

# ADOLESCENT RESILIENCE: A Framework for Understanding Healthy Development in the Face of Risk

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■ **Abstract** Adolescent resilience research differs from risk research by focusing on the assets and resources that enable some adolescents to overcome the negative effects of risk exposure. We discuss three models of resilience—the compensatory, protective, and challenge models—and describe how resilience differs from related concepts. We describe issues and limitations related to resilience and provide an overview of recent resilience research related to adolescent substance use, violent behavior, and sexual risk behavior. We then discuss implications that resilience research has for intervention and describe some resilience-based interventions.

## INTRODUCTION

Resilience refers to the process of overcoming the negative effects of risk exposure, coping successfully with traumatic experiences, and avoiding the negative trajectories associated with risks (43, 65, 72, 84, 106). A key requirement of resilience is the presence of both risks and promotive factors that either help bring about a positive outcome or reduce or avoid a negative outcome. Resilience theory, though it is concerned with risk exposure among adolescents, is focused on strengths rather than deficits. It focuses on understanding healthy development in spite of risk exposure.

The promotive factors that can help youth avoid the negative effects of risks may be either assets or resources (6). Assets are the positive factors that reside within the individual, such as competence, coping skills, and self-efficacy. Resources are also positive factors that help youth overcome risk, but they are external to the individual. Resources include parental support, adult mentoring, or community organizations that promote positive youth development. The term resources emphasizes the social environmental influences on adolescent health and development, helps place resilience theory in a more ecological context, and moves

away from conceptualizations of resilience as a static, individual trait (87). It also stresses that external resources can be a focus of change to help adolescents face risks and prevent negative outcomes.

Adolescents growing up in poverty, for example, are at risk of a number of negative outcomes, including poor academic achievement (2, 96) and violent behavior (34, 37). One approach to understanding why poverty results in negative outcomes is to focus on other deficits to which poverty may be related, such as limited community resources or a lack of parental monitoring. Researchers and practitioners working within a resilience framework recognize that, despite these risks, many adolescents growing up in poverty exhibit positive outcomes. These adolescents may possess any number of promotive factors, such as high levels of self-esteem (21) or the presence of an adult mentor (114), which help them avoid the negative outcomes associated with poverty. Using assets or resources to overcome risks demonstrates resilience as a process. Researchers have also described resilience as an outcome when they identify as resilient an adolescent who has successfully overcome exposure to a risk.

Researchers have suggested that resilience and vulnerability are opposite poles on the same continuum (40), but this may not always be the case. Vulnerability refers to increased likelihood of a negative outcome, typically as a result of exposure to risk. Resilience refers to avoiding the problems associated with being vulnerable. The relationship and distinction between resilience and vulnerability can be depicted in a two-by-two table (104). Table 1 represents four possible combinations of a risk and an outcome. Cell A represents adolescents who are exposed to low levels of a risk factor and who achieve positive outcomes. These adolescents follow trajectories typically considered normative development and are generally not the focus of resilience research. Cell B represents adolescents who are exposed to high levels of risk but who nonetheless achieve positive outcomes. Such adolescents are said to have followed a resilient trajectory. Adolescents in cell C are exposed to low levels of the risk factor and achieve negative outcomes. The adolescents in this cell exhibit an unexpected trajectory. It is likely that these adolescents have been exposed to some risk factor that was either poorly assessed or not measured. Finally, cell D represents adolescents with the expected outcome in risk models because they are exposed to high levels of the risk factor, which results in negative outcomes.

A factor can be considered a risk exposure, or an asset or resource, depending on the nature of the factor and the level of exposure to it. For some constructs, one

**TABLE 1** Depiction of a population of adolescents

	Low risk	High risk
Positive outcome	A (normative development)	B (resilience theory)
Negative outcome	C (inadequate risk assessment)	D (risk models)

Note: Adapted from Reference 104.

extreme may be a risk factor, whereas the other extreme may be promotive. Having low self-esteem, for example, may place an adolescent at risk for developing a number of undesirable outcomes. Having high self-esteem, in contrast, may be an asset that can protect youth from negative outcomes associated with risk exposure. For other constructs, opposite poles may simply mean more or less of the construct. The opposite of positive friend influence is not necessarily bad influence of friends. Rather it may just be limited positive influence of friends. Similarly, involvement in extracurricular or community activities may be related to positive outcomes among adolescents, but this outcome does not mean that not participating in such activities should necessarily be considered a risk.

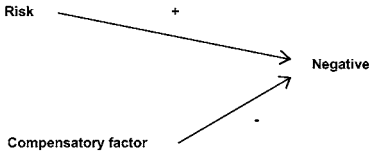
Resilience is sometimes confused with positive adjustment, coping, or competence. Although each of these constructs is related to resilience, they are also distinct. Positive adjustment refers to an outcome of resilience. When youth overcome a risky situation (e.g., the transition to middle school) as evidenced by healthy development (e.g., academic achievement) they have adjusted to their new context. In this case, positive adjustment is a resilient outcome, but the process of overcoming the risk is resilience. Youth may also be considered positively adjusted, however, even though they may not have been exposed to a risk. Resilience processes can have other outcomes as well, such as avoiding a negative outcome or coping successfully with a traumatic event (e.g., the death of a loved one). Resilience is also distinguished from competence. Competence is an asset (i.e., an individual-level promotive factor) that can be a vital component in a resilience process. Competent youth are expected to be more likely to overcome the negative effects of a risk. Competence, however, is only one of many assets that help adolescents overcome adversity. Because resilience models stress the importance of ecological context, external factors in addition to competence may help youth avoid the negative effects of risks.

## Models of Resilience

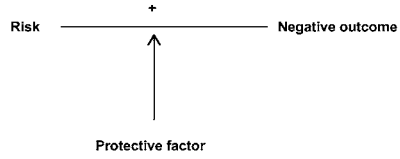
Researchers have identified three models of resilience—compensatory, protective, and challenge—that explain how promotive factors operate to alter the trajectory from risk exposure to negative outcome (43, 84, 113). A compensatory model is defined when a promotive factor counteracts or operates in an opposite direction of a risk factor. A compensatory model therefore involves a direct effect of a promotive factor on an outcome. This effect is independent of the effect of a risk factor (113). Model 1 in Figure 1 depicts how compensatory factors operate to influence outcomes. Youth living in poverty, for example, are more likely to commit violent behavior than are youth not living in poverty (37), but adult monitoring of behavior may help compensate for the negative effects of poverty. This model can be examined using a number of statistical and methodological approaches but is typically tested by examining unique, direct effects in a multiple regression analysis or with structural equation modeling.

Another model of resilience is the protective factor model. In this model, assets or resources moderate or reduce the effects of a risk on a negative outcome.

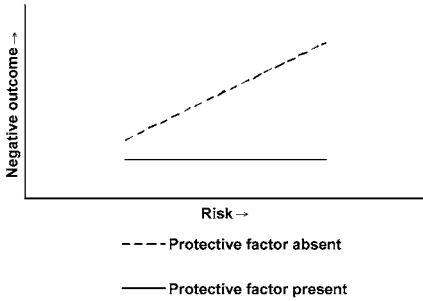
**Model 1: Compensatory**



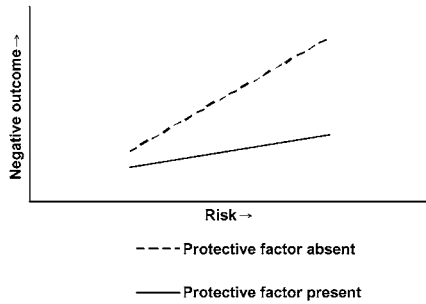
**Model 2: Protective**



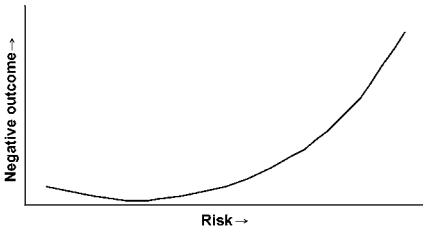
**Model 3: Protective-Stabilizing**



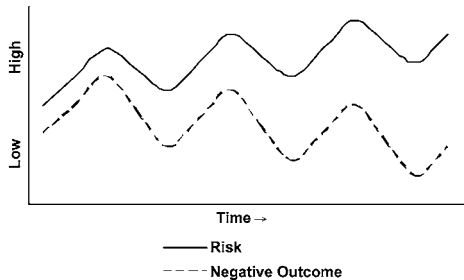
**Model 4: Protective-Reactive**



**Model 5: Challenging**



**Model 6: Inoculation**



**Figure 1** Models of resilience.

A protective model exists if, for example, the relationship between poverty and violent behavior is reduced for youth with high levels of parental support. In this example, parental support operates as a protective factor because it moderates the effects of poverty on violent behavior. Model 2 in Figure 1 shows how a protective factor may influence the relationship between a risk and an outcome. This model can be examined a number of different ways yet is typically tested with an interaction term in multiple regression or with group comparisons in structural equation modeling.

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Protective factors may operate in several ways to influence outcomes. Luthar and colleagues (65), for example, define protective-stabilizing and protective-reactive models. A protective-stabilizing model, depicted in Model 3 in Figure 1, refers to instances when a protective factor helps to neutralize the effects of risks. Thus, higher levels of risk are associated with higher levels of a negative outcome when the protective factor is absent, but there is no relationship between the risk and the outcome when the protective factor is present. Among youth whose parents do not provide adequate support or monitoring (risk factors), for example, those without an adult mentor (a protective resource) may exhibit delinquent behaviors (an outcome), whereas those with a nonparental adult mentor may not.

A protective-reactive model, depicted in Model 4 in Figure 1, refers to instances when a protective factor diminishes, but does not completely remove, the expected correlation between a risk and an outcome. Thus, the relationship between the risk and the outcome is stronger when the protective factor is absent. Adolescents who abuse drugs, for example, may be more likely to engage in sexual risk behavior. The relationship between drug abuse (a risk factor) and sexual risk behavior (an outcome), however, may be weaker among adolescents who are exposed to comprehensive sexuality education in their schools (a protective resource) than among adolescents who do not receive such education.

Brook and colleagues (17, 18) also posit a protective-protective model. In this model, a protective factor enhances the effect of another promotive factor in producing an outcome. Parental support, for example, may enhance the positive effect of academic competence for producing more positive academic outcomes than for either factor alone. Yet, resilience requires the presence of risk, so the protective-protective model may not be a resilience model, unless the two protective factors are studied in a population defined to be at risk for a particular negative outcome (115).

A third model of resilience is the challenge model (43), depicted in Model 5 of Figure 1. In this model, the association between a risk factor and an outcome is curvilinear. This suggests that exposure to low levels and high levels of a risk factor are associated with negative outcomes, but moderate levels of the risk are related to less negative (or positive) outcomes (66). The idea is that adolescents exposed to moderate levels of risk are confronted with enough of the risk factor to learn how to overcome it but are not exposed to so much of it that overcoming it is impossible. A vital point concerning the challenge model is that low levels of risk exposure may be beneficial because they provide youth with a chance to practice skills or employ resources. The risk exposure, however, must be challenging enough to elicit a coping response so the adolescent can learn from the process of overcoming the risk. In challenge models, the risk and promotive factors studied are the same variable—whether it is a risk or is promotive for an adolescent depends on the level of exposure. Too little family conflict, for example, may not prepare youth with an opportunity to learn how to cope with or solve interpersonal conflicts outside of the home. Yet, too much conflict may be debilitating and lead youth to feel hopeless and distressed. A moderate amount of conflict, however, may provide youth with

enough exposure to learn from the development and resolution of the conflict. They essentially learn through modeling or vicarious experience. Challenge models of resilience are typically tested with polynomial terms in multiple regression (e.g., quadratic or cubic terms).

The challenge model of resilience can be considered inoculation or steeling (70, 85, 113) if it includes a developmental (i.e., longitudinal) focus. This model, depicted in Model 6 of Figure 1, suggests that continued or repeated exposure to low levels of a risk factor helps inoculate adolescents so they are prepared to overcome more significant risks in the future. The inoculation model is similar to the challenge model because a factor may be seen as risky when it leads to negative outcomes or promotive when it teaches adolescents to better handle stressors in the future. Yates et al. (112) have described this model of resilience as an ongoing developmental process, in which children learn to mobilize assets and resources as they are exposed to adversity. As youth successfully overcome low levels of risk, they become more prepared to face increasing risk. As people age and mature, and continue to be exposed to adversity, their capacity to thrive despite risks increases. Such models must be tested with longitudinal data. In this way, compensatory, protective, or challenge models can operate within a framework of inoculation, as repeated exposures to compensatory, protective, and/or challenge processes prepare adolescents for dealing with adversities in the future.

## Issues and Limitations

A number of issues related to resilience research have created confusion within the field and fueled criticism of resilience theory. Unfortunately, as several researchers have pointed out (65), differing uses of terminology has seemed to slow down the development of the field, and we need to develop a common language to bring the field to the next level. Some researchers who have criticized resilience research have assumed that resilience is a trait (103). It is vital to note, however, that resilience is not a static trait (58). That is, resilience is not a quality of an adolescent that is always present in every situation. Rather, resilience is defined by the context, the population, the risk, the promotive factor, and the outcome. Thus, the measurement of resilience with a self-report assessment (76, 105) may not be consistent with resilience theory. Part of the confusion may be because some individual-level assets such as self-efficacy, competence, or coping skills may be involved in resilience processes. This should not be interpreted to mean, however, that resilience lies only within the individual or is a static, personal trait. An analytic approach that examines relationships among risk and promotive factors is necessary for understanding adolescent resilience (72, 113).

As a way of stressing that resilience is not a trait, some researchers have suggested that the term resilience be used in place of resiliency (65), a term favored by others in the field. Although this distinction may not be important as both words are synonymous nouns, it is vital to distinguish resilience from a trait-based conception. Further, Luthar & Zelazo (66) point out that the term resilient should

not be used as an adjective describing a person but as a descriptor of profiles or trajectories. This distinction further assures that the construct of resilience is not taken to be an individual trait. The concern in treating or considering resilience as a trait is that it places blame on the adolescent for failing to overcome adversity or risk. It also raises questions about the usefulness of prevention efforts because individual trait-like characteristics may not be amenable to change. Finally, trait conceptions ignore contextual factors, but resilience theory incorporates social and environmental influences.

Another issue to consider is that resilience may be content- and context-specific (26). That is, an adolescent may be resilient in the face of one type of risk but may be unable to overcome other types of risk. Some adolescents, for example, may be resilient against certain negative effects of poverty because they have supportive families, but some of these same adolescents may be less successful overcoming the effects of attending underfunded schools. The risk of an underfunded school may take more than family support to overcome. Researchers have also found that different assets may be associated with different risk and outcome pairings (32, 49). This makes it difficult to identify universal promotive factors and raises concerns that asset lists (7, 60, 73) may be interpreted to operate in the same manner for all groups, all contexts, or all outcomes.

The process of resilience may also vary for different groups of adolescents (28). Resilience for urban and suburban youth, for example, may differ from resilience for rural youth. Similarly, resilience may differ for high- and low-socioeconomic-status youth, for males and females (41), for early adolescents and late adolescents, or for immigrant and nonimmigrant youth. Sameroff et al. (86), for example, describe how parental control may be beneficial in environments characterized by certain risk exposures such as street crime but may be detrimental in environments where such risks do not exist. Similarly, Gutman et al. (49) found among African American adolescents an interaction between number of risk factors and democratic decision making in the family for predicting grade point average and math achievement. Their results indicated that democratic decision making increased the effects of risk factors on the outcomes. This finding suggests that democratic decision making, often considered a resource, may be detrimental in high-risk-exposed environments. This is a critical issue because researchers and practitioners may need to be aware that findings from one context or population may not apply to their given context or population.

Another key point about resilience theory is that, by definition, resilience requires the presence of a risk factor (45). Some have attempted to study resilience among youth not faced with risk (33), but this type of study may be more appropriately defined as research on adolescent development and adjustment more generally. Positive outcomes alone are not sufficient for inferring resilience. Adolescents must have been exposed to some factor or factors (i.e., risks) that increase the likelihood of a poor outcome for promotive factors to be relevant in a study of resilience. Yet, longitudinal research that includes a sample selected on the basis of being at a high level of a risk factor (e.g., poverty) may be problematic because

of the tendency for the sample to regress toward the mean. This phenomenon, and not the presence of a promotive factor, may explain why some youth with a risk factor show fewer negative outcomes over time. In other words, some vulnerable youth may improve simply because of a statistical artifact, regardless of the presence of a promotive factor. Resilience researchers who choose such an approach must be sure to apply designs that will help them eliminate regression to the mean as an explanation of their results.

Adversities facing youth can range from long-term chronic stressors to short-term acute stressors, or to traumatic stressful events (8, 83, 108). Some risk exposures may have immediate, acute effects on adolescents, but the effects may dissipate relatively quickly. Other exposures may not be as dramatic but may be chronic and linger over time. A youth who is HIV positive, for example, faces a number of risk exposures that can lead to poor outcomes. The consistent need to remember to take medications may be considered a long-term chronic stressor, disclosing one's HIV status to a significant other may be a short-term acute stressor, and being hospitalized for a serious opportunistic infection may be a traumatic stressful event. Each of these risk exposures may be responsive to different assets and resources and may be related to different adverse outcomes.

Another issue related to risk exposure is that experiences of the same adverse event or condition may differ across adolescents. For many youth, for example, the divorce of one's parents may be experienced as a negative event. For some youth, however, the same experience may be positive, if it removes family conflict from the home environment (53). Researchers may therefore not always want to assume that because an event is normatively considered negative (or, conversely, normatively considered positive) it is experienced as negative (or positive) by all youth. Researchers may wish to include assessments of how the youth experienced an event in their studies. Failure to consider such a possibility may attenuate research findings, as relationships expected by researchers may operate among some youth in a way that is opposite the hypothesized direction. Similarly, even when an exposure is universally experienced as a risk, the level of adversity may differ. One way to handle this problem is to include measures related to the level of risk exposure in studies. Buckner et al. (21), for example, controlled for variation in experiences of negative events and chronic strains in their study of youth living in poverty. They found that the number of negative events and chronic strains reported was associated with a composite measure of behavior problems, mental health symptoms, functioning and adaptation, and competence.

Resilience research is also somewhat limited because it typically includes single risks and a single promotive factor (111), but most youth are actually exposed to multiple risks, may possess multiple assets, and may have access to multiple resources (45, 86). Several researchers have found that risks (or promotive factors) do not necessarily operate independently in the lives of youth but rather mutually influence each other (49, 65, 74, 75, 86, 94). Masten (71) describes a cascading effect of risks and promotive factors where positive constructs can also be either outcomes or predictors, depending on the situation and when a youth is assessed

(66). A rich understanding of resilience processes therefore necessitates including cumulative risks, assets, and resources studied over time (27, 28, 86, 112).

A final key component of resilience research, though one that is often overlooked, is investigating explanations for how assets or resources interact with risk exposures to produce particular outcomes (65). If researchers find evidence, for example, that parental support (a resource) interacts with negative peer influence (a risk) to predict smoking (an outcome), the next step should be to understand why this is so. The type of parental support provided may be decisive. Parents may provide emotional support necessary to develop the emotional capacity to withstand peer influences, or they may provide informational support related to the health consequences of smoking, increasing the perceived threat of the behavior. Research on the mechanisms by which resilience processes occur, or what Sandler et al. (87) call "small theories," could yield information to be applied in developing interventions. Qualitative studies, like those conducted by Werner and colleagues (106, 107), may also help to answer such questions.

## SELECTED RESEARCH FINDINGS ON ADOLESCENT RESILIENCE

Research on resilience has grown exponentially in the past 10 years. A simple Medline search using PubMed and the key words adolescence, adolescent, resilience, resiliency, and protective factors produced 49 citations from 1975 through 1984, 206 citations from 1985 through 1994, and 756 citations from 1995 through 2004. Consequently, we focus our review on recent articles investigating substance use (alcohol, tobacco, and other drugs), violent behavior, and sexual behavior. We chose these outcomes for several reasons. First, most research on adolescent resilience focuses on psychopathology (75), rather than behavior. Second, these three behaviors pose considerable health risks to adolescents and play a significant role in adolescent development. Finally, these behaviors may be particularly amenable to public health intervention.

### Substance Use

Researchers have found a number of assets and resources that may compensate for or protect against risks for substance use at the individual, peer, family, school, and community levels. Researchers have found adolescents to be protected from the substance use consequences of stressful or negative life events by assets such as self-esteem (22), internal locus of control (92), positive affect (92), and religiosity (110). Wills et al. (109) found among 1702 adolescents followed from age 12 to age 15 that positive affectivity, or feeling happy, interested, and relaxed, was protective against the risk of emotional distress for cigarette, alcohol, and marijuana use. Resources that have been found to compensate for the effects of emotional distress include family connectedness (42, 63) and parental involvement with school (42).

Similar promotive factors, including planning to attend college (19), and resources such as family connectedness (42, 63) and parental involvement with school (42) have been found to compensate for the effects of delinquent behavior on substance use. Scheier et al. (91) found three assets to compensate for the effects of risk-taking on alcohol use among adolescents: self-control, substance-use refusal skills, and academic achievement. Psychological well-being and social competence (47) compensated for the effects of prior cigarette, alcohol, and marijuana use for predicting current use among 1184 junior high school students in New York City. Academic achievement is a consistent protective factor for substance use. This asset helps protect against the risks of low academic motivation (20) and age-related increases in substance use (19). Parental support resources protect youth from the risks of acculturation (50) and low ethnic identification (16, 93) for substance use.

Individual-level assets and family-level resources are consistent promotive factors for substance-use risks associated with peer influences. Participation in extracurricular and community activities (31) have compensated for the negative influences of peer tobacco, alcohol, and illegal drug use. Decision-making skills (12) and positive orientation toward school (30) have also protected youth from the negative effects of peer substance use. Legitimization of parental authority (57), family connectedness (63), parental monitoring (81), and open communication with parents (100) are resources that appear to compensate for peer substance use. Parental support may also protect against the negative effects of peer substance use (39, 59), peer pressure (39), and age-related increases (90). Similarly, decision-making skills (12) protect against having peers with favorable attitudes toward substance use for alcohol and marijuana use.

Parental substance use is also a significant risk factor for adolescent substance use. Among personal assets, social competence helps compensate for the risk posed by parental use (44), and religiosity helps protect youth from the adverse effects of parental substance use on their own use (15). Family connectedness (63) and parental authority (57) are resources that protect youth from the negative influence of parental substance use. Decision-making skills (12) have also protected youth from the negative effects of parental permissiveness on alcohol and marijuana use.

Family connectedness compensated for the risk of low school connectedness on cigarette smoking in a nationally representative sample (63). Parental support protects against the community-level risk factors of drug availability and low community norms for family closeness (16) on marijuana use. Family income has also moderated the relationship between neighborhood problems and adolescent alcohol and marijuana use (35). Higher family income protected youth from adverse neighborhood effects.

Most studies include analysis of one risk and promotive factor at a time, but other approaches are to study multiple risks and promotive factors or to combine multiple risk and promotive factors to form cumulative measures. Researchers have studied cumulative risk measures and adolescent substance use, both with single assets or resources and with cumulative promotive measures. Scal et al. (89), for example, investigated the effects of different combinations of assets and resources

for smoking in the presence of a number of risks at the individual, peer, and parental levels. They found that religiosity, academic achievement, family connectedness, and parental education expectations all compensated for the effects of the risks. Other researchers studied cumulative risk measures with cumulative promotive measures. Cumulative measures made up solely of resources (5) and made up of assets and resources (38, 79, 92, 102) have been protective against cumulative risk measures.

## Violent Behavior

Empirical evidence also supports the compensatory and protective models for adolescent violent behavior. Assets that have compensated for individual-level risk factors include prosocial beliefs compensating for antisocial socialization (56), religiosity compensating for interest in gang involvement (4), and anger control skills compensating for risk-taking behavior (48). Two dimensions of racial identity, public regard and centrality, are assets that Caldwell et al. (23) found to protect against the effects of racial discrimination on violent behavior among 325 African American adolescents studied from ages 14 to 20. Maternal support has both compensated for and protected against the risk factor for violent behavior of getting in a fight, whereas paternal support has been protective (116). Finally, the resource parental monitoring has compensated for the effects of risk-taking behavior on violent behavior (48).

Peer behaviors and attitudes may also pose a risk for violent behavior that promotive factors may compensate for or protect against. Anger-control skills compensate for the effects of peer delinquent behavior for predicting adolescent violent behavior (48). Perceived social status was found to moderate (i.e., a protective factor) the relationship between peer delinquent behaviors and adolescent violent behavior (80). Parental monitoring was also a compensatory factor (48). Adolescents' religiosity also compensated for the risk of peer substance use (55) and exposure to violence for violent behavior (4). Parental factors are also consistent resources to help youth overcome risks for violent behavior. Maternal support protected youth from the negative influences of peer violent behavior (116). Parental monitoring and paternal support were found to compensate for peer violent behavior (55, 116). Parental monitoring also compensated for the risk of living in a risky neighborhood (48). Maternal and paternal support also compensated for and protected youth from the negative consequences of exposure to violence (116).

Researchers have also found assets and resources that compensate for cumulative risk factors for violent behavior. Borowsky et al. (9) found among 13,781 seventh- through twelfth-grade adolescents studied over two years that academic performance, parental presence, parent-family connectedness, and school connectedness, alone and in combination, compensated for the cumulative effects of prior violent behavior, violence victimization, substance use, and school problems on violent behavior. Other researchers have found that cumulative measures of assets and resources compensate for cumulative risk factors (79, 101).

## Sexual Behavior

Sexual behavior among adolescents includes initiation of sex, level of sexual activity, and risky sexual behavior. Substance use is an individual-level risk factor for adolescent sexual behavior that is compensated for by personal assets such as self-esteem (78), participation in extracurricular activities (1), school achievement and attachment (62, 67, 78, 88), religiosity (62, 67), HIV and reproductive health knowledge (67), positive attitudes toward condoms (69), safer sex intentions (69), seeing sex as nonnormative (88), and self-efficacy to refuse drugs and use condoms (88). Resources that have compensated for substance use in predicting sexual behavior include father's education (1), teacher support (1), residence with both parents (1, 62), peer norms for sexual behavior (3), and family socioeconomic status.

Family socioeconomic status (68), parental monitoring (81), and open parental communication (100) have compensated for the risk of peer sexual behavior for adolescent sexual behavior. Paul et al. (78) reported for their 21-year longitudinal study of 1020 participants in New Zealand that school attachment and self-esteem helped compensate for the risk of sexual intercourse before age 16 associated with mothers having had a child before the age of 20. Participation in extracurricular activities and community organizations has also helped counteract the effects of neighborhood poverty on a composite measure of adolescent sexual risk behavior in a study of 370 urban African American adolescents (82).

## Research Findings Summary

Across most risk factors for adolescent substance use, violent behavior, and sexual behavior, parental factors seem to be particularly vital in helping youth be resilient. The compensatory model appears to have more empirical support, but for substance use and violent behavior, several promotive factors are also protective. To date, researchers have not yet tested the challenge or inoculation models for these outcomes.

One limitation in the research literature on adolescent resilience is that most studies focus on individual assets and family-level resources. Research that examines adolescent resilience with the help of school and community-level resources would be useful. Another limitation of this literature is the almost complete reliance on cross-sectional research (1, 3–5, 12, 15, 16, 22, 38, 39, 48, 55, 57, 59, 62, 63, 67–69, 79–82, 92, 102, 116). The studies that are longitudinal typically include only two time points (9, 23, 42, 50, 88, 89). It is necessary to include many waves of observation over longer periods of time to understand more completely the developmental factors associated with resilience processes for adolescent substance use, violent behavior, and sexual behavior.

Although the research described provides empirical support for the resilience models described, the researchers did not necessarily use resilience theory to guide the analyses. Rather, they found that positive factors (what we have called promotive factors) counteract (compensate) or moderate (protect) against risks youth

face. More research that specifically applies resilience theory and tests the models within it will help us further understand how resilience processes operate to help youth overcome the risks they face.

Notably, most research on resilience has focused on either nationally representative samples (9, 19, 35, 63, 89), predominantly white youth (42, 62), or predominantly African American samples (3, 20, 23, 39, 48, 55, 57, 81, 82, 88, 93, 100, 102, 116). Research that focuses on other ethnic groups, such as Latino, Native American, or Arab American youth, or on recent immigrants, would further our understanding of resilience among adolescents. In addition, there are virtually no studies of resilience for gay, lesbian, bisexual, or transgendered youth, leaving a significant void in the literature.

## RESILIENCE-BASED INTERVENTIONS

The concept of resilience and its associated evidence suggest several implications for prevention. A key idea is that interventions may need to focus on developing assets and resources for adolescents exposed to risk (26, 64, 112) instead of the more traditional approach of focusing on risk amelioration. The educational and ecological assessment phase of the widely used health planning model PRECEDE/PROCEED (46), for example, calls for practitioners to catalog the predisposing, enabling, and reinforcing factors associated with the behavior targeted for change. The usual practice is to list deficits that predispose, enable, and reinforce some negative behavior. A resilience approach, however, emphasizes assets and resources as the focus for change. Internal assets that may be particularly critical to develop include social skills for relating to peers, self-efficacy for health-promoting behavior, academic skills, and participation in extracurricular and community activities.

Botvin and colleagues (11, 13, 14) have suggested that skill building for life in general, such as the development of generic social and problem-solving skills, can be just as important as building skills for risk avoidance. External resources that may be developed include opportunities for adult mentorship (51, 114), parenting skills (61), and provision of health-promoting settings for adolescents (36). Another key idea is that, because of the multidimensional nature of resilience, interventions that cut across behaviors may be most effective. Interventions that focus solely on substance use avoidance, for example, may be too narrowly focused to alter the entire context of influences in adolescents' lives. Yet, it may be critical for practitioners to focus attention on those assets and resources that have been found to promote healthy outcomes in their particular populations.

A number of interventions include development of assets and resources in adolescents' lives. Life Skills Training (10, 13) is a classroom-based program that focuses on general adolescent skill development and on developing skills for resisting social influences to use substances. The intervention includes a number of activities such as demonstration, role-playing, and behavioral homework assignments. This intervention's focus on cognitive-behavior skills related to building

self-esteem, decreasing anxiety, communicating effectively, developing relationships with others, and asserting rights suggests a resilience approach because it focuses on vital individual assets for healthy and effective social interaction. These skills are assets that can counteract risks for a variety of outcomes. The Resourceful Adolescent Program (RAP) (95) is another individual-level intervention focused on enhancing adolescents' skills and social resources. It includes sessions on affirming participants' strengths, learning skills for handling stress, developing social support networks, and conducting interpersonal relationships with others, including family members.

Several interventions focus on families as a way to develop both assets and resources. The RAP (95), for example, includes three sessions for participants' parents, with a similar focus as in the adolescent sessions. The Multidimensional Family Prevention project (54) trains counselors to visit participating inner-city families in their homes and to work with the families to identify their existing assets and resources. The program helps the adolescent and parent develop new skills to communicate more effectively in general and with each other. It is also designed to help both parents and youth engage more effectively in their interactions in the community. The Preparing for the Drug Free Years (PDFY) and Iowa Strengthening Families (ISF) programs (97–99) similarly focus on parental skills and adolescent prosocial and peer-pressure resistance skills. PDFY is an intervention with parents of sixth graders; it teaches them how to enhance their relationships with their children, develop appropriate monitoring practices, and manage anger and conflict within the family. Children are included in one of the intervention's sessions. The ISF program includes both parents and children; the parenting content is similar to the PDFY program, and the adolescent content focuses on peer resistance and relationship skills. In some of the sessions, parents and children are brought together to practice the skills they have been learning about. These programs are examples of employing a resilience approach because they focus on building positive relationships as a way to prevent negative outcomes, and they stress the importance of family members as resources for healthy adolescent development. In contrast, a more traditional approach may focus on reducing or eliminating the negative factors in youths' lives.

Some family-based interventions focus on particular racial or ethnic groups so that the intervention stresses risks, assets, and resources unique to the group. The Flint Fathers and Sons program (24, 25) is a family-based intervention focused on strengthening father-son relationships among African American participants. It involves family members in activities to learn skills (e.g., communication skills), participate in community and school activities, and enhance cultural pride and racial/ethnic identity. The focus of the intervention is to prevent or reduce substance use, violent behavior, and sexual risk behavior among the fathers and sons. The Adolescent and Family Rites of Passage program (52) is a similar intervention for African American adolescent males in Washington, D.C., that includes after-school activities, family enhancement, and empowerment activities. The activities include elements of African culture and aim to foster self-esteem, positive peer

relationships, and interpersonal skills among the adolescents. It also includes programs for parents to enhance parenting skills, parent-child bonds, and participation in school and the community. Finally, Familias Unidas is a family-centered intervention for immigrant Latino families in South Florida (29, 77). This program focuses on parents and begins with the development of small parental support networks, which then develop and plan the remaining activities, including family meetings, home visits, parent-child discussion sessions, activities with adolescents, activities with adolescents and peers, meetings with school counselors, and family therapy. These three programs are examples of connecting parents and children in constructive ways so they are both more prepared to address risks for which adolescents are inevitably exposed. Their focus on youths' assets and family resources suggests they use a resilience approach.

## CONCLUSION

The goal of this review is to help provide a common language and understanding to conduct research and interventions that focus on assets and resources. Resilience models help us understand why some youth exposed to risks are able to overcome them and avoid negative outcomes. Although assets and resources that help youth overcome the adverse effects of risks may differ by outcome, context, and population studied, several common themes do emerge. Parental factors are consistent and critical resources for youth. These factors include support, monitoring, and communication skills. Youth who have self-confidence and social skills also are somewhat predisposed to being resilient regardless of the risk or outcome. Nevertheless, it is vital that public health interventions that use a resilience approach pay particular attention to the unique features of the population of interest and the context in which the approach is employed. Resilience theory provides researchers and practitioners with a conceptual model that can help them understand how youth overcome adversity and how we can use that knowledge to enhance strengths and build the positive aspects of their lives.

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## WHY DO POOR PEOPLE BEHAVE POORLY? VARIATION IN ADULT HEALTH BEHAVIOURS AND PSYCHOSOCIAL CHARACTERISTICS BY STAGES OF THE SOCIOECONOMIC LIFECOURSE

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**Abstract**—Attempts to explain socioeconomic inequalities in health have often made reference to the observation that poor health behaviours and psychosocial characteristics cluster in low socioeconomic status (SES) groups. Causal interpretation of the association between SES, health behaviour, psychosocial orientations, and health inequalities has been hampered because these factors and SES have usually been measured at the same point in time. Data from the Kuopio Ischaemic Heart Disease Risk Factor Study were used to examine the associations between measures of SES reflecting different stages of the lifecourse, health behaviours, and psychosocial characteristics in adulthood in a population-based study of 2674 middle-aged Finnish men. Results show that many adult behaviours and psychosocial dispositions detrimental to health are consistently related to poor childhood conditions, low levels of education, and blue-collar employment. Poor adult health behaviours and psychosocial characteristics were more prevalent among men whose parents were poor. Increases in income inequality which place children in low SES conditions may well produce a negative behavioural and psychosocial health dividend to be reaped in the future. Understanding that adult health behaviour and psychosocial orientations are associated with socioeconomic conditions throughout the lifecourse implies that efforts to reduce socioeconomic inequalities in health must recognize that economic policy is public health policy. © 1997 Elsevier Science Ltd. All rights reserved

**Key words**—socioeconomic status, lifecourse, health behaviour, psychosocial factors

The existence of socioeconomic inequalities in health has been well established. Attempts to explain these inequalities have often made reference to the fact that behavioural factors, such as smoking, physical activity, and diet, are differentially distributed by socioeconomic levels. In addition, psychosocial characteristics, such as depression and cynical hostility, have also been considered as potential explanations for why people at the bottom of the social hierarchy have poorer health [1]. Indeed, considerable evidence has accumulated concerning an inverse relationship between socioeconomic status (SES) and the behavioural and psychosocial characteristics which are important risk factors for poor health. Studies in a variety of industrial countries have shown that lower SES is generally associated with higher rates of smoking [2–5], obesity [6–8], poorer dietary habits, [9–11] lower levels of physical activity [12–15], and higher prevalence of psychosocial orientations that are related to poor health outcomes [16–18], while a more complex relationship between SES and

alcohol consumption has been reported [5, 19, 20]. Furthermore, those who occupy lower positions in the socioeconomic hierarchy have also been shown to be more resistant to changing risk behaviours than their more advantaged counterparts [21, 22].

What part should health behaviours and psychosocial orientations play in understanding the graded association between lower SES and poorer health, and informing intervention efforts [23, 24]? Both the explicit and implicit models of behavioural and psychological change that have been used in public health can be broadly classified according to how much emphasis they place on the role of human volition. One model is strongly based on the premise that adult health behaviours are largely intra-individual phenomena which reflect some process involving free choice. This approach, which views unhealthy behaviours as the consequence of poor lifestyle management, gained credence as risk factor epidemiology confirmed that many poor health outcomes were associated with the daily conduct of people's lives [25]. Knowles' ideas on individual responsibility for health [26] provided the sort of theoretical foundation from which the U.S. Surgeon

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General, and his counterparts in other countries, could later chastise their populations for poor health habits and point out the costs to society of such behavioural transgressions [27].

There are many current examples that, at least implicitly, reflect this thinking about SES, behaviour, and disease [28]. McGinnis and Foege [29] present an analysis of U.S. data to demonstrate what they characterize as the "...root determinants of death and disability" (p. 2211). They report that tobacco, alcohol, diet and physical activity are the major contributors to mortality before age 75. While they also point out that SES is an important factor in premature mortality, they argue that it is difficult to quantify the SES effect independent of smoking, alcohol, diet and physical activity, and they conclude that these public health problems are squarely based in behavioural choices.

The other model of health behaviour agrees that while individuals make choices about how they act, those choices are situated within economic, historical, family, cultural and political contexts. According to this view, these contexts exert important influences on both the process of choice and the types of behavioural options which are available and, indeed, appropriate. A number of authors have argued that decontextualizing behaviour from this real-world setting obscures its socioeconomic production and encourages blaming the victims of inequality for their unhealthy lifestyles [30–33]. According to this approach, evidence that health behaviours are differentially distributed by SES should be viewed in a lifecourse perspective, as the cumulative responses of different classes of people to conditions imposed by the social structure [34–36].

The degree to which differences in psychosocial orientations and health behaviours contribute to socioeconomic inequalities in health has usually been assessed in regression models in which the relationship between adult SES and a health outcome is adjusted for a variety of adult risk factors. In other analyses of the same population as the current study, we have shown that statistical adjustment for smoking, physical activity, and alcohol consumption reduced the excess relative hazard of all-cause mortality in the lowest quintile of income earners by 35%, by 50% for cardiovascular mortality, and by 34% for incident myocardial infarction. Similar adjustment for a number of psychosocial orientations, such as depression and hopelessness, decreased the excess relative hazard for the lowest income quintile by 48% for all-cause mortality, 57% for cardiovascular mortality, and by only 3% for incident infarctions [37]. While there is no method to quantify the degree of confounding in such studies, it is commonplace to claim that because only a portion of the increased risk is "explained" by the covariates, there remains some residual "independent" effect of SES. One impli-

cation of this approach would be that if 100% of the increased risk was accounted for by statistical adjustment of covariates, then the effect of SES would be "explained". There are both methodological and conceptual difficulties with this line of reasoning.

A number of studies have demonstrated that in multivariate models containing large numbers of correlated confounders measured with error, both the direction and magnitude of bias in the variable of interest are unpredictable [38–40]. In such circumstances, it is possible that the elevated or diminished risk estimates for SES, observed after adjustment for behaviours and psychosocial characteristics, may be at least partly due to imprecise measures of those factors. Thus, the widespread practice of declaring that any suspected etiologic factor is independent of another may be potentially misleading, unless the underlying measurement error structures of the variables have been assessed. Unfortunately, data of this type are almost never available in epidemiologic studies.

Much more important than the technical difficulties of interpreting such analyses is that they are conceptually uninformative. Most often, SES, health behaviours, and psychosocial characteristics are assessed at the same point in time, making it impossible to disentangle the temporal sequencing of SES and these factors. For instance, if social class position in childhood and educational experiences were important in the adoption and maintenance of adult health behaviours, or influential in the development of psychosocial orientations, then it would be inappropriate to "adjust" for these variables, because the SES exposure would be temporally prior to the behaviours, and so the behavioural and psychosocial characteristics would be in the causal pathway. Consequently, "explanations" of the relationship between SES and health in terms of behaviour and psychosocial characteristics would be limited to describing some pathways through which SES impacts health [37]. In order to sort out these temporal sequences, long-term studies are needed in natural population settings which can trace the development of health behaviours and psychosocial orientations over the entire lifecourse.

However, as data of this type are rarely available, we hope to advance understanding of the relationship between behaviours, psychosocial characteristics, and socioeconomic health inequalities by examining their associations with SES at three temporally distinct stages of the lifecourse—during childhood (measured by parental SES at age 10), adolescence (measured by education) and adulthood (measured by occupation). While many earlier studies have shown associations between adult SES and a limited number of health behaviours or psychosocial orientations, this is the first to simultaneously examine in the same population-based

cohort the SES patterns for a comprehensive array of important behavioural and psychosocial factors, including smoking, alcohol consumption, drunkenness, obesity, physical activity, depression, cynical hostility, hopelessness, sense of coherence, and dietary intake of fruit, vegetables, fat, salt, coffee and vitamins. Moreover, to our knowledge this is the also the first study to examine the SES patterns of these adult behaviours and psychosocial orientations to SES during childhood.

## METHODS

### *Setting*

Kuopio is the major provincial centre in eastern Finland, with about 600,000 people in its sphere of influence. The city and suburbs of Kuopio have a population of 85,000, making it Finland's eighth largest city. The economic base of the region is dominated by wood processing industries, farming and food processing, clothing manufacturing, metal industries, and service industries. The largest individual employers are the regional government, the university and its hospital.

### *Study population*

Subjects were participants in the Kuopio Ischaemic Heart Disease Risk Factor Study, which was designed to investigate previously unestablished risk factors for ischemic heart disease, carotid atherosclerosis and other related outcomes in a population-based sample of Eastern Finnish men [41]. The study population consisted of a 33% random sample of men aged 42 ( $n = 334$ ), 48 ( $n = 356$ ), 54 ( $n = 1589$ ) and 60 ( $n = 398$ ) who resided in the town of Kuopio or its six adjacent rural communities. Of the 3343 eligible men, 198 were not included because of death, serious disease or migration away from the area, and of the remainder, 2682 (82.9%) agreed to participate in the study. Baseline examinations were conducted between March 1984 and December 1989. No marked socio-demographic differences have been found between participants and non-participants [42]. Men were evenly employed in blue- (44%) and white-collar occupations (40%), while 16% were engaged in farming or forestry. Seventy one percent resided in the city of Kuopio or its suburbs, while 30% of men lived in more scattered rural dwellings. Approximately 87% were married, 6.5% were widowed or divorced, while 7% had never married.

## MEASURES

### *Socioeconomic status in childhood, adolescence and adulthood*

At the baseline examination, individuals reported various aspects of their childhood at the age of 10 years, such as their parents' sociodemographic

characteristics, home environment and childhood experiences. An index of childhood socioeconomic conditions was based on six items—father's education and occupation, mother's education and occupation, whether or not the family lived on a farm, the size of that farm, and the degree to which their family was perceived as wealthy. These six items were dichotomized and the scores summed to form an index of childhood socioeconomic conditions. Subjects were classified according to approximate index tertiles as either high ( $n = 557$ ), medium ( $n = 1198$ ) or low ( $n = 928$ ) childhood socioeconomic status. In previous analyses, this scale has been shown to be predictive of an indicator of coronary heart disease [43].

In addition, participants completed detailed questionnaires which included items on education and lifetime occupation. In the present analyses, we used information on the highest level of education received to indicate SES during adolescence and early adulthood. Participants were grouped into those who had received "primary schooling or less" ( $n = 1555$ ), "some high school" ( $n = 939$ ), and those who had "completed high school or better" ( $n = 180$ ). Socioeconomic status in adulthood was measured by occupational type and classified as "farmer" ( $n = 425$ ), "blue-collar" ( $n = 1168$ ), or "white-collar" ( $n = 1042$ ). Both the education and occupation measures have been demonstrated to be associated with atherosclerosis, acute myocardial infarction, and mortality in this population. [44].

### *The adult socioeconomic context*

A variety of other measures which more fully characterized the economic context of different adult SES positions were available for this analysis. These included current income, housing tenure, financial and job insecurity, unemployment, history of work injury, and disability or early retirement. In addition there were 13 questions concerning ownership of material possessions (colour TV, dishwasher, car, telephone, etc.) A summary index of material living conditions was created by counting the number owned [45].

### *Adult health behaviours and psychosocial characteristics*

Extensive behavioural risk factor information was collected as part of the baseline examinations. Smoking was measured by questionnaire and is classified for this analysis as "never smoked", "former smoker", and "current smoker" (measured in pack-years). Alcohol consumption was assessed by dietary record for a four-day period and also for the previous 12 months by self-administered questionnaire [46]. The alcohol consumption distribution was divided into quartiles and a separate category created for abstainers. In addition, participants also reported how often they were drunk. Physical activity was assessed by a 12-month leisure-time his-



Table 2. Adult risk factor behaviour (age-adjusted means or age-adjusted proportion) by stage of the socioeconomic lifecourse

Socioeconomic lifecourse stage	Cigarette smoking			Alcohol		Drunkenness			Physical activity <sup>b</sup>		Obesity	
	% Non-smoker	% former smoker	Mean pack-yrs current	% Abstainer	% High quartile consume	% Drunk > 2-3 times per month	% No condition. activity	% Low quartile condition. activity	% High quartile BMI (≥29)			
Poor (n = 928)	27.7	38.6	176.3	11.7	22.0	19.6 <sup>a</sup>	16.3	29.9 <sup>a</sup>	22.4			
Middle (n = 1198)	25.6	42.2	164.6	14.1	24.3	19.4	17.0	30.4 <sup>a</sup>	25.2			
High (n = 557)	27.5	41.6	147.0	10.6	22.4	14.7	15.9	24.2	22.8			
Primary or less (n = 1555)	22.2 <sup>a</sup>	41.5	195.0 <sup>a</sup>	14.2 <sup>a</sup>	24.2	21.6 <sup>a</sup>	18.7 <sup>a</sup>	35.2 <sup>a</sup>	26.2			
Some high school (n = 939)	31.8	40.8	134.8	11.5 <sup>a</sup>	21.5	15.3 <sup>a</sup>	15.9	22.9 <sup>a</sup>	21.7			
Finished high or better (n = 180)	32.1	39.5	104.4	5.5	22.2	8.7	10.8	15.9	21.0			
Farmer (n = 425)	40.0 <sup>a</sup>	31.1 <sup>a</sup>	159.2 <sup>a</sup>	21.0 <sup>a</sup>	13.2 <sup>a</sup>	9.0	18.5 <sup>a</sup>	45.0 <sup>a</sup>	25.0			
Blue-collar (n = 1168)	20.3 <sup>a</sup>	41.7	218.5 <sup>a</sup>	14.0 <sup>a</sup>	26.2	24.1 <sup>a</sup>	21.0 <sup>a</sup>	32.4 <sup>a</sup>	24.6			
White-collar (n = 1042)	30.1	41.2	129.7	8.2	22.9	14.7	11.4	19.8	23.2			

<sup>a</sup>Differences between specified category and highest category (e.g. high childhood or white-collar) significant at  $P < 0.05$ .

<sup>b</sup>Energy expenditure in conditioning physical activities such as jogging, swimming, cycling and skiing.

likely to have been unemployed in the previous five years, and had work injury rates which were 250% higher.

*Socioeconomic lifecourse stage and adult smoking, alcohol consumption, physical activity and obesity*

Table 2 shows the age-adjusted proportions of men at each stage of the socioeconomic lifecourse who were non-smokers, former smokers and current smokers (age-adjusted mean level in pack-years), who were abstainers from alcohol consumption, or who were in the highest 25% of consumers, and those who reported being drunk more than two to three times each month, who were either completely physically inactive in their leisure-time, or who were in the lowest 25% of energy expenditure in such activities, and who had BMI scores above 29 (highest 25%).

While there were no statistically significant differences in smoking behaviour across levels of childhood SES, the pattern of associations demonstrated that those with the poorest starts in life were more likely to have the highest pack-years of exposure to current smoking. Smoking was strongly associated with education and occupation. The least educated group had significantly fewer non-smokers (22.2%), and the highest mean levels of current smoking (195.0 pack-years). Blue-collar workers had the lowest rates of non-smoking (20.3%), and the highest mean exposure for current smokers (218.5 pack-years).

The amount and pattern of alcohol consumption presented interesting differences at each stage of the socioeconomic lifecourse. Across levels of childhood SES there were no differences between the proportions of men who abstained, or were in the highest 25% of alcohol consumers. However, those who were born into high childhood SES were significantly less likely to report frequent bouts of drunkenness than men from less advantaged homes. When examined according to educational level, those who received only a primary school education were the most likely to be abstainers, but men who consumed alcohol in this group were 2.5 times more likely to report frequent episodes of drunkenness than those who received more than a high school education. Farmers were the most likely to abstain from alcohol (21.0%) and least represented in the highest quartile of consumption. Blue-collar workers reported significantly higher levels of frequent drunkenness (24.1%).

Men born into the most advantaged conditions were significantly less likely to be in the low quartile of conditioning physical activity. Men in the least educated group were significantly more likely to be among those who reported both no activity (18.7%) or being in the lowest 25% of energy expended in conditioning leisure-time physical activity (35.2%). In terms of the occupational association with leisure physical activity, both farmers and blue-collar

Table 3. Adult Diet (age-adjusted mean or age-adjusted proportion) by stage of the socioeconomic lifecourse (also adjusted for total energy intake)

Socioeconomic lifecourse stage	Fruit gm/day	Non-root veges. gm/day	Total fat gm/day	Saturated fat gm/day	Cholesterol mg/day	Salt mg/day	Vitamin C mg/day	Carotene ug/day	% High tertile coffee
Poor (n = 928)	98.0 <sup>a</sup>	83.0 <sup>a</sup>	113.1	55.5	452.8	3433.6 <sup>a</sup>	85.9 <sup>a</sup>	2453.2 <sup>a</sup>	36.9 <sup>a</sup>
Middle (n = 1198)	124.7	89.5 <sup>a</sup>	111.6	54.2	455.1	3302.0	89.6	2666.8	33.8 <sup>a</sup>
High (n = 557)	135.2	97.4	111.9	54.7	440.1	3248.9	93.2	2757.0	27.2
Primary or less (n = 1555)	99.9 <sup>a</sup>	80.4 <sup>a</sup>	113.1 <sup>a</sup>	56.1 <sup>a</sup>	456.8	3412.0 <sup>a</sup>	82.8 <sup>a</sup>	2277.3 <sup>a</sup>	37.6 <sup>a</sup>
Some high school (n = 939)	133.6 <sup>a</sup>	94.9 <sup>a</sup>	111.7	53.8 <sup>a</sup>	446.5	3269.7	93.3 <sup>a</sup>	2914.4	29.3
Finished high or better (n = 180)	163.1	118.4	110.1	51.8	442.8	3188.4	111.8	3309.5	22.3
Farmer (n = 425)	97.7 <sup>a</sup>	73.5 <sup>a</sup>	113.0 <sup>a</sup>	59.5 <sup>a</sup>	456.4	3304.7	83.0 <sup>a</sup>	2217.3 <sup>a</sup>	45.2 <sup>a</sup>
Blue-collar (n = 1168)	103.0 <sup>a</sup>	79.6 <sup>a</sup>	113.3 <sup>a</sup>	55.2 <sup>a</sup>	449.4	3392.6 <sup>a</sup>	82.6 <sup>a</sup>	2298.0 <sup>a</sup>	38.3 <sup>a</sup>
White-collar (n = 1042)	141.9	103.7	111.3	53.0	453.3	3276.5	98.2	3076.1	26.4

<sup>a</sup>Differences between specified category and highest category (e.g. high childhood or white-collar) significant at  $P < 0.05$ .

workers were over-represented in the inactive and barely active groups. There was no clear pattern for obesity by childhood SES, but the least educated men were more likely to be obese adults, although the graded association was not statistically different.

#### Socioeconomic lifecourse stage and adult diet

Table 3 presents age and energy intake-adjusted mean levels of various dietary factors (age-adjusted proportion for coffee consumption) at each stage of the socioeconomic lifecourse. There were significant graded differences across levels of childhood SES for consumption of fruit, non-root vegetables, salt, vitamin C, carotene and coffee. Men born into the poorest childhood circumstances had 28% lower intake of fruit, 15% lower non-root vegetables, 12% lower carotene, 8% lower vitamin C, 6% higher levels of salt and were more likely to be heavy coffee consumers than those most advantaged during childhood. These differences became even more striking when examined by subsequent educational experience. Significant graded differences were evident for fruit, non-root vegetables, total fat, saturated fat, salt, vitamin C, carotene and coffee intake, with the least educated group having the poorest overall dietary profile. Men with only primary education consumed 39% less fruit, 32% fewer non-root vegetables and 8% more saturated fat compared to men with more than a high school education. White-collar workers had significantly better overall dietary profiles than farmers or blue-collar workers, due to significantly higher intakes of fruit, non-root vegetables, vitamin C and carotene, but lower consumption of total and saturated fat, salt, and coffee.

#### Socioeconomic lifecourse stage and adult psychosocial characteristics

Table 4 shows the age-adjusted proportions of men in the highest tertiles of hopelessness, and quartiles of depression, and cynical hostility, and the lowest quintile of sense of coherence at each stage of the socioeconomic lifecourse. At the childhood stage, men whose parents were wealthier had significantly lower rates of hopelessness (6%) and cynical hostility (19.1%) as adults. Similar but statistically insignificant differences were also seen for depression and sense of coherence. Men with only primary schooling had rates of hopelessness which were 10 times higher than men who received more than a high school education. This least educated group also experienced 46% higher rates of depression and 240% higher rates of cynical hostility. Blue-collar workers were consistently more likely to report significantly elevated rates of hopelessness (12.8%), depression (25.2%), cynical hostility (30.0%), and low sense of coherence (21.8%) compared to white-collar workers.

Table 4. Adult psychosocial characteristics (age-adjusted proportion) by stage of the socioeconomic lifecourse

Socioeconomic lifecourse stage	% High tertile hopelessness	% High quartile depression	% High quartile cynical hostility	% Low quintile sense of coherence
<i>Childhood</i>				
Poor (n = 928)	10.7 <sup>a</sup>	20.2	24.7 <sup>a</sup>	19.2
Middle (n = 1198)	10.1 <sup>a</sup>	22.4	25.1 <sup>a</sup>	19.5
High (n = 557)	6.0	18.6	19.1	16.6
<i>Education</i>				
Primary or less (n = 1555)	12.0 <sup>a</sup>	24.0 <sup>a</sup>	28.4 <sup>a</sup>	21.7
Some high school (n = 939)	7.3 <sup>a</sup>	19.6	20.0 <sup>a</sup>	15.4
Finished high or better (n = 180)	1.3	16.4	11.2	16.4
<i>Occupation</i>				
Farmer (n = 425)	8.4	22.9	21.6	18.4
Blue-collar (n = 1168)	12.8 <sup>a</sup>	25.2 <sup>a</sup>	30.0 <sup>a</sup>	21.8 <sup>a</sup>
White-collar (n = 1042)	5.6	17.7	18.8	15.7

<sup>a</sup>Differences between specified category and highest category (e.g. high childhood or white-collar) significant at  $P < 0.05$ .

*Socioeconomic lifecourse pathways—the connections between childhood, education and occupation*

In order to show the important structural relationships between childhood origins, educational experiences, and occupational outcomes, we arrayed childhood SES by education (Fig. 1), and education by occupation (Fig. 2) to illustrate the normative socioeconomic pathways along which this cohort of men moved during their lifecourses. Figure 1 shows that 70% of men whose parents were poor received primary schooling, while 2.1% received more than a high school education. In contrast, 40.2% of men who were born into wealthy families received primary schooling, while 15.8% completed a high school education or better.

The distribution of occupation according to education (Fig. 2) showed that 97.7% of men who completed a high school education took up white-collar employment, while only 20.6% of men with

primary education gained white-collar jobs. Men with the lowest level of education were most likely to find jobs in the blue-collar sector (57.6%)

DISCUSSION

The results presented here show that a large number of adult health behaviours and psychosocial characteristics, which have all been shown to be importantly related to disease risk, exhibit similarly graded associations with SES at temporally distinct stages of the lifecourse. While the observation that adult health behaviours are related to adult SES is not new, data showing that the health-related behaviours and psychosocial characteristics of adult men are associated with the social class of those men's parents has, to our knowledge, only been previously reported for obesity [53]. This observation could be seen as important, given Barker's [54–56] claims that conditions *in utero*, which would be influenced

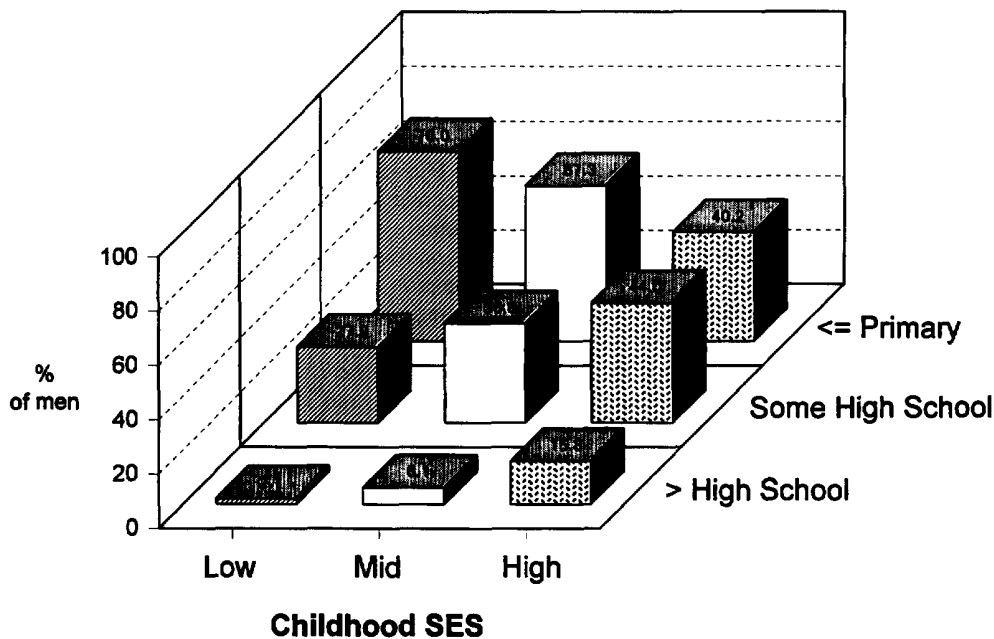


Fig. 1. Socioeconomic lifecourse pathways—childhood SES to education.

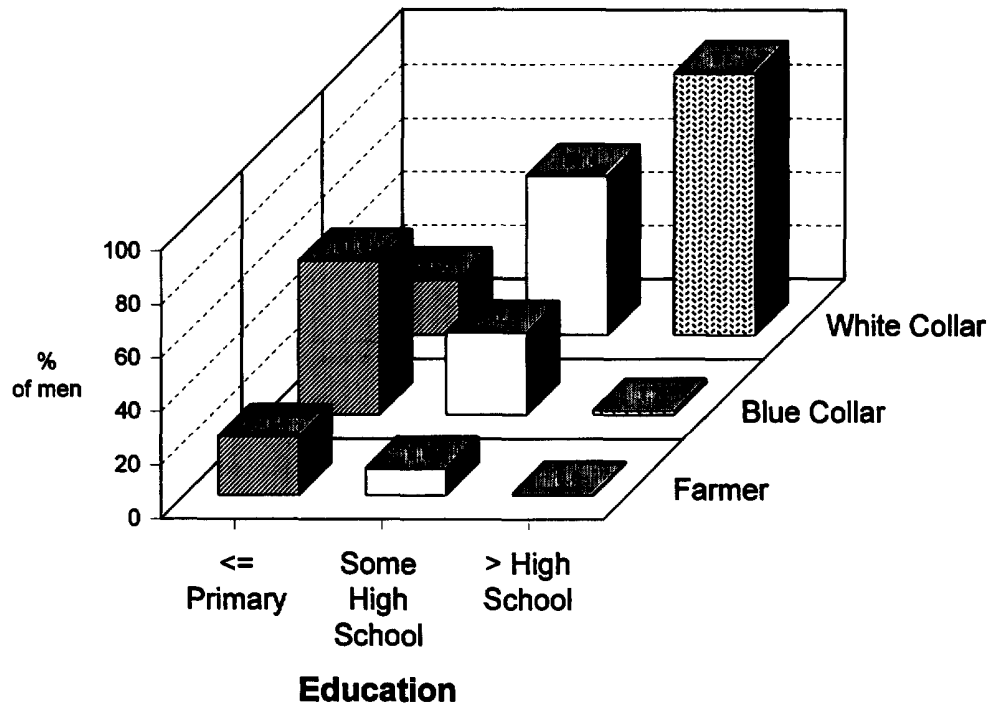


Fig. 2. Socioeconomic lifecourse pathways—education to occupation.

by maternal and paternal SES, are related to adult disease risk.

Some health behaviours such as diet and physical activity were strongly associated with inherited childhood conditions and may track over the life-course [57]. Bourdieu [58] has shown in exquisite detail how tastes for food, music, art, furniture and a whole range of lifestyle factors are symbolic of a particular class position. The observation that diet and physical activity were strongly related to childhood SES may merely reflect the age at which particular behaviours or proclivities became established in the habitual repertoires of these men. Differences in smoking were more strongly associated with education and occupation. Smoking behaviour was adopted later in the lifecourse than dietary practices and so may be more sensitive to different occupational environments and workplace cultures. Farmers and blue-collar workers were more likely to be occupationally engaged in strenuous physical activity, and so it is hardly surprising that they do not engage in strenuous leisure-time pursuits.

The data on the amount and pattern of drinking suggested that the most advantaged groups educationally and occupationally had the lowest rates of abstention and the highest amount consumed. This trend probably reflects the high price of alcohol in Finland, so that those with the lowest incomes may find alcoholic beverages too expensive to purchase. However, the pattern of drinking showed a consistent opposite association, with significantly higher rates of drunkenness in the poorest

childhood group, in the less educated, and in blue-collar workers. Hopelessness, depression and cynical hostility all showed graded, inverse associations with SES at every stage of the socioeconomic life-course.

The association between adult socioeconomic status and health behaviour exists, at least in part, because adult SES destinations depend on childhood SES origins [59]. As we have shown in Figs 1 and 2, men born into high, middle or low socioeconomic circumstances have different probabilities of receiving a certain education, which in turn affects the likelihood of being employed in farming, white- or blue-collar sectors. While these different socioeconomic lifecourse pathways are complicated by mobility both up and down the social hierarchy, normative patterns exist which represent the most common lifecourses.

Beginning with the data on economic milieu showed in Table 1, it is perhaps not surprising that those who were born to poor parents, received little education, and ended up in low paid blue-collar work were the most materially disadvantaged, had higher job and financial insecurity, and experienced more unemployment and work injury. It is not coincidental that these were the same men who tended to smoke more, exercise less, eat less nutritious diets, get drunk more often, have a cynically hostile outlook, and not feel full of hope about the future. While there are a few exceptions, the patterns of adult health behaviours and psychosocial characteristics show remarkably consistent associations with

the childhood, educational and occupational stages of the socioeconomic lifecourse and are supportive of the notion that adult health behaviours and psychosocial characteristics have SES roots early in life.

While our data cannot show which specific factors are responsible for the differences in behaviour and psychosocial orientation we observed at each stage of the SES lifecourse, it seems likely that there are different constraints, supports, opportunities, and encouragements for the development, maintenance and extinction of particular health behaviours and psychosocial orientations. A disadvantaged socioeconomic lifecourse pathway does not necessarily make choices of good health behaviour impossible. However, it may be characterized by different social constructions of which behaviours are associated with "good health", as well as different reinforcements and constraints, all of which exert important influences on the overall adult behavioural and psychosocial profile [60].

Perhaps the most striking aspect of these findings is that adult behaviours and psychosocial orientations are patterned by childhood SES, and so do not provide support for the "free choice" conception of adult behaviour, because in this view adult health behaviour would be unrelated to childhood conditions. Socioeconomic status at birth is not chosen, so it seems difficult to argue that the subsequent normative level of education for any particular childhood SES group is freely "chosen", or in fact that people then "choose" their subsequent occupation. In this light, the concept that individuals somehow choose their socioeconomic pathway through life is too simplistic. Understanding the contribution of health behaviours and psychosocial characteristics to socioeconomic health inequalities requires an acknowledgment of how they are moulded over time by the SES conditions imposed at each stage of the lifecourse.

There are a number of issues which we believe are important in considering the wider implications of these findings for health inequalities, both now and in the future. First, our results indicate that poor adult health behaviours and psychosocial orientations are more prevalent among men whose parents were poor. As Evans *et al.* and others have argued, health behaviours should be conceptualized as responses to environmental conditions, so that a meaningful interpretation can be made of the observation that many poor health behaviours cluster in lower SES groups [61]. Given the disturbing increases in income inequality which have recently been documented in the United States, Great Britain, and other industrial countries, it is vital to consider the health impact of placing ever larger numbers of families with children into low SES groups [62, 63]. In addition to placing children into conditions which are detrimental to their immediate

health status, there may well be a negative behavioural and psychosocial health dividend to be reaped in the future. The impact of these structural adjustments to income distribution is even more frightening in light of reduced commitments to public education and welfare programmes which might offer some opportunities for social advancement. As we have argued previously, the impact of poor childhood conditions on adult disease risk might be ameliorated by upward social mobility [35].

Second, finding that a variety of poor health habits and psychosocial dispositions clusters in lower SES groups has implications for public health policy. It is perhaps tempting to imagine that the behavioural and psychosocial characteristics associated with a lack of education can be remedied by adult health education, but the evidence that health inequalities can be decreased by "targeting" low SES groups for behavioural modification is less than encouraging [64]. This approach already commands much attention in current public health policy and is consistent with the broader logic of market-based economic efficiency. One of the key principles in the idea that markets are the most efficient arbiters of the economy is that individuals will, if unencumbered by regulation, seek to advance their own economic self-interest [65]. Under this system, it is not surprising that placing individuals at the centre of economic rationality also places them as the focus for all aspects of behavioural responsibility. More important than the limitations imposed by the low efficacy of these behavioural interventions is that altering the behaviours of the current adult population will do little to change the socioeconomic conditions which may generate these same behavioural and psychosocial characteristics in the next generation.

While Finland and the other Nordic nations are the most developed welfare states, even they have come under increasing pressure to apply the logic of the free market to their economies [66]. In the Kuopio region of Finland, market globalization, the collapse of the Soviet Union as a trading partner, and the specific changes in farming and manufacturing policies attendant to entry into the European Union have caused increases in unemployment of more than 100% since the 1980s. It is uninformative to conceptually isolate the behaviours and psychosocial orientations which are associated with poor health outcomes from their SES roots [67]. If the desire for economic efficiency is to be a central theme of the processes which shape late industrial societies, then it is entirely possible that the most efficient way to reduce the disease burden associated with poor health behaviours and psychosocial characteristics is to improve the socioeconomic conditions which generate them.

The evidence presented here sheds some light on answering the rhetorical question posed in the title of this paper. We have shown that poor adult

health behaviour and psychosocial characteristics are importantly related to a poor socioeconomic start in life, low levels of education and blue-collar employment. These findings suggest that childhood, adolescence and adulthood are all potentially important stages for attempts to alter the health-related behavioural and psychosocial profiles of adults. Understanding that adult health behaviour and psychosocial orientations are associated with socioeconomic conditions throughout the lifecourse implies that efforts to reduce socioeconomic inequalities in health must recognize that economic policy is public health policy.

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## CUMULATIVE IMPACT OF SUSTAINED ECONOMIC HARDSHIP ON PHYSICAL, COGNITIVE, PSYCHOLOGICAL, AND SOCIAL FUNCTIONING

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**ABSTRACT**

**Background** Although the relation between low income and poor health is well established, most previous research has measured income at only one time.

**Methods** We used income information collected in 1965, 1974, and 1983 from a representative sample of adults in Alameda County, California, to examine the cumulative effect of economic hardship (defined as a total household income of less than 200 percent of the federal poverty level) on participants who were alive in 1994.

**Results** Because of missing information, analyses were based on between 1081 and 1124 participants (median age, 65 years in 1994). After adjustment for age and sex, there were significant graded associations between the number of times income was less than 200 percent of the poverty level (range, 0 to 3) and all measures of functioning examined except social isolation. As compared with subjects without economic hardship, those with economic hardship in 1965, 1974, and 1983 were much more likely to have difficulties with independent activities of daily living (such as cooking, shopping, and managing money) (odds ratio, 3.38; 95 percent confidence interval, 1.49 to 7.64), activities of daily living (such as walking, eating, dressing, and using the toilet) (odds ratio, 3.79; 95 percent confidence interval, 1.32 to 9.81), and clinical depression (odds ratio, 3.24; 95 percent confidence interval, 1.32 to 7.89) in 1994. We found little evidence of reverse causation — that is, that episodes of illness might have caused subsequent economic hardship.

**Conclusions** Sustained economic hardship leads to poorer physical, psychological, and cognitive functioning. (N Engl J Med 1997;337:1889-95.)

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**T**HE relation between low income and poor health is well established.<sup>1-5</sup> Groups whose incomes are low are disproportionately exposed to social and psychological conditions that may have negative effects, while also possessing fewer economic resources to manage these circumstances.<sup>6</sup> Low income may affect health directly through inadequate housing and sanitation or indirectly through threatening, socially disrupted neighborhoods and the promotion of behavior and

psychosocial characteristics that are deleterious to health.<sup>7-9</sup>

Most previous research has measured income at only one time. This method may fail to capture the health effects of sustained exposure to low income or to account for transitions into and out of low-income groups. There is considerable volatility in income over a lifetime, with 26 to 39 percent of people 45 to 65 years of age having income reductions of 50 percent or more at least once in an 11-year period.<sup>10</sup> These rises and falls in income are more pronounced for those at the bottom of the income distribution, who are less likely to have stable employment.<sup>11</sup> Failure to account for such income dynamics could result in an underestimate of the true association between income and health status.<sup>12</sup>

We used income information collected in 1965, 1974, and 1983 as part of the Alameda County Study to examine the cumulative effect of sustained economic hardship among members of the cohort who were alive in 1994. The outcome measures represented important functional aspects of day-to-day living, including physical, psychological, social, and cognitive functioning.

**METHODS****Study Population**

We used information collected from the Alameda County Study, a population-based prospective investigation of predictors of health and functioning in a representative sample of adults in Alameda County, California. Full details of the sample in 1965 (6982 respondents) and 1974 (4864 respondents) have been published previously.<sup>13</sup> In 1983, a random sample of 50 percent of the 1974 respondents who were not known to be dead in 1982 were enrolled in a third period of data collection (a total of 1799 respondents). In 1994, a fourth period of data collection was conducted for the 1974 respondents. Of the 3161 subjects who were known to be alive, 3005 were located; 2935 were able to participate, and 205 refused to participate, leaving a sample of 2730. Response rates for all periods were high, with 86, 85, 87, and 93 percent responding in 1965, 1974, 1983, and 1994, re-

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spectively. The results presented here are based on information on income, risk factors, and prevalent diseases gathered in 1965, 1974, and 1983 and levels of physical, psychological, cognitive, and social functioning in 1994. All analyses are restricted to the 1799 respondents who were part of the random 50 percent sample enrolled in 1983. Of these 1799 subjects, 362 had died and 151 were lost to follow-up between 1983 and 1994, leaving a possible sample of 1286. Analyses excluded subjects who had missing data on income (130 respondents) or covariates (29) in 1965, 1974, or 1983. In addition, between 3 and 43 respondents had missing information on measures of function, leaving a total sample of between 1081 and 1124, depending on the functional outcome. There were no significant differences in demographic, health, or other risk factors between subjects with missing data at base line and those without missing data (data not shown). The maximal sample included 325 women and 263 men who were 45 to 64 years of age in 1994, 273 women and 224 men who were 65 to 84 years old, and 29 women and 10 men who were 85 or older in 1994.

### Assessment of Sustained Economic Hardship

We calculated the number of times between 1965 and 1983 that subjects reported total household incomes that were less than 200 percent of the federal poverty level for that year (the study definition of economic hardship); the results ranged from 0 (never below 200 percent) to 3 (below 200 percent in 1965, 1974, and 1983). The respective income cutoffs for 200 percent of the poverty level in 1965, 1974, and 1983 were \$6,634, \$11,000, and \$20,356 for a four-person household.<sup>14</sup> Income was self-reported, pretax household income from all sources at each period of data collection between 1965 and 1983, recorded in income categories. The category midpoint was adjusted for family size and used to identify households with incomes that were less than 200 percent of the poverty level, based on U.S. Census information for the relevant year.<sup>14</sup>

### Assessment of Functional Outcomes

Physical functioning was assessed with three commonly used measures of functional status: activities of daily living,<sup>15</sup> instrumental activities of daily living,<sup>16</sup> and a physical performance scale developed by Nagi.<sup>17</sup> These scales are based on Likert-type self-reports of the difficulty involved in walking, bathing, brushing hair or teeth, eating, dressing, moving from bed to chair, and using the toilet (activities of daily living); cooking, shopping, doing housework, using the telephone, and managing money (independent activities of daily living); and pushing or pulling, lifting, getting up from a crouch or bend, reaching, stooping, standing up, walking up stairs, and handling small objects (Nagi performance scale). Subjects were classified as having reduced physical functioning in 1994 according to the activities-of-daily-living scale (82 of 1124 subjects, or 7.3 percent) and the independent-activities-of-daily-living scale (164 of 1116 subjects with data available, or 14.7 percent) if they reported difficulties with two or more items, and according to the Nagi scale (130 of 1108 subjects with data available, or 11.7 percent) if they reported difficulties with five or more items.

Psychological functioning was assessed in terms of depression, cynical hostility, and optimism. Depression was measured on the basis of scores on 12 items that used the diagnostic symptom criteria for a major depressive episode outlined in the *Diagnostic and Statistical Manual of Mental Disorders*, third edition, revised (DSM-III-R).<sup>18</sup> Eighty-seven of 1120 subjects with data available (7.8 percent) with the highest scores were classified as clinically depressed. Depressive symptoms were measured with a scale developed in the Alameda County Study that has predictive validity for various outcomes.<sup>19</sup> Subjects with scores of 5 or more on the 18-item scale were classified as having symptoms of depression; 140 of 1121 subjects (12.5 percent) met this criterion. Cynical hostility was measured with a seven-item subgroup of the Cook-Medley hostility scale that is predictive of carotid atherosclero-

sis.<sup>20</sup> Subjects with scores of more than 18 on the 28-point scale (found in 23.4 percent of the subjects) were classified as having cynical hostility. Optimism was measured with the Life Orientation Test.<sup>21</sup> A total of 245 of 1090 subjects (22.5 percent) had scores lower than 17 on a 24-point scale and were categorized as lacking optimism about the future. Both the cynical-hostility scale and the Life Orientation Test have been used in clinical and epidemiologic studies and are internally consistent, reliable, and predictive of health outcomes in a variety of populations.<sup>20,22</sup>

Cognitive function was assessed by four Likert-type questions about self-reported difficulties in remembering things, paying attention, finding the right word, and forgetting where things were placed. Those with difficulties with two or more of these tasks were classified as having cognitive difficulties; 128 of 1116 subjects (11.5 percent) met the criteria. The degree of social isolation was based on the number of contacts with friends or close friends subjects had each month; 204 of 1108 subjects (18.4 percent) with data available who had fewer than two contacts a month were classified as being socially isolated.<sup>23</sup>

### Assessment of Covariates

Smoking history, consumption of beer, wine, and liquor, physical activity, and body-mass index (the weight in kilograms divided by the square of the height in meters) were assessed by questionnaire in 1965, 1974, and 1983.<sup>24</sup> Among all subjects, smoking was calculated in terms of pack-years of exposure. Physical activity was measured according to the frequency of activities such as walking, swimming, participation in exercise programs and vigorous sports, gardening, and fishing. To account for changes in body-mass index, physical activity, and alcohol consumption over time, we measured the number of periods in which participants were in the group with the highest risk. For instance, we divided body-mass index into quartiles for each period and counted the number of periods (0, 1, 2, or 3) in which the subject was in the highest quartile between 1965 and 1983.

Prevalent diseases in 1965, 1974, and 1983 were assessed by self-report; the diseases assessed have been demonstrated in this population to be strongly associated with an increased risk of death.<sup>25</sup> At each period, participants were asked whether they either currently had or had ever had heart trouble, chest pain, hypertension, a stroke, breathing difficulties, chronic bronchitis, diabetes, asthma, arthritis, back pain, or cancer. For each subject we counted the number of periods (0, 1, 2, or 3) in which each condition was present and included it as a continuous variable in all analyses.

### Statistical Analysis

We assessed associations between economic hardship and functioning with multivariate logistic regression,<sup>26</sup> using the Proc Logistic procedure in SAS version 6.12<sup>27</sup> on a Sun workstation. Associations were examined in models that were adjusted for both age and sex and in models that were adjusted separately for age, sex, and risk factors and for age, sex, and prevalent diseases. We assessed the association between mortality and economic hardship with time-dependent proportional-hazards regression using the PHREG procedure in SAS version 6.12.<sup>28</sup>

## RESULTS

Table 1 shows the demographic, behavioral, and health characteristics of the subjects according to the duration of economic hardship (income less than 200 percent of the poverty level). Table 2 presents the results of logistic-regression analyses of the association between the duration of economic hardship and functioning. In models adjusted for age and sex, there were strong, significant, graded associations between the number of periods of econom-

ic hardship and all measures of functioning except social isolation. We found no systematic differences in the pattern of associations between sustained economic hardship and functioning between women and men or between subjects who were 65 years of age or older and those who were under 65 (data not shown).

For physical functioning, subjects whose incomes were less than 200 percent of the poverty level in 1965, 1974, and 1983 had 3.79 times (95 percent confidence interval, 1.32 to 9.81) the odds of having difficulties with activities of daily living in 1994 of those who had no history of economic hardship. Additional adjustment for the number of pack-years of smoking, body-mass index, levels of alcohol consumption, and physical activity reduced the odds ratio to 2.86 (95 percent confidence interval, 1.00 to 8.24). Adjustment for age, sex, and prevalent diseases resulted in an odds ratio of 2.95 (95 percent confidence interval, 1.02 to 8.58). Similar results were obtained when associations between sustained economic hardship and other functional outcomes (e.g., independent activities of daily living and the Nagi performance scale) were adjusted for these risk factors.

There were equally strong, graded relations between sustained economic hardship and measures of psychological and cognitive functioning. For instance, as compared with subjects with no history of economic hardship, subjects with three episodes of economic hardship between 1965 and 1983 had 3.24 times (95 percent confidence interval, 1.32 to 7.89) the sex- and age-adjusted odds of meeting the DSM-III-R criteria for depression. Similarly, the group with the longest history of economic hardship had greater odds of having high levels of depressive symptoms (odds ratio, 4.56; 95 percent confidence interval, 2.07 to 10.07), being cynically hostile (odds ratio, 5.09; 95 percent confidence interval, 2.40 to 10.86), lacking optimism (odds ratio, 5.68; 95 percent confidence interval, 2.73 to 11.83), and having greater self-reported difficulties with cognitive functioning (odds ratio, 4.60; 95 percent confidence interval, 2.06 to 10.32) in 1994.

Table 3 shows complete models for the associations between reduced activities of daily living and economic hardship adjusted separately for risk factors and prevalent diseases. In addition to sustained economic hardship, smoking, body-mass index, and histories of diabetes, arthritis, and back pain were all significantly associated with the outcome.

**Reverse Causation**

We examined reverse causation — the possibility that poor health and functioning caused economic hardship and not vice versa — in three ways. First, we examined associations between sustained economic hardship and functioning in a sample restrict-

**TABLE 1. CHARACTERISTICS OF 1124 PARTICIPANTS IN THE ALAMEDA COUNTY STUDY ACCORDING TO THE DURATION OF ECONOMIC HARDSHIP, 1965 TO 1983.**

CHARACTERISTIC	NO. OF TIMES INCOME WAS <200% OF POVERTY LEVEL			
	0 (N=691)	1 (N=273)	2 (N=125)	3 (N=35)
Mean age (yr)	64.9	63.4	65.2	63.7
Female sex (%)*	51.5	57.5	68.0	82.9
White race (%)*	89.2	81.3	65.6	54.3
Smoking (mean pack-yr)	17.8	17.6	15.5	14.8
No. of times in highest quartile of alcohol consumption (%)*				
0	56.2	68.1	72.0	65.7
1	19.5	15.4	10.4	25.7
2	11.9	10.3	13.6	8.6
3	12.5	6.2	4.0	0.0
No. of times in highest quartile of body-mass index (%)				
0	65.9	61.9	56.8	45.7
1	14.6	16.5	15.2	20.0
2	8.5	9.5	10.4	11.4
3	11.0	12.1	17.6	22.9
No. of times in lowest quartile of physical activity (%)*				
0	62.5	54.6	52.8	34.3
1	21.0	22.0	20.8	14.2
2	10.9	13.9	15.2	31.4
3	5.6	9.5	11.2	20.0
Free of prevalent disease during all 3 periods (%)				
Heart trouble	94.1	93.8	92.0	88.6
Chest pain	80.6	78.4	73.6	68.6
Hypertension	73.4	72.9	68.8	71.4
Stroke	99.3	98.9	96.8	100.0
Diabetes	95.8	95.6	94.4	85.7
Chronic bronchitis	92.2	91.2	89.6	82.9
Breathing difficulties	79.6	78.4	70.4	65.7
Cancer	96.8	97.1	99.2	91.4
Asthma	93.3	93.0	93.6	91.4
Back pain	47.7	44.7	43.6	52.9
Arthritis*	70.8	67.8	60.8	80.0

\*P<0.001 for the overall differences among the groups (by the chi-square test).

ed to 982 subjects who were less than 50 years of age in 1965 and who had no reduction in physical functioning. In this subgroup any association between sustained economic hardship and subsequent poor functioning could not be due to illness at base line, because the prevalence of physical and cognitive dysfunction was extremely low in this age group. The magnitude and pattern of associations with functioning were virtually identical to those in the whole sample (data not shown).

Second, we restricted the sample to 2307 subjects who reported excellent or good health in 1965 and examined the effects of economic hardship in 1965 on levels of functioning in 1994, after adjustment for pack-years of smoking, body-mass index, physical activity, alcohol consumption, and prevalent diseases in 1965. Economic hardship in 1965 was a signifi-

**TABLE 2.** ODDS RATIOS FOR REDUCED LEVELS OF PHYSICAL, PSYCHOLOGICAL, COGNITIVE, AND SOCIAL FUNCTIONING IN 1994 ACCORDING TO WHETHER THERE WAS SUSTAINED ECONOMIC HARDSHIP BETWEEN 1965 AND 1983.

MEASURE ASSESSED AND NO. OF TIMES INCOME WAS <200% OF POVERTY LEVEL*	ADJUSTED FOR AGE AND SEX		ADJUSTED FOR AGE, SEX, AND RISK FACTORS†		ADJUSTED FOR AGE, SEX, AND PREVALENT DISEASES‡	
	ODDS RATIO (95% CI)§	P VALUE	ODDS RATIO (95% CI)§	P VALUE	ODDS RATIO (95% CI)§	P VALUE
<b>Reduced physical functioning</b>						
Difficulty with IADL (n = 1116)						
1	1.41 (0.94–2.13)	0.10	1.29 (0.85–1.97)	0.24	1.34 (0.88–2.05)	0.17
2	1.62 (0.95–2.75)	0.08	1.42 (0.82–2.47)	0.21	1.23 (0.70–2.17)	0.47
3	3.38 (1.49–7.64)	0.003	2.64 (1.14–6.28)	0.03	2.93 (1.24–6.92)	0.01
Difficulty with ADL (n = 1124)						
1	1.49 (0.85–2.59)	0.16	1.39 (0.78–2.47)	0.26	1.37 (0.77–2.44)	0.29
2	1.85 (0.95–3.61)	0.07	1.66 (0.83–3.33)	0.16	1.43 (0.70–2.92)	0.32
3	3.79 (1.32–9.81)	0.01	2.86 (1.00–8.24)	0.05	2.95 (1.02–8.58)	0.05
Difficulty with Nagi performance scale (n = 1108)						
1	1.47 (0.93–2.31)	0.10	1.31 (0.81–2.11)	0.27	1.38 (0.85–2.23)	0.53
2	2.54 (1.50–4.30)	<0.001	2.33 (1.33–4.08)	0.002	2.05 (1.16–3.62)	0.01
3	2.88 (1.20–6.94)	0.02	1.93 (0.77–4.88)	0.16	2.45 (0.93–6.48)	0.07
<b>Reduced psychological functioning</b>						
DSM-III-R criteria for clinical depression (n = 1120)						
1	1.01 (0.58–1.76)	0.96	0.97 (0.55–1.71)	0.92	0.97 (0.55–1.69)	0.91
2	1.72 (0.91–3.29)	0.10	1.68 (0.87–3.23)	0.12	1.54 (0.81–3.00)	0.19
3	3.24 (1.32–7.89)	0.01	2.58 (1.01–6.59)	0.05	3.20 (1.28–8.02)	0.01
Depressive symptoms (n = 1121)						
1	1.75 (1.13–2.70)	0.01	1.65 (1.05–2.59)	0.03	1.72 (1.10–2.68)	0.02
2	4.02 (2.48–6.54)	<0.001	4.00 (2.40–6.62)	<0.001	3.83 (2.31–6.46)	<0.001
3	4.56 (2.07–10.07)	<0.001	3.42 (1.48–7.93)	0.004	3.89 (1.68–9.00)	0.002
Cynical hostility (n = 1081)						
1	2.14 (1.52–3.00)	<0.001	2.05 (1.45–2.90)	<0.001	2.15 (1.52–3.03)	<0.001
2	3.24 (2.10–5.04)	<0.001	3.07 (1.96–4.80)	<0.001	3.16 (2.01–4.96)	<0.001
3	5.09 (2.40–10.86)	<0.001	4.11 (1.87–9.06)	<0.001	4.69 (2.17–10.13)	<0.001
Lack of optimism (n = 1090)						
1	1.48 (1.05–2.09)	0.02	1.35 (0.95–1.92)	0.09	1.41 (1.00–2.03)	0.05
2	2.27 (1.46–3.52)	<0.001	2.08 (1.32–3.26)	0.003	2.05 (1.31–3.23)	0.002
3	5.68 (2.73–11.83)	<0.001	4.32 (2.02–9.24)	<0.001	5.52 (2.59–11.73)	<0.001
<b>Reduced cognitive functioning</b>						
Cognitive difficulties (n = 1116)						
1	1.19 (0.74–1.90)	0.47	1.19 (0.74–1.92)	0.47	1.16 (0.72–1.87)	0.55
2	2.28 (1.35–3.86)	0.002	2.25 (1.31–3.87)	0.003	2.13 (1.23–3.69)	0.007
3	4.60 (2.06–10.32)	<0.001	3.77 (1.62–8.77)	0.002	4.14 (1.79–9.61)	<0.001
<b>Reduced social functioning</b>						
Social isolation (n = 1108)						
1	0.95 (0.65–1.38)	0.79	0.91 (0.62–1.33)	0.63	0.96 (0.66–1.40)	0.84
2	1.33 (0.82–2.15)	0.25	1.31 (0.80–1.93)	0.69	1.27 (0.78–2.08)	0.34
3	1.56 (0.68–3.57)	0.29	1.19 (0.50–2.80)	0.69	1.42 (0.61–3.04)	0.42

\*IADL denotes independent activities of daily living, ADL activities of daily living, and DSM-III-R *Diagnostic and Statistical Manual of Mental Disorders* (third edition, revised).

†The risk factors were the number of periods subjects were in the highest quartiles of body-mass index and alcohol consumption and the lowest quartile of physical activity and the number of pack-years of smoking.

‡Prevalent diseases consisted of heart trouble, chest pain, hypertension, stroke, diabetes, asthma, chronic bronchitis, breathing difficulties, back pain, arthritis, and cancer.

§The reference group is the subjects with no history of economic hardship. CI denotes confidence interval.

cant predictor of reduced physical, psychological, and cognitive functioning in 1994. Although these results mirrored the main findings, the associations were expectedly weaker. Odds ratios for reduced functioning ranged from 1.36 (95 percent confidence interval, 0.99 to 1.85) for depressive symptoms to 1.64 (95 percent confidence interval, 1.19 to 2.27) for cognitive difficulties.

Third, we restricted the sample to 197 subjects whose income was not derived from wages or salaries in 1965 or 1974 and who were in excellent or good health in 1965. Even in this small sample, sustained economic hardship had significant and, in many cases, graded associations with functional outcomes that were very similar to the patterns observed in the whole sample (Table 4). Even among

subjects whose income did not depend on wages, those with economic hardship in 1965, 1974, and 1983 had an odds ratio of 5.91 (95 percent confidence interval, 1.38 to 25.29) for difficulties on the Nagi performance scale (Table 4).

**Mortality**

The associations reported here were evident even though subjects with incomes below 200 percent of the poverty level were more likely than those without economic hardship to have died during the study and therefore not to have been included in the analyses. Selective analysis of mortality with time-dependent covariate proportional-hazards regression, which allowed economic hardship to vary as a function of the length of survival, showed that subjects whose incomes were less than 200 percent of the poverty level were at increased risk for death over the 29-year study period, but this risk differed according to age. As compared with subjects of the same age without economic hardship in 1965, those who were 35 years of age with economic hardship had an age- and sex-adjusted relative risk of death of 1.70 (95 percent confidence interval, 1.29 to 2.22), and those who were 65 years of age with economic hardship had a relative risk of death of 1.29 (95 percent confidence interval, 1.16 to 1.43).

**DISCUSSION**

Our results demonstrate strong, consistent, graded associations between sustained economic hardship from 1965 to 1983 and reduced physical, psychological, and cognitive functioning in 1994. The associations were not greatly attenuated after adjustment for risk factors or prevalent diseases, even though these covariates were related to many of the functional outcomes.

This study has several strengths. First, the findings were based on multiple measures of income over a 17-year period and show dose-response associations between the number of periods of economic hardship and important functional measures. Second, the graded associations were consistent across various measures of functional status, but there was no relation between sustained economic hardship and social isolation. This result is perhaps surprising, given other evidence that has generally shown that lower-income groups have less social support.<sup>29,30</sup> However, the associations may depend on the aspect of support that is measured and the reasons for the initiation of social contact.<sup>13</sup> The absence of an association between economic hardship and social functioning may have been because reduced functioning and higher levels of psychological distress translated into an increased need for social contacts to deliver help. Third, in additional analyses, these results were not sensitive to changes in the cutoff points that defined the functional outcomes (data not shown).

**TABLE 3.** ADJUSTED ODDS RATIOS FOR REDUCED ACTIVITIES OF DAILY LIVING IN 1994 ACCORDING TO THE DURATION OF ECONOMIC HARDSHIP BETWEEN 1965 AND 1983.

VARIABLE	ODDS RATIO	95% CONFIDENCE INTERVAL	P VALUE
<b>Adjusted for age, sex, and risk factors*</b>			
Age†	1.07	1.04-1.09	<0.001
Female sex	1.27	0.75-2.15	0.37
No. of times income <200% of poverty level			
0‡	1.0	—	—
1	1.39	0.78-2.47	0.26
2	1.69	0.83-3.33	0.16
3	2.86	1.00-8.24	0.05
No. of times in lowest quartile of physical activity			
0‡	1.0	—	—
1	1.85	1.02-3.35	0.04
2	1.64	0.83-3.24	0.15
3	1.57	0.71-3.45	0.26
No. of times in highest quartile of alcohol consumption			
0‡	1.0	—	—
1	0.81	0.39-1.69	0.57
2	1.25	0.57-2.76	0.57
3	1.24	0.53-2.89	0.62
No. of times in highest quartile of body-mass index			
0‡	1.0	—	—
1	0.88	0.41-1.90	0.75
2	1.89	0.87-4.10	0.11
3	2.16	1.17-4.00	0.01
Pack-years of smoking†	1.01	1.00-1.02	0.01
<b>Adjusted for age, sex, and prevalent diseases§</b>			
Age†	1.06	1.03-1.08	<0.001
Female sex	1.07	0.64-1.78	0.79
No. of times income <200% of poverty level			
0‡	1.0	—	—
1	1.37	0.77-2.44	0.29
2	1.43	0.70-2.92	0.32
3	2.95	1.02-8.58	0.05
Cumulative index of prevalent diseases†			
Heart trouble	1.05	0.62-1.76	0.86
Chest pain	1.13	0.79-1.61	0.51
Hypertension	1.21	0.89-1.65	0.23
Diabetes	1.74	1.12-2.71	0.01
Stroke	0.84	0.28-2.47	0.75
Breathing difficulties	1.28	0.91-1.79	0.15
Asthma	1.20	0.74-1.94	0.47
Chronic bronchitis	0.86	0.48-1.54	0.60
Arthritis	1.35	1.04-1.75	0.03
Back pain	1.29	1.02-1.64	0.03
Cancer	1.01	0.44-2.31	0.98

\*The risk factors were the number of periods subjects were in the highest quartiles of body-mass index and alcohol consumption and the lowest quartile of physical activity and the number of pack-years of smoking.

†This is a continuous variable.

‡This is the reference group.

§Prevalent diseases consisted of heart trouble, chest pain, hypertension, stroke, diabetes, asthma, chronic bronchitis, breathing difficulties, back pain, and cancer.

**TABLE 4.** ODDS OF REDUCED LEVELS OF PHYSICAL, PSYCHOLOGICAL, COGNITIVE, AND SOCIAL FUNCTIONING IN 1994, ACCORDING TO WHETHER THERE WAS SUSTAINED ECONOMIC HARDSHIP BETWEEN 1965 AND 1983, AMONG SUBJECTS WHOSE INCOME WAS NOT DERIVED FROM WAGES OR SALARIES IN 1965 AND 1974 AND WHO REPORTED EXCELLENT OR GOOD HEALTH IN 1965.\*

MEASURE ASSESSED AND NO. OF TIMES INCOME WAS <200% OF POVERTY LEVEL	ODDS RATIO†	95% CONFIDENCE INTERVAL	P VALUE
<b>Reduced physical functioning</b>			
Difficulty with Nagi performance scale (n = 193)			
1	1.59	0.59–4.27	0.36
2	3.48	1.05–11.44	0.04
3	5.91	1.38–25.29	0.02
<b>Reduced psychological functioning</b>			
Lack of optimism (n = 186)			
1	2.18	1.00–4.76	0.05
2	4.06	1.41–11.66	0.009
3	6.02	1.53–23.67	0.01
<b>Reduced cognitive functioning</b>			
Cognitive difficulties (n = 195)			
1	1.41	0.52–3.82	0.50
2	0.82	0.16–4.08	0.81
3	3.97	0.86–18.33	0.08
<b>Reduced social functioning</b>			
Social isolation (n = 190)			
1	1.16	0.39–3.46	0.79
2	2.21	0.60–8.09	0.23
3	2.53	0.57–11.27	0.22

\*The associations for other measures of functioning not shown in the table were similar.

†The reference group is the group of subjects with no history of economic hardship, adjusted for age and sex.

Although the prospective, dose–response associations we observed may suggest a causal relation between sustained economic hardship and poor functioning, it was important to explore the possibility of reverse causation (i.e., that episodes of illness caused subsequent economic hardship and not vice versa). We addressed this issue in three ways. We examined associations between periods of economic hardship and functioning in a subgroup that was healthy at base line and found almost identical relations between more sustained economic hardship and reduced functioning. However, although this analysis showed that illness at base line could not account for the associations, it did not preclude the possibility that illness after base line caused subsequent economic hardship. We conducted another analysis examining only the effects of economic hardship in 1965 on functioning in 1994 in a subgroup that was healthy at base line. Any association between an income that was less than 200 percent of the poverty level in 1965 and functioning in 1994 would not rely on repeated episodes of low income that could have been the consequence of illness after

base line. In this subgroup, one period of economic hardship in 1965 was a significant predictor of reduced physical, psychological, and cognitive functioning in 1994.

In a more stringent examination of the plausibility of reverse causation as an explanation for the findings, we again restricted the sample to subjects who reported excellent or good health in 1965 and whose income was not derived from wages or salaries. In this sample, episodes of illness were less likely to create economic hardship because the sources and level of income (e.g., partner's income, investments, welfare, and pensions) did not depend directly on the subject's health status. Sustained economic hardship had significant and in many cases graded associations with functional outcomes that were very similar to the patterns observed in the whole sample. Under certain conditions, episodes of illness may affect the ability to generate income,<sup>31</sup> but given the results of these analyses of subgroups we found very little evidence that reverse causation could explain the overall magnitude and pattern of the findings.

The associations were evident even though subjects with more sustained economic hardship between 1965 and 1983 were more likely to have died before the 1994 survey and to have been excluded from these analyses. This increased risk of death should have reduced the likelihood of finding associations between sustained economic hardship and functioning in those who survived until 1994.

Despite the fact that the measures of risk factors and prevalent diseases used in this study were related to the functional outcomes in multivariate models, they failed to attenuate the associations between sustained economic hardship and functioning. Caution should be exercised in interpreting this absence of confounding as evidence that the association between sustained economic hardship and functional status is largely independent of these factors, since it is reasonable to think of them as potentially intervening sources.<sup>1,32</sup> It is possible that the measures used here did not fully capture the cumulative effects of these risk factors and prevalent diseases. Furthermore, without information on the extent of measurement error, the degree and direction of potential bias involved in estimating residual multivariate associations cannot be predicted.<sup>33</sup>

Our results show the cumulative health effects of sustained economic hardship and have potentially important implications for public health, health care, and economic policy. People with sustained economic hardship are more likely to have poorer physical, psychological, and cognitive functioning that might benefit from medical intervention, but recent evidence shows they are less likely to receive such care.<sup>34,35</sup> It will be important to monitor these trends, especially in the light of the uncertainties surrounding welfare reform and transitions to managed care.<sup>36</sup>

Furthermore, increases in income inequality in the United States<sup>37</sup> suggest that larger proportions of the population, especially children, have been pushed into low-income groups, and economic policies that polarize the income distribution may have serious short-term and long-term health consequences.<sup>38,39</sup> In conclusion, sustained economic hardship leaves physical, psychological, and cognitive imprints that decrease the quality of day-to-day life.

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## RESEARCH REPORT

# Explaining the social gradient in coronary heart disease: comparing relative and absolute risk approaches

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**Study objectives:** There are contradictory perspectives on the importance of conventional coronary heart disease (CHD) risk factors in explaining population levels and social gradients in CHD. This study examined the contribution of conventional CHD risk factors (smoking, hypertension, dyslipidaemia, and diabetes) to explaining population levels and to absolute and relative social inequalities in CHD. This was investigated in an entire population and by creating a low risk sub-population with no smoking, dyslipidaemia, diabetes, and hypertension to simulate what would happen to relative and social inequalities in CHD if conventional risk factors were removed.

**Design, setting, and participants:** Population based study of 2682 eastern Finnish men aged 42, 48, 54, 60 at baseline with 10.5 years average follow up of fatal (ICD9 codes 410–414) and non-fatal (MONICA criteria) CHD events.

**Main results:** In the whole population, 94.6% of events occurred among men exposed to at least one conventional risk factor, with a PAR of 68%. Adjustment for conventional risk factors reduced relative social inequality by 24%. However, in a low risk population free from conventional risk factors, absolute social inequality reduced by 72%.

**Conclusions:** Conventional risk factors explain the majority of absolute social inequality in CHD because conventional risk factors explain the vast majority of CHD cases in the population. However, the role of conventional risk factors in explaining relative social inequality was modest. This apparent paradox may arise in populations where inequalities in conventional risk factors between social groups are low, relative to the high levels of conventional risk factors within every social group. If the concern is to reduce the overall population health burden of CHD and the disproportionate population health burden associated with the social inequalities in CHD, then reducing conventional risk factors will do the job.

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There are two apparently contradictory epidemiological perspectives—one concerning the causes of social inequalities in coronary heart disease (CHD) and another concerning the causes of population levels of CHD.

## THE PERSPECTIVE FROM SOCIAL EPIDEMIOLOGY

In social epidemiology it has been established that conventional risk factors—smoking, hypertension, and cholesterol (and by extension the behaviours that influence hypertension and cholesterol such as diet, alcohol consumption, and physical activity)—do not explain social inequalities in CHD. Studies explored this issue from the late 1950s,<sup>1,2</sup> and by 1981 Rose and Marmot concluded that most social inequality in CHD “remains unexplained” (page 13).<sup>3</sup> Exact quantification of how much social inequality in CHD is explained by statistical adjustment for conventional risk factors varies but estimates typically range from 15% to 40%. Such numbers will probably underestimate the role of conventional risk factors because of measurement error, the absence of data regarding conventional risk factors across the lifecourse, and incomplete modelling of their interactions. Nevertheless, the idea that conventional risk factors do not explain social inequalities in CHD has been so widely accepted,<sup>4–10</sup> even by the current authors,<sup>11,12</sup> that over the past 20 years it has become a core concept in understanding the causes of social inequalities in CHD and health inequalities more generally.

Two apparently reasonable conclusions have been drawn from this concept. Firstly, is that there must be other unidentified factors that account for social inequalities in CHD and this has motivated novel avenues of research, especially on psychosocial explanations (for example, stress,

control, and social capital).<sup>13–14</sup> Secondly, some have argued that interventions focused on health behaviours and conventional risk factors are unlikely to appreciably reduce health inequalities.<sup>15–16</sup> The idea that conventional physiological risk factors and behaviours provide only modest explanation of social inequalities in health not only influences research agendas and funding but also intervention strategies and health policy.<sup>16–17</sup>

## THE PERSPECTIVE FROM CHD EPIDEMIOLOGY

In CHD epidemiology it has been established that conventional risk factors do explain most cases of CHD in many populations.<sup>18–22</sup> The INTERHEART study, involving about 15 000 cases and controls from 52 countries, showed that exposure to nine potentially modifiable risk factors accounts for at least 90% of the population attributable risk (PAR) for first myocardial infarction.<sup>23</sup> Exposure to at least one of the four conventional risk factors—dyslipidaemia, smoking, hypertension, and diabetes—had a PAR of 76%, and this is probably a conservative estimate because hypertension and diabetes were based on self report. In the British regional heart study, Whincup and colleagues reported that after accounting for regression dilution bias, smoking, blood pressure, and cholesterol had a PAR of 81%.<sup>24</sup> Data from three large US cohorts involving more than 350 000 people, showed that those with favourable levels (that is, below standard clinical cut off points) of conventional risk factors experienced from 77% to 92% lower relative CHD mortality.<sup>25</sup> Data from the same cohorts showed that exposure to at least one conventional risk factor accounted for 87% to 100% of fatal CHD cases and 84% to 92% of non-fatal CHD among men and women respectively.<sup>26</sup> It is nevertheless possible that

**Table 1** Age adjusted educational inequality in CHD with separate additional adjustment for conventional risk factors and hopelessness

(1) Relative risk adjusted for age only (least v most educated)	(2) Model 1 plus adjustment for smoking, hypertension, dyslipidaemia, diabetes	(3) Model 1 plus adjustment for hopelessness
1.90 (1.38, 2.61)	1.68 (1.22, 2.31)	1.65 (1.19, 2.29)

in some circumstances, other risk factors such as binge alcohol consumption can also make large contributions to the population burden of CHD, albeit via arrhythmic and cardiomyopathic rather than the traditional atherothrombotic processes associated with chronic CHD mechanisms.<sup>27</sup>

### A PARADOX

So how do the risk factors that account for most cases of CHD in a population apparently not account for social gradients in CHD, when social inequalities simply emerge from subgrouping the population according to some indicator of social position? Our objective in this study is to use prospective data from a large well characterised cohort to illustrate how this apparent paradox may arise. We will show that conventional risk factors do account for most cases of CHD but that they do not explain the relative social gradient in CHD. We will then show how this situation arises because of the epidemiological preference for contrasting and explaining risk on a relative rather than absolute scale.

### METHODS

We use prospective data on 2682 Finnish men in the Kuopio ischaemic heart disease risk factor study (KIHD).<sup>28, 29</sup> Baseline examinations were conducted from 1984 to 1989, with ascertainment of events through 1998. Our measure of social inequality was based on education, where we created three groups (primary school or less; some high school; completed high school or better).<sup>30</sup> We stratified the population into lower and higher risk groups<sup>31</sup> based on smoking (current compared with others) hypertension (> 140/90 mm Hg or medications), dyslipidaemia (low density lipoprotein >160 mmol/l) consistent with National Cholesterol Education Project (NCEP)/Adult Treatment Panel III (ATPIII) guidelines,<sup>32</sup> and prevalent diabetes (fasting glucose  $\geq$ 6.1 mmol/l or diabetes drugs). Information on biological and behavioural risk factors was collected by standard procedures and has been described elsewhere.<sup>29</sup> The outcome used here combines fatal (ICD9 codes 410–414) and non-fatal CHD, classified according to MONICA criteria.<sup>33</sup> We calculated the PAR associated with exposure to at least one

risk factor and calculated absolute and relative risks associated with the educational inequalities in the whole KIHD population and a low risk segment of the population free from smoking, hypertension, dyslipidaemia, and diabetes. We used proportional hazard models to calculate the crude and adjusted relative educational inequalities in CHD. Hopelessness, defined as negative expectancies about oneself and the future, was measured by two questionnaire items that asked about the likelihood of reaching goals and the possibility of positive change in the future.<sup>34</sup> The study received ethical approval from the University of Michigan Institutional Review Board.

### RESULTS

In this population, 34.7% were current smokers, 58.7% had hypertension, 42.7% had dyslipidaemia, 6.5% had diabetes, and 84.9% had at least one of these risk factors. There were 425 CHD events (108 fatal, 317 non-fatal) during an average follow up of 10.5 years—402 (94.6%) occurred among men exposed to at least one of the conventional risk factors and more than 70% occurred among men who had at least two risk factors. We calculated the PAR for having at least one conventional risk factor to be 68% but this is an underestimate because they were measured with error,<sup>22</sup> crudely categorised as simple dichotomous exposures, and taken from a single assessment at one point in the lifecourse.<sup>35</sup> Nevertheless, our PAR is similar to the 76% found in the INTERHEART study<sup>23</sup> and that more than 90% of cases occur among those exposed to at least one conventional risk factor is consistent with the findings from the large US cohorts.<sup>26</sup>

In table 1, we show the typical sort of analysis done in social epidemiology. Comparing those with less than a primary education with those with more than high school, the crude relative risk (RR) for CHD was 1.90 (95% CI:1.38 to 2.61). After control for conventional risk factors—smoking, hypertension, dyslipidaemia, and diabetes—the RR was 1.68 (95% CI:1.22 to 2.33)—a reduction in the excess RR of 24%. The crude social inequality adjusted for a psychosocial risk factor (hopelessness) was 1.65 (1.19 to 2.29)—a reduction in the excess RR of 28%.

**Table 2** Distribution of cases of CHD by education in the whole KIHD population and a low risk subset of the KIHD population

Whole population (n=2682)	Number (%)	Number of cases (%)	Risk per 1000 (95% CI)	Crude relative risk*† (95% CI)	Excess risk† (per 1000) (95% CI)
<b>Education</b>					
< Primary school	1121 (41.8)	218 (51.3)	194 (171, 218)	1.83 (1.36, 2.47)	88 (51, 125)
Some high school	1128 (42.1)	161 (37.9)	143 (122, 163)	1.35 (0.99, 1.83)	37 (1, 72)
High school graduate	433 (16.1)	46 (10.8)	106 (77, 135)	1.0	0
Total	2682	425	158	–	–
<b>Low risk population (n=404, 15.1%)</b>					
<b>Education</b>					
< Primary school	122 (30.2)	8 (34.8)	66 (22, 109)	1.61 (0.50, 5.18)	25 (–34, 84)
Some high school	184 (45.5)	11 (47.8)	60 (26, 94)	1.46 (0.48, 4.48)	19 (–33, 71)
High school graduate	98 (24.3)	4 (17.4)	41 (2, 80)	1.0	0
Total	404	23	57	–	–

\*Crude relative risk is calculated directly from this cross tabulation and so is slightly different from the RR of 1.90 mentioned above, which is model based and accounts for survival time. †Compared with the highest education category. All confidence intervals for simple risk calculations and risk differences are asymptotic 95% CI. All confidence intervals for relative risks are Mantel-Haenszel 95% CI.

Based on these analyses, on one hand, we would conclude that most cases of CHD in this population can be attributed to conventional risk factors—consistent with observations from CHD epidemiology. On the other hand, adjustment for conventional risk factors explained only a modest proportion of the relative social inequality in CHD—consistent with observations from social epidemiology.

To illustrate what would happen to the social inequality and levels of CHD, if conventional risk factors were removed from the population, we excluded all men exposed to at least one conventional risk factor (84.9% of the sample and 94.6% of the cases) to create a low risk population.<sup>31</sup> Table 2 shows the calculations of risk and both absolute and relative social inequalities in CHD in the whole population and in the low risk population (that is, not currently smoking, without hypertension, dyslipidaemia, or diabetes). If conventional risk factors were removed from the whole population, CHD risk would reduce by 64% from 158 to 57 per 1000. Among the least educated, risk would reduce by 66% from 194 to 66 per 1000. While a relative social inequality (RR = 1.61) would still remain in the low risk population, the absolute social inequality in CHD—as shown by change in the excess risk—would reduce by 72% from 88 to 25 per 1000 (fig 1).

## DISCUSSION

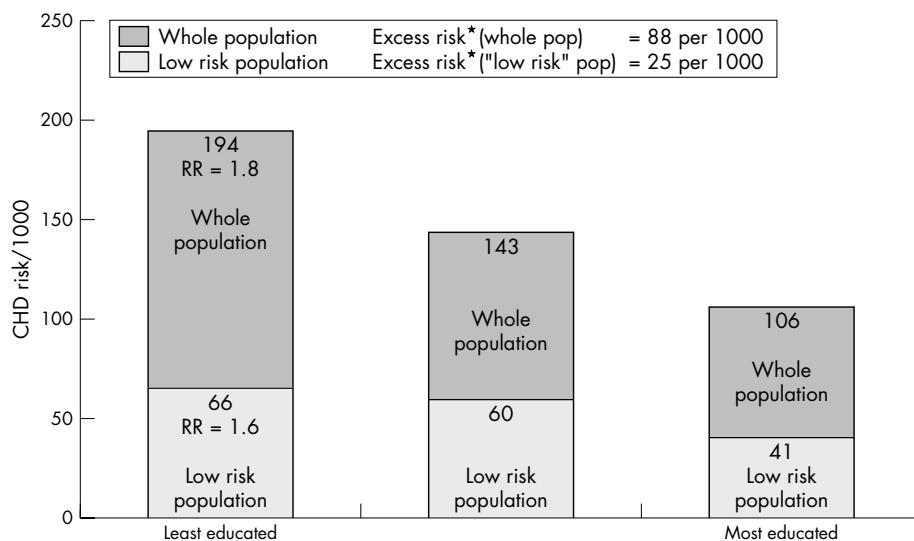
Our intention in this paper was to show that explanations for social inequalities in CHD can depend on whether one is interested in explaining relative or absolute social inequalities. Certainly this approach needs to be examined in other populations. In this Finnish cohort, conventional risk factors account for the vast majority of CHD cases and for a substantial portion of absolute social inequalities in CHD. In a low risk population free from conventional risk factors, the excess risk among the least educated is 72% lower. This 72% reduction in excess risk is probably conservative, given our crude definition of a low risk population. On this point, table 3 shows that in the low risk population, there were no differences in overweight or blood pressure but the least educated men were more likely to be former smokers, have higher total cholesterol and low density lipoprotein (LDL), lower levels of vigorous physical activity and cardiorespiratory fitness, and were 4.5 cm shorter than better educated men. This suggests that there remain residual differences associated with known risk factors that are not captured by our clinically defined categorisation of low risk<sup>36</sup> but that nevertheless may contribute to both overall levels and social

inequalities in CHD risk in this segment of the population. Thus, it is probable that at least some of the residual absolute social inequality in CHD (excess risk = 25/1000) in the low risk population is also partly attributable to increased levels of conventional risk factors that did not meet out clinical cut-points. However, it may also be the case that the residual social inequality in CHD that persists in a population with no conventional CHD risk factors is attributable to the direct effects of other novel mechanisms such as stress, job control, etc, that do not work through conventional risk factors. There are plausible hypotheses concerning direct psycho-neuro-endocrine mediation<sup>37</sup> of social inequality that may bypass conventional risk factor mechanisms. Nevertheless, in this population such non-conventional CHD mechanisms account for a small number of cases compared with those accounted for by conventional risk factor mechanisms. This should not be taken to mean that psychosocial processes are unimportant to CHD.<sup>38</sup> When psychosocial processes influence behaviours and/or conventional risk factors they are extremely important antecedent causes of conventional risk factors. However, if psychosocial processes are hypothesised to not operate through conventional risk factors, then their contribution to population levels and absolute social inequalities in CHD may be small, but they may contribute importantly to relative CHD inequality.

## Limitations

A potential limitation of these analyses is the assumption that all cases exposed to at least one of the conventional risk factors were actually caused by conventional risk factors singly or in combination.<sup>36</sup> Additionally, we are aware of the theoretical limitations of using PAR to assess the contribution of certain risk factors to the amount of disease because it is possible that different combinations of risk factors can mean that the PAR can sum to more than 100%.<sup>39</sup> Nevertheless, it is difficult to propose an alternative set of risk factors that might be the true causal culprits masquerading behind conventional risk factors. Thus, while attributing 94.6% of cases to conventional risk factors may be too high, we also know that the PAR is underestimated at 70%–75%. Wherever the true PAR lies between 70% and 95%, it is reasonable for the purposes of this illustration to conclude that the contribution of conventional risk factors to CHD is large and of major public health importance.<sup>20 21</sup>

A further limitation is that the illustration of overall and social inequality in CHD in the low risk segment of the



**Figure 1** Relative and excess risks associated with social inequality in CHD in the whole KIH population and a low risk subset of that population. \*Excess and relative risk compares least with most educated.

**Table 3** Distribution of selected characteristics (means or percentage) in the low risk population according to education

Risk factor	Education			p Value
	<primary school	Some high school	High school graduate	
Former smokers (%)	58.20	53.26	48.98	p<0.001
HDL cholesterol (mmol/l)	1.42	1.33	1.35	p=0.06
LDL cholesterol (mmol/l)	3.34	3.31	3.17	p=0.06
Total cholesterol (mmol/l)	5.24	5.17	4.99	p=0.02
Diastolic blood pressure (mm Hg)	81.61	81.00	80.53	p=0.41
Systolic blood pressure (mm Hg)	123.47	123.62	122.55	p=0.59
Self report weight at 20 (kg)	68.67	68.11	68.37	p=0.84
Self report heaviest weight (kg)	82.60	82.45	82.42	p=0.99
Current weight (kg)	77.33	78.10	78.02	p=0.84
Height (cm)*	171.42	173.70	176.09	p<0.0001
Vigorous physical activity (log h/y)	3.83	4.28	4.68	p<0.0001
Cardiorespiratory fitness (ml O <sub>2</sub> /kg/min)	33.08	35.04	36.56	p<0.001

\*Not adjusted for age.

population is admittedly based on small numbers as shown by the wide confidence intervals reported in table 2. However, this is unavoidable and results from the fact that the prevalence of conventional risk factors is high and the number of cases attributable to having at least one risk factor is high (94.6% of all cases). Our population is not overly extreme in this regard. Similarly small proportions of individuals with no conventional risk factors have been noted in other studies,<sup>25</sup> and should be expected in populations where chronic CHD is a major cause of death.

Basing our estimates on the low risk population, as defined here, is nevertheless artificial, because it would not be possible in practice to entirely eliminate conventional risk factors from populations. However, our purpose was to show the principle behind examining these issues from both an absolute and relative perspective and so we have presented the most extreme case. A less extreme reduction in conventional risk factors (than the 100% shown here) would still substantially reduce the absolute burden of CHD associated with the social gradient because the prevalence of conventional risk factors is high (exceeds 80%) and the proportion of CHD cases attributable to them is high in each educational group, and this is a focus of future studies. Furthermore, if the change to a low risk population like the one described here could be achieved, this would be considered socially progressive because the absolute and relative reduction in CHD is largest among the least educated.<sup>40</sup>

### Explaining relative and absolute social inequalities in CHD

When investigating relative social inequalities in CHD, the apparent paradox may arise that the factors that explain most cases of CHD do not seem to explain relative social inequalities in CHD. In our example, we showed that adjustment for conventional risk factors only reduced the relative educational inequalities in CHD by 24%. While this is an underestimate, such results are normally interpreted to mean that most of the effects of social inequality on CHD do not work through mechanisms linked to conventional risk factors,<sup>14</sup> and so other potential causes of relative social inequalities need to be investigated. If we pursue this line of reasoning in these data, adjustment of the relative educational inequalities for a single identified psychosocial risk factor, such as hopelessness,<sup>34</sup> reduces the relative educational inequality by 28%, compared with the reduction of 24% achieved by adjusting for four conventional risk factors. If we ignore that this effect of hopelessness on CHD is partly confounded by conventional risk factors, these results would normally be interpreted to mean that hopelessness is at least as important a mechanism for explaining the relative inequality as

conventional risk factors. However, if we could intervene and remove all hopelessness from this population we would eliminate 14% of CHD cases, compared with over 90% of cases eliminated through removing smoking, hypertension, dyslipidaemia, and diabetes.

The extent to which a third variable reduces the RR in an exposure-outcome association (sometimes taken as an indication of confounding of the association) depends on the relative distributions of the exposure over strata of the third variable and the strength of its association with the outcome. In this case there is a more extreme relative distribution of education over strata of hopelessness than over strata of the conventional risk factors (partly because the prevalence of conventional risk factors is high in this population), and so hopelessness seems to be a stronger confounder (in this case interpreted as a mechanism) of the association between education and CHD.

This situation is symmetrical with the point made by Geoffrey Rose two decades ago.<sup>41</sup> If everyone in a population smoked, then relative social inequalities in lung cancer would be associated with social differences in other causes of lung cancer, such as asbestos exposure. However, while intervening on asbestos exposure would reduce relative social inequalities in lung cancer, it would do far less to mitigate the population burden of lung cancer because smoking causes most cases of lung cancer in all social groups. Evaluating the "importance" of different risk factors for elucidating the mechanisms behind social inequalities in CHD should not be based only on reductions in relative risk. It is widely recognised that a singular reliance on relative indicators can be misleading for both clinical practice<sup>42</sup> and public health policy.<sup>43</sup> Geoffrey Rose put it succinctly, "Relative risk is not what decision-taking requires ... relative risk is only for researchers; decisions call for absolute measures." (page 19).<sup>44</sup>

Explaining the mechanisms behind relative social inequalities in CHD is a legitimate and important focus of research because it may lead to an understanding of novel and reversible CHD risk factors that will reduce social inequality in CHD. Nevertheless, explanations for relative social inequalities need to be understood within the context of

### What is already known on this subject

Conventional risk factors seem not to explain social inequalities in CHD. Therefore, there must be other risk factors that generate the social gradient in CHD. This idea has become a powerful influence on how people understand social inequalities in CHD and what should be done to reduce them.

### What this paper adds

Understanding the causes of social inequalities in CHD may depend on whether one is interested in explaining absolute or relative inequalities in CHD. Conventional risk factors account for the vast majority of CHD cases and for a substantial portion of absolute social inequalities in CHD—72% of the excess risk. An absolute risk approach to understand social inequalities in CHD focuses attention on those risk factors that cause most cases of disease attributable to social inequality. If the concern is to reduce the overall population health burden of CHD and the disproportionate population health burden associated with the social inequalities in CHD, then reducing conventional risk factors will do the job.

what causes the population health burden of CHD—that is, what causes most cases of CHD. In populations where the prevalence of conventional risk factors is high, it is possible that there are small or even no differences in their prevalence across social groups such that they cannot account for relative CHD differences across social groups but contribute substantially to the absolute risk of CHD within all social groups.<sup>31</sup> For instance, the Whitehall II study has reported no social inequality in perhaps the primary CHD risk factor—cholesterol.<sup>45</sup> We have shown here that removing conventional risk factors from the population may have little effect on relative social inequalities but a large effect on absolute social inequalities as indicated by a reduction in the excess risk. Importantly, this also means that whatever proximal and distal factors may be proposed as causes of relative social inequalities in CHD, if their behavioural and biological mechanisms do not involve conventional risk factors then they probably account for a small proportion of CHD cases.

The effects of deliberate population interventions to reduce conventional risk factors are limited,<sup>46</sup> but nevertheless, improvements in conventional risk factors have occurred in many countries<sup>47</sup> and in all social groups, albeit unequally.<sup>48–49</sup> Furthermore, their contributions to CHD decline via combinations of secular shifts, primary and secondary prevention has been reported in several countries.<sup>50–51</sup> The determinants of cholesterol levels, blood pressure, and insulin resistance have not been fully explicated, and processes including fetal and early life development may be important. Improved understanding of the genesis of these conventional risk factors could increase ability to intervene effectively especially among children and adolescents.<sup>52</sup>

People make behavioural choices but they do so within layers of social context.<sup>12</sup> Simply admonishing people for their bad habits is at worst victim blaming, at best naive. Health behaviours are influenced over the lifecourse<sup>35–52</sup> at

multiple levels by the material, psychosocial, cultural, and family conditions in which people live.<sup>53</sup> The development and maintenance of smoking, diet, and exercise habits that influence levels of conventional CHD risk factors cannot be construed simply as the result of individual choice because choice is shaped by the physical, cultural, and social environments in which people live and work. A combination of population wide shifts in the distribution of risk factors and individual change strategies for those at high risk, in both early and later life is required. Ultimately this will mean engaging the political and economic forces that have interests in maintaining profits from the sale of products and services that influence conventional CHD risk factors.

The findings of this study in no way alleviate the need to better understand and ameliorate the uneven distribution of conventional CHD risk factors across social groups.<sup>12</sup> However, using an absolute risk approach to understand social inequalities in CHD focuses attention on those risk factors that cause most cases of disease attributable to social inequality.<sup>54–56</sup> So if the concern is to reduce the overall population health burden of CHD and the disproportionate population health burden associated with the social inequalities in CHD, then reducing conventional risk factors will do the job. We should increase efforts to find ways to influence the multiple pathways from international, national, and local policy<sup>46–57–58</sup> through to individual behaviour that will reduce conventional risk factors among current and future generations in richer and poorer countries.

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### CONTRIBUTIONS

All authors contributed to the analysis, interpretation, and writing of the manuscript.

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### Policy implications

The effectiveness of planned interventions to change levels of conventional risk factors and health behaviours has proved to be limited. Nevertheless, reducing levels of these risk factors will reduce the population health burden of social inequalities in CHD because it will reduce absolute risk in all social groups. We should increase our efforts to find ways to influence the multiple pathways from international, national, and local policy through to individual behaviour and treatment that will reduce conventional risk factors among current and future generations in richer and poorer countries.

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## SPECIAL REPORT

## A Potential Decline in Life Expectancy in the United States in the 21st Century

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### SUMMARY

Forecasts of life expectancy are an important component of public policy that influence age-based entitlement programs such as Social Security and Medicare. Although the Social Security Administration recently raised its estimates of how long Americans are going to live in the 21st century, current trends in obesity in the United States suggest that these estimates may not be accurate. From our analysis of the effect of obesity on longevity, we conclude that the steady rise in life expectancy during the past two centuries may soon come to an end.

The trend in the life expectancy of humans during the past thousand years has been characterized by a slow, steady increase<sup>1,2</sup>—a pattern frequently punctuated by a volatility in death rates caused by epidemics and pandemic infectious diseases, famines, and war.<sup>3,4</sup> This volatility was dramatically curtailed in the mid-19th century as infectious diseases yielded swiftly to improved living conditions, advances in public health, and medical interventions. During the past 30 years, the rise in life expectancy at birth in the United States decelerated relative to this historical pattern, and gains in life expectancy at older ages are now much smaller than they were in previous decades.<sup>5</sup>

How much higher can life expectancy rise? This is not just an academic question. The answer formulated today will have substantial influence on the rate at which taxes are levied and on the potential solvency of age-entitlement programs. Some scientists answer this question by extrapolating from historical trends, which has led to the recent prediction that life expectancy at birth will rise to 100 years in the United States and other developed nations by the year 2060.<sup>6</sup> The United Nations used

a similar method but different assumptions to arrive at a projected life expectancy of 100 years for males and females in most countries by the year 2300.<sup>7</sup> The Social Security Administration (SSA) arrived at a more tempered but still optimistic view that life expectancy in the United States will continue its steady increases, reaching the mid-80s later in this century.<sup>8</sup>

A recently convened panel of advisers,<sup>9</sup> and some mathematical demographers who advocate the use of extrapolation,<sup>10</sup> have advised the SSA to project an even more rapid rate of increase in life expectancy for the U.S. population beyond that already anticipated between now and the latter part of this century. The bases for this advice include a demonstration that the maximum life span in Sweden has increased since the mid-19th century,<sup>11</sup> the world record for life expectancy at birth in developed nations has been increasing by three months per year since 1850, mortality declines occurred at older ages in the Group of Seven industrialized nations during the latter half of the 20th century,<sup>12</sup> and the prediction that “negligible senescence” will be scientifically engineered for humans in this century.<sup>13</sup> Negligible senescence is defined as age-specific mortality rates that remain constant throughout life as opposed to rising exponentially after puberty, which is common among humans and most other animals. This last point is important because it is the only “biologic” justification offered for the decision to raise forecasts of life expectancy.

Life-extending technology that might lead to much higher life expectancies does not yet exist and, should it be developed, must be widely implemented before it would influence statistics on population levels. We believe that potential forms of technology do not justify developing or revising forecasts of life expectancy. Extrapolation models fail to consider the health status of people currently

alive and explicitly assume that the past can predict the future. Given that past gains in life expectancy have largely been a product of saving the young, and since future gains must result from extending life among the old, another quantum leap in life expectancy can occur only if the future is different from the past.<sup>14</sup>

An informed approach to forecasting life expectancy should rely on trends in health and mortality that may be observed in the current population. Forecasting life expectancy by extrapolating from the past is like forecasting the weather on the basis of its history. Looking out the window, we see a threatening storm — obesity — that will, if unchecked, have a negative effect on life expectancy. Despite widespread knowledge about how to reduce the severity of the problem, observed trends in obesity continue to worsen. These trends threaten to diminish the health and life expectancy of current and future generations.

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#### THE RISE IN OBESITY

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After remaining relatively stable in the 1960s and 1970s, the prevalence of obesity among adults in the United States increased by approximately 50 percent per decade throughout the 1980s and 1990s.<sup>15</sup> Two thirds of adults in the United States today are obese or overweight. In the United States, 28 percent of men, 34 percent of women, and nearly 50 percent of non-Hispanic black women are currently obese. The distribution of body-mass index (BMI, the weight in kilograms divided by the square of the height in meters)<sup>16,17</sup> has shifted in a skewed fashion such that the proportion of people with extreme obesity has increased at an especially rapid rate. These trends have affected all major racial and ethnic groups, all regions of the country, and all socioeconomic strata,<sup>18</sup> with the largest increases in obesity occurring among children and minorities.<sup>19</sup> It should be noted, however, that data from studies on the relation between BMI and mortality have been interpreted by some to suggest that current tables of ideal height and weight and, by extension, ideal ranges of BMI should be adjusted to include lower ideal weights for height or BMIs before age 40, as well as higher ideal weights for height or BMIs after age 40.<sup>20</sup> Redefining ideal weights for height in this way would increase the projected negative effect of obesity on life expectancy because of the large increases in obesity now observed among people under age 40.

Obesity is a multisystem condition associated with an elevated risk of type 2 diabetes, coronary heart disease, cancer, and other complications.<sup>21,22</sup> The effect of body weight on mortality has been studied extensively. In a study of more than a million U.S. adults, the lowest death rates were found among men with a BMI of 23.5 to 24.9 and among women with a BMI of 22.0 to 23.4.<sup>23</sup> Death rates from cardiovascular diseases were substantially elevated among people with higher BMIs. A prospective study of 6139 subjects in Germany found the greatest obesity-associated excess mortality to be among the young<sup>24</sup> — the standardized mortality ratio for people 18 to 29 years of age with a BMI of 40 or over was 4.2 in men and 3.8 in women. Fontaine et al. estimated the effect of obesity on years of life lost across the lifespan of adults.<sup>25</sup> For any degree of excessive body weight, young age was associated with greater years of life lost. Allison et al. used data from six cohort studies in the United States to determine that obesity causes approximately 300,000 deaths per year,<sup>26</sup> although a study by the Centers for Disease Control and Prevention (CDC)<sup>27</sup> may have overestimated deaths due to obesity.

Being overweight in childhood increases the risk among men of death from any cause and death from cardiovascular disease; it also increases the risk of cardiovascular morbidity among both men and women.<sup>28</sup> The lifetime risk of diabetes among people born in the United States has risen rapidly to 30 to 40 percent — a phenomenon presumably attributable to the obesity “epidemic.”<sup>29</sup> Having diabetes in adulthood increases the risk of a heart attack by as much as having had a previous heart attack,<sup>30</sup> and the life-shortening effect of diabetes is approximately 13 years.<sup>31</sup> Evidence also suggests that at younger ages, disability rates have risen and fitness levels have declined dramatically in the United States, with both trends attributed, at least in part, to the rise in obesity.<sup>32,33</sup> The incidence of type 2 diabetes in childhood in the United States has increased many times over in the past two decades, an increase that is due almost entirely to the obesity epidemic<sup>34,35</sup>; shockingly, life-threatening complications, including renal failure, may develop by young adulthood in at least 10 percent of children with type 2 diabetes.<sup>36</sup>

If left unchecked, the rising prevalence of obesity that has already occurred in the past 30 years is expected to lead to an elevated risk of a range of fatal and nonfatal conditions for these cohorts as they age.<sup>29</sup> If the prevalence of obesity continues to rise,

especially at younger ages, the negative effect on health and longevity in the coming decades could be much worse. It is not possible to predict exactly when obesity among the young will have its largest negative effect on life expectancy. However, in the absence of successful interventions, it seems likely that it will be in the first half of this century, when at-risk populations reach the ages of greatest vulnerability.

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OBESITY AND FUTURE  
LIFE EXPECTANCY

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Obesity has been shown to have a substantial negative effect on longevity, reducing the length of life of people who are severely obese by an estimated 5 to 20 years.<sup>25</sup> Although the life-shortening effect of obesity is evident for people who are obese, its negative effect on the future life expectancy of the population is also critically important to public policy.

We have estimated the effect of obesity on the life expectancy of the U.S. population by calculating the reduction in the rates of death that would occur if everyone who is currently obese were to lose enough weight to obtain an “optimal” BMI, which we defined as a BMI of 24. This calculation can be performed by linking data on the age-, race-, and sex-specific prevalence of obesity in the United States from the Third National Health and Nutrition Examination Survey<sup>15</sup> with estimates of race- and sex-specific rates of death for people 20 to 85 years of age who have BMIs that range from 17 to 45.<sup>37</sup> Complete life tables for the United States according to race and sex for 2000 were obtained from the National Center for Health Statistics.<sup>38</sup>

The calculations begin by estimating the conditional probability of death at age  $x$  of the nonobese population,  $q(x_{no})$ , as follows:

$$q(x_{no}) = [q(x) - q(x_o)P(x_o)] \div P(x_{no}),$$

where  $q(x)$  is the conditional probability of death at age  $x$  for the entire population,  $q(x_o)$  is the conditional probability of death at age  $x$  for the obese population,  $P(x_o)$  is the proportion of the population at age  $x$  that is obese, and  $P(x_{no})$  is  $1 - P(x_o)$ , which is the proportion of the population at age  $x$  that is not obese.<sup>15,37,38</sup>

Because the distribution of BMIs is skewed toward higher levels, and the population is shifting toward higher BMIs, we provide a range of estimates

for the effect of obesity on life expectancy by assuming that everyone who is obese has a BMI of 30 or 35. The effect of eliminating obesity on the life expectancy of the population (encompassed within this range) is then estimated by assuming that everyone who is obese acquires the mortality risk,  $q(x_{24})$ , of those with the optimal BMI of 24. The obesity-adjusted conditional probability of death,  $q(x_a)$ , at age  $x$  may then be obtained as follows:

$$q(x_a) = q(x_{no})P(x_{no}) + q(x_{24})P(x_o).$$

These obesity-adjusted conditional probabilities of death were estimated separately by race and sex. When a BMI of 30 or 35 did not lead to lower death rates than the optimal BMI of 24, it was assumed that there was no change in the conditional probability of death. This occurred for black males at 62 to 85 years of age at a BMI of 30 and at 67 to 85 years of age at a BMI of 35, and for black females at 60 to 85 years of age at a BMI of 30 and at 67 to 85 years of age at a BMI of 35. These calculations yielded a range of age-, race-, and sex-specific rates of death and hypothetical life tables for the year 2000 under the assumption that obesity has been eliminated.

A more detailed analysis of this kind could be performed using the full age distribution of BMIs and integrating these into the calculations at a finer level of detail. However, given that the methods required for such estimates are currently being reviewed by a panel of scientists convened by the Institute of Medicine, and in the interest of identifying plausible estimates rather than precise numbers, we took this simpler approach.

Our approach probably underestimates the negative effect of obesity on life expectancy at BMIs of 30 and overestimates it at BMIs of 35. Clearly, the absence of rates of death for obese people under 19 years of age (which forces the assumption that rates of death in this age range remained unchanged from the levels in 2000) leads to an underestimate of the overall effect of obesity on life expectancy. Finally, it is possible that the use of period (cross-sectional) rather than cohort data may also lead to substantial underestimates of the effects of obesity on life expectancy.<sup>39</sup>

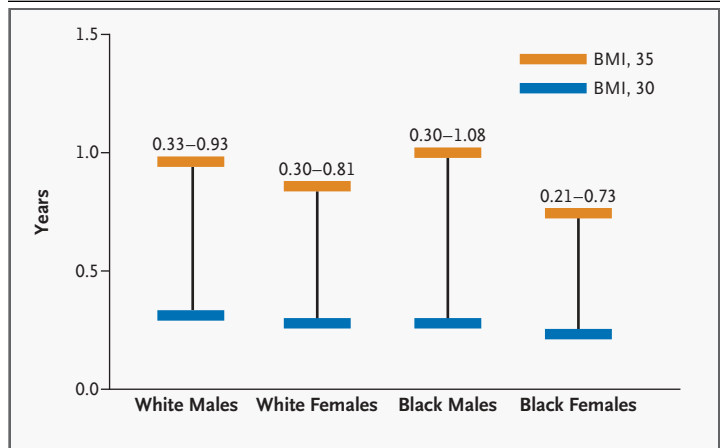
Our conservative estimate is that life expectancy at birth in the United States would be higher by 0.33 to 0.93 year for white males, 0.30 to 0.81 year for white females, 0.30 to 1.08 years for black males, and 0.21 to 0.73 year for black females if obesity did

not exist (Fig. 1). Assuming that current rates of death associated with obesity remain constant in this century, the overall negative effect of obesity on life expectancy in the United States is a reduction in life expectancy of one third to three fourths of a year. This reduction in life expectancy is not trivial — it is larger than the negative effect of all accidental deaths combined (e.g., accidents, homicide, and suicide),<sup>40</sup> and there is reason to believe that it will rapidly approach and could exceed the negative effect that ischemic heart disease or cancer has on life expectancy.

Several facts that suggest that the prevalence and severity of obesity and its complications will worsen and that rates of obesity-induced death will rise are as follows: current estimates of the effect of eliminating obesity are based on past trends, when the prevalence was much lower; the prevalence of obesity, especially among children, is likely to continue to rise; with obesity occurring at younger ages, the children and young adults of today will carry and express obesity-related risks for more of their lifetime than previous generations have done; a significant shift toward higher BMIs throughout the age ranges has occurred; death rates from diabetes have risen steadily in the past 20 years and are expected to rise further as younger cohorts age; and the medical treatment of obesity has been largely unsuccessful.<sup>22</sup> These trends suggest that the relative influence of obesity on the life expectancy of future generations could be markedly worse than it is for current generations. In other words, the life-shortening effect of obesity could rise from its current level of about one third to three fourths of a year to two to five years, or more, in the coming decades, as the obese who are now at younger ages carry their elevated risk of death into middle and older ages.

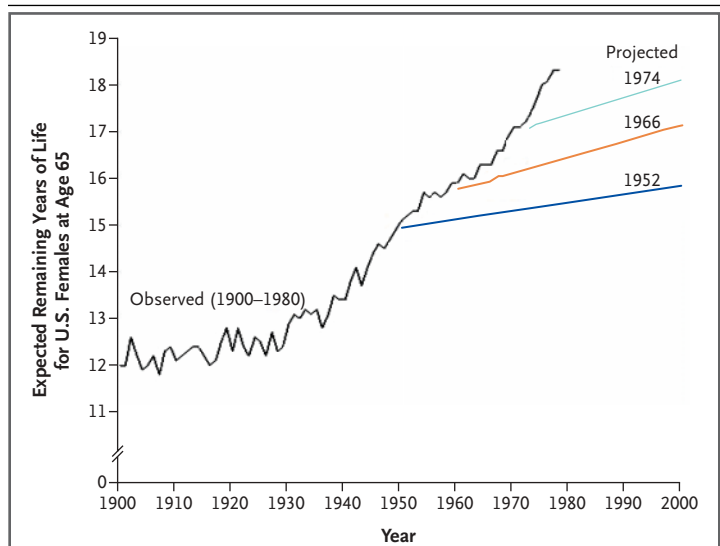
DISCUSSION

A rise in life expectancy to 100 years in the United States in this century would profoundly influence many aspects of society,<sup>41</sup> including the solvency of age-based entitlement programs and tax rates levied by the federal government. Even marginal increases in life expectancy beyond those anticipated by the SSA would markedly increase the number of octogenarians, nonagenarians, and centenarians that the SSA expects. However, in light of the obesity-driven trends in the health status of the U.S. popula-



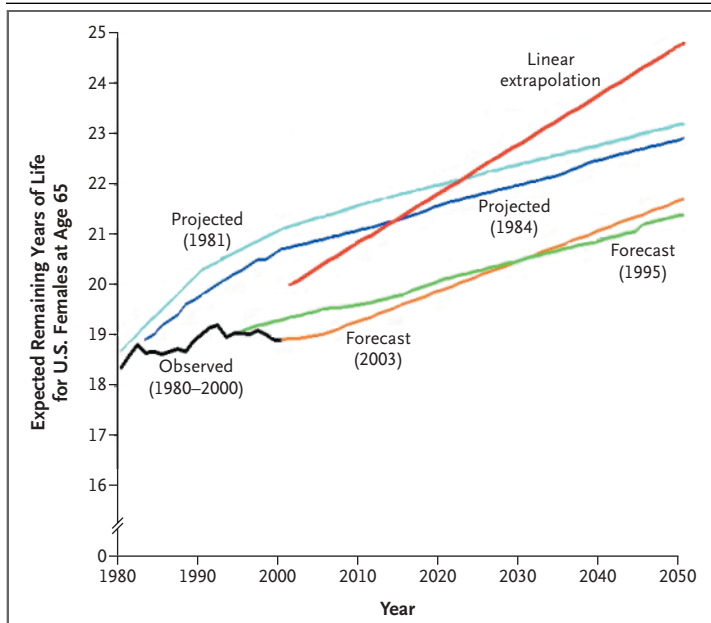
**Figure 1. Life-Shortening Effect of Obesity According to Race and Sex in the United States in 2000.**

This figure shows the potential gain in life expectancy at birth for the U.S. population in 2000, by race and sex, if obesity were eliminated. The range of estimates is shown between the bars on the basis of the assumption that everyone who is obese has a body-mass index (BMI) between 30 (lower bar) and 35 (upper bar) and acquires the risk of death of people with a BMI of 24. The horizontal bars are not error bars.



**Figure 2. Observed and Projected Life Expectancy at Age 65 for U.S. Females (1900 to 2000).**

Shown are observed changes, from 1900 to 1980,<sup>42</sup> in expected remaining years of life at age 65 for females in the United States, and projections of the expected remaining years of life at age 65 made by the SSA in actuarial studies published in 1952,<sup>43</sup> 1966,<sup>44</sup> and 1974.<sup>45</sup>



**Figure 3. Observed and Projected Life Expectancy at Age 65 for U.S. Females (1980 to 2050).**

Shown are observed changes, from 1980 to 2000,<sup>46</sup> in expected remaining years of life at age 65 for females in the United States, projections of the expected remaining years of life at age 65 made by the SSA in actuarial studies published in 1981<sup>46</sup> and 1984,<sup>47</sup> and forecasts based on the SSA's 1995 and 2003 Trustees Reports.<sup>48,49</sup> A forecast of the expected remaining years of life at age 65 for females in the United States, assuming the observed trend from 1940 to 2000 is extrapolated linearly from 2000 to 2050, is shown.

tion (especially the young), the bases for the SSA's recent decision to raise its midrange forecasts of life expectancy beyond the increases anticipated during the next 70 years merit reconsideration.

Figure 2 shows the historical trend in life expectancy at age 65 for females in the United States from 1900 through the inception of the Social Security program, in 1935, and formal projections of the rise in life expectancy at age 65 made by the SSA at various times since then. Before 1980, the SSA consistently underestimated the subsequent rise in life expectancy at age 65 because it assumed that recently observed gains could not be sustained. After 1980, this position was reversed, and the SSA began tracking and extrapolating more recently observed trends in life expectancy at age 65. Ironically, this change in approach occurred just when the rise in life expectancy began to stall (Fig. 3). The fact is, life expectancy at age 65 for females has remained largely unchanged for most of the past 20 years.

A central problem with the SSA's forecast is best

illustrated by its projections for diabetes. From 1979 to 1999, rates of death from diabetes increased annually by an average of 2.8 percent for males and 1.8 percent for females. In 1990, diabetes decreased life expectancy by 0.22 year for males and 0.31 year for females,<sup>40</sup> but the negative effect of diabetes on life expectancy has grown rapidly since then. However, the negative effect of diabetes on the life expectancy of the population could now be several times as great as it was in 1990.<sup>31</sup> Given the rapidly rising prevalence of diabetes and the prospect that childhood obesity today will probably accelerate the rising prevalence of diabetes in the coming decades, it is difficult to justify the SSA's assumption that rates of death from diabetes will decline by 1.0 percent to 3.2 percent annually throughout the 21st century, beginning in the year 2010.<sup>5</sup>

We anticipate that as a result of the substantial rise in the prevalence of obesity and its life-shortening complications such as diabetes, life expectancy at birth and at older ages could level off or even decline within the first half of this century. This is in contrast to both the recent decision by the SSA to raise its forecast of life expectancy and what we consider to be the simple but unrealistic extrapolation of past trends in life expectancy into the future.

There are other realistic threats to increases in life expectancy. From 1980 to 1992 in the United States, the age-adjusted rate of death from infectious diseases rose by 39 percent, an increase fueled mostly by the AIDS epidemic; the overall rate of death from infectious diseases increased 4.8 percent per year from 1980 to 1995<sup>50</sup>; hospital-acquired infections have increased<sup>51,52</sup>; hospital-acquired and antibiotic-resistant pathogens have entered the community and our food supply<sup>53,54</sup>; and recent decreases in mortality related to the human immunodeficiency virus have leveled off.<sup>55</sup>

Infectious diseases could decrease life expectancy substantially if pandemic influenza strikes.<sup>56</sup> Developing and developed nations are far more vulnerable to a global pandemic of influenza today than in 1918, owing to an aging population, resistance to antibiotics, and more rapid transport of microbes, among other reasons. This heightened risk is balanced in part by better global surveillance and interventions already present.<sup>57</sup> Although estimating the negative effects of epidemics on the future course of life expectancy is problematic, it has been established that infectious diseases, when they do emerge, can wipe out a century's worth of gains in health and longevity in less than one generation.<sup>58</sup>

Other forces that could attenuate the rise in life expectancy include pollution, lack of regular exercise, ineffective blood-pressure screening, tobacco use, and stress.

Advances in the medical treatment of major fatal diseases, including the complications of obesity, are likely to continue. Unfortunately, recent trends in the prevalence of cancer and in the rates of death from cardiovascular diseases in the United States reveal only marginal gains in longevity in recent decades,<sup>59,60</sup> and even the gains produced from the elimination of any one of today's major fatal diseases<sup>61</sup> would not exceed the negative effects of obesity that appear to be forthcoming.

A leveling off or decline in life expectancy in the United States is not inevitable. We remain hopeful that the public health community and public policymakers will respond to the impending dangers that obesity poses to both the quality and the length of life. However, the negative effect on health and longevity of unchecked obesity is substantial according to statistics on health and mortality that can be observed for the generations currently alive, as has already been shown in Okinawa, Japan.<sup>62</sup> It is important to emphasize that our conclusions about the future are based on our collective judgment, as are all forecasts, and we acknowledge that forces that influence human mortality can change rapidly.

Finally, our forecast has other public-policy implications. Dire predictions about the impending bankruptcy of Social Security based on the SSA's projections of large increases in survival past 65 years of age appear to be premature. However, this "benefit" will occur at the expense of the economy in the form of lost productivity before citizens reach retirement and large increases in Medicare costs associated with obesity and its complications.<sup>63</sup> Presently, annual health care costs attributable to obesity are conservatively estimated at \$70 billion to \$100 billion.<sup>64,65</sup> With rapid increases in the prevalence of diabetes, and a decrease in mean age at the onset of diabetes, the cost of treating diabetes-related complications, such as heart disease, stroke, limb amputation, renal failure, and blindness, will increase substantially. A similar escalation of health care costs from other complications associated with obesity (e.g., cardiovascular disease, hypertension, asthma, cancer, and gastrointestinal problems) is inevitable. The U.S. population may be inadvertently saving Social Security by becoming more obese, but the price to be paid by obese people themselves and the economy is already high enough to justify

considerably increased spending on public health interventions<sup>66</sup> aimed at reducing the incidence and severity of obesity.

Unless effective population-level interventions to reduce obesity are developed, the steady rise in life expectancy observed in the modern era may soon come to an end and the youth of today may, on average, live less healthy and possibly even shorter lives than their parents. The health and life expectancy of minority populations may be hit hardest by obesity, because within these subgroups, access to health care is limited and childhood and adult obesity has increased the fastest.<sup>67</sup> In fact, if the negative effect of obesity on life expectancy continues to worsen, and current trends in prevalence suggest it will, then gains in health and longevity that have taken decades to achieve may be quickly reversed. The optimism of scientists and of policymaking bodies about the future course of life expectancy should be tempered by a realistic acknowledgment that major threats to the health and longevity of younger generations today are already visible.

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The assumptions and conclusions herein are the sole responsibility of the authors.

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