and adult health at each sibling's level. Consequently, if I found that γ_{33}, or the common family effect of early health on adult health, is significantly different from γ_{11} and γ_{22}, I would argue that the family background does affect the relationship between early and adult health, for instance through genetic or childhood environment effects.

To better illustrate how this model can provide within-family effects that are free of family bias, we can substitute equations 5 and 6 in equation 3 and thus write out the reduced form equation for the adult health of sibling 1 as:

$$Y_1 = \gamma_{33}\xi_3 + \gamma_{11}\xi_1 + \zeta_3 + \zeta_1 \quad \textbf{(8)}$$

and similarly, the reduced form equation for the adult health of sibling 2 is:

$$Y_2 = \gamma_{33}\xi_3 + \gamma_{22}\xi_2 + \zeta_3 + \zeta_2 \quad \textbf{(9)}$$

Note that both of these equations display the same the between-family effect of early health. In consequence, the within-family effects of early health on adult health $\gamma_{11}\xi_1$ and $\gamma_{22}\xi_2$ that are

respectively specific to siblings 1 and 2 are in fact net of the common between-family effects of early health.

Hypothesized structural model

Figure 3 illustrates the full structural model that I will estimate. Note that this model is simplified and presents only one latent variable (and path) for each measure. The actual sibling resemblance model that will be estimated has in fact three latent constructs (and paths) for each dimension of health or SES that is presented in Figure 3, namely one for each sibling and one common family factor.

Thus, this model will examine the recursive pathways[6] that exist between race, early health, education, and household income, hours worked, as well as the number of years uninsured and publicly insured in early and mid-adulthood, and the impact of these pathways on adult health.

In this model, early health is separated into two components, a permanent component, measured by the number of years obese in early adulthood, and a transitory component, measured by the number of years health prevented respondents from working in early adulthood. Early health is hypothesized to have an indirect effect on health through the number of hours worked, household income and health insurance. Similarly, education (measured in 1984) and race are hypothesized to have indirect effects on health through household income and hours worked. The errors of the latent variables of early health and of education of each sibling and of the common family factors are allowed to covary[7].

In turn, the number of hours worked and household income are also hypothesized to have an indirect impact on health through the health insurance measures. Finally, the health insurance measures both have a direct impact on health in this model. This model will be gradually built through a series of nested models beginning with a direct effect of early health on adult health, and the subsequent addition of education, household income and hours worked, and health insurance measures.

DATA

The data used for this study are drawn from the 1979 National Longitudinal Survey of Youth, as it is to my knowledge the only survey to allow such a detailed, prospective study of the parallel evolution of labor-force patterns, provision of health insurance and health.

The NLSY79 is an ongoing longitudinal panel survey that has been following since 1979 a national probability sample of American civilian and military youth aged 14 to 21 years old in 1978 (Zagorsky & White, 1999). The NLSY79, sponsored by the Bureau of Labor Statistics (BLS), was designed principally to gather longitudinal information on the socioeconomic status and labor force experiences of young American men and women. As such, the NLSY79 is particularly well-suited for the study of stratification outcomes, as it includes data about social

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[6] Although this is not strictly speaking a recursive model, since some errors are allowed to covary among the endogenous variables.

[7] For instance, the number of years obese and education for sibling 1 are allowed to covary, but there are no correlated errors between sibling 1 and 2, or between either sibling and the common family factor.

origins and traces a comprehensive, prospective and continuous work history spanning 21 years of the life course of its respondents, from ages 14-21 to 37-44.

Recent additions to the survey also make it an important source for the study of health over the life course. Up until 1998, data on respondents' health was collected at several points in time through the years, but mainly restricted to health concerns limiting the ability to work and to health behaviors (body mass index, smoking, drinking). In 1998 and 2000 this data collection became broader and more systematic, as all respondents age 40 and over were asked a supplementary battery of questions on general health concerns, including mental health, diagnosed conditions, and health-related quality of life.

The NLSY79 also provides detailed and continuous information on health insurance coverage. In addition to information on employer provided health insurance that was collected for most years, these data provide information on yearly health insurance coverage from any source at the time of survey from 1989 to 2000. Moreover, beginning in 1993 and until 2000, the uninsured/insured status was reported monthly.

Finally, a crucial and underused feature of the NLSY79 is its household sampling scheme that included in the study all the eligible individuals in a household. This sampling strategy has yielded 596 clusters of siblings, making it an optimal dataset for the use of models with siblings pairs.

The most important limitation of the NLSY79 for the purposes of this research is that the respondents are still relatively young at the latest wave (40-44), and thus are unlikely to report serious ailments in significant numbers. However, the NLSY79 has detailed measures of self-reported health-related quality of life. In addition to being strong predictors of mortality and morbidity (Mossey and Shapiro 1982), these measures follow socioeconomic differentials even in early and mid-adulthood (Power et al. 1999).

The NLSY79 thus presents a remarkable opportunity for studying the unfolding work history and health of a nationally representative sample of Americans born in the late 1950s and early 1960s, and residing in the United States when the survey began. Very few surveys presently allow such a detailed follow-up of the parallel evolution of labor-force patterns, provision of health insurance and health.

SAMPLE

The sample under study here was limited first by the fact that the dependent variable of interest (health in mid-adulthood) was asked only of respondents 40 years or over in 1998 or 2000. In addition, the NLSY79 was originally composed of three probability samples, one nationally representative of the noninstitutionalized civilian youth population, one oversampling economically disadvantaged youth and one oversampling the military. The military oversample was mostly dropped in 1991. The white males and females of the economically disadvantage sample were dropped in 1991. Thus, the sample was limited to those who were 18 to 22 years old at baseline in 1979 and who were part of the probability sample for the whole period of observation (N=5,026).

In addition, siblings were identified as full siblings if they were listed as brothers or sisters in 1979, had the same race/ethnicity, were in agreement as to whether their biological mother and father were deceased as of 1998 or 2000 and were confirmed as full siblings by a kinship linking algorithm described in greater detail in Rodgers, Buster and Rowe (2001).

Among these respondents, 596 clusters of siblings were identified in 1979. However, attrition over the 19 to 21 years that these individuals were followed brought the number of

sibling clusters who responded to the health supplement in 1998 or 2000 to 287. Deletion of all the missing values on the variables of interest yielded between 227 to 235 clusters of siblings, or from 476 to 511 individual respondents (depending on the explanatory variables included in the model).

While this seems like a large drop in the sample under study, it is important to remember that attrition will hit the study of sibling clusters hardest, as the loss of only one sibling can lead to the loss of a cluster. Moreover, the age restriction in the population responding to the health supplement may have lead to the loss of clusters if one of the siblings was not aged 40+ in 1998 or 2000.

The remaining siblings (stayers) differed significantly from those who dropped out of the survey (attriters) only in that they were in 1978 on average older by two years, had nine more months of education and a higher body mass index and that they came from smaller families ($\mu_{stayers} = 5.23; \mu_{attriters} = 5.51$) who were less likely to be in poverty. In contrast, there were no significant differences on race, gender, parents' education, household income in 1978, income to needs ratio in 1978, number of siblings in 1978, obesity in 1980, and health-related work limitations in 1978 between these two groups.

Beyond the concerns specific to sibling data, possible biases resulting from survey attrition must also be addressed, particularly with data covering such a long time span. In an analysis of attrition in the NLSY79, MaCurdy, Mroz and Gritz (1998) found that, despite being nonrandom, attrition did not introduce biases in the estimation of earnings and other labor-market variables. However, no comparable studies have been conducted as of yet on the impact of attrition on health in the NLSY79. My own analyses indicate that a marginally significantly greater proportion of NLSY79 siblings reported being in fair or poor health than respondents from the same age bracket in the nationally representative National Health Interview Survey 2002 (NHIS 2002: 8.52%, 95% CI[7.86; 9.19]; NLSY79: 9.91%, 95% CI[7.96; 11.86]), which may be due to stayers being older on average.

This pattern contrasts with previous studies that found that attriters were more likely to be in poor health (see the five-paper series in the Journal of Clinical Epidemiology, 2002, volumes 55 and 56, introduced by Deeg 2002, as well as Norris 1987). It is possible that this is due to the fact that the NLSY79 makes every attempt to, and is quite successful at bringing back attriters into the survey. For instance, MaCurdy et al. (1998) found that attriters were more likely to be unemployed before leaving the survey, but that those who were brought back into the survey at a later period also exhibited lower employment levels than the stayers. A parallel process may be at play with health, with the least healthy attriters being brought back in the survey at later periods.

More analyses are obviously needed to assess the impact of attrition on health in the NLSY79. However, given the slight, yet conceptually consistent differences between stayers and attriters in the NLSY79 exposed here, and based on prior studies of health-related attrition bias in panel studies, it is likely that this impact of nonrandom attrition on descriptive statistics will not be associated with discernable bias in the estimation of the relationships between those variables (Deeg 2002; Norris 1987).

MEASUREMENT

The NLSY79 provides longitudinal information on the respondents' health, health insurance coverage and socioeconomic status. The main outcome, general health, was measured only among individuals who were 40 years and over in 1998 or 2000. Respondents who had answered the health supplement in 1998 were not asked the questions again in 2000. Thus the outcome of interest was measured in 1998 for some respondents and in 2000 for others.

In order to ensure a time lag between the measurement of work experience and health insurance coverage that was equal between these two groups, health insurance variables were measured until 1996 for respondents whose health had been assessed in 1998, and in 1998 for those whose health had been assessed in 2000. Household income was reported for the past calendar year and so the last measures of income come from the same survey year as the health measures.

The measurement of variables will be described in the following section.

Dependent variables

Self-reported general physical health 1998/2000.

The main measure of health in these analyses comes from a single question assessing the respondent's general health ("In general, would you say your health is – Excellent, Very good, Good, Fair, Poor"). One of the reasons to use this measure is that it permits to replicate results of prior studies. When necessary, this variable may be dichotomized into fair or poor health versus good, very good or excellent health.

The primary reason for using measures of health-related quality of life is that they circumvent a limitation of the NLSY79, namely that the respondents are still young at the latest waves (40-44). While the respondents are unlikely to suffer from serious ailments in significant numbers, they are already reporting health-related limitations to their quality of life. More specifically, about 12% or the respondents reported having fair or poor health even at these young ages.

Moreover, research indicates that these health-related limitations are strong predictors of mortality and morbidity, mainly because poor self-reported health is – even at these young ages – not transitory (Mossey and Shapiro 1982; Power et al. 1999). In other words, for the main part, those who are reporting poorer health at these ages are already on a downward slope in terms of health, and thus these findings will have substantial implications for the future health of those aging populations (Power et al. 1999).

In addition, there are also strong substantive reasons for relying on this measure in the study of health insurance and health. First, it provides an evaluation of respondents' health status that is relatively independent from their propensity to seek medical care and use formal services (Zagorsky & White 1999). As such, this measure is in great part free of the endogeneity problems inherent in the study of diagnosed conditions among insured and uninsured individuals that stem from the differential propensity of these two groups to have regular access to care, and thus to diagnosis and awareness of medical conditions (Seccombe and Amey 1995). Finally, measures of health-related limitations may also be theoretically more appropriate than the use of mortality rates. Indeed, while access to medical care may have played a limited role in limiting mortality, it has undoubtedly increased individuals' health-related quality of life, and yet very few studies have paid attention to this dimension of health (Robert and House 2000).

Independent variables: The process of stratification

Respondent's highest grade completed.

The highest education level completed is considered here the first level of achieved status attained by the respondents. This measure ranges from 0 to 20 years of education. This variable measures the highest grade completed in 1984 so that the period of observation financial resources may begin in 1985.

Thus, the year of assessment of education was chosen to respect temporality in the measurements, but in fact, this measure of educational attainment very closely approximates the highest degree attained in 2000, as three-fourths (75.02%) of the respondents had attained their final (2000) educational status in 1985. Average values (of education in 2000 by education in 1985) show even greater congruence and do not exceed two years. All analyses were replicated with measures of education in 2000 and yield comparable results.

Hours worked annually.

A variable measuring a logarithmic transformation of the average number of hours (in 100 of hours) worked annually (measured in any given survey year for the past calendar year) from 1986 to 1997/99 was included in the analyses.

Financial resources.

Financial resources from 1986 to 1997/99 were operationalized by household-level variables, namely *household income* and the *income to needs ratio*. These measure were reported for the past calendar year, so 1998 and 2000 reports were used with respondents who reported their health in the same years respectively. Household income is measured as the total household income of all family members in $10,000s constant dollars, inflated to 2000 price levels using the Consumer Price Index. Less than 1% of the cases in any given year had incomes of $200,000 or more. These cases were examined for possible miscoding and were found to be legitimate. However, these high values did pull the average up to $40,199 (individual sample) and to $45,755 (sibling sample) while the respective medians were $30,368 and $32,733, so the data were topcoded at $200,000, given that average household income is a measure of interest here.

The income to needs ratio is obtained by dividing household income by the poverty levels provided by the NLSY79 for each respondent. These poverty levels were based on the official U.S. poverty thresholds and take into account family size and region of residence.

A household-level variable was preferred over individual measures of SES in these analyses for several reasons. First, as the Institute of Medicine (2002b) put its in a recent eponymous report, "Health insurance is a family matter". While this report deals more with consequences of lack of coverage, it also points at the importance of taking into account the household determinants of coverage. Indeed, a number of individuals rely on their spouses for health insurance (Dushi and Honig 2003), and thus household measures are appropriate precursors of the likelihood of obtaining coverage in later years.

In addition, this is a prime childbearing period for the women in this sample, and using a household measure circumvents the fact that they may temporarily leave the labor force due to a pregnancy. Finally, household measures of resources may be more valuable in understanding socioeconomic inequalities in health (Chandola et al. 2003b), as they are argued to be for national mobility regimes (DiPrete 2002).

Health insurance.

A number of problems in measuring the impact of health insurance on health emerge from the instability of health insurance coverage over time in the United States. For instance, the

median length of time that nonelderly adults remain on Medicaid is five months (Tin and Castro 2001). Even in longitudinal studies, health insurance status is often only measured at baseline (IOM 2002a). Thus, changes in status or continuity of coverage cannot be assessed even though they may lead to overlap in group membership, as when for instance an individual moves from being covered under Medicaid to being uninsured during the period of observation (IOM 2002a). In consequence, the current study goes beyond much of previous literature by assessing the health insurance status of all respondents repeatedly over up to nine years, as health insurance coverage and the source of that coverage were measured at every survey between 1989 and 2000 (except for 1991).

Prior to 1989, only workers were asked if they had access to health insurance through their employer. While these measures were available in almost every survey year from 1979 to 2000, they were not used here because they provides information only for working individuals, and only about employer-provided health insurance, whereas one of the goals of these analyses was to contrast the effects of public and private health insurance.

Starting in 1989, respondents were asked if they were covered by any health or hospitalization insurance plan. If they acquiesced, they were then further queried as to the source(s) of this plan. Possible sources included the respondent's current employer, past employer, the spouse's current employer, past employer, privately bought insurance, or public insurance (Medicaid, Medi-Cal, Medical Assistance, welfare, medical services).

Respondents could name multiple sources for type of health insurance coverage. Respondents who obtained health insurance through their current or past employer, their spouse's current or past employer, or who purchased it privately were coded as having private insurance, regardless of whether they also reported another type of coverage.

In order to ensure an equal time lag between the measurement of work experience and health insurance coverage, health insurance was measured until 1996 for respondents whose health had been assessed in 1998, and in 1998 for those whose health had been assessed in 2000. Therefore, variables were created to measure the number of years uninsured, privately insured, and publicly insured between 1989 and either 1996 or 1998.

While there are in fact up to nine calendar years of observation between 1989 and 1998, health insurance was not assessed in 1991, and the NLSY79 was conducted biannually beginning in 1994. Thus, health insurance status in 1994, 1996 and 1998 was measured as contributing only one year and the number of years insured through any type of insurance therefore ranges from 0 to 7 years. . However, results did not change in analyses with an alternative measure where each biennial coverage assessment contributed two years (results not shown here).

Finally, because the number of years uninsured, privately insured and publicly insured would sum up to the whole period of observation for any given respondent, years privately insured or years uninsured will be successively omitted from the models to avoid perfect multicollinearity. Moreover, this omission will allow the effects of being uninsured or privately insured to be evaluated in contrast with the other sources of health insurance coverage.

Control variables

The capacity to work and, even more importantly, to secure health insurance, are both influenced by prior health. In consequence, in addition to race/ethnicity (White, non-Hispanic; Black; Hispanic) and gender, variables measuring health prior to 1985 (when measurement of achieved status begins) were also included as controls. The NLSY79 included no measure of general health prior to 1998. However, some indirect measures of health were collected that

permit to control at least minimally for prior health. Two early health measures will be used that are hypothesized to capture respectively the permanent and transitory components of health, namely the number of years obese and the number of years unable to work because of a health reason. By taking the number of years respondents report these health problems, acute limitations such as accidents from which the respondents recover can be differentiated from more chronic and persisting conditions.

Early health: Permanent and transitory components

Health can be conceptualized as having two components, a permanent one and a transitory one (Jasso, 2003), and consequently two measures of early health were chosen that can be thought of as each reflecting primarily one of those components. The permanent component of health is more closely related to genetic and developmental influences that set the stage for adult health, while the transitory component of health is affected by proximate environmental conditions and may change rapidly. In this sense, obesity in early adulthood perhaps best reflects the permanent component of health, as it is related both to genetic predispositions and environmental factors in utero and during childhood (nutrition and exercise for instance). In turn, being unable to work because of health reasons in young adulthood may be more related to random shocks to health that affected the transitory component of health in individuals. Of course, neither measure exclusively represents one component or the other.

Years obese in early adulthood. This variable is based on the respondents' body mass index (BMI), and covers the years 1981 to 1985. The BMI was calculated with the following formula (Willett, Diez & Colditz 1999):

$$BMI = \frac{Weight(lb) * 703}{Height(in.)^2} \qquad \textbf{(10)}$$

Height was measured in 1981, 1982 and 1985 in the NLSY79, while weight was measured more often, namely in 1981, 82, 85, 86, 88, 90 and from 92 to 2000. Height and weight from the same years were used if available, but height in 1985 was used for calculations of the BMI for later years (86-90). Following the National Heart, Blood and Lung Institute guidelines (NHBLI 1998), individuals were considered obese if their BMI was greater than or equal to 30. A high BMI (>25) is associated with an increased risk of diabetes, hypertension, coronary heart disease, and all-cause mortality, including death from cardiovascular disease and cancer (Calle et al. 1999; Willett, Dietz & Colditz 1999). This measure ranges from 0 to 3 years.

A measure of years overweight (24<BMI<30) was also considered, but it did not predict health beyond years obese, and so was not included in the analyses due to its high correlation with the former variable. In addition, a measure of years underweight was not used here given the debate about the predictive value of this factor for health. Researchers argue that it is still unclear what this group of individuals represent, as they include both individuals who are healthy, watch their weight and exercise and those who weigh less but not because they are healthier (smokers for instance, or those who just lost weight as the result of an illness) (Willett et al. 1999).

Years unable to work in early adulthood. This variable measures the number of years respondents reported that their health prevented them from working altogether between 1978 and 1984. The measures for socioeconomic status begin in 1985, and years with a health limitation should provide some measure of the confounding in securing employment – and thus health insurance – due to prior health status and capacity to work.

While more detailed information on the specific ailments was provided from 1979 to 1982, only the general questions were used here for comparability purposes (as these questions were asked in virtually every NSLY79 survey from 1979 to 2000), and because they are conceptually more relevant to general health. Relying on health-related work limitations may not permit the detection of minor chronic health problems or of slowly progressing illnesses (Zagorsky and White 1999). However, it should screen out the most extreme cases of ill-health or disability. Moreover, by taking the number of years respondents report a work-related limitation, I can differentiate acute limitations such as accidents from which the respondents recover from more chronic and persisting conditions. This measure ranges from 0 to 7 years.

RESULTS

Family effects on health, schooling, financial resources and health insurance

Table 1 presents the estimates of between- and within- family components of variance for each latent construct. The first line for each variable shows the absolute estimate of the between-family, within-family for siblings 1 and within-family for siblings 2 variance components. For instance, the between-family component of variance for adult health is 0.232, while the corresponding within-family components for siblings 1 and siblings 2 are respectively 0.805 and 1.089. The second line for each latent construct first presents the percentage of total variance accounted for by each within-sibling component. Thus, the within-family variance in adult health of siblings 1 is 77.6% of their total variance (100*0.805/[0.805+0.232]), and similarly, the within-family variance in adult health of siblings 2 is 82.4% of their total variance (100*1.089/[1.089+0.232]).

Following Hauser et al. (1999), these percentages can be understood as inverse measures of the correlation between hypothetical siblings who are either like siblings 1, or like siblings 2. In this sense, for siblings 1, 22.4% of the variance lies between families, which corresponds to a correlation of 0.224 for two "siblings 1" from the same family. For two "siblings 2" this correlation would be 0.176.

Taken together, this means that we can calculate the correlation between two siblings' health implied by the model by taking the geometric average of those two siblings correlations. As such, the model implies a correlation of $(0.224*0.176)^{1/2}=0.198$ between the adult health of the two siblings, which is the congruent with the estimate for the intra-class correlation reported in Quesnel-Vallée (2004). These correlations are in the fourth column, second line for each variable of Table 1.

These results show that, with the exception of education and household income, the within-family component of variance generally accounts for more of the total variance than the between-family component. This suggests that the factors observed here are overall more variable within than between families.

However, there are also theoretically coherent and substantial differences in the extent to which the factors show more or less variance between families. Consistent with the hypothesis that this measure is closely related to a transitory component of health, work limitations barely exhibit any correlation between the siblings, while the number of years obese shows a much greater between-family component of variance.

The log of hours worked in early and mid-adulthood also varies quite modestly between families, suggesting that this variable depends more on sibling-specific characteristics such as

gender or local labor markets. Interestingly, adult health exhibits a similarly limited amount of variance between families, which may be due to the young age of the respondents.

The health insurance variables exhibit moderate between-family effects, as the within-family component of these variances accounts for between two-thirds and three-fourths of the total variance. In addition, as hypothesized, public health insurance suggests a slightly larger correlation between siblings than the number of years uninsured does.

Finally, status attainment variables, i.e. education and household income, have the strongest between-family component, which in both cases accounts for more than half of the total variance. This strong between-family component was to be expected for education, especially since it is measured early on in the life course. However, it is somewhat more surprising that household income should be so strongly related to the family of origin, as previous studies have shown that within-family variance increases across the life course (Hauser et al. 1999; Warren et al. 2002). This substantial between-family component may be due to the fact that the respondents are still young even at the last wave[8].

In sum, to the exception of the number of hours worked, status attainment variables and obesity in early adulthood exhibit the greatest between-family component of total variance. Health insurance variables and adult health have moderate between-family components of total variance, consistent with their measurement later in the life course. Finally, as hypothesized, work limitations in early adulthood have substantial within-family components that suggest that this indicator truly measures a transitory and highly individual component of health.

Model fit

To arrive to the final model, which is graphically represented in Figure 3, I followed a two-step approach in comparing hierarchically nested models. I started with a model with only the latent variables, but no path between them (this was necessary to ensure that the same covariance matrix was compared throughout). Then, I sequentially added the block of paths and covariances relevant to a given predictor of health and time period.

Within these conceptual blocks, I then simplified the models to make them more parsimonious. First, I tested for equality of the siblings' regressions. Then, I set paths to 0 when it was theoretically warranted, or for non-significant paths that were more exploratory. Lastly, in the final model including the health insurance measures, I also tested for the equality of the siblings regressions to the family regressions. A subset of fit indices for these analyses are presented in Table 2.

I used tests of chi-square difference to assess fit. The p-value for the Satorra-Bentler scaled chi-square must be non-significant to accept that the model has good fit. Therefore, a significant decrease in chi-square indicates an increase in fit. Two nested models can be compared by computing the absolute difference in their chi-squares values, which itself follows a chi-square distribution with the difference in degrees of freedom between the two models as the degrees of freedom.

In addition, I also report the following fit indices: the root mean square error of approximation (RMSEA), the Akaike information criterion (AIC), the comparative fit index (CFI), and the standardized root mean square residuals (SRMR).

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[8] Considering for instance that Hauser et al.'s (1999) WLS respondents are 50 years and over.

The RMSEA must be below 0.05 to accept the model, and smaller values for both the AIC and the SRMR indicate better fit. Thus, decreases in RMSEA, AIC and SRMR suggest a better fitting model. In contrast, the CFI must be above 0.90 to accept the model, and so increases in CFI point to better fit.

The first block of variables to be added was early health, along with a direct effect of race (white vs. non-whites) on health. Early health is measured in these analyses by two conceptually distinct set of factors, obesity and the incapacity to work due to health reasons in young adulthood. As was discussed earlier, health can be conceptualized as having two components, a permanent one and a transitory one (Jasso, 2003), and these two measures can be thought of as each respectively reflecting primarily one of those components. Of course, neither variable will exclusively measure one component or the other, but if these hypotheses are true on average, then we should find that obesity exhibits a stronger family bias than work limitations.

Now, turning to the analyses, we see that the first model estimated is found not to fit the covariance structure well. This is not surprising, given that the covariance matrix relied on here includes all the latent variables in the models but constrains most paths between them to 0. The same model estimated with a covariance matrix including only sample moments between early health, adult health and race yields the same parameter estimates but actually fits the data, and this will be true of all the models discussed here that do not estimate paths between all the latent variables.

In turn, both the decline in the RMSEA and AIC and the difference of chi-square test indicates that setting the siblings' paths of early health on adult health equal for each health measure in Model 2 yields a significant increase in fit ($\chi^2_{df=2} = 7.12$; $p = 0.028$). In Model 3, the non-significant covariances of obesity and the work limitations are set to 0 for each sibling, without any loss of fit ($\chi^2_{df=2} = 1.02$; $p = 0.600$). This change highlights the fact that obesity and work limitations may in fact be distinct conceptual entities that are not systematically related to one another at the individual level. The covariance between the common family factors for each of these measures remains significant, suggesting that even work limitations may have a slight part of family variation (as we would see for instance with young adults with early onset genetic or congenital diseases).

In Model 4, the block of paths and covariances pertaining to education is then added to Model 3, which results in smaller RMSEA, AIC and SRMR, a greater CFI and a significant decrease in chi-square ($\chi^2_{df=10} = 63.55$; $p = 0.000$). Here the covariances are estimated between the common family factors for education and obesity, education and work limitations, and education and race. Similarly, covariances are estimated between each siblings' unique latent factors for education and obesity, and education and work limitations. Again, setting the paths of education on health equal between siblings in Model 5 results in decreased RMSEA and AIC and a significant decrease in chi-square ($\chi^2_{df=1} = 7.15$; $p = 0.000$).

Model 6 adds the block of paths and covariances related to the variables measuring the impact of financial resources in early and mi-adulthood on adult health. Financial resources are measured here by the logarithm of average income over the period 1985 to 1997/99. This conceptual block also includes a variable to control for the individuals' attachment to the labor force, namely the logarithm of the average hours worked over the same period. Covariances are estimated between the common family factors for household income and hours worked, as well as between those same sibling-specific latent variables. Finally, paths are also drawn between all the variables existing in Model 6 and both household income and hours worked.

The addition of all these paths in Model 6 increases fit relative to Model 5, as evidenced by the decrease in the RMSEA, CFI and SRMR, increase in CFI, and the concomitant significant decrease in chi-square ($\chi^2_{df=29} = 211.50$; $p = 0.000$). Model 7 sets the paths of financial resources on adult health equal for the siblings, and while this does not result in a significant decrease in chi-square ($\chi^2_{df=1} = 0.82$; $p = 0.999$), it does improve fit as indicated by the decline in RMSEA and AIC. Models 8, 9, 10 and 11 respectively test whether the household income and hours worked factors mediate the direct impact of race, education, obesity and work limitations on adult health. All of these modifications result in lower RMSEA and AIC and a significant decrease in chi-square for work limitations (Model 10: $\chi^2_{df=2} = 6.94$; $p = 0.031$), and non-significant increases for race (Model 9: $\chi^2_{df=1} = 0.29$; $p = 0.590$), education (Model 12: $\chi^2_2 = 0.94$; $p = 0.625$), or obesity (Model 11: $\chi^2_{df=2} = 0.58$; $p = 0.748$). Thus, the hypotheses that early health and education should have both direct and indirect effects on health are not supported here.

Finally, some non-significant paths were set to 0 because they highlighted theoretically plausible mechanisms of status attainment. More specifically, Model 12 set the paths between education and hours worked to 0 under the hypothesis that education will affect more the type of job (and thus of income) that one attains, and not as much the number of hours worked. This modification did not significantly increase the chi-square ($\chi^2_{df=2} = 3.49$; $p = 0.175$), or change the fit measures, except for the AIC, which does decline slightly. In turn, even though some of the paths between health and the number of hours worked were non-significant, those were all left in the model, under the hypothesis that the number of hours worked may at least partly reflect an individual's health-related capacity to work. In contrast, household income may not be as dependent on individual health, as the other members of the household may be able to increase their work output to compensate for a drop in income. Thus, the paths from early health to household income were set to 0 in Models 13 and 14, without substantial changes in the fit measures or significant change in chi-square (Model 13: $\chi^2_{df=2} = 2.16$; $p = 0.340$; Model 14: $\chi^2_{df=2} = 4.67$; $p = 0.097$). These modifications suggest that the cumulative effects of household income and hours worked do in fact mediate the impact of race, early health, and education on adult health.

Model 15 adds all the paths and covariances relevant to health insurance in mid-adulthood to Model 14. In this case, covariances are estimated between the common family factors for years without insurance and years publicly insured, and between those latent factors for each sibling. In addition to the direct effects of health insurance on adult health, paths were also drawn between early health and financial resources variables and both sets of health insurance variables. Neither education nor race were hypothesized to affect health insurance variables directly. Compared to Model 14, the addition of this block of paths resulted not only in a significant decrease in chi-square ($\chi^2_{df=34} = 146.02$; $p = 0.000$), but also in a Model chi-square that is non-significant, to an RMSEA below 0.05, smaller AIC and SRMR, and a CFI well above 0.90.

Model 16 sets the paths to be equal between siblings, which does not significantly increase the chi-square value ($\chi^2_{df=10} = 7.49$; $p = 0.679$), nor the RMSEA, but does decrease the SRMR and the AIC. These fit indices thus designate Model 16 as the preferred, most parsimonious

model (compared to Model 15). Models 17 and 18 constrain to 0 paths affecting the number of years uninsured that were non-significant, namely those leading from health-related work limitations and obesity. These modifications did not significantly increase the chi-square values (Model 17: $\chi^2_{df=2} = .66$; $p = 0.883$; Model 18: $\chi^2_{df=2} = 1.17$; $p = 0.557$), nor change the RMSEA, but they did result in a decrease in AIC. This suggests that the number of years uninsured is not so much a function of prior health as of financial resources and employment. In the case of the number of years publicly insured, all paths were maintained even if not significant, due to the strong theorized bias in prior health and SES in this variable.

Finally, a set of models were estimated to test the constraint of equality of the family and siblings effects in Model 18. If those effects were equal, it would indicate that family background does not affect the endogenous variable under consideration. All of those models resulted in poorer fit (results not shown) and Model 18, which suggests that the common, between-family effects differ significantly from the siblings' within-family effects, therefore remains the final model.

The analyses in this section highlight a structural relationship where the impact of race, early health and education on adult health is not direct, but rather mediated by household income and the number of hours worked, as well as by health insurance. Moreover, the best fitting models constrain the within-family paths to be equal, while fit indices do not support the hypothesis of equality of between- and within-family effects.

THE CONTRIBUTION OF HEALTH INSURANCE TO THE RELATIONSHIP BETWEEN FINANCIAL RESOURCES AND ADULT HEALTH

In this section, I will first discuss the change in the total effects on adult health of race, early health, and financial resources that emerged from the nested models presented in Table 2 and secondly, I will cover the results of two variants of the final model.

NESTED MODELS OF TOTAL EFFECTS ON ADULT HEALTH

Table 3 presents the total effects and standard errors of the latent variables in the different nested models on adult health. In Models 3 and 5, these total effects are also the direct effects. In Models 14 and 18, the total effects are also the direct effects only for household income and hours worked and health insurance variables. Two columns are presented for each model; the column Between stands for the between-family (common family) effects, while the column Within stands for the within-family (sibling-specific) effects, which were all constrained to be equal for siblings 1 and 2 for a given latent variable.

The first thing to note in these results is that Model 18 is the only one to exhibit marginally statistically significant effects of the family of origin on health. These between-family effects indicate that being white (relative to non-white) works through the family background and has a positive effect on health.

Turning now to the within-family effects, only work limitations emerge in Models 3 and 5 as significantly related to adult health. However, this effect declines substantially in magnitude (by 66%) and is no longer significant with the addition of household income and hours worked. This suggests that later socioeconomic achievement may reduce the negative effects of early shocks to health. Finally, the addition of health insurance variables in Model 18 slightly increases this effect, though not significantly so relative to Model 14.

Contrary to what was hypothesized, education has no direct significant effect on health in Model 5, but the addition of household income and hours worked in Model 14 both decreases the

magnitude of this effect by more than two thirds, and renders it marginally significantly. As such, in Model 14, education and household income both exhibit positive, marginally significant total effects on adult health. Given that we would hypothesize that both of the these factors should have positive effects on health, we could evaluate the significance of those effects using one-tailed t-tests, and find them to be significant at the 0.05 level. Substantively, both of these effects are moderate, as each year of education is associated with a 0.02 increase on the scale of health – this would mean that 20 years of education, the maximum number of years reported in these data, would not even yield an increase of half a point on the health scale of 1 to 5. Similarly, a doubling of household income (100% increase) is only associated with 0.24 point increase in health (ln(200/100)*0.351=0.243).

These total effects of education and household income are no longer significant in Model 18 of Table 3, and the effect of household income slightly declines in magnitude with the addition of health insurance to the model. As in Quesnel-Vallée (2004), public health insurance has no significant effects on health once the unobserved effects of family background are controlled for, while the number of years uninsured does have a significant negative effect that is comparable to that estimated by Quesnel-Vallée (2004). In addition, note that, while the point estimate of years publicly insured appears to suggest a greater negative effect in these analyses, it is well within the 95% confidence interval of the effect estimated by Quesnel-Vallée (2004). Thus, the results presented in Table 3 provide added confidence that public health insurance does not have negative effects on health relative to private insurance, while the number of years uninsured does.

Status attainment, health insurance and adult health

In this section I will address more explicitly the issue of the contribution of health insurance to socioeconomic inequalities in health over the life course. I will begin by discussing the pathways of the main model already exposed, and follow with a discussion of a model measuring the effects of an alternative specification of health insurance. More specifically, while I first address the impact of lack of insurance and of public insurance in contrast to private insurance, the second model instead looks at the contribution of the source of coverage relative to being uninsured.

Years with private insurance as the reference group

In order to build a substantive understanding of the contribution of health insurance to socioeconomic inequalities in health and their intergenerational reproduction, I will first examine the structural equations with financial resources and health insurance as endogenous latent variables and subsequently interpret the pathways from these variables to health in light of these relationships. Figure 4 summarizes these relationships graphically, with Panel A presenting the between-family paths, and Panel B the within-family paths[9]. Table 4 presents the total effects (equivalent in this instance to direct effects) of race, early health and education on the log of average hours worked between 1985 and 1997/99 and the log of average household income in $10,000s adjusted for 2000 CPI, while Table 5 presents the direct, indirect and total effects of race, early health, and financial resources on the number of years uninsured and the number of years publicly insured between 1989 and 1997/99. Finally, Table 6 presents the direct, indirect and total effects of those variables on adult health.

[9] Note the absence in Panel B of race, as this variable was only estimated as a between-family effect, since clusters where siblings did not have the same race were excluded from the sibling sample.

Table 4 indicates that being white (relative to non-white) has significant, positive effects on both the average number of hours worked and average household income.
More specifically, compared to non-whites, whites' work hours are 15% greater, and their household income is 25% higher. In both cases, race affects financial resources through the common family effects, as both siblings are of the same race. But the similarities end there, as the effects of education are constrained to 0 on the average number of hours worked, and in turn, the effects of early health are constrained to 0 on the average household income.

Work limitations, or the number of years that health prevented work in early adulthood, negatively and significantly affects the number of hours worked, through both the common family effect and the sibling specific effects. This indicate that, while family background factors did not appear to bias the relationship between work limitations and adult health, they do affect the relationship of this early health factor with the number of hours worked in early and mid-adulthood. In addition, the effect of common family factors is actually stronger than the sibling-specific effect, as each additional year spent unable to work because of health reasons in young adulthood is related to a decrease in hours worked of about 77% at the family level ($1-(e^{-1.460})=0.767$), and of 35% at the sibling level ($1-(e^{-0.426})=0.347$). The same pattern is apparent for the relationship between education and household income, but education in this case has positive, significant effects. Thus, each additional year of education increases household income by 14% at the family level ($1-(e^{0.127})=0.135$), and by 5.7 % at the sibling level ($1-(e^{0.055})=0.057$).

Table 5 presents the results for the structural equations with health insurance measures as the endogenous latent variables. Here, the total effect of race is once again significant, but it does not impact health insurance directly. In addition, this effect is negative, and indicates that whites were uninsured or publicly insured for about a third of a year less than non-whites. Apart from race, there were no other significant family background total effects on the number of years uninsured.

In contrast, education and household income also have significant family effects on public insurance, which points to the origin of the family bias that this variable exhibits with adult health. More specifically, each doubling (100% increase) of household income is associated with a decrease of one year in the number of years publicly insured ((ln(100+100)/100)*-1.482 = -1.027), and each increase of one year of education is associated with a decrease of about two months in the number of years publicly insured (12*-0.188=2.256).

The number of years with work limitations has no family effect on either measures of health insurance. In addition, this measure does not exhibit a significant total effect on the number of years uninsured nor does it have a direct effect on the number of years publicly insured. However, the number of years health prevented work does have significant positive indirect and total effects on the number of years publicly insured. The total effect is relatively strong, as one additional year of work limitations is associated at the sibling level with a 4-month increase in the number of years publicly insured (12*0.361=4.332).

Education has positive effects at the sibling level on both measures of health insurance, but these effects are only marginally significant for public insurance. In both cases these effects are relatively small substantively, as one additional year of education is associated with a decrease in the number of year publicly insured of a little more than a week (52*-0.025=-1.300), and a decrease in the number of years uninsured of about 7 weeks (52*-0.043=-6.536).

Similarly, household income has negative effects at the sibling level for both measures of health insurance. These effects are significant in both cases, but while it is stronger for the number of years publicly insured, the magnitude of these effects is once again moderate

substantively. For instance, each doubling of income (100% increase) is associated with a 7-month decrease in the number of years uninsured ((ln(100+100)/100)* - 0.782 = - 0.542), and a 4-month decrease in the number of years publicly insured ((ln(100+100)/100)* - 0.459 = - 0.318). Finally, the average number of hours worked over this period has a direct significant impact at the sibling level on the number of years publicly insured, but not on those spent uninsured. This relationship indicates that for each doubling (100% increase) of the numbers of hours worked, the number of years publicly insured decreases by about 7 months ((ln(100+100)/100)*-0.847 = -0.587).

Table 6 presents the direct, indirect and total effects of the exogenous variables on adult health. As noted above, the only between-family effect that is even marginally significant is that of race, which indicates that whites report better self-rated health in adulthood than non-whites. The totality of this effect is indirect, working notably through positive effects on hours worked and household income, and negative effects on health insurance. In turn, only two within-family effects are significant, namely household income and years uninsured.

In fact, household income has in this instance a significant positive indirect effect on adult health, which necessarily works through health insurance by decreasing the likelihood of being either publicly insured or uninsured. In addition, the indirect effect going through the number of years uninsured ($-0.782 * -0.116 = 0.091$) is 33% stronger than that going through public health insurance ($-0.459 * -0.135 = 0.062$), suggesting that the lack of private insurance is an important mechanism through which household income impacts adult health. As discussed in the previous section, controlling for family bias, the number of years uninsured has a significant negative effect on health when contrasted with private insurance, while the number of years publicly insured does not differ significantly from private insurance.

In sum, Model 18 with private insurance as the reference does point to the presence of bias from the family of origin in the effects of many of these variables. More specifically, as hypothesized, public health insurance was significantly affected by factors from the family of origin working through household income, and by extension, race and education. In turn, family background also had an impact on the lack of insurance through race, and the number of hours worked. However, it does not appear that these family inequalities result in between-family adult health inequalities, since neither health insurance measure had a significant direct effect on health at that level.

In contrast, at the sibling, within-family level, similar pathways were found, which resulted in a significant direct negative effect of the number of years uninsured. More specifically, controlling for family bias, work limitations, obesity in early adulthood, the number of hours worked and household income all had a significant impact on the number of year publicly insured, which in turn did not significantly differ from private insurance in its impact on health. The lack of health insurance was affected by household income and education, and had itself a significant negative impact on adult health. This indicates that one of pathways from inequalities in status attainment to health inequalities goes through the contrast between the lack of insurance and private insurance.

Years uninsured as the reference group

The findings presented above, while telling, do not yield a complete picture of the extent to which health insurance contributes to socioeconomic inequalities in health, since they compare the lack of insurance and being publicly insured to being privately insured. Another comparison of interest contrasts the number of years with either private or public insurance to the number of years uninsured. That model was estimated, and the results are presented in Figure 5. The

model is not strictly comparable to Model 18, since the number of hours worked had to be omitted from the model because of multicollinearity with private insurance. In addition, the disturbance variance of the between-family factor for both measures of health insurance had to be set to very small positive values, because LISREL yielded statistically insignificant negative values for these inherently positive values (also see Warren et al. 2002). This situation has been noted to occur as a consequence of sampling fluctuation when sample size is small, and this correction has been found to have no adverse effects on the estimates (Anderson and Gerbing 1984; Gerbing and Anderson 1987). Still, while the model in Figure 5 appears to fit the covariance data well, ($\chi^2_{df=73} = 75.90$; $p = 0.385$; RMSEA=0.013) it should be considered more suggestive than definitive.

The effects in Figure 5 are relatively similar to those of Figure 4, so I will not discuss them in much detail, and will rather focus on the conclusions that we can draw from this alternative parameterization for the contribution of health insurance per se to socioeconomic inequalities in health.

As in Figure 4, the model in Figure 5, Panel A shows that both race and education have significant and positive between-family effects on household income, which itself also has significant effects on the health insurance variables. Race and education thus exhibit respectively positive and negative total effects on private and public health insurance that are totally mediated by household income. The early health variables have no significant between-family effects in this model. In turn, while the health insurance variables do not have significant between-family effects on adult health either, the positive direction of the effect for public insurance is noteworthy.

This positive effect indicates that, when contrasted with being uninsured, public health insurance could actually *reduce* the family background total effects of socioeconomic status on adult health. In fact, as shown in Table 7, the indirect effects of race, education, and income that go through private insurance are reduced by 40% when the indirect effects of these variables going through public health insurance are taken into account.

In turn, the same relationships are evident in Panel B: education has a direct positive effect on income, and indirect positive and negative effects on private and public health insurance, respectively. Household income also has a direct effect on health insurance in the same direction as that of education. In addition, contrary to Panel A, early health variables now have a significant direct effect on public insurance, though still not on private insurance.

The within-family effect of years with public insurance on health is negative, but negligible in magnitude such that it is in fact not significantly different from that of years uninsured. However, compared with being uninsured, the number of years with private insurance have a significant cumulative, positive effect on adult health. As such, we can formulate the results of the previous section alternatively by saying that, contrary to the between-family effect of public health insurance, the number of years privately insured is found to *contribute* to sibling-specific total effects of socioeconomic background on adult health.

Summary – Results

To the exception of the number of hours worked, status attainment variables and obesity in early adulthood exhibit the greatest between-family component of total variance. Health insurance variables and adult health have moderate between-family components of total variance, consistent with their measurement later in the life course. Finally, as hypothesized,

work limitations in early adulthood have substantial within-family components that suggest that this indicator truly measures a transitory and highly individual component of health.

Beyond these associations, the model fitted highlights a structural relationship where the impact of race, early health and education on adult health is not direct, but rather mediated by household income and the number of hours worked, as well as health insurance variables. In addition, while within-family paths were all found to be equal, this was not true of between- and within- family effects, suggesting that the pathways observed here are affected by family background biases.

However, the results concerning the mediating effects tested in Model 14 must still be interpreted with caution, as the direct effects of education and obesity that were estimated in Models 5 were only marginally significant. This means that setting those paths to 0 is not likely to affect the chi-square value to a great extent. In turn, the direct effects of work limitations were also fully mediated by later SES, which cannot evidently in this case be explained by a lack of significance. In fact, these results suggest that later socioeconomic achievement may reduce the negative effects of early shocks to health.

Moreover, while these were not formally constrained to 0, neither hours worked nor household income had significant direct effects on adult health, which is contrary to what was hypothesized earlier. On the other hand, Table 4 and Table 5 showed that significant paths were found between education and financial resources, from financial resources and hours worked to health insurance variables, and those were all as hypothesized, which would tend to validate the model.

According to the final structural model, which is schematically represented in Figure 4, social background appears to significantly affect the status attainment process, but these effects are not translated into health inequalities. Indeed, factors from the family of origin affect the number of hours worked primarily through race and work limitations in early adulthood, while they affect household income through race and education. These effects carry over to public health insurance and the number of years uninsured, but there are no significant between-family effects from these variables to adult health.

In contrast, there are significant within-family effects on adult health as well as on the status attainment process. These show that education has a positive effect on household income, while early work limitations have a negative effect on the number of hours worked. In turn, household income and hours worked decrease the number of years publicly insured. Yet, the number of years publicly insured does not have significant within-family effects on adult health. In contrast, household income also has a significant negative impact on the number of years uninsured, which itself has a significant negative effect on adult health. In addition to this direct effect of years uninsured, household income also had a significant, indirect effect on adult health, which necessarily works through the direct effect of health insurance on health.

Yet, when lack of insurance is the reference group, as presented in Figure 5, I find that, in contrast with being uninsured, public health insurance actually may *reduce* the family background total effects of socioeconomic status on adult health. In turn, compared with being uninsured, the number of years with private insurance have a marginally significant cumulative, positive within-family effect on adult health. As such, contrary to the between-family effect of public health insurance, the number of years privately insured is found to *contribute* to sibling-specific total effects of socioeconomic background on adult health.

In sum, these analyses indicate that the number of years uninsured, and privately or publicly insured are associated with adult health through very different pathways. While the two

former have an impact on health that is mainly due to sibling-specific processes of status attainment and achieved status in adulthood, the latter appears to not differ from private insurance in its impact on health. Thus, the results presented provide added confidence that public health insurance does not have negative effects on health relative to private insurance within families when the between-family bias is controlled for, while the number of years uninsured does, but they also suggest that health insurance is a mediator of the effects of household income on adult health. Moreover, while private insurance appears to contribute to social inequalities in health net of family background, public insurance has the potential to reduce inequalities from the family of origin when lack of insurance is the alternative.

DISCUSSION

Overall, the results highlighted two structural relationships. First, that the impact of race, early health and education on adult health was not direct, but rather mediated by household income and the number of hours worked. In turn, the effects of household income and the number of hours worked appeared themselves to be primarily mediated by the effects of health insurance on adult health.

In sum, these analyses indicate that the number of years uninsured, privately and publicly insured are associated with adult health through very different pathways. While the two former have an impact on health that is mainly due to sibling-specific processes of status attainment and achieved status in adulthood, the latter appears to not differ from private insurance in its impact on health. Thus, the results presented provide added confidence that public health insurance does not have negative effects on health relative to private insurance when the between-family bias is controlled for, while the number of years uninsured does, but they also suggest that health insurance is a mediator of the effects of household income on adult health. Moreover, while private insurance appears to contribute to social inequalities in health net of family background, public insurance has the potential to reduce inequalities from the family of origin when lack of insurance is the alternative.

In this section, I will first discuss the results regarding the between- and within-family components of total variance of the latent variables modeled and interpret these in light of previous sibling studies on status attainment, and of twin studies on the variance decomposition of self-rated health. In turn, I will expose the main findings regarding the pathways between the family of origin, status attainment and health, follow with a discussion of the contribution of health insurance to these relationships, and finally conclude by outlining certain limitations of these analyses.

Family effects on health, schooling, financial resources and health insurance

In these analyses, to the exception of the number of hours worked, status attainment variables and obesity in early adulthood exhibited the greatest between-family component of total variance. Health insurance variables and adult health had moderate between-family components of total variance, consistent with their measurement later in the life course. Finally, as expected, work limitations in early adulthood had substantial within-family components suggesting that this indicator truly measures a transitory and highly individual component of health.

Whereas this strong between-family component was as hypothesized for education, especially since it is measured early on in the life course, it was somewhat more surprising that household income should be so strongly related to the family of origin, as previous studies have

shown that within-family variance in attained SES increases across the life course (Hauser et al. 1999; Warren et al. 2002). A first explanation for this finding is that this notable between-family component of household income is related to the substantial effect of income in the late twenties substantiated in previous studies of the same data. In addition, a number of differences between samples of the Wisconsin Longitudinal Study (WLS) and the NLSY79 could account for these discrepancies. First, the sampling frame of the WLS may have increased heterogeneity between siblings by selecting the main respondent by educational status but imposing no such requirement for siblings that were later incorporated into the survey (Hauser 1988). In contrast, NLSY79 siblings were all part of the same household in 1978 by sampling design, and were in the current analyses no more than four years apart in age. Therefore, in the absence of these restrictions, siblings in the WLS may not have shared as much family environment as the NLSY79 siblings did. In addition, in the NLSY79, respondents were only 40 years old at the last wave (and thus last period of income measurement), while respondents from the WLS were 50 years and over, which adds ten more years of diverging life course occupational experiences. Finally, income may not exhibit the same patterns as the occupational measures that Hauser and his colleagues considered.

In contrast, the relatively moderate between-family component of self-rated health also merits consideration, as it is in fact congruent with recent findings from twin studies investigating differences in genetic and environmental sources of variation for self-rated health. Quesnel-Vallée (2004) found with the same data that the intra-family correlation in self-rated health was about 20%, with a 95% confidence interval of (0.071; 0.320). Thus, the correlation of 0.199 between siblings' health estimated here corresponds to that reported by Quesnel-Vallée (2004).

In the study with the population most comparable to the NLSY79, Romeis et al. (2000) used the Vietnam Era Twin Registry, and found that among male-male twin pairs who were on average 38 years old, the best fitting model suggested that 40% of the variance in self-rated health was due to additive genetic effects, while the remaining 60% were attributed to non-shared environmental factors. In turn, using the Swedish Twin Registry, Harris et al. (1992) and Svedberg, Lichtenstein and Pedersen (2001) found that individual differences in self-rated health were primarily due to individual-specific environmental influences at all ages. Unfortunately, the age groupings that these authors used were quite broad, due to smaller sample sizes at younger ages, but their findings are still indicative. More specifically, Harris et al. (1992) also found that among respondents under 50 years of age, non-shared environmental factors accounted for 77% of the total variation in self-rated health, while shared environmental influences constituted the remaining 23%. In contrast, Svedberg et al. (2001) found in their best fitting model that among 17 to 44 year olds, 92% of the total variance in self-rated health between twins came from non-shared environments, and 8% from shared environments, while among 45 to 64 year olds, 44% of the total variance was accounted for by genetic factors, and 56% by non-shared environmental influences.

Therefore, these findings suggest that we should expect around 40% of the variation in self-rated health among twins to be due to family background[10]. However, these studies did not agree unequivocally on the genetic or environmental attribution of the shared variance. Thus,

[10] Since I cannot distinguish between genetic and shared environmental influences in these data, the intra-class correlation I report should be understood as amalgamating both of these dimensions.

while two of them suggest that this variation can be wholly attributed to genetic factors among 40-year old American veterans (Romeis et al. 2000) and 45 to 64 year-old Swedes (Svedberg et al. 2001), Harris et al.'s (1992) and Svedberg et al.'s (2001) findings for Swedes respectively under 50 years of age and 14 to 44 year old suggest that the shared family environment may also contribute to family background variation. Still, these studies do agree on one point, namely that the greater part of variation in self-rated health is due to non-shared environments. In sum, these estimates are consistent with those reported here, especially considering that they bore on twin pairs, who bear much greater genetic resemblance than sibling pairs.

Pathways between status attainment and adult health

The final model fitted here highlights a structural relationship where the impact of race, early health and education on adult health is not direct, but rather mediated by household income, as well as health insurance variables. In addition, household income did not have significant direct effects on adult health either, but it did exhibit significant indirect effects through health insurance.

Moreover, while the within-family effects were all found to be equal, further constraints of equality of the between- and within-family paths resulted in poorer model fit, which suggests that factors from the family of origin do bias the within-family relationships observed here. Yet, in spite of a number of significant between-family effects on other endogenous variables, race was the only variable to have a marginally significant between-family total effect on adult health. This suggests that the family bias in the status attainment process carried over only marginally to adult health.

These findings contrast with those of Hauser and his colleagues (Hauser et al. 1999; Warren et al. 2002), who did not find significant differences in the between and within slopes and thus concluded that there was no family bias in the relationship between education and occupation. It is plausible that these distinct findings are again due to sample differences between the NLSY79 and the WLS. In addition to those mentioned earlier, it is important to note that the primary WLS respondents were all high school graduates in Wisconsin and mostly white, while the NLSY79 is representative of the U.S. population[11], with an oversample for disadvantaged minority groups. Thus, while the NLSY79 is likely to count siblings with more shared family experiences, the WLS may include more homogenous family units, which should depress the amount of between-family variation or even of bias. As such, the contingency of status attainment on the family of origin, and particularly on race, may be more evident in the NLSY79.

In addition, another difference between these analyses and those of Hauser and his colleagues is that the impact of early health on status attainment was estimated here, while they focused more on the effects of cognitive ability. It is possible that these factors have a different impact on status attainment where early health (and particularly work limitations) is more heterogeneous between families, while cognitive ability accounts for much of the bias associated with the family of origin. Finally, another possibility is that the latent between-family factor for early

[11] More specifically, regarding siblings, the NLSY79 is representative of siblings living in the same household and satisfying the age restriction of having been 18 to 21 years old as of December 31, 1978. Thus, the NLSY79 does not constitute a nationally representative sample of siblings of all ages and living arrangements (Zagorsky and White 1999).

health does not measure the same variable as the siblings' latent early health, in which case we could never expect these paths to be equal even with complete exogeneity (i.e. a complete lack of bias from family effects).

Concerning the pathways between the family of origin and health, the lack of direct effects of early health, education and household income all contradict the formal hypotheses earlier, and therefore deserve further consideration. In fact, this finding of a full mediation by household income and hours worked of early health and education effects on adult health must be interpreted with caution, as the direct effects of education and obesity on health were not significant. This signifies that setting those paths to 0 was not likely to affect the chi-square value to a great extent, and thus made it more likely to accept this modification. It is possible that greater statistical power, notably from an increased sample size, may not allow the replication of those results. On the other hand, highly significant paths were found notably between education and financial resources, and from financial resources and hours worked to health insurance variables, and those were all as hypothesized, which would tend to suggest that statistical power was not a substantial issue in this case.

Therefore, we can also surmise that these findings are highlighting true relationships between family background and adult health in these data. The twin studies reviewed in the previous section suggest that much of the variation in self-rated health is due to non-shared environments. Yet, as shown above, both education and the number of years obese exhibited strong between-family components of variation. Thus, while many previous studies have found evidence of significant direct effects of education, in an analysis like this one, which decomposes between- and within-family effects on adult health, there may not be enough within-family variation in early educational attainment to have a direct effect on adult health. Similarly, early obesity was hypothesized to have a direct effect on adult health as a reflection of a more permanent, and partly genetic component of health. However, obesity may in fact reflect in this sample a greater component of the shared environment, which played a more trivial role in explaining the variation in self-rated health between twins in the aforementioned studies.

In contrast, the number of years during which respondents were unable to work because of health problems in early adulthood exhibited, as expected, very little between-family variation, and in turn, this variable had strong direct significant within-family effects on adult health. This pattern of effects is congruent with findings from the twin studies, as it underscores the impact of non-shared environmental factors on adult self-rated health. Yet, these direct effects were also fully mediated by later SES, which cannot evidently in this case be explained by a lack of significance. In fact, the total effects of work limitations on adult health declined in magnitude and became insignificant when mediated by later SES, which suggests that later socioeconomic achievement may reduce the negative effects of early shocks to health.

Finally, household income had no significant direct effects on health, which may, as in the case of education and obesity, be related to the large between-family component of variance of this variable. However, household income did have indirect effects on health through health insurance, which will be discussed in the following section.

The contribution of health insurance to socioeconomic inequalities in health and their intergenerational reproduction.

While processes of status attainment did not appear to have much significant direct effects on adult health, they did exhibit significant indirect effects through health insurance variables.

More specifically, private insurance – or the lack thereof – was found to contribute to inequalities in health, net of family influences.

As mentioned above, the final structural model indicated that social background appears to significantly affect the status attainment process, but these effects were not translated into health inequalities. Thus, factors from the family of origin affected the number of hours worked primarily through race and work limitations in early adulthood, while they affected household income through race and education. These effects carried over to public health insurance and the number of years uninsured, but there were no significant between-family direct effects from these variables to adult health.

In contrast, there were significant within-family effects on adult health as well as on the status attainment process. These showed that education had a positive effect on household income, while early work limitations had a negative effect on the number of hours worked. In turn, household income and hours worked decreased the number of years publicly insured. Yet, the number of years publicly insured did not have significant within-family effects on adult health. In contrast, household income also had a significant negative impact on the number of years uninsured, which itself had a significant negative effect on adult health.

Similarly, when lack of insurance was the reference group, I found that compared with being uninsured, the number of years with private insurance had a marginally significant cumulative, positive within-family effect on adult health. As such, the number of years privately insured was found to *contribute* to sibling-specific total effects of socioeconomic background on adult health. These findings are at odds with those of Ross and Mirowsky (2000), which suggests that, as asserted earlier, their estimates may have been affected by selection biases.

In turn, still with lack of insurance as the alternative, public health insurance had the potential to *reduce* the family background total effects of socioeconomic status on adult health. The lack of significance of these effects could be due to the fact that public health insurance coverage is highly unstable over time and that publicly insured individuals often face substantial barriers to care (The Medicaid Access Study Group 1994). If the Medicaid and Medicare programs for nonelderly adults were extensive enough in terms of the population covered, length of time with coverage and services reimbursed, we may find that these services in fact eliminate the role of private insurance as a pathway.

In sum, these analyses indicate that health insurance and the source of coverage contribute to social inequalities in health through very different pathways. Three contrasts are of interest here: first, the number of years privately insured was found to compound the positive sibling-specific processes of status attainment and achieved status in adulthood on health when contrasted with the lack of insurance; second, public insurance was not found to differ in its effects on health from those of private insurance; and third, public insurance may have the potential to reduce socioeconomic inequalities from the family of origin when lack of insurance is the alternative.

Limitations

While this study filled a gap in the literature by looking at early and mid-adulthood in the United States, a longer period of observation of financial resources in adulthood may help model these processes better, as it would allow for more periods of observation and more detailed trajectories. Moreover, the fact that there are already substantial differences in the social gradient in health among these young respondents is of course telling, but the analyses should be replicated with older respondents, as many life course studies indicate that social inequalities in

health are greatest between the ages of 55 and 64 (Frytak, Harley and Finch 2003). Finally, with older respondents, the contribution of wealth to these processes could be assessed, which would certainly add to the evidence regarding the impact of cumulative processes on health.

Another limitation of this study is that it did not consider the contribution of psychosocial resources to the relationship between SES over the life course and self-rated health in adulthood. For instance, self-efficacy or mastery may impact the performance of individuals on the job market (Mirowsky and Ross 2003), which is strongly related to the type of health insurance coverage one can secure. Moreover, it is not clear that the effects highlighted here apply to mental health, given that the coverage of mental health services is highly inconsistent, both between and within insurance sources. Future studies should therefore examine the significance of these mechanisms with psychosocial predictors and outcomes.

Moreover, the current analyses did not consider the impact of cognitive ability on the status attainment process, which has been shown by many to be substantial (see Warren et al. 2002), and of particular interest in the study of between- and within-family effects because highly heritable (Nielsen 2004). In addition, recent evidence indicates that the association between cognitive ability and mortality is mediated by a greater cumulative exposure to lifetime low socioeconomic conditions (Kuh et al. 2004). Cognitive ability may thus constitute an important pathway through which the family of origin impacts status attainment and adult health that merits further attention.

Similarly, these analyses did not control for the marital status of the respondents. One of the reasons for this omission was that it was unclear when marital status should be measured, given the length of observation of this study. Moreover, the potential impact of marital status on income or health insurance coverage was incorporated in the measurements to the extent that they consisted of household measures. Beyond marital status, other dimensions of social support and social networks have been shown to affect both status attainment process (Lin 1999) and health and mortality (Berkman and Syme 1979; Cohen and Syme 1985; Lin 1986a, 1986b). While these levels of the social structure that are more proximate to the individual were not under study here, their contribution to the interplay between status attainment and health should be explored in future studies. A last class of factors that was not controlled for in these analyses are health-related behaviors in mid-adulthood. Whereas Hayward and Gorman (2004) show that controlling for these factors does not substantially affect the impact of income on mortality, thus confirming previous results on this matter (Lantz et al. 2001), they could nevertheless constitute a pathway from the family of origin to health that would compete with that of health insurance.

Finally, sibling models also have their limitations, the first and most obvious of which is the exclusion of half-siblings and only children from the analyses. This exclusion necessarily results in a substantial drop in sample size, and, whereas this was not the case here, may also yield estimates that are not replicated in the sample including the full range of kinship links. Second, as was evident in Quesnel-Vallée (2004), fixed effects models are less efficient than other designs in the absence of significant unobserved heterogeneity at the family level, and thus should not be used as a panacea in the absence of such heterogeneity. Third, an important conceptual limitation is that these models also assume that siblings share the same home environment. While the narrow age range of the sample respondents limits the extent to which they might have experienced very different family environments, it remains impossible to assess whether families may have voluntarily shifted their resources to favor one child over the other (Conley 2004). Yet, despite the fact that a number of childhood and family factors could not be captured because they were not shared between siblings (Solon 1999), this strategy is still to a

certain extent validated by the fact that it does account for 20% of the variance in adult health. Fourth, whereas this was avoided here notably by including measures of early health that vary across siblings and affect both health insurance (X) and adult health (Y), Hauser (1988) notes that siblings resemblance models may not provide unbiased estimates of the relationship between X and Y unless factors that are sibling-specific and jointly determine X and Y are included in the analyses.

Further limitations of these analyses pertain to choices made in model estimation. A first limitation is that the current models did not estimate a measurement model for the latent variables. This significant advantage of SEM has been used early on in models of sibling resemblance (see for instance Hauser and Mossel 1985). Thus, this entails notably that the analyses did not rely on observed indicators of the family of origin for the estimation of the between-family effects, while a number of previous studies used such measures (See for instance Hauser et al. 1999 and Warren et al. 2002). However, given the small sample size, adding any additional variables to the model would have negatively affected both the stability of the estimates and statistical power. A promising alternative to consider in future studies would be to follow Warren et al. (2002), who fixed the latent variables' error variances in their sibling model to values that were estimated in separate measurement models.

Gender was also absent from the model in these analyses, as it had null covariance with too many factors and consequently caused the model to become unstable. Another strategy used by Hauser and his colleagues (Hauser et al. 1999; Warren et al. 2002) would have been to split the sample into same gender sibling pairs, but it was impossible to do so in these analyses, due to the small sample size and large number of factors. Indeed, sample size should be at least 50 cases more than 8 times the number of variables (Garson 2004), and analyses meet this requirement, but only with 99 additional cases. Moreover, $k(k+1)/2$ observations (k being the number of observed variables) are needed to estimate the asymptotic covariance matrix (Garson 2004), which in the present case means that the analyses required 153 observations (sibling pairs) or much more than would be left after splitting the sample in less than half (because a number of pairs of siblings are not of the same gender). Still, sensitivity analyses (not shown) indicate that the only gender difference in the effects of the variables on health was for the number of hours worked. Thus, this suggests that the number of hours worked may be the only variable affected by the omission of gender in these analyses.

In conclusion, sample size may have been an issue in these analyses. While this is usually an insurmountable obstacle, in the case of the NLSY79 it is not, as this is an ongoing survey. More specifically, the 2002 wave of data should be available shortly, which will increase the total sample size by about 50%. However, this addition may result in more than a 50% increase in the sibling sample size, as it will not only add clusters of siblings who all turned 40 between 2000 and 2002, but also will "reunite" siblings who had turned 40 in previous waves with the 2002 wave of 40 year olds. In addition, this new wave of data will also lengthen the period of observation and therefore the variation in number of years insured. These increases in sample size and variation should add to the statistical power of the analyses presented here, and may allow for different model specifications.

CONCLUSION

This research made several contributions to the study of socioeconomic inequalities in health, which were all noted successively in the previous discussion. However, one finding stands out from the others for its provocative revision of widely held – though rarely tested –

assumptions about the contribution of health insurance to socioeconomic inequalities in health. More specifically, contrary to what had been found before by Ross and Mirowsky (2000), I have shown that net of family influences, education, and cumulative financial resources over a 15-year period in early and mid-adulthood, the lack of private health insurance has a cumulative negative effect that contributes to socioeconomic inequalities in adult health.

Now, I was not attempting in this research to set us back fifty years and argue that access to the medical institution is the only – or even the most important – determinant of health in modern societies. However, I found evidence here that health insurance has the potential to play a non-trivial role in the perpetuation of socioeconomic inequalities in health in the United States that may have been overlooked or downplayed in the past because of methodological limitations. Thus, I would argue that the United States' organization of health care services is unique among G8 countries in not ensuring universal health insurance coverage for all its citizens (Veney 2002), and that this American exceptionalism is threatening the health of its population.

The United States already spends more than any other country in the world on health care, both per capita (US$4,887 in 2001) and as a share of total economic activity (13.9% of the GDP in 2001), and its comparatively higher growth rate of spending (4.4% from 1999 to 2001) suggests that this trend is unlikely to abate in the near future. Yet, in spite of these substantial expenditures, the United States lags behind other OECD countries in terms of many population health indicators, such as number of physicians per capita, increases in life expectancy, and decreases in infant mortality (OECD 2003). Of course, there is no reason to expect a strict dose-response relationship between more expenditures and better population health, as many other institutional factors come into play to affect the latter, such as diet, physical activity levels, births to teenager mothers and deaths from violence (Alliance for Health Reform 2002). Nevertheless, the extent of the discrepancy between the resources expanded and population health demands that we examine whether the organization of care in the United States may also play a role.

In fact, whatever their cause, rising costs have had at least one consistent consequence over the past few years, namely that employers have increasingly limited the availability and scope of health insurance coverage for employees and their dependents (Holahan and Wang 2004; Mills 2002; Mills and Bhandari 2003). While this affects most strongly employees in small firms (3-199 workers), which have seen the greatest increases in premiums (Gabel and Pickreign 2004), it is also increasingly true of employees in large firms (500 or more employees; Glied, Lambrew and Little 2003). In addition, the economic downturn has compounded these effects by reducing profit margins for employers who are then unable to bear the full burden of coverage for their employees, and by increasing the number of unemployed individuals who may not have the option, or are able to afford continuation coverage (Miller 2001). Given that employer-sponsored insurance is the single most common source of coverage among adult non-elderly individuals, it is not surprising given these patterns that the uninsured population has been growing steadily over the past few years[12].

Quesnel-Vallée (2004) showed that the lack of insurance is associated with poorer health outcomes, while public and private insurance have no distinct effects on health. However, while

[12] Another problem of note that I will not discuss here because it reaches beyond the scope of this argument is the increase in the underinsured population as well (Short and Banthin 1995).

the growth in the uninsured population is problematic in and of itself, the impact of this situation on the health of the American population is heightened by another structural feature of the U.S. labor market. Contrary to prevailing economic theories, the lack of employer-provided insurance is not compensated in the United States by a wage premium (O'Brien 2003). In fact, "good jobs" that offer health insurance also tend to offer higher salaries, other fringe benefits such as pensions, as well as opportunities for promotion (Farber and Levy 2000; Kalleberg et al. 2000; O'Brien 2003).

Accordingly, an even more consequential question is whether health insurance contributes to those inequalities and their impact on health. In these results, I have shown that health insurance, and particularly the lack of private coverage, does add to those inequalities in health. Moreover, results suggested that, when lack of insurance is the alternative, public health insurance may, if wide-ranging enough, actually reduce inequalities stemming from the family of origin.

Thus, the findings of this research add to a growing body of literature showing the deleterious effects on individual health of the lack of insurance (see IOM 2002a for the most thorough review to date) that culminated most recently with the recommendation by the Institute of Medicine that health care coverage should be universal in the United States (IOM 2004). Similarly, providing coverage for the uninsured is part of the strategies of the United States Department of Health and Human Services' *Healthy People 2010* (U.S.D.H.H.S. 2000), which states as one of its two overarching goals the elimination of health disparities.

Table 1 Between- and within-family variance components

	Between-Family	Within-Siblings 1	Within Siblings 2	Correlation
Adult health	0.232	0.805 (77.63)	1.089 (82.44)	0.198
Work limitations	0.027	0.689 (96.23)	0.387 (93.48)	0.050
Years obese	0.370	0.608 (62.17)	0.467 (55.80)	0.409
Education	3.054	3.012 (49.66)	2.126 (41.04)	0.545
Household income	0.157	0.154 (49.52)	0.135 (46.23)	0.521
Hours worked	0.134	0.596 (81.64)	0.449 (77.02)	0.205
Years uninsured	0.713	1.378 (65.90)	2.401 (77.10)	0.279
Years publicly insured	0.684	1.591 (69.93)	1.148 (62.66)	0.335

Note: Percentages in parentheses

Table 2 Fit indices used in model building

	χ^2	DF	RMSEA	AIC	CFI	SRMR
Early health and race						
1. Paths and covariances	472.11	118	0.113	542.11	0.55	0.20
2. Equality of paths	464.99	120	0.110	530.99	0.50	0.20
3. Set covariances of within effects to 0	466.01	122	0.110	528.01	0.51	0.20
Education in early adulthood						
4. Paths and covariances	402.46	112	0.105	484.46	0.57	0.19
5. Equality of paths	395.31	113	0.103	475.31	0.57	0.19
Financial resources in early and mid-adulthood						
6. Paths and covariances	183.79	84	0.071	321.79	0.77	0.15
7. Equality of paths	184.61	92	0.066	306.61	0.77	0.15
8. Race -> Family factor for adult health = 0	184.32	93	0.065	304.32	0.77	0.15
9. Education -> Adult health = 0	183.38	95	0.063	299.38	0.77	0.15
10. Work limitations -> Adult health = 0	176.44	97	0.059	288.44	0.77	0.15
11. Obesity -> Adult health = 0	177.02	99	0.058	285.02	0.77	0.15
12. Education -> Hours worked 1985-1997/99 = 0	180.51	101	0.058	284.51	0.77	0.15
13. Obesity -> Household income = 0	182.67	103	0.057	282.67	0.77	0.15
14. Work limitations -> Household income = 0	187.34	105	0.058	282.34	0.77	0.15
Health insurance in mid-adulthood						
15. Paths and covariances	41.32	71	0.000	205.32	0.98	0.05
16. Equality of paths	48.52	81	0.000	192.52	0.98	0.05
17. Work limitations -> Lack of insurance = 0	49.47	83	0.000	189.47	0.98	0.05
18. Obesity -> Lack of insurance = 0	50.64	85	0.000	186.64	0.98	0.05

Table 3 Total effects of nested models on adult health

	Model 3		Model 5		Model 14		Model 18	
	Between	**Within**	**Between**	**Within**	**Between**	**Within**	**Between**	**Within**
Adult health								
Race	0.218		0.145		0.165		0.173^{+}	
	(0.158)		(0.226)		(0.108)		(0.104)	
Work limitations	0.742	-0.206**	1.665	-0.198**	1.671	-0.064	1.506	-0.077
	(4.434)	(0.077)	(6.826)	(0.076)	(2.288)	(0.045)	(2.123)	(0.053)
Yrs obese	-0.347	-0.136	-0.346	-0.131	-0.254	0.001	-0.215	-0.017
	(0.702)	(0.097)	(0.719)	(0.097)	(0.338)	(0.008)	(0.321)	(0.018)
Education			0.154	0.064	0.180	0.020^{+}	0.178	0.018
			(0.350)	(0.047)	(0.133)	(0.011)	(0.133)	(0.012)
Hours worked					-1.106	0.148	-1.407	0.153
					(1.424)	(0.094)	(1.403)	(0.118)
Household income					1.425	0.351^{+}	1.398	0.330
					(1.049)	(0.186)	(1.040)	(0.203)
Yrs. pub. insured							-0.215	-0.135
							(0.433)	(0.100)
Years uninsured							0.549	-0.116*
							(0.955)	(0.059)

Note: Standard errors in parentheses
$^{+}$ $p < 0.10$ * $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$ (two-tailed tests)

Table 4 Direct effects for financial resources variables as endogenous, Model 18

	Between-Family	Within-Family
Hours worked		
Race	0.138**	--
	(0.045)	--
Yrs. health prevented work	-1.460*	-0.426***
	(0.627)	(0.070)
Yrs obese	0.218	0.005
	(0.179)	(0.051)
Household income		
Race	0.227***	--
	(0.026)	--
Education	0.127***	0.055***
	(0.017)	(0.016)

Note: Standard errors in parentheses. Direct effects are also total effects in this case.
$^{+}$ $p < 0.10$ * $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$ (two-tailed tests)

Table 5 Direct, indirect and total effects on health insurance, Model 18

	Direct effects		Indirect effects		Total Effects	
	Between	**Within**	**Between**	**Within**	**Between**	**Within**
Years publicly insured						
Race					-0.343***	
					(0.080)	
Yrs. w/ work limitations	0.104	0.083	0.079	0.361***	0.183	0.444*
	(1.006)	(0.164)	(1.245)	(0.085)	(1.105)	(0.186)
Yrs obese	-0.058	0.132*	-0.012	-0.005	0.070	0.127^{+}
	(0.283)	(0.054)	(0.183)	(0.043)	(0.200)	(0.100)
Education					-0.188*	-0.025^{+}
					(0.080)	(0.013)
Hours worked	-0.054	-0.847***				
	(0.855)	(0.144)				
Household income	-1.482**	-0.459*				
	(0.566)	(0.201)				
Years uninsured						
Race					-0.349***	
					(0.073)	
Yrs. w/ work limitations					1.757	-0.045
					(1.322)	(0.045)
Yrs obese					-0.262	0.001
					(0.272)	(0.005)
Education					-0.103	-0.043*
					(0.071)	(0.019)
Hours worked	-1.203^{+}	0.105				
	(0.691)	(0.110)				
Household income	-0.807	-0.782**				
	(0.557)	(0.267)				

Note: Standard errors in parentheses
$^{+}$ $p < 0.10$ * $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$ (two-tailed tests)

Table 6 Direct, indirect and total effects on adult health, Model 18

	Direct effects		Indirect effects		Total Effects	
	Between	**Within**	**Between**	**Within**	**Between**	**Within**
Race					0.173+	
					(0.104)	
Yrs. w/ work limitations					1.506	-0.077
					(2.123)	(0.053)
Yrs obese					-0.215	-0.017
					(0.321)	(0.018)
Education					0.178	0.018
					(0.133)	(0.012)
Hours worked	-0.397	0.052	-0.649	0.102	-1.407	0.153
	(2.089)	(0.143)	(1.270)	(0.083)	(1.403)	(0.118)
Household income	1.523	0.178	-0.125	0.152*	1.398	0.330
	(1.229)	(0.218)	(0.962)	(0.074)	(1.040)	(0.203)
Years publicly insured	-0.215	-0.135				
	(0.433)	(0.100)				
Years uninsured	-0.549	-0.116*				
	(0.955)	(0.059)				

Note: Standard errors in parentheses
$^{+}$ $p < 0.10$ * $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$ (two-tailed tests)

Table 7 Indirect between-family effects going through private and public insurance, from Figure 5, Model 18 with years uninsured as the reference group

Indirect effect of:	**Race**		**Education**		**Household income**	
Going through:	**Income and private insurance**	**Public insurance**	**Income and private insurance**	**Public insurance**	**Income and private insurance**	**Public insurance**
race -> income	0.237	0.237				
education -> income			0.114	0.114		
income -> private insurance	3.151		3.151		3.151	
income -> public insurance		-1.464		-1.464		-1.464
private insurance -> health	0.228		0.228		0.228	
public insurance -> health		0.333		0.333		0.333
income -> health	0.504		0.504		0.504	
Indirect effects	0.290	-0.116	0.139	-0.056	1.222	-0.488
Percentage of public to private indirect effect		(0.399)		(0.399)		(0.399)

Note: Percentages are in parentheses.

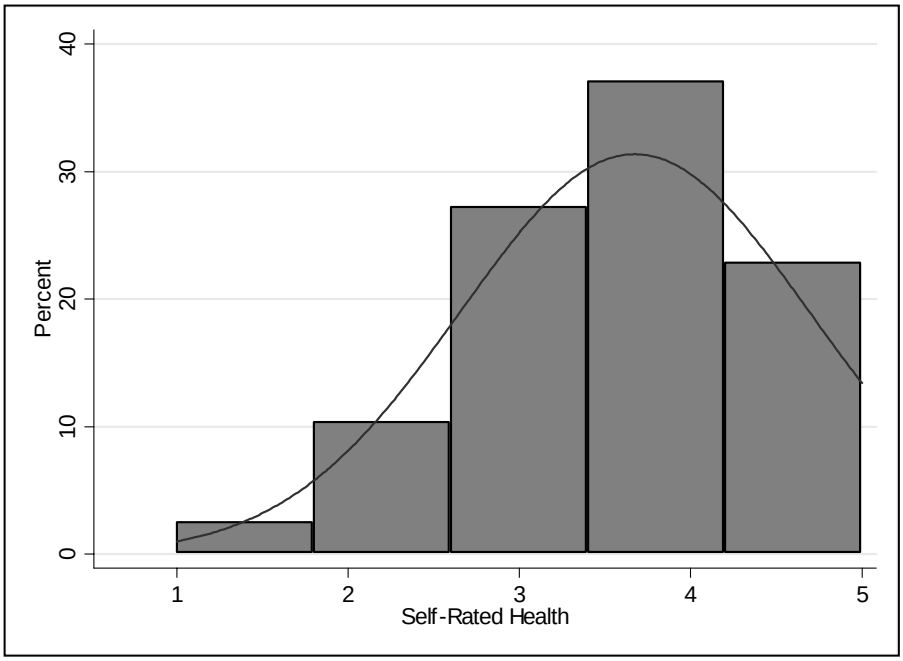


Figure 1 Distribution of self-rated health with normal curve overlaid, NLSY79

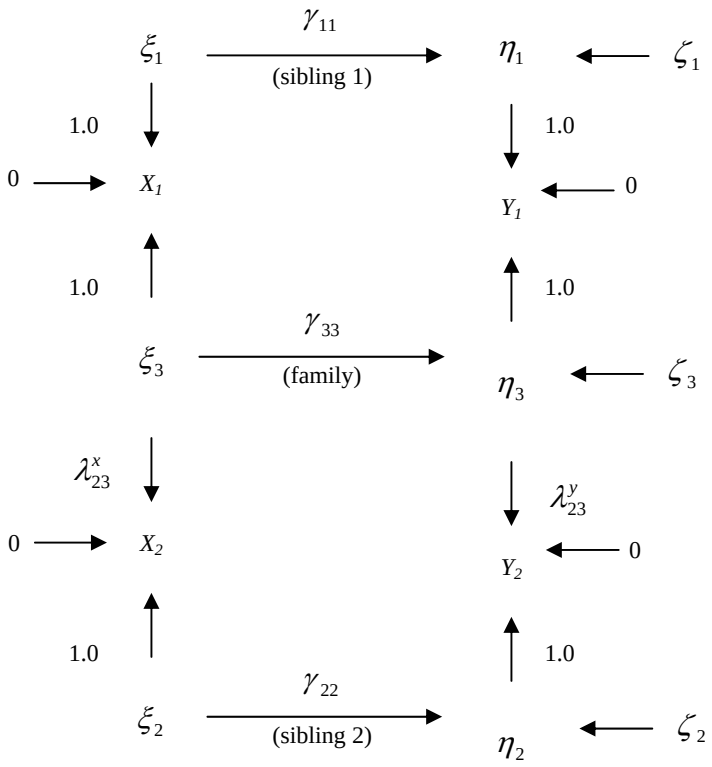


Figure 2 Simple Structural Model of Adult Health on Early Health

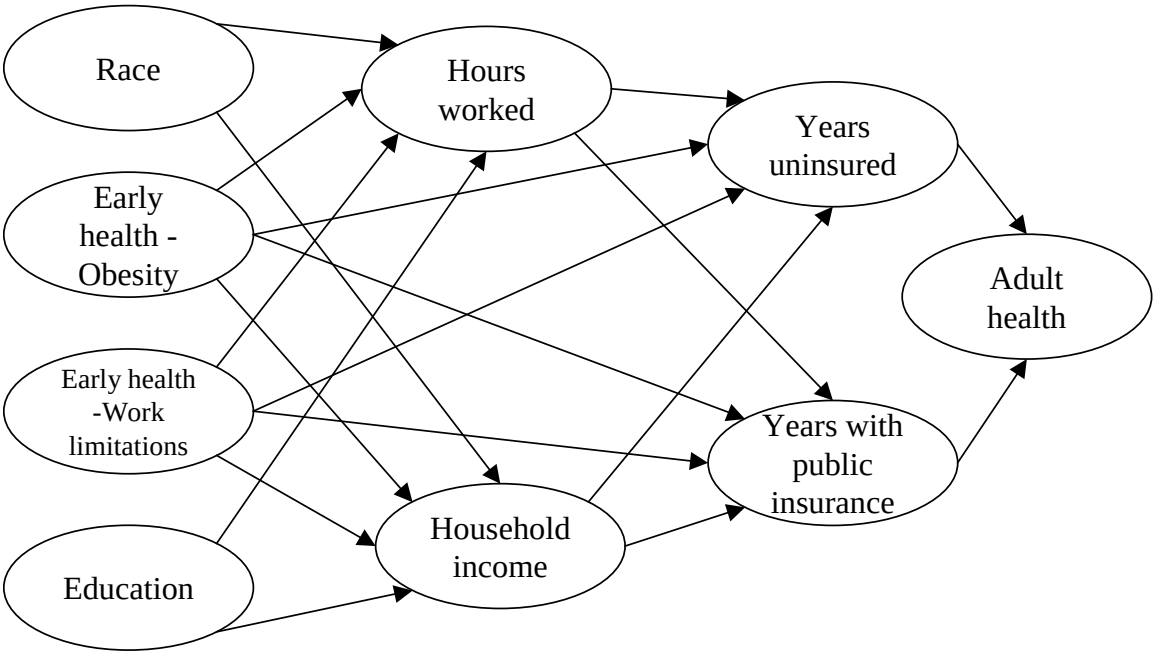
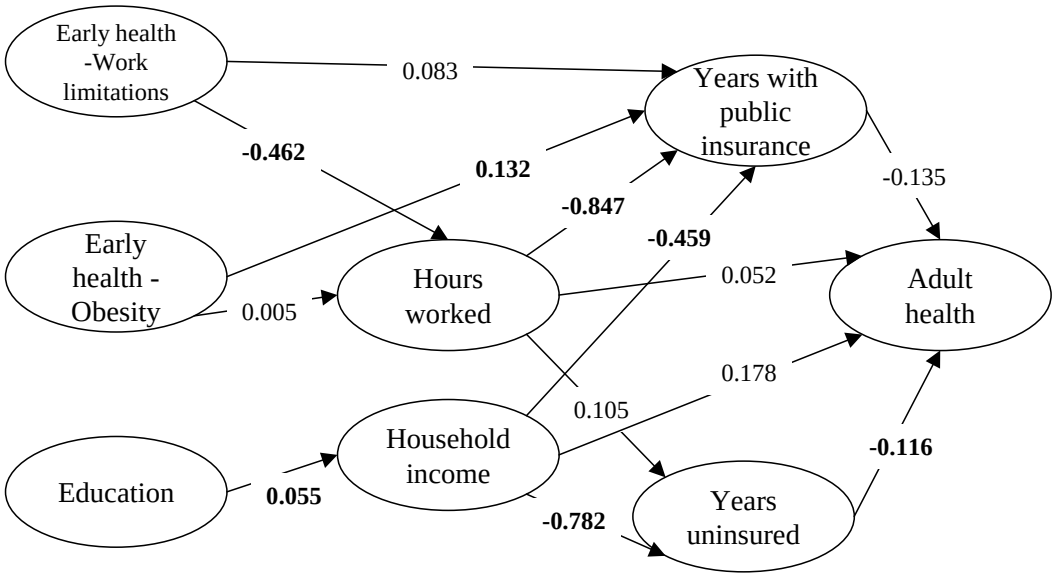


Figure 3 Full hypothesized sibling resemblance model

Note: With the exception of race, which is only estimated as a between-family factor, this structural model actually counts within-siblings 1, between-family and within-siblings 2 effects of each latent variable.



Panel A. Between-family effects

Panel B. Within-family effects

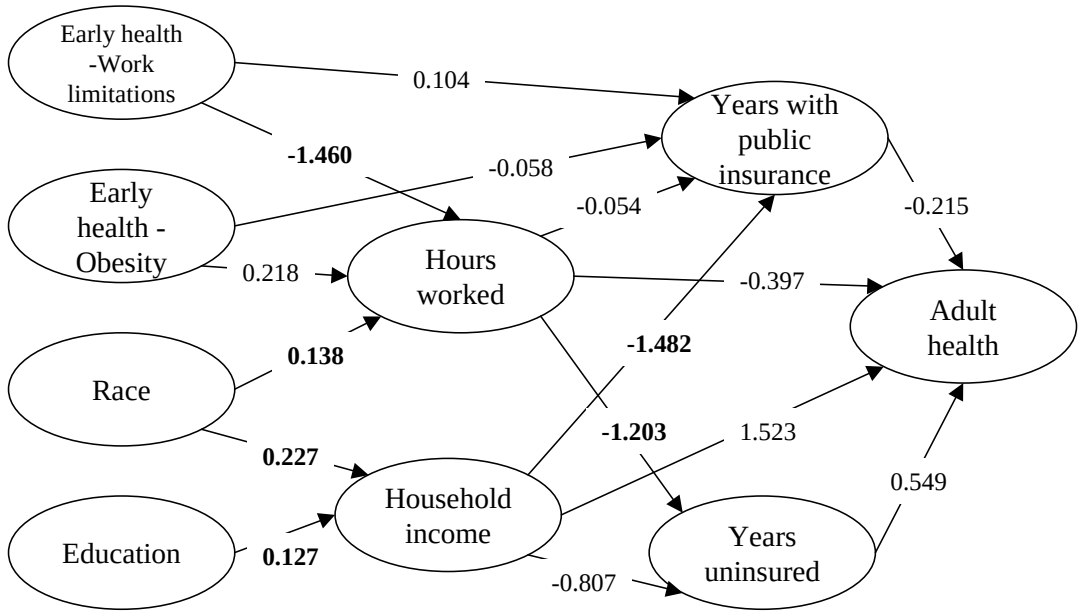
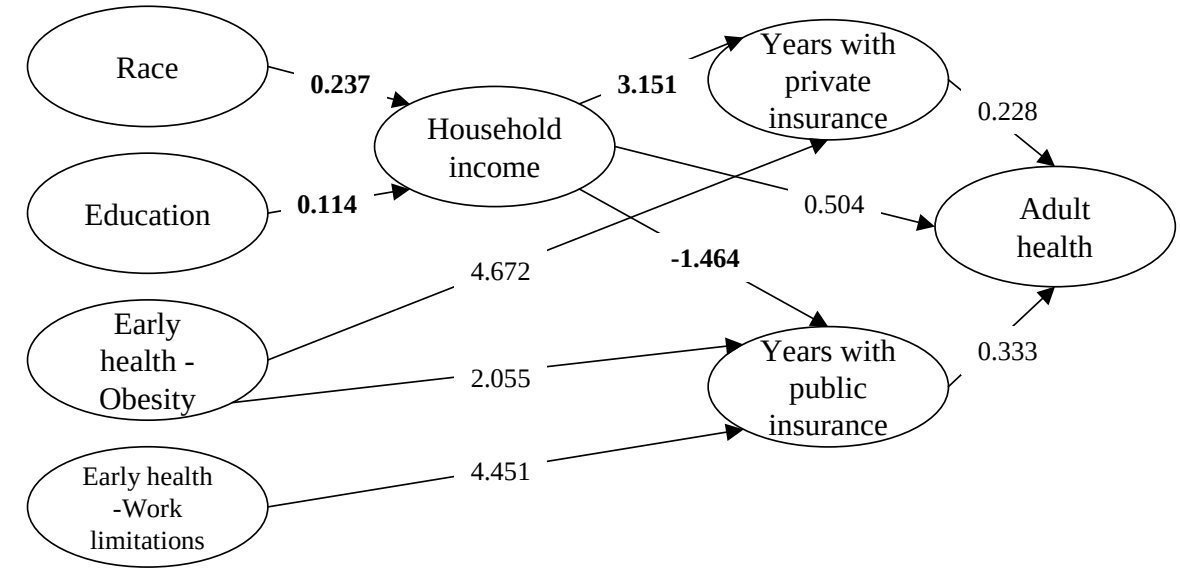
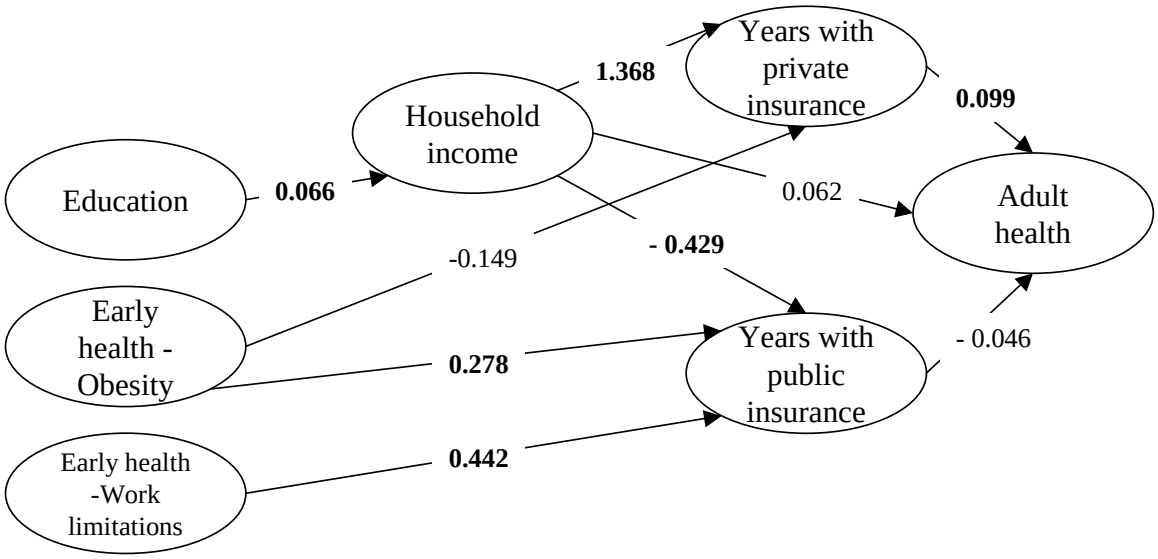


Figure 4 Estimated sibling resemblance model, with years privately insured as the reference

Note: Significant paths at the 0.05 level for one-tailed t-tests are in bold.



Panel A. Between-family effects



Panel B. Within-family effects

Figure 5 Estimated sibling resemblance model, with years uninsured as the reference

Note: Significant paths at the 0.05 level for one-tailed t-tests are in bold.