Childhood Socioeconomic Position and Disability in Later Life: Results of the Health and Retirement Study

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Objectives. We used a life course approach to assess the ways in which childhood socioeconomic position may be associated with disability in later life.

Methods. We used longitudinal data from the nationally representative Health and Retirement Study (1998–2006) to examine associations between parental education, paternal occupation, and disabilities relating to activities of daily living (ADLs) and instrumental activities of daily living (IADLs).

Results. Respondents whose fathers had low levels of education and those whose fathers were absent or had died while they were growing up were at increased risk of disability in later life, net of social, behavioral, and pathological health risks in adulthood. Social mobility and health behaviors were also important factors in the association between low childhood socioeconomic position and ADL and IADL disabilities.

Conclusions. Our findings highlight the need for policies and programs aimed at improving the well-being of both children and families. A renewed commitment to such initiatives may help reduce health care costs and the need for people to use health and social services in later life. (*Am J Public Health.* 2010; 100:S197–S203. doi:10.2105/AJPH.2009.160986)

The disablement process model has been used to show that disability is a long-term process varying largely according to disease type and severity but also according to social status and health behaviors.[1] For example, the health status of people with few socioeconomic resources is consistently worse than that of their better-off counterparts.[2,3] Poor health behaviors such as smoking and physical inactivity also vary according to socioeconomic position (SEP), and these behaviors are associated with an increased risk for a variety of disabling conditions and other adverse health outcomes in adulthood.[4] As such, most disability studies have focused on health processes occurring during the midlife to later life period,[5–7] and less is known about earlier life course processes that may be associated with accumulated risk.

Life course researchers have increasingly suggested that exposure to adverse health risks in childhood may have long-term effects.[8,9] According to the life course approach, disability and other poor health outcomes may be the product of a range of adverse social and behavioral health risks incurred across the life course and stemming from early life conditions and experiences. For example, the Whitehall studies of British civil servants reported relationships between low childhood SEP and cardiovascular disease, morbidity, and mortality.[10,11] Although these relationships have been contested,[12,13] other life course studies have similarly suggested relationships of morbidity and mortality with poor maternal and fetal nutrition,[14] low parental education, nativity,[15,16] and manual paternal occupation.[17] The reason may be that children from poor families are at risk for low birthweight and poor nutrition and are exposed to more adverse health risks (e.g., toxins, pollution, lead, secondhand smoke) than their peers.[18] As a result, respiratory disorders, infectious diseases, chronic conditions, and functional limitations are typically more common among children from such families.[19]

The adverse effects of low childhood SEP may increase the risk of disability in several ways. Children from poor families have fewer opportunities for socioeconomic achievement in adulthood than do their counterparts from more advantaged families.[11,15] As such, low childhood SEP may begin a "chain of risk"[8] leading to further disadvantages across the life course. This idea is similar to cumulative disadvantage[20] (the accrual of adverse social, behavioral, and other health risks over time) and allostatic load[21] (the cumulative negative effects incurred when the body adapts to various challenges and adverse environments). Low childhood SEP may also initiate a pattern of poor health behaviors.[22] Children from disadvantaged families have poorer nutrition and lower physical activity levels than their counterparts,[23] and this situation may continue into adulthood, adversely affecting health and functioning.[24]

There is additional evidence that some of the adverse effects of low childhood SEP on health may be independent of these social and behavioral pathways. For example, low childhood SEP has been associated with adult morbidity, poor health, and mortality independent of socioeconomic achievement and health behaviors.[25–28] These findings are consistent with the notion of biological embedding,[29] the process through which health risks interact to create systematic differences in host resilience, and the fetal origins hypothesis,[30] according to which childhood is a critical period of development in which exposure to adverse health risks has long-term and enduring effects on adult health outcomes.

We used a life course approach to explore the relationship between low childhood SEP (assessed via parental education and father's occupation) and severe disabilities in later life self-care (e.g., bathing, eating) and moderate disabilities in other areas of functioning (e.g., taking medications, preparing meals). We also examined the ways in which factors such as social mobility (education, income, and wealth) and behavioral transitions (smoking, drinking, exercising, and body weight) in adulthood may alter this relationship over time. We used a nationally representative sample of older Americans and accounted for predisposing characteristics (age, gender, and race) and intraindividual changes in pathology (heart problems, diabetes, stroke, hypertension, and lung disease) over a 9-year period. We hypothesized that low childhood SEP would increase disability risk in later life through

a variety of adverse socioeconomic and behavioral factors.

METHODS

Our data were derived from the Health and Retirement Study (HRS), a prospective cohort study conducted by the University of Michigan with support from the National Institute on Aging. In 1992, a probability cohort sample of individuals aged 51 to 61 years took part in the first wave of this multistage nationally representative study; this first-wave cohort sample was merged with the Asset and Health Dynamics of the Oldest Old Study cohort (born 1890–1923) in 1998. Two other cohorts, the Children of the Depression (born 1924–1930) and War Babies (born 1942–1947) cohorts, were added in 1998 to fill in between-group age and cohort gaps, resulting in a sample design nationally representative of US residents aged older than 50 years in 1998. Further details on the HRS design and methods have been previously published.[31]

We used 5 waves of data (1998–2006) from the HRS combined with data prepared by the RAND Center for the Study of Aging (RAND–HRS). The benefits of using the RAND–HRS data included the availability of detailed health, behavioral, and socioeconomic information and the use of bracketing methods to minimize nonresponse for these data.[32] Data were weighted via respondent-level sampling weights to account for the HRS sample design.[31] After exclusion of respondents with sampling weights of zero, indicating that they had been unable to answer survey questions at baseline, were institutionalized, or had died, our final sample consisted of 18 465 individuals. By the final survey year included in our study (2006), approximately 22.8% of respondents had died and 8.2% had been lost to follow-up. HRS attrition rates are comparable to those of other panel surveys,[33] and sample attrition has not significantly influenced the representativeness of the remaining sample.[34]

Measures of Interest

We used scales assessing respondents' abilities with respect to activities of daily living (ADLs) and instrumental activities of daily living (IADLs) to predict disabilities in self-care and other forms of functioning.[1] In the case of ADLs, respondents were asked whether they had severe difficulty walking across a room, bathing, eating, dressing, and getting in and out of bed. For IADLs, respondents were asked whether they had moderate difficulty using the phone, managing money, taking medications, shopping for groceries, and preparing meals. Each scale ranged from zero to 5, with 5 indicating the highest level of difficulty.

Predictor Variables

Respondents' reports of parental educational levels (both mother and father, each ranging from zero to ≥17 years) and father's occupation (main occupation when the respondent was aged 16 years) were used to assess childhood SEP. Although retrospective reports of childhood SEP may underestimate childhood disadvantage,[35] there is evidence that retrospective reports of father's education and other familial characteristics are reliable.[36] Paternal occupational categories were as follows: professional (manager or administrator; reference category), craftsman, farmer or farm manager, clerical or sales worker, operative (e.g., machine or transport worker), and service worker or laborer.

Respondents who reported that their father was disabled or had never worked, or whose father was absent while they were growing up or had died during this period were grouped into 2 categories (father disabled or never worked and father absent or deceased). Respondents with missing data on mother's (10.0%) and father's (14.4%) educational level and father's occupation (4.8%) were excluded from our analyses. These respondents were more likely to be older, female, and Black and to have completed fewer years of education.

We also examined social mobility and behavioral transitions in adulthood to determine potentially mediating relationships between these variables and childhood SEP and disability.[11,15,24] Social mobility was assessed in terms of education (zero to ≥17 years), income (log-transformed total household income), and wealth (log-transformed value of assets). The health behaviors examined were smoking (current, never, former smoker), alcohol use (yes or no), and exercise; body mass index (BMI; defined as weight in kilograms divided by height in meters squared) was also assessed. The exercise category included activities such as physical labor on the job, heavy housework, aerobics, bicycling, running or jogging, and swimming 3 or more times per week; we used this measure because it was used consistently over the different waves of the HRS.

To account for pathology, the leading cause of self-care and functioning difficulties,[1] respondents were asked whether a doctor had ever told them that they had heart problems (including coronary heart disease, heart attack, congestive heart failure, and heart surgery), diabetes (or high blood sugar), lung disease (excluding asthma, chronic bronchitis, and emphysema), or hypertension (or high blood pressure) and whether they had had a stroke (or a transient ischemic attack). Self-reported health conditions have shown substantial agreement with both survey and medical record reports.[37]

In applicable instances, variables were measured at baseline (1998) and subsequently every 2 years over the course of the study. As such, age, social mobility (income, wealth), health behaviors, pathology, and disability varied over the course of time, allowing for an examination of intra-individual changes over the 9-year study period. Gender, race/ethnicity (White versus non-White), educational level, and childhood SEP variables were fixed.

Statistical Analysis

In conducting our statistical analyses, we used generalized linear latent and mixed-model commands[38] available for the analysis of complex sample survey data in Stata version 9.2.[39] All of the statistical analyses were design based, accounting for the complex HRS sampling design and the subset analyzed. Our multilevel models incorporated information from 5 waves (or 9 years; 1998–2006) of data simultaneously in the same model.

We used 2-level generalized latent and mixed models. The first level examined intra-individual changes in disability related to social mobility, health behaviors, and pathology. The second level examined individual variations in these factors across groups categorized according to characteristics such as gender, race/ethnicity, and childhood SEP. Many health and functioning distributions are non-normal, reflecting the higher frequency of intact functioning among the general, community-dwelling older adult population. As such, nonlinear models that modeled Poisson

distributions of the ADL and IADL data fit better than the linear models. We present the results of these nonlinear analyses.

To examine the cumulative effects of low childhood SEP on disability through temporally ordered social and behavioral health risks across the life course, we entered childhood SEP (model 1), social mobility (model 2), and health behaviors (model 3) sequentially into multilevel models. All models were adjusted for predisposing characteristics (age, gender, race/ethnicity), and model 4 accounted for pathology in addition to these characteristics.

RESULTS

Consistent with a community-dwelling sample of older adults, our respondents reported relatively few ADL and IADL disabilities at baseline (Table 1). On average, mothers' educational levels were higher than fathers' educational levels, and most respondents reported their father's main occupation as craftsman, farmer or farm manager, or operative.

Activities of Daily Living

As can be seen in model 1 of Table 2, which accounted for age, gender, and race/ethnicity, each additional year of parental education was associated with a decreased risk of ADL disabilities. For example, respondents whose parents had completed high school (12 years of education) had a lower risk of ADL disabilities than did respondents whose parents had completed only secondary school (8 years of education). Relative to respondents whose fathers were professionals, respondents whose fathers were farmers or farm managers, operatives, or service workers also were at increased risk for ADL disabilities. Respondents whose fathers had never worked or were disabled, or were absent or had died were at increased risk as well.

The addition of social mobility variables to the model weakened the effects of parental education on ADL disabilities and statistically explained much of the relationship between father's (manual) occupation and such disabilities. Having a father who was an operative, who had never worked, or who was disabled was no longer associated with increased risk for ADL disabilities in this model. In addition, having a father who was a farmer or farm manager was associated with a decreased risk for ADL disabilities.

Additional analyses (data not shown) were conducted to further examine the pathways linking childhood SEP, social mobility, and ADL disabilities. An examination of childhood SEP and social mobility interactions showed that education mediated the relationship between ADL disabilities and having a father who was an operative. Also, income lowered ADL disability risk among respondents with fathers who were farmers or farm managers or operatives and respondents whose fathers were absent or had died while they were growing up.

As can be seen in model 3 of Table 2, the addition of behavioral variables did not affect the relationship between parental education and ADL disabilities. However, the strength of the relationship between father's occupation and ADL disabilities was reduced for respondents whose fathers were service workers or laborers and respondents whose fathers were absent or had died while they were growing up.

Finally, the addition of pathology variables statistically explained the relationship between mother's education and ADL disabilities and reduced the strength of the relationship between father's education and ADL disabilities. Also in this model, having a father who was a farmer or farm manager or an operative was associated with a decreased risk for ADL disabilities, and having a father who was absent or had died was associated with an increased risk for such disabilities. The additions of social mobility, health behaviors, and pathology accounted for some of the between-person variability in initial status, meaning that these adult health risks accounted for a portion of the between-person variability in ADL disabilities (Table 2).

TABLE 1—Characteristics of Respondents: Health and Retirement Study, United States, 1998–2006

Characteristic	Sample (n = 18 465)
Demographic characteristics	
Age, y, mean (SE)	64.3 (0.15)
Female, % (SE)	57 (0.01)
White, % (SE)	87 (0.00)
Childhood socioeconomic position	
Mother's education, y, mean (SE)	9.46 (0.03)
Father's education, y, mean (SE)	9.17 (0.03)
Father's primary occupation, % (SE)	
Professional (manager or administrator)	15 (0.00)
Craftsman	21 (0.00)
Farmer/farm manager	21 (0.00)
Clerical/sales worker	11 (0.00)
Operative (machine or transport worker)	20 (0.00)
Service worker/laborer	4 (0.00)
Father never worked/disabled	<1 (0.00)
Father absent/deceased	1 (0.00)
Adult characteristics	
Disability score, mean (SE)	
ADLs	0.26 (0.00)
IADLs	0.18 (0.01)
Social mobility, mean (SE)	
Education, y	12.4 (0.02)
Income (log transformed)	4.48 (0.00)
Wealth (log transformed)	4.65 (0.02)
Health behaviors, % (SE)	
Current smoker	18 (0.00)
No history of smoking	40 (0.00)
Former smoker	42 (0.00)
Drinks alcohol	52 (0.01)
Exercises 3 or more times/wk	45 (0.00)
BMI, mean (SE)	25 (0.05)
Health conditions, % (SE)	
Heart problems	19 (0.00)
Diabetes	12 (0.00)
Stroke	6 (0.00)
Hypertension	42 (0.00)
Lung disease	7 (0.00)

Note. ADL = activities of daily living; BMI = body mass index (defined as weight in kilograms divided by height in meters squared); IADL = instrumental activities of daily living. Data are weighted. Values were calculated at baseline (1998).

Instrumental Activities of Daily Living

Model 1 in Table 3 shows that, after control for age, gender, and race/ethnicity, each additional year of parental education was associated with a reduced risk for IADL disabilities. Also, relative to respondents whose fathers were professionals, respondents whose fathers were farmers or farm managers were at increased risk for IADL disabilities, and respondents whose fathers were clerical or sales workers had a decreased risk for IADL disabilities.

TABLE 2—Results of Multilevel Models Examining the Relationship Between Activities of Daily Living Disabilities and Childhood Socioeconomic Position: Health and Retirement Study, United States, 1998–2006

	Model 1, b (SE)	Model 2, b (SE)	Model 3, b (SE)	Model 4, b (SE)
	Childhood socioeconomic position			
Mother's education, y	–0.04*** (0.00)	–0.01* (0.00)	–0.01** (0.00)	–0.02 (0.00)
Father's education, y	–0.06*** (0.01)	–0.04*** (0.01)	–0.04** (0.01)	–0.03** (0.01)
Father's primary occupation				
Professional (manager or administrator; Ref)	1.00	1.00	1.00	1.00
Craftsman	0.14 (0.12)	0.03 (0.12)	–0.02 (0.11)	–0.07 (0.08)
Farmer/farm manager	0.16*** (0.01)	–0.05*** (0.04)	–0.05*** (0.01)	–0.03* (0.01)
Clerical/sales worker	0.02 (0.07)	–0.00 (0.09)	–0.02 (0.06)	0.02 (0.03)
Operative (machine or transport worker)	0.18* (0.09)	0.01 (0.10)	0.08 (0.07)	–0.12* (0.06)
Service worker/laborer	0.25*** (0.01)	0.17*** (0.04)	0.11*** (0.03)	0.03 (0.02)
Father never worked/disabled	0.72* (0.33)	0.51 (0.34)	0.24 (0.41)	0.19 (0.36)
Father absent/deceased	0.43*** (0.10)	0.21** (0.12)	0.19** (0.07)	0.16* (0.07)
		Adult characteristics		
Social mobility				
Education, y		–0.09*** (0.01)	–0.07*** (0.01)	–0.06*** (0.01)
Income (log transformed)		–0.17*** (0.04)	–0.20*** (0.03)	–0.19*** (0.02)
Wealth (log transformed)		–0.07*** (0.00)	–0.08*** (0.00)	–0.07*** (0.00)
Health behaviors				
No history of smoking (vs current smoker)			–0.30*** (0.00)	–0.22*** (0.00)
Former smoker			0.19*** (0.00)	0.16*** (0.00)
Drinks alcohol			–0.22*** (0.00)	–0.19*** (0.00)
Exercises 3 or more times/wk			–0.30*** (0.00)	–0.29*** (0.00)
BMI			–0.04*** (0.00)	–0.09*** (0.00)
Random effect estimates				
Within person	0.18	0.18	0.18	0.19
Rate of change	0.07	0.07	0.07	0.06
Initial status	13.71	13.23	11.93	10.49

Note. BMI = body mass index (defined as weight in kilograms divided by height in meters squared). The sample size was n = 18 465. Data are weighted. Respondents were interviewed at baseline (1998) and at 2-year intervals thereafter. All models adjusted for age, gender, and race, and model 4 also adjusted for pathology (heart problems, diabetes, lung disease, hypertension, and stroke). *P < .05; **P < .01; ***P < .001.

The addition of social mobility variables to the model statistically explained the relationship between mother's education and IADL disabilities and weakened the effects of father's education on IADL disabilities. With these additions, having a father who was a farmer or farm manager or a clerical or sales worker was associated with a decreased risk for IADL disabilities. As such, social mobility in adulthood may mediate the adverse effects of having a father who was a farmer or farm manager on IADL disabilities. As with the ADL model, additional analyses (data not shown) were conducted to further examine the pathways linking childhood SEP, social mobility, and IADL disabilities. In these analyses, income mediated the relationship between IADL disabilities and having a father who was a farmer or farm manager.

It can be seen in model 3 of Table 3 that the addition of health behavior variables did not affect the relationship between parental education and IADL disabilities, although it did statistically explain the relationship between having a father who was a clerical or sales worker and IADL disabilities. The addition of pathology variables in the final model reduced the strength of the relationship between IADL disabilities and father's education as well as the relationship between IADL disabilities and having a father who was a farmer or farm manager. Also, each additional year of father's education was associated with a reduced risk for IADL disabilities net of the other adult health risks considered.

The addition to the model of social mobility, health behaviors, and pathology accounted for some of the between-person variability in initial status, and the addition of health behaviors and pathology accounted for some of the between-person variability in the rate of change in disability across the survey period. These findings suggest that unhealthy behaviors and pathology in adulthood result in

TABLE 3—Results of Multilevel Models Examining the Relationship Between Instrumental Activities of Daily Living Disabilities and Childhood Socioeconomic Position: Health and Retirement Study, United States, 1998–2006

	Model 1, b (SE)	Model 2, b (SE)	Model 3, b (SE)	Model 4, b (SE)
		Childhood socioeconomic position		
Mother's education, y	–0.04*** (0.00)	–0.01 (0.01)	–0.01 (0.01)	–0.01 (0.02)
Father's education, y	–0.06*** (0.01)	–0.04*** (0.00)	–0.04*** (0.00)	–0.03*** (0.01)
Father's primary occupation				
Professional (manager or administrator; Ref)	1.00	1.00	1.00	1.00
Craftsman	0.08 (0.17)	–0.06 (0.17)	–0.09 (0.18)	–0.13 (0.15)
Farmer/farm manager	0.10*** (0.02)	–0.13* (0.06)	–0.12* (0.06)	–0.09** (0.03)
Clerical/sales worker	–0.24* (0.10)	–0.26* (0.10)	–0.23 (0.12)	–0.17 (0.11)
Operative (machine or transit worker)	0.04 (0.16)	–0.13 (0.17)	–0.19 (0.13)	–0.22 (0.12)
Service worker/laborer	0.12 (0.16)	0.03 (0.18)	–0.02 (0.11)	–0.08 (0.10)
Father never worked/disabled	0.44 (0.53)	0.23 (0.51)	0.19 (0.46)	0.11 (0.44)
Father absent/deceased	0.18 (0.17)	–0.05 (0.20)	–0.09 (0.10)	–0.11 (0.11)
		Adult characteristics		
Social mobility				
Education, y		–0.09*** (0.02)	–0.08*** (0.02)	–0.08** (0.02)
Income (log transformed)		–0.19*** (0.06)	–0.23*** (0.06)	–0.22*** (0.04)
Wealth (log transformed)		–0.07*** (0.01)	–0.08*** (0.01)	–0.07*** (0.01)
Health behaviors				
No history of smoking (vs current smoker)			–0.01 (0.05)	0.03 (0.05)
Former smoker			0.32*** (0.05)	0.26*** (0.05)
Drinks alcohol			–0.14*** (0.02)	–0.46*** (0.02)
Exercises 3 or more times/wk			–0.34*** (0.00)	–0.70*** (0.03)
BMI			0.13*** (0.02)	–0.00 (0.01)
Random effect estimates				
Within person	0.18	0.18	0.18	0.19
Rate of change	0.13	0.13	0.11	0.09
Initial status	24.85	24.32	21.4	17.26

Note. BMI = body mass index (defined as weight in kilograms divided by height in meters squared). The sample size was n = 18 465. Data are weighted. Respondents were interviewed at baseline (1998) and at 2-year intervals thereafter. All models adjusted for age, gender, and race, and model 4 also adjusted for pathology (heart problems, diabetes, lung disease, hypertension, and stroke).
*P < .05; **P < .01; ***P < .001.

variations in levels of IADL disability risk over time (Table 3).

DISCUSSION

In this nationally representative prospective study of community-dwelling older Americans, low childhood SEP was associated with both severe ADL disabilities and moderate IADL disabilities, although the childhood indicators and pathways predicting these disability levels varied over time. Social mobility and health behaviors in adulthood mediated much of the impact of low childhood SEP on severe and moderate disability risk; however, low paternal educational level and having a father who was absent or had died during one's childhood (in the case of ADL disabilities only) remained a significant predictor of disability net of these adult health risk factors.

As such, low paternal educational level and absence of one's father during childhood may have long-term health consequences. Policies aimed at reducing these childhood risks may help decrease the high health care costs associated with severe disablement in old age. Our findings are particularly strong given that we accounted for temporally ordered social and behavioral risks and intra-individual changes in pathology and disability during a period in the life course in which pathology and disability are manifest.

Consistent with the results of traditional disability studies, we found that social mobility, health behaviors, and pathology were important factors in the disablement process, leading to variations in the severity of disability in later life.[1] For example, health behaviors significantly altered the disability trajectories of our participants, accounting for some of the between-person variability in initial status (in terms of both ADL and IADL disabilities) and rate of change (in terms of IADL disabilities only) over the course of the 9-year study period. As such, interventions

aimed at improving health behaviors in adulthood may reduce the prevalence of disability in elderly populations. Most important for this and other life course research, however, our findings suggest that adverse socioeconomic conditions in early life may have long-term effects on disability and that a life course approach may provide additional insight into disablement processes over time.[8,9] Although the findings of life course studies have been mixed,[12,13] most of these studies have reported both direct and indirect pathways between childhood social conditions and adult health outcomes.[11,15,25]

Consistent with the fetal origins hypothesis and biological embedding, individuals whose fathers were absent during their childhood or died during that period may be at risk for severe disability years later.[29,30] The reason may be that childhood is a critical period of development in which access to material and social resources is important for later life health outcomes. Although such hypotheses have largely focused on the role of childhood health, it is likely that childhood health and SEP effects are interwoven.[40] As mentioned, poverty exposes children to a variety of harmful health risks, ranging from environmental pollutants and toxins to poor nutrition and health problems.[19,41]

Low childhood SEP may also initiate a cumulative "chain of risk," predisposing at-risk children to further socioeconomic, behavioral, and pathological disadvantages across the life course.[11,15,42] In this study, social mobility mediated much of the relationship between low childhood SEP and severe disability and was a particularly important factor among respondents whose fathers had worked in manual occupations, had never worked, or had been disabled while they were growing up.

In addition, low childhood SEP may increase the risk for severe disability by initiating a pattern of poor health behaviors. This relationship was most evident among respondents whose fathers were service workers or laborers and respondents whose fathers were absent or had died while they were growing up, possibly because children from these families have restricted access to the information necessary to promote good health.[23,43] As described earlier, poor health behaviors such as physical inactivity and smoking are associated with a number of debilitating conditions in later life, including cardiovascular and lung disease.[4]

Some childhood factors may also have protective effects. For example, we found that each additional year of father's education was associated with a reduced risk for severe and moderate disabilities. Although some studies have reported similar benefits from mother's education,[44] we found that the relationship between mother's education and severe and moderate disabilities was mediated largely by social mobility. Our findings might differ because we accounted for multiple family structures (e.g., absent and deceased fathers) and temporally ordered social mobility indicators across the life course in attempting to capture socioeconomic potential (e.g., education) and the ability to purchase the goods and services necessary for good health (e.g., income and wealth).

We also found that having a father who was a farmer or farm manager or an operative (in the case of ADL disabilities only) was associated with a decreased risk for disabilities. The initial disadvantages associated with these manual backgrounds were mediated by educational level and income in adulthood, suggesting that social mobility may be particularly important in reducing disability risk among these groups. Previous life course research has similarly reported benefits of growing up in a rural area or having a father in a farmer or farm manager position.[16,45]

Limitations

There are several limitations to consider when interpreting our results. First, studies comparing childhood records with reports of childhood SEP in adulthood suggest that retrospective reports may be more favorable.[35] Although we used multiple measures of childhood SEP, our findings may represent an underestimate of the relationship between childhood SEP and disability. Second, poor childhood health may affect a variety of health outcomes,[14,46,47] but the HRS includes only limited measures of childhood health, and we were unable to examine its effects on disability risk.

Third, attrition is a persistent problem in longitudinal studies.[33] Attrition has not significantly influenced the representativeness of the HRS sample in terms of demographic, economic, and health measures.[34] Nevertheless, given that the healthiest adults are more likely to be selected into and remain in the study over time, this study's findings may err toward an underestimation of the relationship between childhood SEP and disability.

Conclusions

In terms of public policy, programs such as Medicaid and the State Health Insurance Program may provide needed health benefits to children and their families and promote good health across the life course. Unfortunately, however, these programs are threatened by rising health care costs, unemployment rates, and poverty rates and declining tax revenues.[48] Even with these programs in place, millions of poor children and adolescents have restricted access to the goods and services needed to maintain and promote health.[49]

Programs aiming to increase access to higher education and promote healthy behaviors across the life course are also needed. Educational loans have helped young adults from poor families afford college tuition. However, these loan programs are also threatened. As such, short-sighted health policies—focused on end-of-life care or treatment of disease—are not likely to be as effective as preventative health care measures geared toward long-term health goals. Reducing SEP differences in health will require policy initiatives that address early-, middle-, and later-life components of socioeconomic status (income, education, and occupation) as well as the pathways through which these factors affect health. ■

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This article was accepted February 20, 2009.

Contributors

M.E. Bowen was responsible for the study design, analysis, and interpretation of findings. She also prepared the article for publication. H.M. González assisted with preparing the article for publication.

Acknowledgments

Mary Elizabeth Bowen is supported by a National Institutes of Health postdoctoral training grant (HS

013819). Hector M. González is supported by a Career Development Award from the National Institute of Mental Health (K08 MH67726). The Health and Retirement Study is sponsored by the National Institute on Aging (grant U01AG009740).

The data used in this article were made available by the Inter-University Consortium for Political and Social Research at the University of Michigan. We acknowledge the anonymous reviewers for their valuable editorial comments.

Human Participant Protection

No protocol approval was needed for this study.

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