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## **“Inter-Cohort Trends in Age- Specific Health Inequalities: A Test of the Theory of “Fundamental Causes”\***

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**Inter-Cohort Trends in Age-Specific Health Inequalities:  
A Test of the Theory of “Fundamental Causes”**

**ABSTRACT**

Link and Phelan’s (1995; 1996) “Fundamental Causes” theory posits that social and economic inequalities in morbidity and mortality have persisted over historical time --- and will continue to persist --- because people with access to more social and economic resources are always better able to avoid health risks (even if the nature of those risks change over time). Despite the prominence of this theory and its inherent challenge to mainstream epidemiological, public health, and social science research, the core empirical fact that the theory is designed to explain has not been well established by Link and Phelan or others. Have socioeconomic inequalities in morbidity and mortality remained essentially constant over time? To investigate, we examine inter-cohort trends in age-specific health inequalities using data from the 1972 through 2002 General Social Surveys. Our results reproduce findings of inter-cohort improvements in health outcomes, but we find that socioeconomic differences in health have remained unchanged across the bulk of the 20<sup>th</sup> century. Although Americans’ health has improved over time, inequalities in health have persisted.

## **Inter-Cohort Trends in Age-Specific Health Inequalities: A Test of the Theory of “Fundamental Causes”**

### **INTRODUCTION**

Epidemiologists, public health researchers, social scientists, and others have spent a great deal of time investigating the behavioral and biological mechanisms that account for well-documented associations between socioeconomic status (SES) and morbidity and mortality (Stockwell 1961; Antonovsky 1967; Fingerhut 1980; Williams 1990; Williams and Collins 1995). Although this line of research has undoubtedly improved public health and improved morbidity and mortality rates, its central premise --- that SES rarely affects health directly, but usually does so indirectly via mechanisms or “risk factors” that need to be identified and studied --- has been questioned in recent years. First, even net of known risk factors there are still sizable SES-health associations --- and researchers seem to be running out of major new mechanisms to identify and study (Fox 2003). Second, there has been renewed interest in the “basic causes” (Lieberson 1985) of morbidity and mortality, including a focus on the role of broad social and economic circumstance in shaping patterns and trends in population health (e.g., McKeown 1976; Williams 1990). The result is renewed interest in conceptualizing SES as a central and primary force driving population health rather than as just an individual-level attribute.

In this paper we test a central assumption of one prominent theoretical approach to understanding the socioeconomic stratification of health outcomes that has arisen in response to the dominant “risk-factor” approach: Link and Phelan’s theory of “Fundamental Causes” (Link and Phelan 1995; Link and Phelan 1996). The prominence of this theory in recent research on public health --- as well as the fundamental challenge that that the theory poses to much mainstream research in epidemiology, public health,

the social sciences, and elsewhere --- motivates our investigation. As we describe below, the theory of Fundamental Causes is built on the largely untested assumption that socioeconomic inequalities in morbidity and mortality have remained essentially constant over historical time. We begin our paper with a review of the theory of Fundamental Causes and the role of this untested assumption in the theory. We then offer a discussion of the difficulties involved in testing this assumption, and conclude with a preliminary investigation of inter-cohort trends in SES differences in age-specific morbidity across the 20<sup>th</sup> century.

## **THEORY OF FUNDAMENTAL CAUSES**

### *Historical Context*

It is important to consider the association between socioeconomic status and morbidity and mortality within the context of the dramatic overall decline in morbidity and mortality rates. Both crude and age-adjusted mortality rates declined over the course of the 1900s; similarly, morbidity rates (a measure of illness) also declined (McKinlay and McKinlay 1977; Fingerhut et al. 1980). Moreover, the leading causes of death have shifted from infectious to chronic over the same time period (McKeown 1976; McKinlay and McKinlay 1977; Fingerhut 1980; Williams 1990). Explanations for the decline in morbidity and mortality rates include improvements in nutrition, national and worldwide immunization programs, and medical advances. Others counter that immunization programs and medical advances have had a limited impact on declining rates (McKinlay and McKinlay 1977; McKeown 1976).

### *Socioeconomic Status and Health*

The inverse relationship between socioeconomic status and morbidity and mortality has been recognized for centuries (Antonovsky 1967; McKeown 1976). However, it is not very clear why socioeconomic inequalities in health have purportedly persisted over historical time, despite declining rates of mortality and morbidity. Link and Phelan (1995; 1996) note that broad improvements in public health and increasing life expectancies have not led to a narrowing of SES gradients in health. To explain this observation Link and Phelan (1996: 472)...

...propose that such enduring associations between sociodemographic factors and disease are predictable and perhaps unavoidable, because many social conditions are what [they] have called "fundamental social causes" of disease. As [they] define it, a fundamental social cause involves resources like knowledge, money, power, prestige, and social connections that strongly influence people's ability to avoid risks and to minimize the consequences of disease once it occurs. Because of the very general utility of these social and economic resources, fundamental causes are linked to multiple disease outcomes through multiple risk-factor mechanisms. Moreover, because these resources can be used differently in different situations, fundamental causes influence disease even when the profile of risk factors changes dramatically. It is this persistent effect on health in the face of dramatic changes in mechanisms that leads [them] to call such factors "fundamental" social causes...

The theory of Fundamental Causes is built on the recognition that the risk factor mechanisms that link SES to morbidity and mortality in one generation may not be the same risk factor mechanisms to make that link in subsequent generations. Whereas issues like sanitation, water quality, and food safety may have been the key mechanisms linking SES to morbidity and mortality several decades ago, researchers now focus on factors like smoking, exercise, and access to health insurance as explanations for associations between SES and morbidity and mortality. As a result, the associations

between SES and morbidity and mortality persist despite changes over time in intervening mechanisms.

How does this shift in mechanisms come about? The theory posits that people with superior economic, social, and knowledge-based resources will be better able to avoid health risks. These resources may be individual in nature --- for example, personal income or knowledge of the risks of obesity --- or they may be more contextual in nature --- for example, neighborhood and working conditions. In any case, these resources place socioeconomically advantaged individuals in a better position to avoid health risk factors, whatever those risk factors might be at a particular point in historical time. As Link and Phelan (2002:730) note, “when a population develops the wherewithal to avoid disease and death, individuals’ ability to benefit from that wherewithal is shaped by resources of knowledge, money, power, prestige, and beneficial social connections.” In the mid 20<sup>th</sup> century, children of socioeconomically advantaged parents were more likely to be immunized, in part because their parents had better access to medical care and greater knowledge of the health benefits of immunization. Towards the end of the century, immunization rates approach their maximum and so the socioeconomic gradient declined. However, at the same time there was increasing evidence of the health risks of smoking. While there were smaller socioeconomic group differences in smoking behaviors in preceding decades, more recently we have witnessed much larger differences as individuals with more resources and knowledge have been able to avoid smoking. One causal mechanism linking SES and health --- patterns of immunization --- became less important while another --- patterns of smoking --- became more import; in the end, the association between SES and health may not have changed.

### *Public Health Policy Implications*

The theory of Fundamental Causes has a number of important implications for research and policy. From a research perspective, focusing on SES as a fundamental cause is more of an “upstream” approach to health disparities (Robert and House 2000). For instance, educational level influences income, which then influences employment. Since health care insurance is largely an employer-based system in the U.S., this employment status impacts access to health care. Finally, unequal access to health care leads to disparities in morbidity and mortality (e.g. unequal rates of screening lead to disparities in stage-at-diagnosis for cancer; Lerman 1990; Shattner and Gilad 2002). In this example, providing health insurance in the form of government programs (such as Medicaid), is more of a downstream approach --- it provides immediate access to health care, but it does not address the root cause of the inequalities (e.g. education, income).

Understanding the upstream causes of inequalities directly influences policy. In a study of the mortality of low SES men, Duleep (1989) did not find improvements between 1960 and the mid-1970s, yet Medicaid was introduced in 1965. The implication is that a nearly exclusive focus on uncovering and eradicating the mechanisms that currently link SES and particular health outcomes will not move us much closer to understanding more broadly and sociologically how health outcomes are persistently stratified. Thus, from a research and policy perspective, it is imperative to understand the upstream and downstream causes for inequalities in health.

### **HAVE SES INEQUALITIES IN HEALTH CHANGED OVER TIME?**

The central tenets of the theory of Fundamental Causes have been tested on a number of occasions (Link et al. 1998; Phelan et al. 2004), and the theory has held up well. However, a close reading of Link and Phelan’s work reveals a surprising lack of sound empirical evidence for the core assumption that underlies and motivates the theory of Fundamental Causes: SES gradients in morbidity and mortality have persisted at essentially constant levels over a broad historical period.

As Link and colleagues note in a recent manuscript (2004), “The basis for the argument [for Fundamental Causes] rests on the remarkably consistent association between SES and health across time and place.” However, to demonstrate the remarkable consistency of this association the authors typically take one of two approaches. In most cases, they simply site evidence that observers in the 19<sup>th</sup> century (or earlier) also witnessed socioeconomic gradients in health. For example, Link and Phelan (1996: 471) note that...

...if one returns to the volumes of the [*American Journal of Public Health*] published 25 or 50 years ago, one will find studies focused on problems such as the relationship of socioeconomic status to health and age and sex variations in the prevalence and onset of diabetes mellitus. If one journeys back even farther to the roots of social medicine in the works of people like Rudolf Virchow, one will find something very similar: a predominant concern with associations between disease and social conditions like poverty and substandard housing.”

Because concerns about socioeconomic inequalities in morbidity and mortality have been voiced for more than a century, the reader is led to believe that the magnitude of the association between SES and health has remained more or less constant.

In other cases, Link and Phelan site historical and contemporary evidence about associations between SES and particular health outcomes. In their 1998 article, Link and colleagues (Link et al. 1998) cite Villerme’s (1840) evidence from France in the 1830s of



occupation category-differences in life expectancy alongside more recent evidence of SES gradients in health (Antonovsky 1967; Chaplin 1924; Coombs 1941; Pappas et al. 1993). Again, the reader is led to believe that the magnitude of socioeconomic gradients in morbidity and mortality has remained essentially unchanged over time.

We argue below that neither approach provides good evidence for the assertion that the strength of the association between SES and health has persisted over time. Unless there is sound evidence that socioeconomic gradients in health have not declined substantially over time, then the motivation for the theory of Fundamental Causes seems weak. It may be that the empirical association between SES and health has declined substantially over time. If this is the case, then a continued focus on understanding intervening mechanisms between SES and health and a continued de-emphasis of broader social and economic environmental factors may help to erode SES-health associations even further.

## **CONCEPTUAL ISSUES**

As described above, Link and Phelan tend to cite historical evidence of SES-health associations --- sometimes alongside contemporary evidence --- to support the assertion that socioeconomic gradients in health have persisted over time. However, knowing that SES-health associations also existed in the past provides no meaningful evidence about trends over time in the magnitude of those associations. Often, Link and Phelan provide anecdotal evidence that amounts to comparing “apples to oranges.” For example, as noted above Link and colleagues (1998) cite Villerme’s (1840) evidence alongside contemporary evidence from Chaplin (1924), Pappas et al. (1993) and others.

Villerme demonstrated that life expectancy was higher for “managers, merchants and directors etc” than for factory workers in early 19<sup>th</sup> century France. Chaplin showed that tax-payers (i.e., property owners) had lower mortality rates than non-taxpayers (i.e., non-property owners) in Providence, Rhode Island in 1865. Pappas et al. (1993) showed that mortality rates varied by race, sex, income, and education in the United States in 1960 and 1986. All three pieces of evidence speak to socioeconomic inequalities in health, but because the measures of SES (and even of mortality) vary so widely, it is impossible to say anything meaningful from this evidence about trends over time in SES-health associations.

How can we assess temporal trends in the association between SES and morbidity and mortality? As a first step, we need to measure both SES and health or mortality in a consistent manner over time. The basic structure of the U.S. education system has not changed dramatically in the last several decades; nor has the basic racial classification system. It seems possible, therefore, to use major educational credentials or crude racial classifications as proxies for SES in studying long-term trends in the association between SES and health or mortality. One could imagine, for example, pooling more than 100 years of U.S. Census data and modeling inter-cohort changes in racial differences in age-specific mortality rates since Reconstruction.

The problem with this approach is that even if we measure SES in a consistent manner over time, the social meaning and consequences of particular components of SES change in important ways over time. Chaplin’s (1924) comparison of tax-payers to non-taxpayers made good sense for 1865, because it served as a good proxy for property ownership and because property ownership was an important indicator of social and

economic standing at that time. However, comparing tax-payers to non-payers would mean something completely different in 2005. In a similar fashion, the social meaning of race has changed in important ways over time. In our hypothetical model (above) of inter-cohort changes in racial differences in age-specific mortality rates since Reconstruction, we might measure race in a crude but consistent manner (e.g., “White,” “Black,” or “other”), but the social and economic implications of falling into one of these racial categories has changed over time. Although blacks and whites most certainly do not enjoy equal social and economic rewards in American society, the black-white divide in political, economic, and social power looks quite different today than it did 100 or even 50 years ago. The same is true for education: Even if we were to measure education (as a proxy for SES) in a consistent way over time, the social and economic advantages associated with completion of different levels of formal schooling have changed over time. In the early 20<sup>th</sup> century the lack of a high school diploma did not necessarily carry the dire lifetime economic consequences that it does today. Even Pappas et al.’s (1993) examination of trends between 1960 and 1986 in educational differences in mortality may be biased by changes over that time period in the social and economic consequences of completing particular levels of schooling; for example, this period witnessed declining rates of high school dropout and a marked expansion of post-secondary education.

Our point is that in order to come to a meaningful assessment of temporal trends in the association between SES and health, at least two things must be true. First, both SES and health must be measured in a consistent manner over time. Second, the measure of SES must carry the same social and economic meaning over time. Beyond this, it is worth noting that the populations from which the data are drawn must be historically

comparable. It is not clear to us, for example, what we can learn by comparing 1830s France to Providence, Rhode Island in 1865.

We do not claim to have a perfect solution to the methodological quandary presented by the need to utilize measures of SES that mean the same thing over historical time. We hope that the preceding discussion inspires others to think more carefully about how to assess temporal trends in SES-health associations. In the sections that follow, we offer an attempt to model inter-cohort trends in SES differences in self-assessed overall health using a subjective measure of childhood SES. As we describe below, this subjective measure is closely related to more objective measures of childhood SES; more importantly for the present discussion, its social and economic meaning is also relatively stable over time.

## **DATA AND MEASURES**

The General Social Survey (GSS) is administered by the National Opinion Research Center (NORC) at the University of Chicago. The multi-purpose survey of members of households across the United States was conducted annually between 1972 and 1994 (except in 1979, 1981, and 1992) and bi-annually between 1994 and 2004; because the 2004 have not yet been released, the present analyses use data collected between 1972 and 2002. Each survey year NORC selects a fresh cross-sectional random sample of Americans living in households. Prior to 1994, the GSS was conducted in-person with about 1,500 respondents each year; beginning in 1994, NORC began interviewing approximately 3,000 people every other year. Respondents range in age from 18 to 89. The content of the interviews ranges broadly from attitudes about a

variety of social issues to questions about socioeconomic and demographic characteristics to a variety of behavioral measures. Core questions --- including those at the heart of our analyses --- have been asked the same way in each survey year in order to facilitate comparisons over time.

Our measure of childhood SES is derived from a survey item that asks: “Thinking about the time when you were 16 years old, compared with American families in general then, would you say your family income was--far below average, below average, average, above average, or far above average?” Although it is a purely subjective measure of childhood socioeconomic circumstances, this measure is tightly associated with more objective measures of respondents’ parents’ educational attainment and occupational prestige in the GSS data.<sup>1</sup> What is more, as we demonstrate below, this measure is strongly associated with self-assessed overall health across the life course. This measure was obtained from the full GSS sample each year through 1993, was asked of a randomly

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<sup>1</sup> For example, here are the percentages of respondents who reported their relative family income at age 16 to be below average by the occupation that their father held when they were growing up: Managerial and Professional Specialty Occupations: 12%; Technical, Sales, and Administrative Support Occupations: 16%; Service Occupations: 34%; Farming, Forest, and Fishing Occupations: 45%; Precision Production, Craft, and Repair Occupations: 28%; and Operators, Fabricators, and Laborers: 34%. Likewise, and perhaps more strikingly, here are the percentages of respondents who reported their relative family income at age 16 to be below average by the highest level of schooling completed by their father: Less than high school: 35%; High school graduate: 17%; Attended some college: 15%; College Graduate: 8%; Some graduate schooling: 8%.

selected half of the sample in 1994, and was then asked of a randomly selected two thirds of the sample in 2002. Over these years, 32,831 respondents were presented with this question; 32,444 (or 98.8%) gave valid responses. In our analyses, we dichotomize this measure such that 0 equals average or above average family income and 1 equals below average family income.

Our measure of adults' self-assessed overall health is derived from a survey item that asks: "Would you say your own health, in general, is excellent, good, fair, or poor?" Entirely subjective measures of self-assessed overall health are closely linked to more objective measures of morbidity and mortality (Idler and Benyamini 1997). Fingerhut (1980) notes, "one of the best single indicators of general health status is self-assessment of health" (p.3). This item was asked of all respondents between 1972 and 1987 (except that it was not asked in 1978, 1983, or 1986) and of a random two thirds of respondents since 1988. In these years, 33,722 respondents were presented with this question; 33,620 (or 99.7%) gave valid responses. In our analyses, we also dichotomize this variable such that 1 equals excellent or good health and 0 equals fair or poor health. Alternate specifications (e.g., dichotomizing the measure such that 1 equals excellent and 0 equals other response) yield results that closely parallel those presented below.

In all survey years, NORC ascertained each respondent's age at the time of their interview. Using this information, we have computed each respondent's year of birth. Because we have a wide age range in each survey year and because we have almost 30 years of cross-sectional survey data, respondents' years of birth range from 1883 to 1984. Since we have relatively few respondents born in the 1880s or 1980s we restrict these analyses to individuals born between 1890 and 1979. Likewise, because we have

relatively few respondents in their teens or in their 80s we restrict the sample to individuals between ages 20 and 79.

## **DESCRIPTIVE RESULTS**

Table 1 reports the percentage of individuals who report their health as excellent or good, separately by age group, birth cohort, and childhood socioeconomic circumstances. One purpose of this table is to demonstrate the obvious: We do not observe members of all age groups in all birth cohorts, and we do not observe members of all birth cohorts in all age groups. The empty cells in the upper left portion of Table 1 are a function of the GSS not beginning until 1972; people born in the 1890s could only be in the 20 to 29 age group if they were interviewed in the 1910s. The empty cells in the lower right portion of the table cannot be filled in yet; 70 to 79 year olds born in the 1970s will not exist until the 2050s. Another purpose of this table is to demonstrate that we have a reasonable number of observations --- at least 146 --- in each age group by birth cohort cell.

The estimates in the table --- which are also depicted in Figure 1 --- provide some reassurance about the quality of the GSS data for this purpose. First, as expected we observe that respondents' self-reported overall health declines with age within every birth cohort. Second, and as expected, we observe that respondents' self-reported overall health improves across birth cohorts within specific age groups. It is reassuring to us to replicate these basic and expected findings using these data and measures.

The diagrams in Figure 1 are designed to give some preliminary answer to our basic research question. If the core empirical assumption underlying the theory of

Fundamental Causes is correct, then we should observe that SES differences in age-specific health outcomes remain the same (or even increase) across birth cohorts. If these age-specific SES differences decline across birth cohorts, then this would present a serious challenge to the theory. Among people in their 20s, 30s, 60s, and 70s the SES difference in the percentage of respondents who report their health as excellent or good remains essentially unchanged across birth cohorts. Yet, among people in their 50s, we see some visual evidence that SES differences in this outcome actually increase across birth cohorts. Finally, only among people in their 40s do we see declining SES differences in this outcome across birth cohorts. It is important to note that Figure 1 simply presents age-group and birth cohort-specific means. Whether the patterns described above are statistically significant requires multivariate analyses of these data.

## **MULTIVARIATE RESULTS**

In Table 2, we present results from logistic regression models in which health is a function of relative childhood family income and birth cohort. We estimate separate models for each of the six age groups. Each model includes a covariate indicating whether the respondent reported that their childhood family income was below average and series of dummy variables representing birth cohorts. As described above and in Table 1, each age group is observed in different subsets of birth cohorts. The relative childhood family income coefficient represents the association between this variable and respondents' health. The birth cohort coefficients reflect inter-cohort changes in age group-specific health outcomes. The most substantively interesting coefficients are those representing interactions between birth cohorts and relative childhood family income.



The goal of our paper is to test the hypothesis that age group-specific SES differences in health outcomes have remained stable over time. These interaction terms allow us to test this hypothesis directly. If they are statistically different from zero, then we would conclude that age group-specific SES differences in health outcomes have changed across birth cohorts.

The results in Table 2 indicate that respondents whose relative childhood family incomes were below average were substantially less likely to report their health as excellent or good. These coefficients are statistically significant for four of six age groups. The coefficients for birth cohorts re-iterate what was clear in Figure 1: Age group-specific health outcomes generally improve across birth cohorts. Most importantly, in only three cases are the birth cohort by relative childhood family income interaction terms statistically significant. In two of these three cases (in the models for people in their 30s and 50s) the statistically significant coefficients indicate growing age-specific SES difference in health outcome. As a result, in only one instance do we observe a statistically significant interaction term that would contradict the assumption that age group-specific SES differences in health outcomes have remained stable over time. We cannot say with certainty whether this single coefficient represents something distinctive in the experiences of people in their 40s who were born in the 1950s, or whether this anomalous finding is due to sampling variability.

## **DISCUSSION**

Link and Phelan's (1995; 1996) "Fundamental Causes" theory posits that social and economic inequalities in morbidity and mortality have persisted over historical time -

-- and will continue to persist --- because people with access to more social and economic resources are always better able to avoid health risks (even if the nature of those risks change over time). Despite the prominence of this theory and its inherent challenge to mainstream epidemiological, public health, and social science research, the core empirical fact that the theory is designed to explain has not been well established by Link and Phelan or others. Have socioeconomic inequalities in morbidity and mortality remained essentially constant over time?

To investigate, we examined inter-cohorts trends in age-specific health inequalities using data from the 1972 through 2002 General Social Surveys. Our results reproduce findings of inter-cohort improvements in health outcomes, but we find that socioeconomic differences in health have remained unchanged across the bulk of the 20<sup>th</sup> century. The results presented here indicate that although Americans' health has improved over time, inequalities in health have persisted.

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Table 1. Percentage of Respondents in Excellent or Good Health, by Decade of Birth and Age Group  
1972 to 2002 General Social Surveys

Age Group	Relative Family Income at Age 16	Decade of Birth								
		1890s	1900s	1910s	1920s	1930s	1940s	1950s	1960s	1970s
<b>20 to 29</b>	Avg. or Above Avg	<i>% in Excellent or Good Health</i> →				90%	88%	89%	85%	
	Below Avg.	<i>Sample Size</i> →				716	1,708	1,340	323	
<b>30 to 39</b>	Avg. or Above Avg				83%	88%	87%	88%		
	Below Avg.				462	1,323	1,465	394		
<b>40 to 49</b>	Avg. or Above Avg				79%	79%	85%	82%		
	Below Avg.				454	894	987	374		
<b>50 to 59</b>	Avg. or Above Avg			68%	71%	77%	85%			
	Below Avg.			393	846	621	213			
<b>60 to 69</b>	Avg. or Above Avg		56%	61%	68%	79%				
	Below Avg.		342	726	582	113				
<b>70 to 79</b>	Avg. or Above Avg	56%	55%	56%	62%					
	Below Avg.	161	487	474	90					
		45%	39%	51%	54%					
		96	258	322	56					

Figure 1. Percentage of Respondents in Excellent or Good Health, by Decade of Birth and Age Group, 1972 to 2002 General Social Surveys

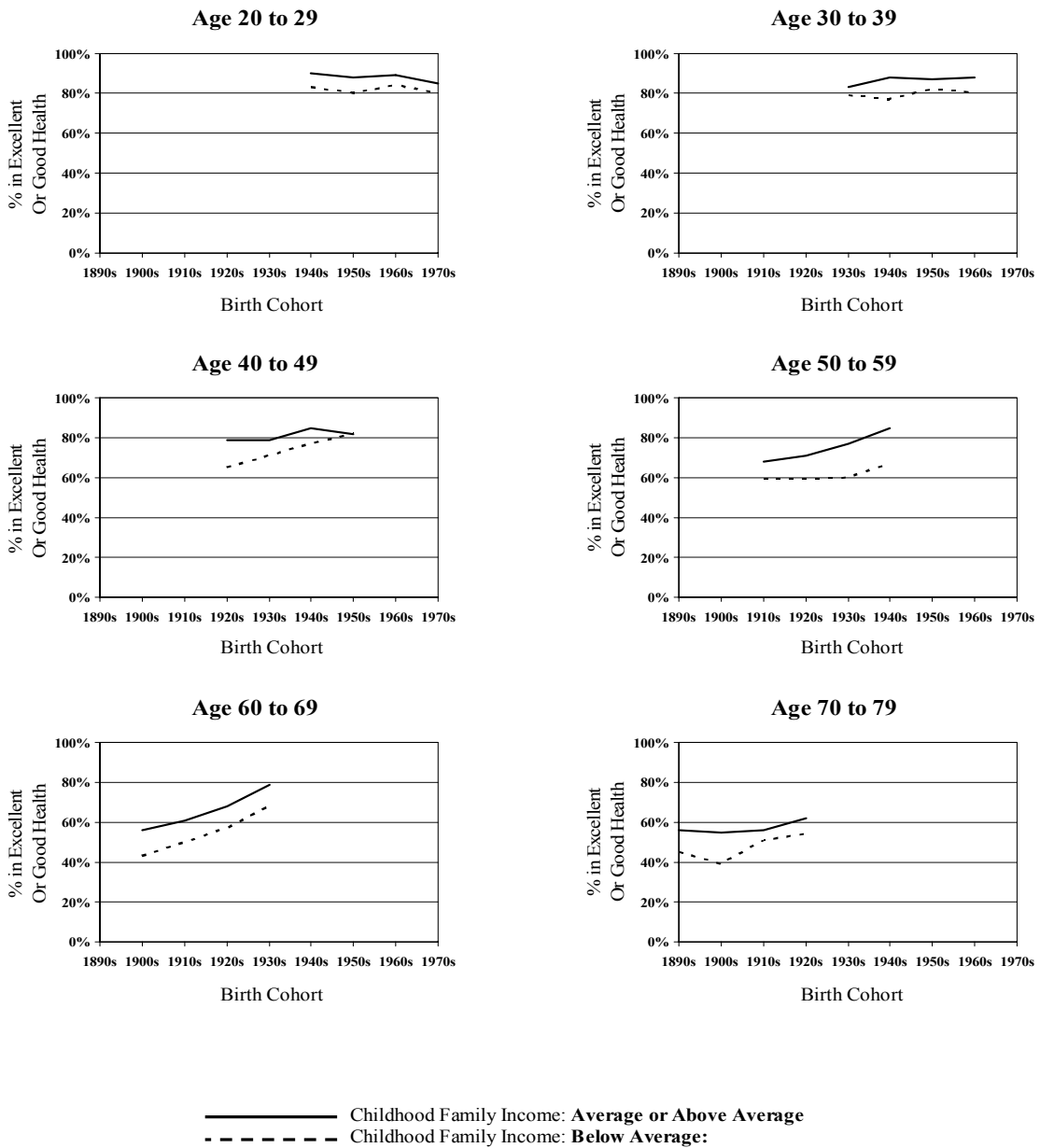




Table 2. Logistic Regression Models of Excellent or Good Health on Relative Childhood Family Income and Birth Cohort, by Age Group

	Age 20 to 29		Age 30 to 39		Age 40 to 49		Age 50 to 59		Age 60 to 69		Age 70 to 79	
	<i>exp</i> <sup>b</sup>	b/s.e.	<i>exp</i> <sup>b</sup>	b/s.e.	<i>exp</i> <sup>b</sup>	b/s.e.	<i>exp</i> <sup>b</sup>	b/s.e.	<i>exp</i> <sup>b</sup>	b/s.e.	<i>exp</i> <sup>b</sup>	b/s.e.
Childhood Family Income Was Average or Above	<i>Reference Group</i>											
Childhood Family Income Was Below Average	0.59	(2.53) *	0.80	(1.14)	0.51	(3.73) **	0.67	(2.38) *	0.60	(2.70) **	0.64	(1.72)
1890 Birth Cohort	---		---		---		---		---		<i>Reference Group</i>	
1900 Birth Cohort	---		---		---		---		<i>Reference Group</i>		0.96	(0.24)
1910 Birth Cohort	---		---		---		<i>Reference Group</i>		1.22	(1.51)	1.00	(0.00)
1920 Birth Cohort	---		---		<i>Reference Group</i>		1.12	(0.89)	1.62	(3.46) **	1.30	(0.97)
1930 Birth Cohort	---		<i>Reference Group</i>		1.00	(0.00)	1.57	(3.11) **	2.90	(4.18) **	---	
1940 Birth Cohort	<i>Reference Group</i>		1.49	(2.66) **	1.53	(2.93) **	2.51	(4.22) **	---		---	
1950 Birth Cohort	0.82	(1.38)	1.41	(2.35) *	1.27	(1.34)	---		---		---	
1960 Birth Cohort	0.92	(0.55)	1.56	(2.23) *	---		---		---		---	
1970 Birth Cohort	0.69	(1.88)	---		---		---		---		---	
1890 Cohort x Below Avg. Inc.	---		---		---		---		---		<i>Reference Group</i>	
1900 Cohort x Below Avg. Inc.	---		---		---		---		<i>Reference Group</i>		0.81	(0.68)
1910 Cohort x Below Avg. Inc.	---		---		---		<i>Reference Group</i>		1.06	(0.27)	1.28	(0.83)
1920 Cohort x Below Avg. Inc.	---		---		<i>Reference Group</i>		0.88	(0.64)	1.06	(0.24)	1.09	(0.21)
1930 Cohort x Below Avg. Inc.	---		<i>Reference Group</i>		1.30	(1.18)	0.65	(1.97) *	0.97	(0.08)	---	
1940 Cohort x Below Avg. Inc.	<i>Reference Group</i>		0.59	(2.19) *	1.14	(0.56)	0.56	(1.76)	---		---	
1950 Cohort x Below Avg. Inc.	0.97	(0.11)	0.81	(0.90)	1.86	(2.03) *	---		---		---	
1960 Cohort x Below Avg. Inc.	1.10	(0.36)	0.67	(1.25)	---		---		---		---	
1970 Cohort x Below Avg. Inc.	1.12	(0.30)	---		---		---		---		---	
Constant	8.55	(17.58) **	4.85	(12.77) **	3.68	(11.38) **	2.17	(7.13) **	1.28	(2.27) *	1.27	(1.49)
Sample Size	5,293		5,193		4,061		3,393		2,960		1,944	