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Issue: *The Biology of Disadvantage***Multiple risk exposure as a potential explanatory mechanism for the socioeconomic status–health gradient**Gary W. Evans<sup>1</sup> and Pilyoung Kim<sup>2</sup><sup>1</sup>Cornell University, Ithaca, New York. <sup>2</sup>National Institute of Mental Health, Bethesda, Maryland

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The social patterning of disease and mortality provokes a search for explanation. One potential underlying explanation for socioeconomic status (SES) gradients in health is exposure to multiple risk factors. Income and class tend to sort individuals into different settings that are often accompanied by systematic differences in environmental quality. Housing and neighborhood quality, pollutants and toxins, crowding and congestion, and noise exposure all vary with SES. Persons lower in SES also experience more adverse interpersonal relationships with family members, friends, supervisors, and community members. Furthermore, exposure to these multiple risk factors is associated with worse health outcomes. Thus, the convergence of exposure to multiple physical and psychosocial risk factors accompanying disadvantage may account for a portion of SES gradients in health in both childhood and adulthood.

Keywords: multiple risk; cumulative risk; SES; health; poverty

**Introduction**

Other papers in this volume document the ubiquitous relationship between socioeconomic status (SES) and health. This paper addresses the question—how does this happen? Why does one's social address play such a formidable role in health, virtually from conception to death? One potentially powerful mechanism underlying the SES–health gradient is multiple risk exposure.

Multiple risk exposure refers to experiencing more than one risk at a time. Some of the exposure to multiple risk factors is simultaneous—crowded living environments are frequently noisy; high conflict families tend to be harsh and unresponsive in their parenting. Some forms of risk covariation are sequential, triggering a cascade of other adverse events and circumstances (e.g., job loss, divorce).<sup>1</sup> Other salient examples of sequential risk proliferation are teenage pregnancy, dropping out of high school, residential relocation, trauma or a major illness. Each of these circumstances can set off a chain of subsequent events and circumstances capable of compromising health. Note that each of these simultaneous (e.g., crowding and noise) and sequential (e.g., teenage pregnancy) examples of multiple

risk are inversely related to SES. Lower SES households are more crowded and noisier in comparison to higher SES households. Lower SES teenage girls are more likely to become pregnant than their more affluent counterparts. Most research on risk factors and health examines singular risk factors in order to understand their unique contribution to health. But what happens when risks are experienced together? We know across a wide range of risk factors that the adverse health effects of multiple risk exposure exceed singular risk exposure.

In this paper, we examine whether multiple risk exposure could account in part for the SES:health gradient (Fig. 1). In order for multiple risk exposure to be a viable underlying, explanation of the SES:health gradient, several conditions must be met. First, there needs to be a linear relation between SES and multiple risk exposure. The evidence for this is sparse. There is reasonable evidence of linear relations between singular risk factors and SES, but much less is known about multiple risk exposure across varying levels of SES. Moreover, most research on SES and multiple risk exposure compares disadvantaged to advantaged populations. Two data points do not allow us to draw firm conclusions about the degree of linearity in the SES: multiple



**Figure 1.** Multiple risk exposure as a mediating mechanism for social gradients in health.

risk exposure gradient. One aspect of this question of particular interest is whether multiple risk exposures accumulate nonlinearly at the low end of the SES ladder or are they distributed linearly as one moves from the bottom to the top of the ladder? Although there is not much data, we will see that nearly all of the studies that do have three or more levels of SES suggest a linear relation between SES and multiple risk exposure.

A second prerequisite for investigating the multiple risk model of SES and health depicted in Figure 1 is that the putative mediator, multiple risk exposure, be linearly related to health outcomes. This requirement raises several challenges that we will discuss herein. To begin with, most of the data on multiple risk exposure and human reactions is not on health, instead focusing on cognitive and socioemotional outcomes, primarily in children. A second more complex challenge is the issue of how best to operationalize multiple risk exposure. Two issues that we will discuss herein are additive compared to interactive multiple risk metrics and how best to represent each risk factor in multiple risk models.

A third criterion for evaluating Figure 1 is evidence showing that the association between SES and health is attenuated when multiple risk exposure is in the model. As we shall see, there is precious little data on this and what does exist is focused on behavioral rather than health outcomes. Multiple risk exposure offers an intriguing and potentially viable explanation for the SES:health gradient, but at present there are major gaps in the evidence base to evaluate this idea.

### Socioeconomic status and multiple risk

There is a large literature showing an inverse relation between SES or its constituent components (income, education, and occupation) and singular indicators of environmental quality including exposure to toxins and hazardous wastes, ambient pollutants, noise, crowding, substandard housing, greater residential mobility, sub par neighborhood conditions including higher traffic volume, abandoned lots, and poorer municipal services such as

sanitation, along with more physically hazardous working conditions on the job.<sup>2–5</sup> We also know for children that the lower the household income, the greater the exposure to each of several, major psychosocial risk factors including being raised by a teenage mother, family turmoil and conflict, maternal partner changes (including divorce), child separation from family, and exposure to violence.<sup>3,6</sup> Family income is also inversely related to the degree of structure and predictability in household routines for children (e.g., regular bedtime, eating family meals together).<sup>7</sup> Furthermore, lower SES families reside in neighborhoods with fewer suitable places to engage in physical exercise and have less access to purchase healthy foods.<sup>8</sup> Each of the above papers covers research on exposure to singular physical or psychosocial risk factors but omit work on SES and multiple risk exposure.

Unfortunately, there is much less data documenting SES and exposure to *multiple* rather than *singular* risk exposure. The largest amount of evidence for SES and multiple risk exposure comes from stressful life events studies. Stressful life events methodology measures the total number of stressful life events persons are exposed to. Some examples of life events are divorce, residential relocation, or job loss. The sum of the number of different events one is exposed to over some period of time is the index of multiple risk exposure. Thus stressful life events are an additive model of multiple risk exposure. Studies consistently reveal that low SES children experience more stressful life events and hassles in total than their wealthier counterparts.<sup>9–13</sup> To gain a sense of the magnitude of differences in exposure to stressful life events in low- versus nonlow-income households, low-income children in grades two to seven were three times more likely (18%) than non low-income children (6%) to experience two or more stressful life events.<sup>13</sup>

SES is related to the number and severity of stressful life events exposure in adults.<sup>14–17</sup> A standardized total stress estimate revealed evidence of linearity in exposure across three levels of SES (–0.277, 0.051, 0.227) for upper, middle, and lower SES adults, respectively among a sample of more than one thousand 18–22 year olds.<sup>18</sup> Table 1 shows data from

**Table 1.** Mean number of lifetime events as a function of income and age

Age (years)	Education (years)			Income (\$1000)		
	<12	12–15	>15	<10	10–29	>29
25–44	0.76	0.53	0.33	0.61	0.57	0.44
46–64	0.84	0.68	0.55	1.11	0.71	0.56
65+	1.00	0.77	0.82	1.07	0.83	0.59

Note: Adapted from Tables 2, 3, and 4 [Wave 1 Stress/Life Event Variables by Socioeconomic Indicators, Ages 25–44 years; Ages 45–64 years; Ages 65 years and older] in Ref. 19. Copyright 2005 by American Sociological Association, adapted with permission.

Lantz *et al.*<sup>19</sup> on a large sample of adults at three different ages in relation to three levels of income. As in the case of Turner and Avison's data with younger adults, there is relatively good evidence of linearity between exposure to life events over one's lifetime and income levels. However, since only three cut points are available, the evidence on linearity is limited.

The stressful life event studies above relate SES to the number of stressful life events individuals are exposed to. There are also a set of studies on multiple risk exposure and SES that incorporate other risk variables in addition to discrete stressful life events. SES among 5–10 year olds was significantly correlated with 15 out of 18 risk factors (stressful life events, single parenthood, high child:adult ratio, teenage pregnancy, unplanned pregnancy, maternal social isolation, low parental involvement of father, marital conflict, violence, harsh discipline, physical harm, lack of positive parenting, maternal attitudes toward aggression, peer rejection, and unstable peer group).<sup>20</sup> Although the distinction between stressful life events and other risk factors is not always clear, in general the stressful life events measures count up the number of major, discrete events that a person has been exposed to such as marital breakup, loss of a spouse or parent, or loss of a job. As can be seen in Table 1, since many of these events are rather major and catastrophic their occurrence is rare.

On the other hand, by adding other less severe lifetime disruptions as well as more enduring, ongoing situations that are demanding (e.g., teenage pregnancy, marital conflict, peer rejection), many multiple risk measures capture a wider and more common set of stressors and hassles that impinge upon individuals throughout daily life. Greenberg *et al.*<sup>21</sup> found that both parental education and

occupation were directly related to the quality of the home and neighborhood environments encountered by kindergarten children across four different geographic sites. Maternal education in a predominantly low-income sample of African American infants was inversely related to household crowding, life events, and positively correlated with maternal responsiveness and the quality of the home environment.<sup>22</sup> From preschool to late adolescence, SES was inversely related to the total number of different caretakers, changes in residential location, and changes in schools.<sup>23</sup>

In a sample of middle school children, Felner *et al.*<sup>24</sup> examined multiple risk factors at home and school in relation to parental education. These data are particularly valuable in the present context because they are one of the few to display multiple risk exposures beyond stressful life events across more than two levels of SES. As shown in Table 2, there is evidence of linearity in multiple risk exposure across three levels of parental education.

In a study of civil service grade and health among a large sample of adults between 35 and 55 years in the United Kingdom<sup>25</sup> showed that social support, job control, and job variety, were related to civil service grade. Two aspects of these data are noteworthy compared to previous descriptive information on SES and multiple risk exposure. First and importantly, Marmot and Wilkinson<sup>25</sup> show evidence across finer gradients of SES, in this case occupational status. The few other studies as detailed above that have information on the linearity of SES and multiple risk exposure are restricted to three levels of SES. Here we have it at six levels (Table 3).

The second noteworthy aspect of these data is the nonlinearity of service grade and risk exposures. On the other hand since the SES index is categorical,

**Table 2. Multiple risk exposure and parental education**

Measure of proximal environmental experiences	Parental educational level		
	No high school	High school	College
Family intellectual/cultural climate	4.21	4.58	5.42
Family active-recreational climate	4.90	5.36	5.97
Mother rejection	4.13	3.46	2.94
Family social support	14.73	15.26	16.59
Belonging at school	2.19	2.41	2.64
Negative life events	4.74	3.63	3.27
Daily hassles	151.76	139.00	138.67

Note: Adapted from Table 5. [Significant effects for parent educational level on proximal environmental experience] in Ref. 24. Copyright 1995 by the Society for Research in Child Development, Inc., adapted with permission.

Occupational Level, the nonlinearity of the trends has to be interpreted with some caution. Nonetheless, the Whitehall civil servants data suggest that risk factors may clump together more at the low end.

Bendersky and Lewis<sup>10</sup> formed a composite risk score by standardizing individual risk factors and then forming a mean. The multiple risk composite was made up of minority status, number of children in household, single parenthood, stressful life events, parenting quality, social network size and mother-child interaction. Among 175 preterm infants, average SES over the first year highly correlated ( $r = 0.49$ ) with their composite multiple risk index exposure during the same period.<sup>10</sup>

A smaller number of multiple risk exposure studies have examined SES in relation to *cumulative risk* exposure. Cumulative risk metrics sum exposures to a series of dichotomous risk factors where 0 = no to moderate risk and 1 = high risk. The risk designation in cumulative risk indices is typically accomplished in two ways. For factors where there is some well-established category of risk such as low birth weight, then all persons with a low birth weight would be assigned = 1, all else = 0. For risk factors that are continuous such as family conflict, a statistical cutoff (e.g., upper quartile) is used for the assignment of 1 = risk and 0 = no risk.

Rutter *et al.* examined 10-year-old children in inner city London and in a working class area. As

**Table 3. Control and variety at work and social support in relation to sex and social class**

	Social class					
	I	II	IIINM	IIIM	IV	V
Men (age ≥ 16)						
Low control over work	6	6	21	22	40	47
Low variety at work	9	18	35	35	62	66
Sever lack of social support	10	12	16	18	21	26
Women (age ≥ 16)						
Low control over work	14	10	36	31	50	46
Low variety at work	8	22	56	52	74	92
Sever lack of social support	6	9	12	13	14	15

Note: Values are percentages for men and women in each social class. Social class I and II are professional and managerial, IIINM is other nonmanual, IIIM is skilled manual, and IV and V semi-skilled and unskilled manual. From a table [Low control and variety at work and severe lack of social support in relation to sex and social class] in Ref. 25. Copyright 2001 by BMJ Publishing Group Ltd, reprinted with permission.

**Table 4.** Cumulative risk exposure in 10-year-olds in relation to SES

	Neighborhood	
	Working class	Innecity
≥ 2 or more family risk factors	19%	52%
Single parent	20%	33%
Father been imprisoned	1%	8%
Crowded household	10%	50%
Public housing	33%	71%
Ever in foster care	7%	10%
High family conflict	7%	10%

Note: Adapted from Tables I [Family disturbance and child psychiatric disorder], II [Current marital status of parents of children not living with both natural parents], III [Parental deviance and child psychiatric disorder], IV, VIII in "Attainment and adjustment in two geographical areas: III—some factors accounting for Area Differences" by Rutter, M *et al.* 1974 Br. J. Psych. 125: 522–5. Copyright 1974 by the Royal College of Psychiatrists. Adapted with permission.

shown in Table 4, the inner city children were more likely to experience two or more risk factors.<sup>26</sup> In a national sample of high risk infants, 35% of low-income toddlers in follow-ups had been exposed to six or more risk factors (<1500 g birth weight, poor neonatal health, ethnic minority, unemployed parent, maternal high school dropout, low IQ, maternal depression, high maternal stressful life events, low maternal social support, teenage motherhood, father absence, high residential density, or negative parenting values). This figure of 35% exposure to high levels of cumulative risk (>6 risk factors) among low income toddlers contrasted markedly with only 5% of middle income toddlers at similar levels of high cumulative risk.<sup>27</sup>

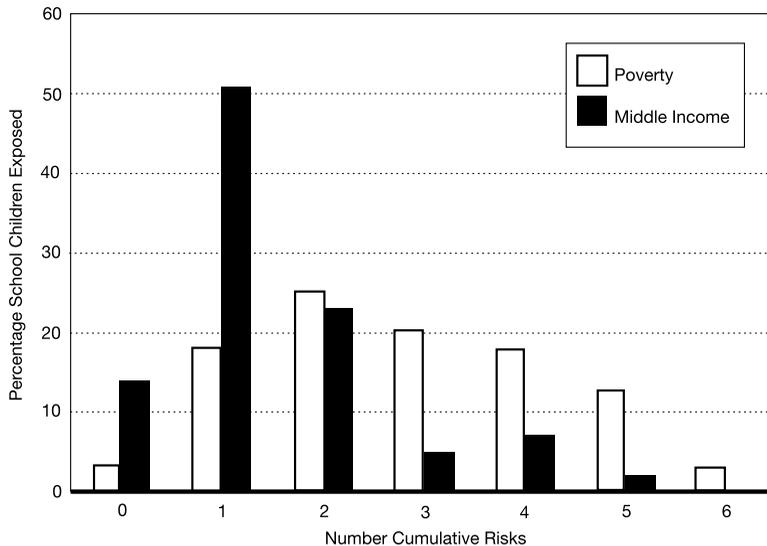
In a summary analysis of nine different national surveys of U.S. households, low-income families were exposed to significantly higher levels of cumulative risk (see Table 5).<sup>28</sup> For example, nearly 20% of low-income families had to contend with three or more risk factors whereas less than 2% of nonpoor families were exposed to comparable high risk. Finally, the mean level of risk factors (0–9) encountered by low-income families was five times greater than the number of risk exposure of wealthier families.

**Table 5.** Cumulative risk exposures from a nationally representative U.S. sample of families with children

	Nonpoor	Poor
Percent of persons in families with		
No risk	87	45
One risk or more	13	55
Two or more	3	27
Three or more	1	12
Four or more	0.3	4
Five or more	0.1	1
Mean (0–9 risks)	0.2	1

Note: Risk factors are evicted in past year, utilities disconnected in past year, telephone disconnected in past year, housing with upkeep problems, not enough food in past 4 months, >1 person/room, no refrigerator, no stove, no telephone. From Table 8 [Overall deprivation in 1992] in Ref. 28. Published by the Bureau of Labor Statistics of the U.S. Department of Labor.

In a nationally representative sample of American families with children, 13% of children below the poverty line experienced multiple indices of chaotic living compared to 4.5% of families living at more than two times the poverty line.<sup>29</sup> Chaos was defined as experiencing two or more of the following changes in the prior 12 months (moved to another state, changed residence, moved in with another family, two or more changes in employment by either parent, two or more school changes, or significant decline in health of a family member). In a sample of 4-year-olds,<sup>30</sup> both parental occupation and education were negatively correlated with an index of cumulative risk exposure (minority status, large family size, father absence, stressful life events, poor parenting beliefs, maternal anxiety, poor maternal mental health, or low levels of positive maternal child interaction). Finally, as shown in Figure 2, low-relative to middle-income, 9-year-olds living in rural areas are exposed to more cumulative risk.<sup>31</sup> Risks included residential crowding, high noise levels, substandard housing quality, family turmoil, child separation from family, and exposure to violence. Note that Evans and English<sup>31</sup> as well as all of the other cumulative risk and SES studies do not permit any assessment of linearity. In each case means are presented for two levels of SES or just the correlation of SES and cumulative risk is provided.



**Figure 2.** Percentage of poor and nonpoor children exposed to cumulative physical and psychosocial environmental risks. Note: Risk factors were family turmoil, community violence, early childhood separation, substandard housing, noise, crowding. From Figure 5 in Ref. 3, Copyright 2004 by the American Psychological Association, Inc. Reprinted with permission.

Summarizing, several studies document an inverse correlation between income or SES and exposure to multiple risk factors. Most of the evidence comes from stressful life events studies that count the degree of exposure to discrete, major adverse events such as divorce or death. These associations are robust and have been shown across many different samples and across the life course. Additional multiple risk studies show similar SES-related patterns for a larger spectrum of risk factors going beyond stressful life events. A smaller number of studies reveal consistent elevations of cumulative risk exposure, regardless of the specific risk factors incorporated, in lower relative to higher SES households.

There is insufficient data to resolve the issue of the linearity of the SES:multiple risk function. Most studies either report correlations or when descriptive data are provided they compare high versus low SES households. As shown in the tables and figures, there is some evidence when more than two SES data points are available, as one moves down the SES gradient, the degree of exposure to multiple risk factors increases linearly. However, almost all of this evidence comes from three data points which requires caution in inferring the linearity of the functions.

### Multiple risk exposure and health

An important reason to examine the potential mediating role of multiple risk exposure in health inequalities (see Fig. 1) is because of the potential power of multiple risk exposure to drive health outcomes. A large literature in child development documents that multiple risk exposure has greater negative impact on child development compared to singular risk exposure.<sup>32–37</sup> Unfortunately, there are two major drawbacks of this literature with respect to examining whether multiple risk might function as an underlying explanation for SES gradients in health. First, nearly all of the research on multiple risk examines behavioral rather than physical health outcomes. Second, this literature has relied on additive models of multiple risk exposure, with many studies using the cumulative risk metric to operationalize multiple risk exposure. As a reminder, the cumulative risk metric defines each risk dichotomously as 1 = risk, 0 = not risk based either on theory (e.g., low birth weight) or using a statistical criterion such as upper quartile of the distributions of exposure (e.g., family conflict). These dichotomous risks are then summed.

Before reviewing what we have learned about multiple risk exposure and physical health

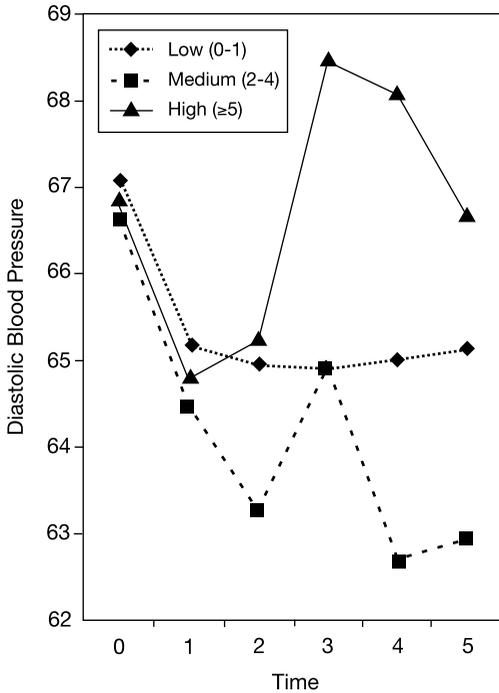
outcomes, we briefly discuss some of the strengths and weaknesses of cumulative risk metrics since most of the work on SES and health has relied on this type of multiple risk index. As an additive model, cumulative risk metrics assume no unique effects of being exposed to Risk Factor A and Risk Factor B above and beyond their independent effects. Thus in the additive formulation of multiple risk exposure there are no statistical interactions between respective risk factors. There is no synergistic impact of exposure to the combination of two risks, the impact is assumed to be the simple summation of each of the singular risks. For example, if a low income-child was exposed to harsh, unresponsive parenting plus poor housing quality, in the additive formulation used in cumulative risk assessment, the effects of these two risk factors is simply added together. However, risks that share some common pathways of health impacts would be expected to have combined effects that differ from risks that are unique in their impacts. To put it differently, additive models of multiple risk make no distinctions between different domains of risk that might vary in what types of health impacts they have. Cumulative risk models also imply that it is the sheer volume of risk factors one is exposed to that matters, no distinctions are made between different risk exposure profiles. The relative contribution of each risk factor to outcomes is not reflected in cumulative risk models.

Why then would so many investigators rely on simply additive models like cumulative risk to represent multiple risk exposures? Why not examine the main and interactive effects of a group of singular risk factors while maintaining their continuous values? There are several reasons why cumulative risk metrics have endured. From a statistical perspective, it takes a large sample in order to examine the effects of a large number of singular risk variables. Furthermore, the loss of information when one reduces the continuous variable to a dichotomous indicator is offset somewhat by gains in reliability of measurement. Less error of measurement is likely to occur if one makes a simply binary decision about risk: no risk compared to estimating the actual level of risk exposure. If one also wants to examine the interactions of each of the risk factors, the growth in required sample size is exponential. Moreover even if one has the requisite statistical power to detect interaction effects, it becomes impossible to interpret the meaning of higher order

interaction terms. One way around the limitation of losing information by dichotomizing variables is to standardize singular risk variables and then form an additive composite. Note that either one of these approaches, however, precludes examination of potential synergistic interactions between singular risk factors—both are additive models. The difference between using a composite of standardized scores compared to a cumulative risk index lies in whether the relative contribution of each particular risk variable matters.

On the plus side, there is a theoretical argument favoring cumulative risk. If it is correct that many risks covary, then an additive formulation is a better fit to the ecology of risk than an interactive model. An interesting additional question is whether the extent of ecological covariation of risk varies in a systematic fashion with SES. We do not have the data to answer this question. We do know from several of the studies reviewed in the first section of this paper that across SES, many singular risk factors are indeed correlated with one another. On the other hand, most are intercorrelated within the 0.2–0.4 range.

A few studies have examined cumulative risk and physical health in children. As a reminder, in order for Figure 1 to be a viable model of the SES:health gradient, multiple risk exposure needs to predict health. Evans *et al.*<sup>38–40</sup> uncovered evidence of cross sectional and longitudinal relations between cumulative risk and various indices of physiological stress in children at age 9 and at age 13. The cumulative risk index incorporated housing quality, crowding, noise, family turmoil, child separation from family, and violence. Blood pressure and overnight catecholamines and cortisol levels rose with increasing levels of cumulative risk exposure. In 9 year olds allostatic load also increased with more cumulative risk exposure. Allostatic load is an index of overall physiological dysregulation across multiple response systems (see McEwen & Gianaros in this volume). For 13 year olds, the same pattern emerged but only among youth with unresponsive mothers. Furthermore, among 13 year olds exposed to higher levels of cumulative risk, blood pressure recovery to baseline levels following exposure to an acute stressor (mental arithmetic) was slower (see Fig. 3). Thus these two studies reveal evidence across several different physiological indices of stress that higher levels of cumulative risk exposure elevate stress.



**Figure 3.** Cumulative risk exposure (as measured by the cumulative risk index) and diastolic blood pressure recovery (in millimeters of mercury). The x-axis depicts the timing of the blood pressure measures taken every 2 min throughout the recovery period. Note: From Figure 3 in Ref. 40. Copyright 2007 by the American Psychological Association. Reprinted with permission.

As shown in Table 6, Felitti *et al.*<sup>41</sup> uncovered a graded relationship between retrospective reports of childhood risk exposures (0–7) and the 10 major causes of death in adults. Risks incorporated into the cumulative index included psychological, physical, or sexual child abuse, violence against mother, parental substance abuse, family member with mental illness, or criminal behavior in household. Furthermore, exposure to accumulated risk factors over the life course was associated prospectively with elevated allostatic load in late adulthood.<sup>42</sup> Risks over the life course included bonding with mother and father in childhood, spousal intimacy, and quality of intellectual and recreational relationships with spouse.

Carmody *et al.*<sup>32</sup> showed that exposure to multiple risks as indicated by a composite index during early childhood was related to lower brain activation (fMRI) in the parietal cortex and the temporal lobes. This study and others have documented that this in

**Table 6.** Childhood risks and adult morbidity

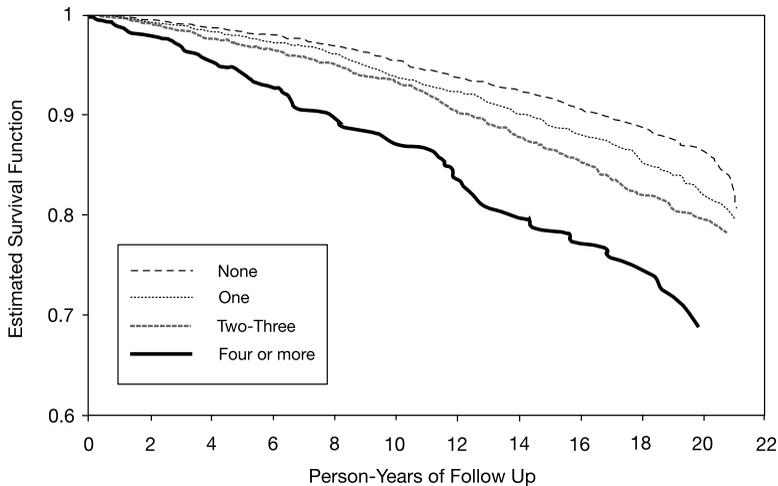
Disease condition	Cumulative risk				
	0	1	2	3	≥ 4
Obesity	1	1.1	1.4	1.4	1.6
Depression	1	1.5	2.4	2.6	4.6
Current smoker	1	1.1	1.5	2.0	2.2
Ischemic heart disease	1	0.9	0.9	1.4	2.2
Diabetes	1	1	0.9	1.2	1.6

Note: Values are odds ratios adjusted for age, gender, race and educational attainment. Adapted from Table 4 [Number of categories of adverse childhood exposure and the adjusted odds of risk factors including current smoking, severe obesity, physical inactivity, depressed mood and suicide attempt] and Table 7 [Number of categories of adverse childhood exposure and the prevalence and risk (adjusted odds ratio) of heart attack, cancer, stroke, COPD, and diabetes] in Ref. 41. Copyright 1998 by Elsevier. Adapted with permission.

turn is related to poorer executive control. The multiple risks incorporated into their composite index included: a set of medical complications associated with prematurity (e.g., respiratory distress) and a series of environmental factors. The environmental factors were parental SES, family structure, life stress, social support, minority status, and mother–child interaction. Because their multiple risk composite incorporated both medical complications as well as environmental factors, it is difficult to tease out the contributions of exposure to multiple risk factors from medical complications.

A few studies also reveal evidence of multiple risk exposure and ill health in adults. Based on a national sample of 25–74 year olds, Thurston and Kubzansky<sup>43</sup> examined the incidence of coronary disease and psychosocial risk factors over a 23 year follow up period. Risk factors included unemployment, single parenthood, loss of spouse, depression, and high anxiety. As indicated in Figure 4, they found a linear association between the number of risk factors and incidence of coronary heart disease.<sup>43</sup>

There is a large literature on multiple effects of two or occasionally three physical and/or psychosocial stressors at work on employee health and well being.<sup>44–48</sup> Typically, these studies find the adverse health impacts of two particular work stressors are



**Figure 4.** Number of psychosocial risk factors and risk of incident coronary heart disease. Note: From Figure 1 in Ref. 43. Copyright 2007 by Lippincott Williams & Wilkins. Reprinted with permission.

worse than the effect of either individual stressor in isolation. Both additive and interactive models have been used. Fewer studies have examined multiple risk composites representing a larger number of multiple risk exposures. Melamed *et al.*<sup>49</sup> developed an index of cumulative risk exposure at work consisting of safety (e.g., fall hazards), job demands (e.g., physical effort), and ambient stressors (e.g., noise levels). Among both blue- and white-collar workers exposure to higher levels of cumulative risk was associated prospectively with occupational injuries.<sup>49</sup> Devereux and associates<sup>50,51</sup> conducted a similar study. Workers with high physical (e.g., heavy lifting) and psychosocial stressors (e.g., low job control) were more likely to suffer from musculoskeletal problems than workers with only one set of workplace stressors. Matthews and Gump<sup>52</sup> investigated a large sample of middle aged men for risk factors at home (marital separation) and at work (job change, demotion, business failure, personal trouble with someone at work, missing work due to disability, job loss, or problems getting new job) in relation to all-cause mortality and cardiovascular mortality. Individuals exposed to three or more risk factors at work had nearly double the rate of mortality in a 9-year period. Marital stress had an independent adverse impact on health as well. Of particular interest, these two domains of risk, marital and work, interacted such that the adverse impacts of work stress on health were found only among divorced men. This final result illustrates another

drawback of a purely additive model of multiple risk measurement. Matthews and Gump<sup>52</sup> show that different domains of multiple risk exposure may interact in a multiplicative manner to affect health. Accumulated exposure to work stress only mattered if the male worker had undergone a marital breakup.

Summarizing, studies with children and adults show that exposure to multiple risk factors is associated with negative health outcomes. Most of these studies have relied upon an additive, cumulative risk index. Some, but not, all of these studies also suggest the effects are linear in that the functions plotting health against the number of risk factors tend to be linear. Although we have not reviewed the evidence in any detail here, these same linear trends dominate the literature on multiple risk and behavioral outcomes.<sup>30,34–38</sup>

### SES and health: the mediating role of multiple risk exposure

The two sections immediately above document each of the intervening pathways displayed in Figure 1, showing that SES is related to multiple risk and that multiple risk is related to health. Thus we have evidence indicating the plausibility of multiple risk exposure as a mediating mechanism that could explain some of the SES gradients in health. What remains is evidence showing that the zero-order correlations between SES and health are accounted for, at least in part, by multiple risk exposure. Little research has

explored the full mediational pathway depicted in Figure 1. Unfortunately, there are two major limitations of several of these studies that render the current state of the evidence equivocal. First as has been the case throughout, most of the studies on multiple risk exposure have focused on behavioral outcomes rather than physical health. A second problem is more troubling vis-a-vis our objective. Some of the SES mediational studies have included constructs such as unemployment or educational attainment in their multiple risk metric. This makes it more difficult to show evidence for the SES → Multiple Risk → Health pathway since part of the multiple risk measure includes aspects of SES.

Approximately 125 elementary school children<sup>31</sup> and middle school<sup>53</sup> who lived in poverty were compared on multiple physical health indices as well as behavioral outcomes with their middle class counterparts. Cumulative risk exposure (family turmoil, child separation from parent, violence, crowding, noise, substandard housing quality) mediated the relations between poverty and multiple cardiovascular and neuroendocrine biomarkers of stress among 9 year olds<sup>31</sup> (see Table 7). Evans and Kim<sup>39</sup> showed that the duration of life time poverty exposure among the sample 4 years later predicted HPA activation (overnight cortisol) and cardiovascular reactivity to an acute stressor (mental arithmetic). Childhood cumulative risk exposure over the two waves of data collection (ages 9 and 13) mediated the poverty—cardiovascular reactivity link but not overnight cortisol levels. Furthermore, at both ages, the negative relations between poverty and multiple indices of socioemotional distress were significantly

attenuated by cumulative risk exposure. Outcomes included parental, teacher, self-report as well as behavioral indicators of distress and self-regulatory difficulties.

Power *et al.*<sup>54,55</sup> showed social class gradients in an overall health index among a large, representative sample of 23 and then 10 years later among 33 year olds in the United Kingdom. These social health gradients were largely explained by exposure to multiple risk factors throughout the life course. For example among 33 year old men the odds ratio of being in fair or poor health was 3.15 times higher in manual laborers compared to professional or managerial males. For women the OR equaled 2.30. Inclusion of a series of risk factors diminished these ratios to 2.06 and 1.34, respectively for men and women. One interesting aspect of Powers *et al.* data was their use of life course theory to stage the order of entry of risk factors from prenatal (e.g. maternal smoking) to factors at age 33 (e.g. marital status). In general early risk factors persisted throughout, but for men especially, later in life risk exposures also contributed to social gradients in adult health. Because Power *et al.* included factors indicative of social class throughout the life course as part of their multiple risk indices, it is difficult to gauge the independent impact of accumulated risk exposures to the SES health gradient. However their analyses reveal that even when factors such as social class at birth were in the model, a multitude of individual risk factors still contributed to the class gradients in health. These risk factors included indices of social support, housing characteristics, and health-related behaviors. Parallel trends were also uncovered for

**Table 7. Poverty, cumulative risk, and elementary school children’s physiological stress**

	Poverty <i>b</i> ( <i>SE</i> )	Poverty $\Delta R^2$	Poverty <i>b</i> partialing out multiple stressors	% Shrinkage in poverty, <i>b</i>	$\Delta R^2$ , Partialing out multiple stressors	% Shrinkage in $\Delta R^2$
Diastolic blood pressure	-2.14 (0.99)*	0.02*	-0.98	54	0.00	100
Systolic blood pressure	-2.35 (1.20)*	0.02*	-1.73	26	0.00	100
Cortisol	-0.009 (0.003)**	0.04***	-0.005	44	0.02	50
Epinephrine	-1.81 (0.58)**	0.04**	-1.31	28	0.01	75
Norepinephrine	-2.18 (2.68)	0.00				

\*  $P < 0.05$ ; \*\*  $P < 0.01$ .

Note: Adapted from Table 4 [Preliminary Mediational Analyses] in Ref. 31. Copyright 2002 by the Society for Research in Child Development, Inc. Adapted with permission.

multiple risk exposure mediating social class gradients in mental health.<sup>56,57</sup>

Wills *et al.* uncovered an inverse association between parental educational attainment and adolescent substance abuse among a large sample of racially and socioeconomically heterogeneous eighth graders.<sup>58,59</sup> Composite risk was operationalized with structural equation models indicating that the best fitting model included multiple mediating constructs. The parental education link to youth substance abuse was indirect via reduced parental support, high negative life events, deficient youth competency, and contact with friends who were substance abusers. This final model was superior to both the direct model and models with only a single mediating variable in the structural equation.

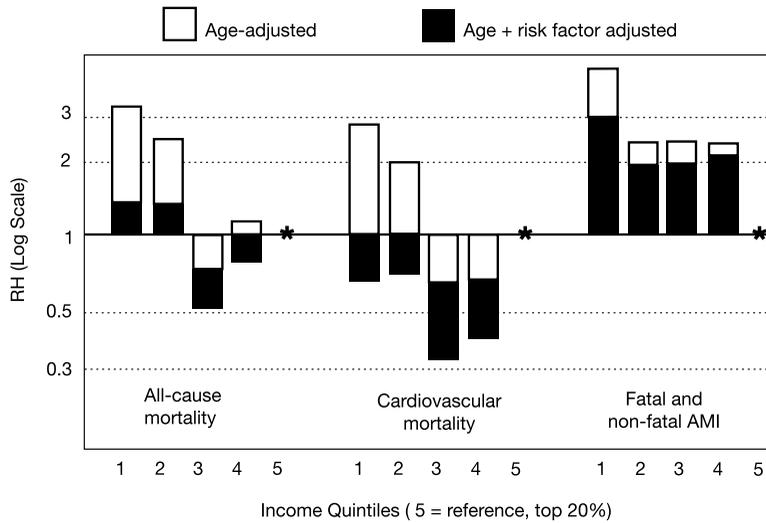
In addition to these three studies on SES, multiple risk exposure, and physical health among children, several studies have also uncovered evidence for parallel trends among adults. House *et al.* studied a large, nationally representative sample longitudinally across three waves beginning at age 25, then 2.5 years later, and again 7.5 years afterward.<sup>19,60</sup> Income at wave 1 was prospectively related to health outcomes (mortality, activities of daily living, overall perceived health) at waves 2 and 3. However when multiple risk factors from prior waves were incorporated into the model, previous income levels no longer predicted self report measures of health, and the prediction of mortality from prior income was cut nearly in half. Risk factors included parental, financial, and marital stress along with recent and lifetime stressful life events. An additive cumulative risk index mediated the relationship between SES and an overall health index among elderly adults as well. Evans *et al.*<sup>61</sup> showed that income among persons over 65 living independently in the community was prospectively linked to health effects 2 years later. The prospective association with income was reduced 58% by the inclusion of a cumulative risk index. This cumulative risk index included death of a close friend, care giving burden, housing quality problems, and low social integration.

Cohen *et al.*<sup>62</sup> studied the role of multiple risk exposure and perceived health in the United States and Finland. In both countries, lower income individuals engaged in riskier health behaviors and were exposed to greater perceived stress and life events, experienced lower mastery and social support, were more angry and hostile, and had elevated levels of

depression. Odds ratios comparing the lowest to highest income quintiles for perceived ill health were cut from 4.6 to 2.0 and from 5.3 to 3.1 in the United States and Finland, respectively, by the inclusion of these risk factors as a composite term in the models. Robert<sup>63</sup> provided evidence that neighborhood SES could also predict overall adult health independently of household SES. This association was mediated by about 33% from exposure to a composite risk index consisting of health related behaviors (e.g., smoking) and psychosocial factors such as mastery, life events, and social integration. Looking at neighborhood SES and mental health instead of physical health, Koster *et al.*<sup>64</sup> showed similar mediational pathways between SES and mental health.

Marmot *et al.*<sup>65</sup> examined incidence of coronary heart disease over a 5-year period among a large sample of British civil servants. Civil service grade showed a linear relationship to incidence of heart disease. Of particular interest herein, inclusion of multiple risk factors in the model: job control, effort-reward imbalance, social support, and health related behaviors such as smoking, altered the odds ratio from 1.5 to 0.95 for men in the lowest versus highest civil service for incidence of heart disease. Similar data were found in women.<sup>65</sup> Stansfeld *et al.*<sup>66</sup> following up this same cohort over an additional 5-year period examined general overall health as well as mental health outcomes. Composite risk included job control, job demands, effort-reward balance, social support, negative life events, housing tenure, marital status, and material problems (e.g., housing, financial difficulties). There was a 35% reduction for men and a 27% reduction for women in civil service grade health gradients.<sup>66</sup> Stronger mediation was shown for mediation of social gradients in mental health outcomes.<sup>66</sup> Because the longer Whitehall cohort study included in the multiple risk metric a measure of financial difficulties, it is difficult to interpret the status of the multiple mediators. On the other hand, several of the individual risks made independent contribution to lowering the SES:physical and mental health gradients.

Finally, Lynch *et al.*<sup>67</sup> studied cardiovascular mortality and all-cause mortality in a large, nationally representative sample of Finnish men, beginning at ages 42–60 and then 7 years later. As expected, income levels showed a prospective, graded relationship with both causes of death. The investigators also collected data on a large number of risk factors



**Figure 5.** Relative hazards (RH), on the log scale, of all-cause mortality, cardiovascular mortality, and acute myocardial infarction (AMI) by quintile of income. Height of the hatched bars reflects RH adjusted for age. *Black bars* represent relative hazard after adjustment for all risk factors in a population-based sample of 2272 (mortality analyses) and 1707 (AMI analyses) eastern Finnish men aged 42–60 years (1984–1993)\*, reference category. Note: From Figure 1 in Ref. 67. Copyright 1996 by the Johns Hopkins University School of Hygiene and Public Health. Reprinted with permission.

including biological (e.g., cholesterol), behavioral (e.g., smoking), and psychosocial (e.g., social support) variables. As can be seen in Figure 5, all cause mortality (OR = 3.14) as well as cardiovascular mortality (OR = 2.66) for the lowest versus highest income quintile were markedly reduced when multiple risk factors were incorporated into the equations (1.32, 0.7 for all-cause and cardiovascular mortality, respectively).<sup>67</sup>

In addition to the above studies testing the mediational mechanism of multiple risk exposure between SES and physical health (see Fig. 1), several studies have also examined parallel pathways for behavioral outcomes. Because our focus herein is on physical health, we simply note these studies. The positive links between SES and adolescent<sup>24,35</sup> as well as primary school aged children<sup>68,69</sup> and socioemotional and cognitive outcomes are mediated by cumulative risk exposure. Some of the gains in early childhood intervention programs for children at risk are due to reductions in cumulative risk exposure.<sup>70</sup>

**Summary and conclusions**

There is abundant evidence that SES is inversely related to exposure to singular physical and social risk

factors, particularly when comparing low-income children to their wealthier counterparts or when contrasting lower to upper SES adults. We also know that SES is inversely related to multiple risk exposure. Very consistent and strong evidence reveals that multiple risk exposures elevate physical health problems. The degree of linearity in the SES—risk functions is less clear since many studies have relied on two or occasionally three levels of SES. Thus we cannot at this time definitively analyze the nature of the gradient between SES or income and exposure to multiple risks. An interesting issue worthy of scrutiny is the degree of convergence of multiple risk exposures with respect to SES. Perhaps one of the reasons why lower SES is unhealthier is not only because higher levels of risk occur but perhaps the degree of convergence across different risk factors mounts as well.

Not all investigators have operationalized multiple risk in a similar manner. Some have shown that SES is significantly correlated with counts of stressful life events, whereas others have used various composite indicators to operationalize exposure to multiple risk factors (e.g., block of singular risk factors, summation of singular risk variables into one index, latent index of multiple individual risk

factors). Finally, cumulative risk metrics that sum exposure to dichotomously defined risk factors are associated with SES.

Unfortunately of the small handful of studies that have actually tested the mediating mechanism of multiple risk exposure for SES and health, several have constructed risk indices that incorporated factors that are components of SES (e.g., poverty status, employment, and perceived financial difficulty). This renders conclusions moot about multiple risk exposure as an underlying explanation of the SES and health gradient. In addition, some of these studies combined biological and environmental risk factors, making it difficult to disentangle the role of environmental risk exposures in health inequalities.

Multiple risk exposure is a plausible, hypothetical mechanism that could account for a significant portion of the SES:health gradient (see Fig. 1). We do not expect that multiple risk exposure is the sole, underlying mechanism for the robust and complex interplay between disadvantage and ill health. There are likely multiple, underlying explanations for health inequalities. Multiple risk exposure is a viable candidate for one of the major pathways by which disadvantage leads to ill health. We need more research that operationalizes multiple risk exposure incorporating both physical and psychosocial risk factors and making sure these factors are not direct constituents of SES in order to test this model more rigorously. Ideally multiple risk factors would be measured over time, particularly during early childhood with multiple health outcomes assessed over time. This would enable health effects to manifest and afford an opportunity to examine how chronic risk exposure operates both in terms of developmental timing and as risks accumulate over time as the organism matures. Biomarkers (e.g., allostatic load, neurological function, and architecture) that may help account for the adverse effects of multiple risk exposure would ideally be assessed as well. These should be treated as outcomes, not part of the cumulative risk metric as they are biological pathways that may help us understand how chronic stress leads to ill health.

It is important that studies incorporate samples that are heterogeneous with respect to income or SES in order to test the model in Figure 1. This would entail large samples or samples that over represent low SES participants in order to represent the full spectrum of SES. Finally, it is premature to decide

on which multiple risk metric is most appropriate to best represent exposure to more than one singular risk factor. The evidence is strong that multiple risk exposures leads to worse health outcomes compared to singular exposure, and data are promising that multiple risk exposure may account for some of the SES:health gradient. It is less clear at this point whether additive or multiplicative models of multiple risk best capture the underlying dynamics of multiple risk exposure. Samples of sufficient size that enable us to maintain the continuous nature of singular risk factors rather than having to condense them into dichotomous variables are preferable, but cumulative risk indices may be an acceptable proxy when this is not possible.

Income and social class matter for health. Exposure to the confluence of physical and social risk factors accompanying deprivation is a plausible model of the SES:health gradient worthy of continued scientific investigation. Should this model of health inequalities prove accurate, it also raises critical challenges to the design of effective interventions to combat the ill effects of low human capital on health. Interventions targeting singular risks accompanying deprivation are likely to be less effective than those that tackle multiple risk factors.<sup>71,72</sup> It also follows that families facing the greatest amount of multiple risk exposure should be prioritized for interventions.

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## Conflicts of interest

The authors declare no conflicts of interest.

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