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Does Cognitive Ability Buffer the Link Between Childhood Disadvantage and Adult Health?

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Objective: Individual differences in childhood cognitive ability have been neglected in the study of how early life psychosocial factors may buffer the long-term health consequences of social disadvantage. In this study, we drew on rich data from two large British cohorts to test whether high levels of cognitive ability may protect children from experiencing the physical and mental health consequences of early life socioeconomic disadvantage. ***Method:*** Participants from the 1970 British Cohort Study (BCS; N = 11,522) were followed from birth to age 42, and those from the 1958 National Child Development Study (NCDS; N = 13,213) were followed from birth to age 50. Childhood social disadvantage was indexed using 6 indicators gauging parental education, occupational prestige, and housing characteristics (i.e., housing tenure and home crowding). Standardized assessments of cognitive ability were completed at ages 10 (BCS) and 11 (NCDS) years. Psychological distress, self-rated health, and all-cause mortality were examined from early adulthood to midlife in both cohorts. ***Results:*** Early social disadvantage predicted elevated levels of psychological distress and lower levels of self-rated health in both cohorts and higher mortality risk in the NCDS. Childhood cognitive ability moderated each of these relationships such that the link between early life social disadvantage and poor health in adulthood was markedly stronger at low (−1 *SD*) compared to high (+1 *SD*) levels of childhood cognitive ability. ***Conclusions:*** This study provides evidence that high childhood cognitive ability is associated with a decrease in the strength of socioeconomic status–driven health inequalities.

Keywords: socioeconomic status, cognitive ability, psychological distress, health, mortality

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Early life socioeconomic disadvantage is the social factor most consistently linked to adverse mental (Everson, Maty, Lynch, & Kaplan, 2002; Repetti, Taylor, & Seeman, 2002) and physical (Adler et al., 1994; Galobardes, Lynch, & Davey Smith, 2008; Pollitt, Rose, & Kaufman, 2005; Stafford et al., 2015) health outcomes, including physiological dysfunction, disease, and death. Those from disadvantaged backgrounds tend to be exposed to a potent combination of risk factors (e.g., overcrowding, family conflict, food insecurity, less responsive parenting; Evans, 2004), and the current scientific consensus is that health disparities in adulthood emerge because of the cumulative impact or biological embedding of exposure to these risk factors over time (Matthews & Gallo, 2011; Shonkoff, Boyce, & McEwen, 2009). Despite the robustness of this phenomenon across cohorts and health measures (Adler et al., 1994; Matthews & Gallo, 2011), it nonetheless remains the case that not all individuals who grow up in difficult life circumstances go on to experience poor health later in life (e.g., Chen & Miller, 2013).

A range of contemporaneous adaptive psychological resources (e.g., perceived control, optimism, self-esteem) have been proposed as key factors that may weaken the relationship between socioeconomic status and health (Matthews & Gallo, 2011; Turiano, Chapman, Agrigoroaei, Infurna, & Lachman, 2014). Yet, consistent evidence for a specific psychological buffer against the health consequences of deprivation has yet to be uncovered (Matthews, Gallo, & Taylor, 2010). In this article we test the idea that one psychological resource that may protect against the health consequences of early disadvantage is cognitive ability. Measures of cognitive ability (used interchangeably with the term *intelligence*) tap a range of cognitive resources and processes, including reasoning, memory, processing speed, and spatial ability, and although performance on subtests varies, correlations across domains are uniformly positive (Deary, Weiss, & Batty, 2010). Cognitive ability functions chiefly to foster effective adaptation to the environment (Godfrey-Smith, 2001), and premorbid childhood cognitive ability is known to have a range of health-protective effects (e.g., Deary, 2010). Although there has been considerable focus on whether intelligence represents a fundamental cause of health inequalities (Link, Phelan, Miech, & Westin, 2008), the possibility that intelligence could attenuate the link between socioeconomic status (SES) and health remains relatively underexplored.

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Indeed, initial evidence has suggested that higher levels of cognitive ability may play a protective role in reducing mortality risk chiefly among the most deprived (Hart et al., 2003). Further, the resilience literature has suggested a role for cognitive ability in protecting young people from the adverse psychological effects of disadvantage (Fergusson & Lynskey, 1996; Masten et al., 1999; Riglin et al., 2016). Emerging from the discipline of developmental psychopathology, resilience refers to positive adaptation in the face of adversity, and research in this area has sought to characterize the child, family, and community characteristics that confer protection against the negative impact of environmental stressors (Masten, 2001). Although the search for reliable resilience factors has generally focused on parenting and community factors, there has been some evidence that cognitive ability may also play a role (Luthar, 2006). For example, teenagers exposed to high levels of family adversity have been shown to exhibit fewer externalizing problems (e.g., substance abuse, juvenile offending) if they had higher cognitive ability than their similarly disadvantaged peers (Fergusson & Lynskey, 1996). Similarly, cognitive ability has been shown to interact with life stress to buffer the adverse impact of stressful life events on externalizing behavioral problems (Flouri, Mavroveli, & Panourgia, 2013) and internalizing depressive symptoms (Riglin et al., 2016) in adolescence. However, it remains to be seen whether high cognitive ability plays a similar role in diminishing the long-run mental and physical health consequences of social disadvantage, as observed in the general population.

In the current report, we tested this idea using data from two large representative British cohorts that include rich records of both socioeconomic status and cognitive ability during childhood coupled with measures of psychological distress, general health, and mortality throughout adulthood. Across both cohorts, social disadvantage was operationalized by combining a broad range of socioeconomic characteristics of each household, including parents' education and social class and housing factors such as crowding and housing tenure. By integrating several domains of information assessed at multiple time points across childhood, we could produce a reliable composite measure of early life disadvantage. Our approach also capitalized on the rich family background data available in the cohorts and captures several distinct routes through which social disadvantage is thought to impact on health (Evans, 2004). We anticipated that higher levels of social disadvantage would be associated with worse health throughout adulthood, as assessed by measures of psychological distress and self-rated health throughout adulthood and mortality by midlife. Further, we hypothesized that individual differences in cognitive ability would moderate the association between early social disadvantage and subsequent health such that those with higher levels of childhood cognitive ability would be protected against the long-run detrimental health outcomes of background disadvantage.

Method

Participants

This study uses data from two nationally representative ongoing British birth cohort studies: the 1958 National Child Development Study (NCDS; Power & Elliott, 2006) and the 1970 British Cohort Study (BCS; Elliott & Shepherd, 2006) cohorts. Both the NCDS and BCS began as surveys designed to examine factors associated with stillbirth and death in early infancy (the Perinatal Mortality Survey and British Births Survey, respectively) for which information was gathered from almost 17,500 babies born in a single week (in 1958 and 1970, respectively). These surveys have gone on to form the basis of continuing national longitudinal studies with multiple follow-up data collection exercises at regular intervals from birth to midlife (age 50 in the NCDS; age 42 in the BCS). Anonymized data from all follow-up waves are currently made available by the U.K. Data Service.

At the time of data collection, ethical approval was attained via internal review boards until 1997, after which point approval was granted by the London Multicenter Research Ethics Committee (MREC). Parental consent was sought for data collected during childhood, and all consent for participation once cohort members became adults was gained by respondents' agreeing to be interviewed and returning completed questionnaires. Written consent, however, was obtained only for data collected after 1997, as was required by MREC approval. Access to the data set for the purposes of secondary analysis was subject to the terms of an end-user license agreement, and further ethical approval was not needed. The current study includes participants who provided data on all key variables: social disadvantage, cognitive ability data, and adult health. Both samples are particularly homogeneous in terms of ethnicity: Where data are available (n = 5,658 in the BCS; n = 8,122 in the NCDS), they show that both samples were overwhelmingly White (97.9% in the BCS; 99.1% in the NCDS). Because of this homogeneity, ethnicity was not included within the principal analyses. The sample size was 13,213 (48.8% female) in the NCDS and 11,522 (48.6% female) in the BCS.

Measures

Childhood social disadvantage. A composite measure of social disadvantage during early childhood was derived from six measures collected via parental interviews at birth and early childhood (age 7 in the NCDS and age 5 in the BCS). These were (a) social class based on the father's occupation at birth and (b) social class in early childhood, both measured by using the Registrar General's Social Class scheme (where I = professional occupations, II = managerial or technical occupations, III = skilled workers, IV = semiskilled workers, V = unskilled workers; Office of Population Census & Surveys, 1980); (c) the age at which the participant's father left education; (d) age at which the participant's mother left education; (e) parental housing tenure in early childhood (ranked as 1 = *owner occupied or being bought,* 2 = *private rented furnished or unfurnished,* 3 = *council rented,* 4 = *rent free* [NCDS] or *tied to occupation* [BCS]); and (f) persons per room in early childhood (in the online supplemental materials, see Table S1 for specific details on these measures and Table S2 for descriptive statistics for each indicator). To maximize the sample size, we included cohort members in the analyses if they provided data on at least two of the key measures. On average, participants included in the study had complete data on 5.5 disadvantage measures (SD = 1.12) in the BCS and 5 (SD = 1.15) in the NCDS. Each measure was standardized and subsequently averaged and restandardized to form a normally distributed, internally reliable social disadvantage measure with a mean of 0 and standard deviation of 1 (Cronbach's alpha was .77 for both the NCDS and the BCS; see Table S2 and Figure S1 in the online supplemental materials).

Childhood cognitive ability. At age 11, NCDS cohort members completed an 80-item general ability test (Pigeon, 1964). Children were tested individually by their teacher and were presented with 40 verbal and 40 nonverbal items. For verbal items, children were presented with a set of four words linked either logically, semantically, or phonologically. They were then given another set of three words with a blank and were required to select the missing item from a list of five alternatives. The task was comparable for the nonverbal items except that stimuli were shapes and symbols. Each correctly responded to item was awarded 1 mark, giving a final score between 0 and 80, which was standardized for inclusion in all analyses here. The general ability test has shown high levels of test–retest reliability (Cronbach's alpha = .94) and has been shown to correlate strongly with tests employed for secondary-school level selection in England (r = .93) indicating a high degree of validity (Douglas, 1964). In the BCS, ability was measured at age 10 using the 120-item British Ability Scales (BAS), which comprised two verbal subscales (Word Definitions, Word Similarities) and two nonverbal subscales (Recall of Digits, Matrices; Elliott, Murray, & Pearson, 1978). The word definitions test required cohort members to indicate the meaning of 37 words of increasing complexity, whereas the word similarity test consisted of 21 three-word lists (e.g., *orange, banana, strawberry*) for which the child was requested to name a word consistent with the theme (e.g., *apple, cherry*) and to provide a group name that united the items (e.g., *fruit*). Digit recall required the recall of 34 series of digits of increasing difficulty. In the matrices test, each child was presented with 28 incomplete patterns and asked to complete the missing section of the pattern. The BAS has shown high levels of internal reliability (Cronbach's alpha = .93) and convergent validity with established measures of cognitive ability such as the Wechsler Intelligence Scale for Children and the Stanford-Binet Intelligence test (Elliott et al., 1978; McCallum & Karnes, 1987). Scores in both cohorts were standardized to have a mean of 0 and standard deviation of 1 (see Table S2 and Figure S2 in the online supplemental materials).

Psychological distress. Understood to reflect emotional suffering characterized by anxiety and depression within the general population, psychological distress was measured using nine items drawn from the Malaise Inventory (Rutter, Tizard, & Whitmore, 1970) examined at each time point throughout adulthood (BCS: ages 26, 30, 34, and 42; NCS: ages 23, 33, 42, and 50; see Table 1). The Malaise Inventory has been shown to have acceptable internal consistency and validity (Rodgers, Pickles, Power, Collishaw, & Maughan, 1999) and good psychometric properties (McGee, Williams, & Silva, 1986). The nine items employed here relate to the psychological subscale which consists of a set of yes–no self-completion questions gauging a range of negative feelings related chiefly to feelings of anxiety and depression (e.g., "Do you often feel depressed?"; "Do you often get worried about things?"; and "Do you suddenly become scared for no good reason?"), yielding a final score from 0 to 9. The nine-item scale has been shown to have good psychometric properties (McGee et al., 1986; Ploubidis, Sullivan, Brown, & Goodman, 2017) and showed a high level of reliability in the current study (e.g., Cronbach's alphas = .77 in the BCS and .81 in the NCDS at age 42). Further, the cross-cohort measurement equivalence of the scale has previously been established in the BCS and the NCDS, providing evidence that the same construct is being assessed in each study (Ploubidis et al., 2017).

Self-rated health. General self-rated health was assessed at each time point throughout adulthood (see Table 1). Participants were asked to rate their current health on a scale from 1 (*poor*) to 4 (*excellent*).[1] This single-item indicator provides a global summary of general health that produces predictions of hospitalizations and health care usage, similar to estimates derived using multi-item subjective health measures (DeSalvo, Fan, McDonell, & Fihn, 2005). This measure also predicts mortality more strongly than do physical measurements or clinical indicators derived from blood assays (Ganna & Ingelsson, 2015).

Mortality data. All-cause mortality and month of death was assessed using information on deaths drawn from deaths certificates of the National Health Service Central Register and from information ascertained from relatives or friends as part of cohort maintenance activities. Mortality was tracked from ages 10 to 42 in the BCS (N = 200 deaths; 1.7% of the sample) and from ages 11 to 50 in the NCDS (N = 466 deaths; 3.5% of the sample).

Childhood health. The NCDS includes rich data on both physical and psychological health from a number of points in childhood. Specifically employed here are data from extensive medical examinations taken at age 7 that documented the presence of over 40 health problems, including digestive problems, epilepsy, headaches or migraines, speech defects, hearing and vision defects, emotional maladjustment, intellectual disability, asthma, diabetes, respiratory problems, urinary problems, heart disease, and other physical abnormalities. Objectively recorded measures of birth weight as well as head circumference and body mass index at age 7 were also included. Hospital admissions by age 7 and their stated cause (e.g., road accidents, operations, adenoids removal) were also assessed. Together these variables were used to estimate the sensitivity of our main analyses to adjustment for health during childhood.

Adult social disadvantage. Four measures of social disadvantage were selected from the age 42 wave of both the NCDS and the BCS to enable the sensitivity of the study results to adjustment for adult disadvantage to be compared directly across the two cohorts. The four measures were (a) social class based on current or most recent job measured by the Registrar General's Social Class scheme, (b) housing tenure, (c) persons per room, and (d) number of years in education (see Table S3 in the online supplemental materials). Each of these four measures was standardized at age 42.

Data Analysis

We first used Cox proportional hazards models to calculate the hazard ratios (HRs) and associated 95% confidence intervals (CIs) for the main effect of social disadvantage and cognitive ability in predicting mortality. We then examined the interaction between early disadvantage and cognitive ability to ascertain whether the potential contribution of disadvantage to premature mortality occurs chiefly among those with lower levels of childhood cognitive ability. Linear mixed models were then used to identify how early life disadvantage and cognitive ability relate to subsequent psychological distress and self-rated health (Model 1). Following this, we tested whether the interaction between cognitive ability and early life social disadvantage predicted measures of psychological distress and self-rated health measured across adulthood (Model 2). Both dependent variables were standardized within each wave to enable the magnitude of the associations of interest to be directly compared across individual waves.

[1] At ages 34 and 42 in the BCS and age 50 in the NCDS a 5-point scale was used, and responses were standardized to provide comparability with responses to the 4-point scale used in other waves.

Table 1
Descriptive Statistics for Health Outcome Variables at Each Wave for the NCDS and BCS Cohorts

Sample and characteristic	Wave 1	Wave 2	Wave 3	Wave 4
NCDS				
Age (years)	23	33	42	50
Psychological distress				
M (*SD*)	1.25 (1.53)	.99 (1.54)	1.50 (1.78)	1.47 (1.92)
n	10,295	9,325	9,257	7,956
Completion rate (%)[c]	78	71	70	60
Self-rated health				
M (*SD*)	3.35[a] (.69)	3.20[a] (.70)	3.09[a] (.76)	3.48[b] (1.11)
n	10,302	9,262	9,329	8,031
Completion rate (%)[c]	78	70	71	61
BCS				
Age (years)	26	29	34	42
Psychological distress				
M (*SD*)	1.76 (1.76)	1.54 (1.74)	1.66 (1.89)	1.86 (1.98)
n	6,577	8,199	7,120	6,324
Completion rate (%)[c]	57	71	62	55
Self-rated health				
M (*SD*)	3.25[a] (.65)	3.15[a] (.71)	4.04[b] (.89)	3.61[b] (1.07)
n	6,571	8,265	7,142	7,184
Completion rate (%)[c]	57	72	62	62

Note. NCDS = National Child Development Study; BCS = British Cohort Study.
[a] Rated on a scale ranging from 1 (*poor*) to 4 (*excellent*). [b] Rated on a scale ranging from 1 (*poor*) to 5 (*excellent*). [c] Percentage of baseline sample.

Linear mixed models are capable of handling missing repeated-measures data, thus enabling the average effect of the predictor variables to be estimated over all available adult waves and data points simultaneously. Employing random-intercept models also allowed the presence of nonindependent error terms (resulting from the existence of clustering or multiple measurements nested within participants) to be adjusted for.

Next, we examined the simple slopes for associations between social disadvantage and the health outcomes at different levels of the moderator cognitive ability. Specifically, we defined those scoring 1 standard deviation below the mean on the standardized cognitive ability measure as low cognitive ability; medium cognitive ability was defined as scoring at the mean on this measure, and those with high cognitive ability were defined as scoring at 1 standard deviation above the mean.

The formal model specifications were as follows:

$$\textit{Model 1}: \text{Adult health}_{ti} = \beta_{0i} + \beta_1\text{social disadvantage}_i + \beta_2\text{cognitive ability}_i + \beta_3\text{female}_i + \varepsilon_{ti}$$

and

$$\textit{Model 2}: \text{Adult health}_{ti} = \beta_{0i} + \beta_1\text{social disadvantage}_i + \beta_2\text{cognitive ability}_i + \beta_3\text{female}_i + \beta_4\text{social disadvantage}_i \times \text{cognitive ability}_i + \varepsilon_{ti}$$

A number of additional analyses were included to gauge the sensitivity of our results to adjustment for childhood health, to ascertain the stability of the predicted interaction effects from early adulthood to midlife, and to identify the extent to which the associations observed may be explained by adult socioeconomic status. First, the role of childhood health was examined in the NCDS by adding the rich array of early health variables collected at age 7 (see the Measures section) to Model 2. Second, ordinary least square regressions were conducted for self-rated health and psychological distress at each wave to determine the stability of the buffer effect across adulthood. Finally, we examined changes in the Disadvantage × Cognitive Ability interaction coefficient after adjustment for four different measures of adult social disadvantage in midlife. These analyses were restricted to health outcomes at age 42 because comparable indicators of adult social position were available at this time point for both the BCS and NCDS. For analyses controlling for adult social disadvantage and childhood health, missing adult disadvantage data were imputed with the average of existing data, and an additional dummy variable coding for the presence of replaced data was included to adjust for differences in the outcome variable between those with and missing adult disadvantage data.

Results

Descriptive Statistics

Table S2 in the online supplemental materials reports the descriptive statistics for each of the six measures employed to derive a composite measure of social disadvantage and for the cognitive ability measures included in the NCDS (M = 43.26, SD = 16.00) and the BCS (M = 59.64, SD = 13.37). Table 1 presents the descriptive variables for the psychological distress and self-rated health measures and the rate of completion for both measures at each wave. On average, the portion of the baseline sample who provided outcome data at a given survey wave was higher in the NCDS than in the BCS (70% vs. 62% per wave), and completion rates remained relatively stable throughout the period of follow-up (ages 23–50 in the NCDS; ages 26–42 in the BCS). Mean levels of distress were higher in the BCS than in the NCDS for similar age groups, as has been shown previously (Ploubidis et al., 2017), and were relatively stable over time, particularly in the BCS (mean values ranging from 1.54 [SD = 1.74] to 1.86 [SD = 1.98]).

Table S4 in the online supplemental materials shows correlations between all key study variables across waves and provides further evidence that psychological distress levels were relatively stable across waves and cohorts (NCDS: mean r = .53, min r = .46, max r = .59; BCS: mean r = .49, min r = .40, max r = .58). The average strength of the correlation between self-rated health measures across waves was slightly lower in both cohorts (NCDS: mean r = .42, min r = .35, max r = .47; BCS: mean r = .41, min r = .29, max r = .54). This marginally greater stability over time for psychological distress may have arisen because distress is gauged using a multi-item well-being measure that has been shown to be stable across multiple time points (Furnham & Cheng, 2015), whereas the physical health measure remains a single-item measure. Multi-item scales are thought to have increased reliability because multiple items help overcome and average out errors inherent within single-item scales (DeVellis, 2003). Table S4 also provided initial evidence that higher levels of childhood social disadvantage were predictive of raised distress levels (mean r = .1 in both the NCDS and the BCS) and worse self-rated health (mean r = −.13 in the NCDS and −.14 in the BCS) throughout adulthood as anticipated.

Main Regressions

Tables 2 and 3 present mixed-model estimates for psychological distress and self-rated health for the BCS and NCDS cohorts, respectively (see Table S4 in the online supplemental materials for correlations between all variables across waves). There were highly statistically significant main effects of social disadvantage and cognitive ability on health in both cohorts. In the NCDS and BCS, respectively, a 1-*SD* increase in social disadvantage was associated with a .064/.042-*SD* increase in psychological distress and a .104/.092-*SD* decrease in self-rated health. Higher levels of childhood disadvantage were linked to an increased risk of all-cause mortality by midlife in the NCDS (HR = 1.16, 95% CI [1.04, 1.28], $p < .01$) and unrelated to early mortality in the BCS. High cognitive ability was linked to low levels of psychological distress, high levels of self-rated health, and a reduced risk of mortality in both cohorts (see Tables 2 and 3).

Next, we sought to identify whether cognitive ability moderated the associations we observed between social disadvantage and risk of later distress and low self-rated health (BCS and NCDS) and premature mortality (NCDS only). As anticipated, social disadvantage interacted with cognitive ability to predict psychological distress (NCDS: $\beta = -.052$, $p < .001$; BCS: $\beta = -.049$, $p < .001$), self-rated health (NCDS: $\beta = .022$, $p < .01$; BCS: $\beta = .031$, $p < .001$), and mortality (NCDS: HR = .90, 95% CI [.82, .98]). An examination of the simple slopes indicated that greater social disadvantage was more closely related to higher levels of psychological distress and self-rated health chiefly at low levels of cognitive ability, as illustrated in Figure 1. The lower sections of Tables 2 and 3 show the effect of social disadvantage on distress and self-rated health for each level of cognitive ability. On average across the two cohorts, a 1-*SD* increase in social disadvantage predicted a .120-*SD* increase in psychological distress at low levels of cognitive ability (−1 *SD*) compared to a .019-*SD* increase at high levels (+1 *SD*). Moreover, a 1-*SD* increase in social disadvantage predicted a .131-*SD* decrease in self-rated health at low levels of cognitive ability (−1 *SD*) compared to a .079-*SD* decrease for high cognitive ability (+1 *SD*). Finally, in the NCDS we found that high levels of disadvantage were associated with a higher risk of death at low (−1 *SD;* HR = 1.27, 95% CI [1.11, 1.43], $p < .001$) but not high (+1 *SD;* HR = 1.02, 95% CI [.88, 1.17], $p = .81$) cognitive ability.

Table 2
Regression Models Assessing the Cognitive Ability × Social Disadvantage Interaction in Predicting Psychological Distress, Self-Rated Health, and Mortality in the BCS Cohort

Model and predictor	Psychological distress b (SE)	Self-rated health b (SE)	Mortality: HR [95% CI]
Model 1			
Social disadvantage (z score)	$.064^{***}$ (.010)	$-.104^{***}$ (.009)	.954 [.81, 1.13]
Cognitive ability (z score)	$-.089^{***}$ (.009)	$.087^{***}$ (.009)	$.744^{***}$ [.64, .86]
Female	$.259^{***}$ (.017)	−.006 (.016)	$.404^{***}$ [.30, .55]
Constant	$.144^{***}$ (.012)	$.031^{**}$ (.011)	
Model 2			
Social disadvantage (z score)	$.083^{***}$ (.010)	$-.116^{***}$ (.010)	.954 [.81, 1.13]
Cognitive ability (z score)	$-.090^{***}$ (.009)	$.087^{***}$ (.009)	$.744^{***}$ [.64, .86]
Social disadvantage × Cognitive ability	$-.049^{***}$ (.009)	$.031^{***}$ (.008)	1.016 [.89, 1.16]
Female	$.259^{***}$ (.017)	−.006 (.016)	$.404^{***}$ [.30, .55]
Constant	$-.127^{***}$ (.012)	.021 (.011)	
n	9,686	9,807	11,522
Association between disadvantage and health for those with:			
Low cognitive ability (−1 *SD*)	$.133^{***}$ (.016)	$-.145^{***}$ (.014)	
Medium cognitive ability (mean)	$.083^{***}$ (.010)	$-.116^{***}$ (.010)	
High cognitive ability (+1 *SD*)	$.035^{**}$ (.010)	$-.085^{***}$ (.010)	

Note. BCS = British Cohort Study; HR = hazard ratio; CI = confidence interval.
** $p < .01$. *** $p < .001$.

Table 3
Regression Models Assessing the Cognitive Ability × Social Disadvantage Interaction in Predicting Psychological Distress, Self-Rated Health, and Mortality in the NCDS Cohort Prior to and After Adjustment for Childhood Health

	Psychological distress		Self-rated health		Mortality	
	Prior to	After	Prior to	After	Prior to: HR	After: HR
Predictor	*b* (*SE*)	*b* (*SE*)	*b* (*SE*)	*b* (*SE*)	[95% CI]	[95% CI]
Model 1						
Social disadvantage (*z* score)	.042*** (.008)	.041*** (.008)	−.092*** (.008)	−.090*** (.008)	1.16** [1.04, 1.28]	1.14* [1.03, 1.27]
Cognitive ability (*z* score)	−.167*** (.008)	−.164*** (.008)	.141*** (.008)	.136*** (.008)	.77*** [.70, .85]	.82*** [.74, .91]
Female	.395*** (.015)	.386*** (.015)	−.078*** (.014)	−.070*** (.015)	.62*** [.52, .76]	.64*** [.52, .77]
Constant	.215*** (.008)	.557* (.247)	.056*** (.010)	−.214 (.241)		
Model 2						
Social disadvantage (*z* score)	.053*** (.008)	.052*** (.008)	−.096*** (.008)	−.094*** (.008)	1.14* [1.02, 1.26]	1.13* [1.02, 1.25]
Cognitive ability (*z* score)	−.164*** (.008)	−.161*** (.008)	.140*** (.008)	.134*** (.008)	.79*** [.71, .87]	.83*** [.75, .92]
Social disadvantage × Cognitive ability	−.052*** (.008)	−.052*** (.008)	.022** (.007)	.020** (.007)	.90* [.82, .98]	.93† [.85, 1.01]
Female	.392*** (.015)	.383*** (.015)	−.076*** (.014)	−.069*** (.015)	.62*** [.51, .75]	.63*** [.52, .77]
Constant	−.194*** (.011)	.525* (.247)	.047*** (.011)	−.201 (.241)		
n	11,900	11,900	11,915	11,915	13,213	13,213
Association between disadvantage and health for those with:						
Low cognitive ability (−1 *SD*)	.107*** (.013)	.105*** (.013)	−.116*** (.013)	−.112*** (.013)	1.27*** [1.11, 1.43]	1.22** [1.07, 1.38]
Medium cognitive ability (mean)	.053*** (.008)	.053*** (.001)	−.096*** (.008)	−.092*** (.008)	1.12* [1.01, 1.24]	1.13* [1.02, 1.25]
High cognitive ability (+1 *SD*)	.002 (.009)	.001 (.009)	−.072*** (.009)	−.071*** (.009)	1.02 [.88, 1.17]	1.05 [.90, 1.21]

Note. NCDS = National Child Development Study.
† $p < .1$. * $p < .05$. ** $p < .01$. *** $p < .001$.

Additional Analyses

Table 3 also shows that controlling for childhood health did not markedly impact on the strength of the interaction between social disadvantage and cognitive ability in the NCDS cohort: There was no change in the interaction predicting psychological distress, and only small reductions were evident in the interaction effect for self-rated health and mortality. This was despite adjustment for an array of variables, some of which are likely to overlap substantially with cognitive ability levels (e.g., intellectual disability, head circumference). Tables 4 and 5 show the outcomes of the individual wave analyses. A significant interaction between disadvantage and cognitive ability was present in 13 of 16 individual wave analyses, which indicated that the anticipated buffering effect of cognitive ability was consistently evident across adulthood. The conditional effect of social disadvantage at each level of cognitive ability for each individual wave is also presented in Table S5 in the online supplemental materials and provides further support for this conclusion.

Finally, Tables S6-S9 in the online supplemental materials depict the outcomes of simple regressions predicting psychological distress and self-rated health at age 42 when each of four measures of social disadvantage in midlife were included in the model. The interaction remained significant when any form of adjustment for social disadvantage in adulthood was made. On average, the inclusion of adult social disadvantage measures reduced the magnitude of the interaction coefficient for psychological distress by 6.8% and 14.7% for the BCS and the NCDS, respectively. The average reduction of the coefficient for self-rated health was 10% in the BCS and 10.8% for the NCDS. These analyses suggest that the majority of the interactive associations between early disadvantage and cognitive ability in predicting adult health cannot be explained by adult socioeconomic status.

Discussion

Using two longitudinal British cohorts, the present report showed that childhood cognitive ability moderated the impact of early life exposure to social disadvantage on psychological distress, self-rated health, and mortality risk in adulthood. Across both cohorts, the adverse health consequences of early social disadvantage were found to be attenuated among those with high levels of childhood cognitive ability and most pronounced among those with low ability levels. Our results converge with prior evidence suggesting that those with lower cognitive ability may be most vulnerable to the mental and physical health effects of adversity (e.g., Fergusson & Lynskey, 1996; Hart et al., 2003; Riglin et al., 2016). However, in this study we provide the first evidence demonstrating that cognitive ability appears to have a persistent protective role in buffering against the long-run psychological and physical health consequences of early life disadvantage.

From young adulthood to middle age, those with high levels of childhood cognitive ability showed remarkable resilience to the stress of background disadvantage. For example, the longitudinal link between early disadvantage and adult psychological distress was over six times greater among those with low (−1 *SD*) as opposed to high (+1 *SD*) cognitive ability. Similarly, high cognitive ability appeared to promote resilience against the physical health consequences of familial disadvantage as gauged by personal ratings of health from young adulthood to midlife in both

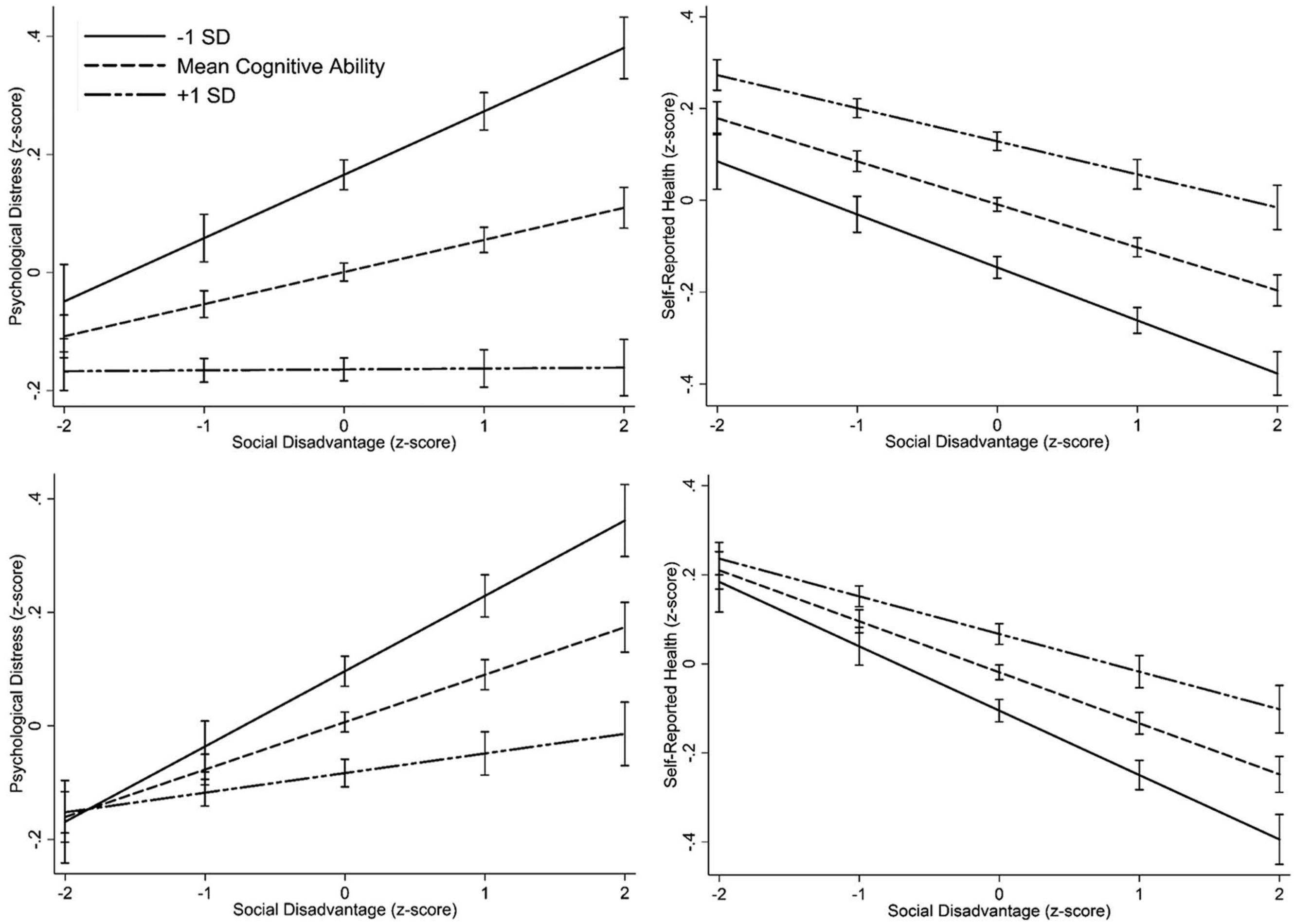

Figure 1. Association between social disadvantage and psychological distress (left) and self-rated health (right) at low (−1 *SD*), mean, and high (+1 *SD*) levels of cognitive ability. Upper panels: National Child Development Study cohort; lower panels: British Cohort Study cohort.

cohorts and premature mortality, at least in the longer running NCDS cohort. Perhaps due to the younger age of the BCS sample, there was no association between childhood disadvantage and mortality by age 42 and thus no detrimental impact of cognitive ability to modify. As such, our findings provide suggestive evidence that the protective role of cognitive ability in attenuating mortality risk may become increasingly evident as people age and the cumulative life span health effects of disadvantage become more apparent. In support of this idea, a previous study of 938 individuals born in 1921 and tracked for 25 years beyond midlife (Hart et al., 2003) found that the highest rates of all-cause mortality occurred among those who were both highly deprived and whose cognitive ability scores fell in the bottom quartile. Taken together, our findings coupled with existing work suggest that cognitive ability may be a key psychological resource that buffers the long-term health consequences of the stress of socioeconomic deprivation.

There are a number of potential explanations for why cognitive ability might confer protection in this way. Cognitive ability may foster successful adaptation to adversity by enabling people to respond fast, flexibly, and strategically to environmental challenges and demands, particularly in contexts where novel or complex problems must be addressed with limited resources (Godfrey-Smith, 2001). For example, high cognitive ability and the associated strong problem-solving capacities might better equip individuals to successfully avoid or negotiate potentially harmful stressors—ranging from daily hassles to stressful life events—which may pose a greater threat in conditions where financial resources are scarce (Almeida, Neupert, Banks, & Serido, 2005; Baum, Garofalo, & Yali, 1999; Dowd, Palermo, Chyu, Adam, & McDade, 2014). In this way, higher cognitive ability may diminish the likelihood of encountering adverse experiences and their downstream biological effects. Further, when stressful experiences cannot be avoided, cognitive ability may also confer mental resources such as greater executive functioning (e.g., working memory capacity, cognitive flexibility) that can support emotion regulation (Schmeichel & Tang, 2015) and thus directly mitigate the psychological and biological impact of exposure to stressors.

Supporting evidence for this notion has come from daily experiences data showing that higher cognitive ability is associated with smaller increases in negative mood in response to daily stressors (Stawski, Almeida, Lachman, Tun, & Rosnick, 2010).

Table 4
Regression Analyses Predicting Psychological Distress at Each Wave in the NCDS and BCS Cohorts

Cohort and predictors	Wave 1	Wave 2	Wave 3	Wave 4
NCDS				
Social disadvantage (*z* score)	.081*** (.010)	.046*** (.011)	.040*** (.011)	.039* (.012)
Cognitive ability (*z* score)	−.191*** (.010)	−.173*** (.011)	−.115*** (.011)	−.134*** (.012)
Social disadvantage × Cognitive ability	−.059*** (.019)	−.040*** (.011)	−.045*** (.011)	−.056*** (.012)
Female	.522*** (.019)	.361*** (.020)	.330*** (.020)	.316*** (.022)
Intercept	−.279*** (.013)	−.190*** (.015)	−.179*** (.015)	−.166*** (.016)
R^2	.118***	.064***	.043***	.046***
n	10,295	9,325	9,257	7,956
BCS				
Social disadvantage (*z* score)	.121*** (.015)	.053*** (.013)	.073*** (.017)	.072*** (.015)
Cognitive ability (*z* score)	−.077*** (.013)	−.088*** (.012)	−.090*** (.013)	−.081*** (.014)
Social disadvantage × Cognitive ability	−.059*** (.012)	−.042*** (.011)	−.042*** (.012)	−.048*** (.013)
Female	.372*** (.024)	.251*** (.022)	.252*** (.023)	.205*** (.025)
Intercept	−.216*** (.018)	−.147*** (.016)	−.142*** (.017)	−.109*** (.019)
R^2	.058***	.029***	.032***	.024***
n	6,577	8,199	7,120	6,324

Note. Standard errors appear in parentheses. NCDS = National Child Development Study; BCS = British Cohort Study.
* $p < .05$. *** $p < .001$.

Further, the idea that cognitive ability could protect against the affective consequences of exposure to socioeconomic adversity corresponds well with influential life span models of SES-related health disparities that posit that psychosocial resources exercise salutary effects at the point between stress exposure and its emotional impact (e.g., Matthews & Gallo, 2011). The current findings suggest that childhood cognitive ability could be viewed as an intrapersonal psychological resource of this kind, particularly in light of the observation that the protective effect of high ability was strongest for psychological distress outcomes and that evidence for protection against potential downstream health effects was also uncovered. However, additional evidence is needed to understand the extent to which higher cognitive ability enables individuals to avoid stressors associated with disadvantage or to dampen the consequential behavioral and physiological stress responses to such stressors that may produce vulnerability to disease (Matthews & Gallo, 2011).

This study has several key strengths. We examine background disadvantage in early childhood and follow the same individuals into adulthood observing their health across decades of follow-up. As such, we can rule out the possibility of reverse causality, whereby one's socioeconomic status is partially a result of the impact of one's previous physical and mental health (e.g., Goodman, Joyce, & Smith, 2011), a problem that is commonplace in studies of health inequalities conducted in adulthood (Smith, 1999). Further, social disadvantage was measured using a reliable continuous composite index comprised of the same set of key indicators capturing diverse elements of background deprivation in both cohort studies. In addition, validated and reliable tests of cognitive ability were used to identify a consistent moderating role

Table 5
Regression Analyses Predicting Self-Rated Health at Each Wave in the NCDS and BCS Cohorts

Cohort and predictors	Wave 1	Wave 2	Wave 3	Wave 4
NCDS				
Social disadvantage (*z* score)	−.052*** (.014)	−.109*** (.011)	−.105*** (.011)	−.127*** (.012)
Cognitive ability (*z* score)	.102*** (.011)	.138*** (.011)	.154*** (.011)	.160*** (.012)
Social disadvantage × Cognitive ability	.024* (.010)	.013 (.011)	.035** (.011)	.020 (.011)
Female	−.158*** (.019)	−.073*** (.020)	−.035 (.020)	−.026 (.022)
Intercept	.090*** (.014)	.031* (.015)	.022 (.015)	−.005 (.016)
R^2	.022***	.040***	.043***	.052***
n	10,302	9,262	9,329	8,031
BCS				
Social disadvantage (*z* score)	−.078*** (.015)	−.114*** (.013)	−.104*** (.014)	−.153*** (.014)
Cognitive ability (*z* score)	.050*** (.014)	.081*** (.012)	.069*** (.013)	.117*** (.013)
Social disadvantage × Cognitive ability	.009 (.013)	.038** (.011)	.024* (.012)	.040** (.012)
Female	−.077** (.025)	.024 (.022)	−.073** (.023)	.041 (.023)
Intercept	.032 (.019)	−.012 (.016)	.033 (.018)	−.019 (.017)
R^2	.021***	.022***	.019***	.043***
n	6,571	8,265	7,142	7,184

Note. Standard errors appear in parentheses. NCDS = National Child Development Study; BCS = British Cohort Study.
* $p < .05$. ** $p < .01$. *** $p < .001$.

of intelligence across cohorts and health variables. Finally, in the NCDS sample it was also possible to employ detailed data collected as part of medical examinations during childhood to demonstrate that this protective effect was unrelated to childhood health.

The current findings are not without their limitations. Two of the key measures of physical and psychological health employed here are dependent on participant self-report, which is vulnerable to various sources of bias. Nonetheless, the key claims made here are not dependent upon self-report measures and extend to an objective indicator of health—mortality. Further, because our key explanatory variables, cognitive ability and disadvantage, do not rely on subjective self-reports, our estimates are unlikely to be inflated by common method variance typically observed when both predictor and health outcome variables rely on self-reports measured on similar scales (e.g., Watson & Pennebaker, 1989). It is also the case that the current data remain observational in nature, and it was not possible to demonstrate a causal role of cognitive ability in decoupling the effect of social disadvantage on health over the life span. Within the field of cognitive epidemiology, it has been suggested that childhood cognitive ability may be a marker for the general integrity of multiple bodily systems (Deary, 2012). As such, we cannot rule out the noncausal explanation that cognitive ability may act as a proxy indicator of initial multisystem "fitness," which is the true protective factor that shapes effective adaptation to the environmental challenges of social deprivation. It is also important to note that individuals from Black, Asian and minority ethnic backgrounds constitute a very small portion of the current samples, and future research is needed to ensure these findings can be replicated in more ethnically diverse samples.

A final point to acknowledge here is the limited ability of the current study to speak to mechanisms by which the buffer effect might operate. As outlined earlier, we propose that cognitive ability may play a key role in reducing the extent to which individuals are exposed to and affected by the stress of adversity. Another possibility is that because higher cognitive ability predicts increased educational and status attainment in adulthood (Damian, Su, Shanahan, Trautwein, & Roberts, 2015; Strenze, 2007) those with high cognitive ability as children may go on to live in socioeconomic environments in adulthood that are more conducive to better health. However, our sensitivity tests examining the impact of adult socioeconomic status on the key interaction results provide only limited support for this explanation. On average, adjusting for adult SES led to a small reduction in the strength of the interaction between cognitive ability and early disadvantage in predicting distress and health ratings (8.4% in the BCS and 12.75% in the NCDS), suggesting that mobility may be a mechanism that can account for a minority of the protective effect of cognitive ability.

Conclusions

In summary, across two large United Kingdom samples, we found that childhood cognitive ability buffered the longitudinal link between early social disadvantage and distress, poor health, and mortality from early adulthood to midlife. The long-range protective effect of cognitive ability remained strong when health during childhood and socioeconomic variables in adulthood were adjusted for. The present findings suggest that whilst those with high cognitive ability levels appear to experience few ill effects of their disadvantaged upbringing, those with low childhood cognitive ability may be particularly vulnerable to the health consequences of early adversity. Conversely, those with low cognitive ability may be may be particularly likely to benefit from efforts to ameliorate the long-run economic and health effects of initial disadvantage through investment in preschool intervention programs, housing mobility programs, or the provision of family supports (e.g., Campbell et al., 2014; Leventhal & Brooks-Gunn, 2003). The current research provides initial support for a life-course account of childhood cognitive ability as a key psychological resource that shapes the development of health in disadvantaged circumstances. This work also sets the stage for future studies to test these relationships further and to identify the psychosocial processes through which such protective effects are likely to occur.

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