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ORIGINAL ARTICLE

Bullying victimization in childhood predicts inflammation and obesity at mid-life: a five-decade birth cohort study

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Background. We aimed to test whether childhood bullying victimization increases risk for age-related disease at mid-life using biological markers including inflammation and adiposity, independent of other childhood risk factors and key adult variables.

Method. The present study was a 50-year prospective longitudinal birth cohort study of all births in Britain in 1 week in 1958. Exposure to bullying was assessed prospectively when participants were aged 7 and 11 years (27.7% occasionally bullied; 14.6% frequently bullied). Blood inflammation biomarkers [C-reactive protein (CRP) and fibrinogen] and adiposity [body mass index (BMI) and waist:hip ratio] were measured at age 45 years.

Results. Participants who had been frequently bullied in childhood showed increased levels of CRP at mid-life [$\beta = 0.07$, 95% confidence interval (CI) 0.04–0.10] and higher risk for clinically relevant inflammation cut-off [CRP > 3 mg/l: 20.4% *v.* 15.9%, odds ratio (OR) = 1.35, 95% CI 1.12–1.64]. Women who were bullied in childhood had higher BMI than non-bullied participants and were at increased risk of being obese (BMI ≥ 30 kg/m^2: occasionally bullied: 26.0% *v.* 19.4%, OR = 1.45, 95% CI 1.18–1.77; frequently bullied: 26.2% *v.* 19.4%, OR = 1.41, 95% CI 1.09–1.83). Findings remained significant when controlling for childhood risk factors (e.g. parental social class; participants' BMI and psychopathology in childhood) and key adult variables (e.g. adult social class, smoking, diet and exercise).

Conclusions. Bullied children show increases in risk factors for age-related disease in middle adulthood, independent of co-occurring childhood and adult risks. Given the high prevalence of bullying victimization in childhood, tackling this form of psychosocial stress early in life has the potential of reducing risk for age-related disease and its associated burden.

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Introduction

As predicted life expectancy continues to rise (Christensen *et al.* 2009), age-related illnesses such as cardiovascular disease and type 2 diabetes are of increasing public health concern (Murray *et al.* 2012). Efforts aimed at reducing the burden of ill health in later years are focusing earlier in development because the pathophysiological processes underlying age-related disease appear to start much earlier in life (Berenson *et al.* 1998). Animal models show that early stressful experiences influence the ontogenesis of the immune and metabolic systems that promote ill health (Cole *et al.* 2012; Conti *et al.* 2012). Epidemiological surveys show that psychosocial stress in childhood plays a part in humans (Shonkoff *et al.* 2009; Brent & Silverstein, 2013); evidence indicates that maltreated children grow up to have higher levels of circulating inflammation proteins (Danese *et al.* 2007; Matthews *et al.* 2014) and higher risk of obesity (Danese & Tan, 2014) than their non-maltreated peers. Identifying childhood risks for age-related disease is especially important because readily available interventions focusing on risk factors in adulthood (e.g. smoking, inactivity and poor diet) have limited long-term efficacy (Braunwald, 1997; Ebrahim *et al.* 2006).

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Targeting potentially malleable risk factors early in life may provide alternative strategies to limit the burden of disease in an ageing population.

We explored these issues in relation to bullying victimization, a stressor that is common in childhood (Nansel *et al.* 2001) and is increasingly considered a form of maltreatment alongside abuse and neglect by adults (Gilbert *et al.* 2009). Bullying victimization occurs between people in the same age groups (e.g., children, adolescents, adults), where an imbalance of power makes it difficult for victims to defend themselves (Olweus, 1993). Victimization by bullies is associated with an increased risk of mental health disorders in childhood and adolescence (Arseneault *et al.* 2010); young victims of bullying show higher levels of anxiety and depression (Salmon *et al.* 1998; Bond *et al.* 2001; Arseneault *et al.* 2010) and also severe problems such as self-harm (Fisher *et al.* 2012), psychotic symptoms (Schreier *et al.* 2009; Arseneault *et al.* 2011) and suicidality (van Geel *et al.* 2014). Using the sample reported on here, we have shown that these risks persist to mid-life, and that they extend beyond mental health to affect physical and cognitive health and socio-economic outcomes (Takizawa *et al.* 2014). Bullying victimization has also been found to be associated with low-grade systemic inflammation in young adults in a US epidemiological sample (Copeland *et al.* 2014) and obesity (Midei & Matthews, 2011; Qualter *et al.* 2015). At this stage, however, it is not known whether exposure to bullying is associated with other markers of age-related disease and whether any such effect persists to mid-life, when markers are commonly used to predict disease. An answer to this question would improve understanding of the mechanisms involved in the ageing process and would further our knowledge on the outcomes associated with childhood bullying victimization.

We aimed to extend previous findings by testing whether childhood bullying victimization, assessed prospectively in a 50-year British birth cohort study, exerts effects on biological risks for age-related disease at mid-life. Following other research (Whitlock *et al.* 2009; Wormser *et al.* 2011; Kaptoge *et al.* 2012; Singh *et al.* 2013), we focused on indicators of inflammatory processes [e.g. C-reactive protein (CRP) and fibrinogen] and adiposity [body mass index (BMI) and waist:hip ratio] as our main indicators of such risks. We controlled for potential confounders of bullying victimization in childhood and known correlates of the selected outcomes in adult life. We hypothesized that cohort members who had been the victims of bullying in childhood would have higher levels of inflammatory markers and adiposity at mid-life, compared with participants who were not bullied, indicating a greater liability to later age-related disease.

Method

Participants

Data were from the National Child Development Study (NCDS), the 1958 British birth cohort study (Power & Elliott, 2006). Information was collected on 98% of all births in 1 week in 1958 in England, Scotland and Wales ($n = 17\,416$). Subsequent follow-ups took place at ages 7, 11 and 16 years in childhood, and at ages 23, 33, 42, 45, 50 and 55 years in adult life. We report on data collected at birth, from the age 7 and 11 years childhood contacts, and from the age 45 years biomedical survey. Ethical approval for the biomedical survey was given by the South East Multi-Centre Research Ethics Committee.

Assessment of bullying

Exposure to bullying was assessed via parental interviews when participants were 7 and 11 years. At each age, parents were asked if their child was bullied by other children 'never', 'sometimes' or 'frequently'. We combined responses from both interviews ($n = 11\,500$) to create a three-level indicator of exposure to childhood bullying: 0 = never bullied ('never' at both 7 and 11 years); 1 = occasionally bullied ('sometimes' at either 7 or 11 years); 2 = frequently bullied ('frequently' at either 7 or 11 years, or 'sometimes' at both ages). Where only one parental interview was available ($n = 2405$ at age 7 years; $n = 1311$ at age 11 years), responses from that interview were used, providing bullying assessments on 86% of cohort members. Consistent with findings in contemporary cohorts (Nansel *et al.* 2004), bullying victimization was common in this 1950s sample: 27.7% of children had been exposed to occasional bullying, and 14.6% had been frequently bullied.

Reports of bullying victimization from mothers and children have been shown to be similarly associated with emotional and behavioural problems (Shakoor *et al.* 2011). Although agreement between informants is typically low (Ronning *et al.* 2009; Wienke Totura *et al.* 2009), this suggests that both informants provide a unique and meaningful perspective on bullying victimization.

Measures of adult biomarkers

The biomedical survey was undertaken by trained nurses and included venepuncture, physical measurements and an in-home interview (Power & Elliott, 2006). Venous blood samples were centrifuged and the aliquots of plasma stored at −70 °C. CRP was measured by high-sensitivity nephelometric analysis of latex particles coated with CRP monoclonal antibodies (BN ProSpec protein analyser; Dade Behring, Germany).

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Inter- and intra-assay coefficients of variation were <10%. We excluded participants ($n = 210$) with CRP of 10 mg/l or more. We used two indicators of CRP levels: (i) a continuous measure (log-transformed to reduce skewness); and (ii) a dichotomous low *versus* high risk indicator based on the Centers for Disease Control and Prevention/American Heart Association definition of high cardiovascular risk (CRP > 3 mg/l) (Pearson *et al.* 2003). Fibrinogen was determined by the Clauss method (MDA 180 coagulometer; Biomerieux, UK). All analytes were monitored for internal quality control by Levey–Jennings plots during the assay period.

An anthropometric assessment was also conducted at the biomedical survey, providing measures of BMI [weight (kg)/height (m)^2] and waist:hip ratio [waist circumference (cm)/hip circumference (cm)]. We used two indicators of BMI: (i) a continuous measure; and (ii) a dichotomous indicator identifying obese participants with a BMI of 30 kg/m^2 or more.

Childhood confounders

Childhood physical status

Birth weight was taken from medical records and childhood BMI was calculated from height and weight measured at age 7 years.

Childhood cognition/behaviour

Childhood intelligence quotient (IQ) was assessed at age 11 years using a standardized 80-item general ability test (Douglas, 1964) with verbal and non-verbal components, and the values were standardized (mean=100, S.D.=15) in the whole sample. Childhood internalizing and externalizing problems were derived from teacher ratings on the Bristol Social Adjustment Guides (Stott, 1969) at 7 and 11 years (Clark *et al.* 2007). We used the mean of scores summed across these two ages where both measures were available ($n = 12\,366$), and single-age measures for the remainder of the sample ($n = 3136$).

Childhood environment

Family social class in childhood was classified on the basis of the father's occupation at age 7 years, and categorized as professional/managerial/technical, other non-manual, skilled manual, and unskilled manual (Office of Population Censuses and Surveys, 1980). Childhood adversity was assessed from both prospective and retrospective reports. Information prospectively collected from parents and teachers was used to create an eight-item scale of low parental involvement, including indicators of the child's physical appearance and the parents' activities with the child at ages 7 and 11 years (Power *et al.* 2012). Retrospectively at age 45 years, participants completed a 16-item questionnaire (Rosenman & Rodgers, 2004) about their exposure to a range of childhood adversities including poverty, parental mental health and drug/alcohol problems, family conflict, and physical and sexual abuse.

Adult correlates

We also took account of a range of adult factors (health-related behaviours and other correlates) with established associations with inflammatory markers and obesity in adult life. Data from the age 42 years interviews provided a range of sociodemographic and health behaviour indicators, including: (i) adult social class of cohort member (categorized in a similar way to parental social class in childhood); (ii) smoking status (0 = never or ex-smoker; 1 = current smoker); (iii) regular exercise (0 = no; 1 = yes, regularly); (iv) diet (eating fruits and vegetables, coded from 0 = never to 6 = more than once per day).

Depressive and anxiety disorders were assessed at age 45 years using the Depression and Anxiety modules of the Revised Clinical Interview Schedule (Lewis *et al.* 1992). Diagnoses were derived according to standard algorithms for International Classification of Diseases (ICD)-10 diagnoses. We used summary measures of: (i) mild, moderate and severe depressive disorders; (ii) any anxiety disorders (including generalized anxiety disorder, specific and social phobias, panic disorder and agoraphobia); and (iii) any anxiety or depressive disorders. The age 45 years assessments also included the Alcohol Use Disorders Identification Test (AUDIT; Babor *et al.* 2001), a 10-item screening questionnaire designed by the World Health Organization to identify mild alcohol dependence.

At the biomedical survey, participants were assessed for their use of medications for cardiovascular, respiratory and central nervous systems, infections, endocrine system and other problems, including systemic steroids, respiratory steroids, non-steroidal anti-inflammatory drugs, prophylactic aspirin, anti-gout medications, anti-rheumatic medications, statins and oestrogens.

Attrition

Sample retention in childhood was high (92% at age 7 and 11 years) (Power & Elliott, 2006). Retention rates were lower in adulthood, with data available on 78% of those invited for the mid-life biomedical survey (9426/12 069). For the current analyses, we took a conservative approach and report on 7102 cohort members with complete data on bullying victimization at age 7 or 11 years, and also inflammatory markers at the biomedical survey. Non-participation was unrelated to exposure to childhood bullying (online Supplementary Table S1), but was predicted by male gender, low

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Table 1. *Associations between bullying victimization in childhood and adult risk for cardiovascular disease*[a]

		Bullied at ages 7 and 11 years			Group difference	
Risks for cardiovascular disease	Total ($n = 7102$)	Never ($n = 4190$)	Occasionally ($n = 1919$)	Frequently ($n = 993$)	F/χ^2 (df)	p
Inflammation						
CRP >3 mg/l, *n* (%)	1178 (17.2)	646 (15.9)	332 (18.1)	200 (20.4)	11.01 (2)	0.004
CRP (log_{10}), mg/l	−0.02 (0.48)	−0.03 (0.48)	−0.01 (0.49)	0.04 (0.47)	9.44	<0.001
Fibrinogen, g/l	2.93 (0.57)	2.91 (0.56)	2.94 (0.58)	2.97 (0.58)	3.88	0.021
Adiposity						
Obesity, *n* (%)[b]						
Total	1613 (23.2)	886 (21.4)	471 (25.5)	256 (25.5)	13.43 (2)	0.001
Males	861 (24.3)	471 (23.6)	247 (25.2)	143 (25.0)	0.89	0.640
Females	752 (22.0)	415 (19.4)	224 (26.0)	113 (26.2)	17.95	<0.001
BMI, kg/m^2[c]						
Total	27.23 (4.76)	27.03 (4.66)	27.50 (4.86)	27.51 (4.90)	7.39	<0.001
Males	27.71 (4.15)	27.67 (4.07)	27.76 (4.11)	27.76 (4.48)	0.18	0.837
Females	26.72 (5.27)	26.41 (5.09)	27.20 (5.56)	27.20 (5.37)	8.18	<0.001
Waist:hip ratio	0.871 (0.085)	0.866 (0.084)	0.876 (0.084)	0.880 (0.088)	14.04	<0.001

Data are given as mean (standard deviation) unless otherwise indicated.

df, Degrees of freedom; CRP, C-reactive protein; BMI, body mass index.

[a] We report unweighted *n*, but weighted percentage, mean and standard deviation. We present results separately by gender where an interaction between bullying victimization and gender was found to be significant. Associations with categorical variables were estimated with ordinal logistic regression analyses, whereas associations with continuous variables were estimated with one-way analyses of variance.

[b] Group differences for obesity were not significant for males (Wald $\chi^2 = 0.89$, $p = 0.640$), but significant for females (Wald $\chi^2 = 17.95$, $p < 0.001$).

[c] Group differences for adult BMI were not significant for males ($F = 0.18$, $p = 0.837$), but significant for females ($F = 8.18$, $p < 0.001$).

birth weight, low IQ, low childhood social class, low parental involvement, and childhood internalizing and externalizing problems. We derived inverse probability weights (Seaman & White, 2013) from a logistic regression analysis predicting availability of complete data on childhood bullying and biological data of blood sample, including the variables listed above. These weights were included in all analyses.

Statistical analyses

We used linear regression and ordinal logistic regression analyses to test bivariate associations between childhood bullying victimization and adult risk factors for age-related disease. To test their robustness, multivariate models were used to adjust, in separate steps, for childhood confounders (child physical status, cognition and behaviours, and child environmental risk exposure) and adult covariates (including indicators of health-related behaviours). When not considered as an outcome, adult BMI was included as a covariate. All multivariate analyses controlled for gender (males = 1; females = 2) and medication use (no = 0; yes, any = 1). We tested for gender-specific effects in all models. We present results separately by gender where an interaction between bullying victimization and gender was found to be significant. We used robust variance (sandwich-type) estimates to adjust the standard errors of the parameter estimates for the sampling weights applied to observations. All analyses were conducted in STATA version 12.1 (USA).

Results

Is childhood bullying victimization associated with mid-life risks for age-related disease?

Individuals who had been frequently bullied in childhood showed higher levels of inflammation at mid-life than non-bullied participants (Table 1). We observed significant differences in CRP levels for both the clinically relevant categorical measure of CRP and also for the continuous measure. In addition, children who were frequently bullied showed elevated levels of fibrinogen. Women who had been either occasionally or frequently bullied in childhood showed greater

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adiposity in mid-life compared with women who had not been bullied. Differences in adiposity for bullied women were observed both for obesity and also across the full distribution of BMI. The effect of bullying generalized to a measure of central adiposity: both men and women who had been bullied in childhood showed greater waist:hip ratio at mid-life than non-bullied individuals.

Is bullying victimization associated with childhood risk factors and adult correlates of age-related disease?

Bullying victimization was associated with several potential confounders assessed in childhood and also with adult correlates of age-related disease risks (Table 2). In terms of child characteristics, bullied participants had lower birth weight and BMI at age 7 years than those who had not been bullied; they also had elevated levels of internalizing and externalizing problems and lower IQ scores; and they experienced greater socio-economic disadvantage as they were growing up. In mid-life, participants who had been bullied in childhood were in lower social class occupations and showed higher rates of affective disorders than their non-bullied peers. They were more likely to smoke than non-bullied participants and less likely to take regular exercise or eat healthily; bullied women were also more likely to report taking medications (Table 2).

Is childhood bullying victimization independently associated with adult risk for age-related disease after controlling for childhood confounders and adult correlates?

Controlling for childhood confounders and adult covariates did not modify the observed group differences in inflammation markers. Compared with those who had not been bullied in childhood, participants who had been frequently bullied showed higher CRP levels in mid-life after statistical adjustment for child physical status, cognition and behaviour, and environment, and also for adult health-related behaviours and other adult correlates (Table 3). Adjusted analyses revealed similar results for the effect of bullying on fibrinogen levels, although the association was reduced to marginal significance when we statistically controlled for childhood behavioural problems and IQ, childhood adversities, and adult social class and BMI.

Compared with women who had not been bullied in childhood, women who had been occasionally or frequently bullied showed significantly higher rates of adult obesity and higher BMI in mid-life after childhood confounders and adult correlates were statistically accounted for (Table 3). In relation to waist:hip ratio, associations with occasional bullying were less consistent, but links with frequent bullying remained largely significant.

When childhood confounders and adult correlates were statistically controlled for simultaneously, findings remained significant for the continuous measures of both CRP [$\beta = 0.04$, 95% confidence interval (CI) 0.00–0.07, $p = 0.034$] and BMI ($\beta = 0.54$, 95% CI 0.08–1.00, $p = 0.022$).

Discussion

We report evidence that childhood bullying victimization is associated with increased risks for age-related disease at mid-life. Our study using data from a large prospective birth cohort shows that study participants who experienced bullying victimization have higher inflammation levels than non-bullied peers, and women who had been bullied are more likely to be obese decades later. Findings are consistent across two different measures of inflammation and two different measures of adiposity. They are also independent of the effects of correlated childhood risks, and of key adult risk factors targeted by current preventive interventions.

These findings are innovative in three ways. First, they add to the growing body of evidence from human and non-human primates showing the adverse effects of early stress on inflammatory and adiposity markers. Second, they widen the spectrum of poor outcomes associated with childhood bullying victimization to risks for age-related disease. Third, they show that early psychosocial stress exerts enduring effects that remain significant into mid-life, when inflammation and obesity are used clinically to predict age-related disease risk. Bullying victimization is a concern for policy and intervention because of the extensive evidence that it contributes to suffering in childhood and adolescence (Arseneault *et al.* 2010). Recent research findings have been supporting initiatives aimed at reducing bullying behaviours. Findings from this study further suggest that preventing bullying in childhood could reduce risks for age-related disease. Our study also implies that early interventions with the victims could not only limit poor outcomes associated with being bullied in childhood but also contribute to the prevention of health problems in adulthood.

The effects of childhood bullying victimization on biological risks for age-related disease at mid-life were observed on both clinically relevant categorical measures and also across the whole continuum of the distribution of inflammation and adiposity. Therefore, tackling the effect of bullying victimization on risks for age-related disease may not only reduce the prevalence of problems at the clinical level, but

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Table 2. *Associations between bullying victimization in childhood and childhood confounding factors and key adult variables*[a]

		Bullied at ages 7 and 11 years			Group difference	
	Total ($n = 7102$)	Never ($n = 4190$)	Occasionally ($n = 1919$)	Frequently ($n = 993$)	F/χ^2 (df)	*P*
Male gender, *n* (%)	3573 (51.1)	2012 (49.1)	1005 (52.5)	556 (56.2)	15.83 (2)	<0.001
Childhood						
Physical status						
Birth weight, g	3335.3 (510.1)	3352.2 (508.0)	3317.6 (506.0)	3302.9 (523.4)	4.62	0.010
Child BMI, kg/m^2	15.84 (1.66)	15.87 (1.68)	15.86 (1.62)	15.70 (1.65)	2.93	0.020
Cognition/behaviour						
Internalizing problems	1.94 (0.91)	1.85 (0.88)	2.03 (0.92)	2.14 (0.97)	46.59	<0.001
Externalizing problems	1.95 (0.96)	1.89 (0.92)	2.00 (1.01)	2.10 (1.02)	19.81	<0.001
IQ	100.71 (14.17)	102.01 (13.89)	99.44 (14.24)	97.99 (14.52)	38.89	<0.001
Environmental exposure						
Parental social class, *n* (%)					35.94 (2)	<0.001
Professional/managerial	1555 (19.6)	1030 (22.0)	369 (17.5)	156 (14.1)		
Skilled non-manual	677 (9.3)	406 (9.4)	190 (9.7)	81 (7.9)		
Skilled manual	3091 (45.0)	1763 (44.3)	846 (44.5)	482 (48.9)		
Semi-skilled/unskilled manual	1740 (26.1)	957 (24.3)	509 (28.3)	274 (29.0)		
Low parental involvement	1.14 (1.43)	1.03 (1.34)	1.25 (1.50)	1.36 (1.62)	25.50	<0.001
Child adversity	1.50 (2.18)	1.38 (2.08)	1.59 (2.25)	1.80 (2.37)	15.59	<0.001
Adulthood						
Health-related behaviours						
Smoking, *n* (%)	1926 (28.1)	1113 (27.2)	504 (27.5)	309 (32.1)	6.64(2)	0.036
Regular exercise, *n* (%)	5213 (75.3)	3108 (76.2)	1413 (75.2)	692 (72.1)	6.04(2)	0.049
Eating fruits	4.03 (1.74)	4.09 (1.70)	4.01 (1.76)	3.81 (1.82)	9.59	<0.001
Eating vegetables	3.07 (1.48)	3.11 (1.47)	3.01 (1.50)	3.01 (1.51)	3.90	0.020
Other adult correlates						
Adult social class, *n* (%)					49.67 (2)	<0.001
Professional/managerial	2914 (38.5)	1834 (41.4)	740 (36.3)	340 (31.3)		
Skilled non-manual	1438 (20.1)	857 (20.4)	390 (20.1)	191 (19.1)		
Skilled manual	1341 (20.3)	754 (19.7)	364 (19.6)	223 (23.9)		
Semi-skilled/unskilled manual	1409 (21.1)	745 (18.5)	425 (24.0)	239 (25.8)		
Depression/anxiety diagnosis, *n* (%)	403 (6.0)	217 (5.3)	112 (6.3)	74 (8.0)	9.06 (2)	0.011
Alcohol dependence, *n* (%)	429 (6.6)	267 (6.8)	105 (6.5)	57 (6.3)	0.46 (2)	0.793
Medications, *n* (%)[b]						
Total	2602 (36.8)	1493 (35.8)	718 (37.4)	391 (39.8)	5.14 (2)	0.076
Males	1022 (28.5)	576 (28.6)	275 (27.2)	171 (30.4)	1.56 (2)	0.459
Females	1580 (45.5)	917 (42.7)	443 (48.7)	220 (51.8)	14.94 (2)	<0.001

Data are given as mean (standard deviation) unless otherwise indicated.

df, Degrees of freedom; BMI, body mass index; IQ, intelligence quotient.

[a] We report unweighted *n*, but weighted percentage, mean and standard deviation. Associations with categorical variables were estimated in ordinal logistic regression analyses, whereas associations with continuous variables were estimated in one-way analyses of variance.

[b] Gender-specific associations were found only for use of medication only.

also across the wider population. Clinical studies are needed to establish if the effects of childhood bullying on risks for age-related disease can be remediated before the onset of clinical symptoms. For example, in previous research we reported that bullied children are at higher risk of psychiatric disorders in adult life (Takizawa *et al.* 2014), and psychiatric disorders have been linked to prospective increase in obesity (Luppino *et al.* 2010). It is unclear if interventions that can relieve mental health problems in bullied individuals can also affect the biological vulnerability for age-related disease.

Future research also needs to investigate the molecular mechanisms through which exposure to psychosocial

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Table 3. *Multivariate models for the associations between childhood bullying victimization and adult risk for cardiovascular disease, controlling for childhood confounders and adult key variables*[a]

	Unadjusted	Model 1 – controlling for child physical status[b]	Model 2 – controlling for child cognition/ behaviour[b]	Model 3 – controlling for child environment[b]	Model 4 – controlling for adult health-related behaviours[b]	Model 5 – controlling for other adult correlates[b]
Inflammation						
CRP > 3 mg/l						
Occasionally bullied	1.17 (0.99 to 1.37)†	1.15 (0.97 to 1.37)	1.12 (0.95 to 1.32)	1.13 (0.96 to 1.33)	1.14 (0.96 to 1.33)	1.11 (0.93 to 1.33)
Frequently bullied	1.35 (1.12 to 1.64)**	1.33 (1.09 to 1.64)**	1.27 (1.04 to 1.54)*	1.29 (1.07 to 1.57)**	1.27 (1.05 to 1.55)*	1.29 (1.04 to 1.59)*
CRP (log_{10})						
Occasionally bullied	0.02 (−0.01 to 0.05)	0.02 (−0.01 to 0.05)	0.01 (−0.02 to 0.04)	0.01 (−0.02 to 0.03)	0.01 (−0.02 to 0.04)	0.00 (−0.03 to 0.02)
Frequently bullied	0.07 (0.04 to 0.10)***	0.07 (0.03 to 0.11)***	0.05 (0.02 to 0.09)**	0.05 (0.02 to 0.09)**	0.05 (0.02 to 0.09)**	0.05 (0.02 to 0.09)**
Fibrinogen						
Occasionally bullied	0.03 (−0.01 to 0.06)	0.02 (−0.02 to 0.05)	0.01 (−0.02 to 0.05)	0.01 (−0.02 to 0.05)	0.03 (0.00 to 0.06)	0.01 (−0.02 to 0.04)
Frequently bullied	0.06 (0.02 to 0.10)**	0.05 (0.01 to 0.10)*	0.04 (0.00 to 0.08)†	0.04 (0.00 to 0.08)*	0.05 (0.00 to 0.09)*	0.04 (0.00 to 0.08)†
Adiposity						
Obesity (only for females)						
Occasionally bullied	1.45 (1.18 to 1.77)**	1.43 (1.18 to 1.74)**	1.39 (1.17 to 1.67)**	1.39 (1.17 to 1.66)**	1.41 (1.18 to 1.69)***	1.39 (1.15 to 1.67)**
Frequently bullied	1.41 (1.09 to 1.83)**	1.39 (1.08 to 1.79)**	1.26 (1.00 to 1.59)*	1.26 (1.00 to 1.59)*	1.33 (1.06 to 1.68)*	1.28 (1.00 to 1.64)*
BMI (only for females)						
Occasionally bullied	0.87 (0.44 to 1.31)**	0.86 (0.41 to 1.32)**	0.76 (0.33 to 1.19)**	0.74 (0.30 to 1.17)**	0.86 (0.42 to 1.30)***	0.77 (0.33 to 1.22)**
Frequently bullied	0.73 (0.17 to 1.30)**	0.87 (0.30 to 1.44)**	0.54 (−0.03 to 1.11)†	0.52 (−0.05 to 1.10)†	0.75 (0.18 to 1.32)**	0.52 (−0.05 to 1.10)†
Waist:hip ratio (×100)[c]						
Occasionally bullied	0.41 (0.08 to 0.74)**	0.41 (0.05 to 0.76)*	0.22 (0.10 to 0.55)	0.27 (−0.06 to 0.60)	0.37 (0.04 to 0.70)*	0.30 (−0.04 to 0.64)†
Frequently bullied	0.70 (0.27 to 1.14)**	0.80 (0.34 to 1.25)***	0.39 (−0.04 to 0.83)†	0.47 (0.04 to 0.91)*	0.56 (0.12 to 1.00)**	0.50 (0.05 to 0.95)*

Data are given as odds ratio or β coefficient (95% confidence interval).

CRP, C-reactive protein; BMI, body mass index.

[a] All models controlled for effects of gender and use of medications. We present results separately by gender where an interaction between bullying victimization and gender was found to be significant.

[b] We report the adjusted odds ratio/β values in model 1 [child physical status (birth weight and child BMI)], in model 2 [child cognition/behaviour (internalizing problems and externalizing problems and childhood intelligence quotient)], in model 3 [child environment (parental social class, low parental involvement and child adversity)], in model 4 [adult health-related behaviours (smoking, regular exercise, diet and exercise] and in model 5 [other adult correlates (adult social class, depression/anxiety disorders, alcohol dependence and adult BMI)].

[c] Waist:hip ratio values were multiplied by 100 for these analyses.

Significant findings: *** $p \leq 0.001$, ** $p \leq 0.01$, * $p \leq 0.05$, † $p \leq 0.10$ (trend level).

adversity, including bullying in childhood, can be translated to biological risks for disease (biological embedding; Danese & McEwen, 2012). Bullied children undergo epigenetic changes (Ouellet-Morin *et al.* 2013) associated with blunted neuroendocrine response to psychosocial challenges (Ouellet-Morin *et al.* 2011*a*). We have previously reported that these neuroendocrine abnormalities were linked to greater internalizing and externalizing problems in childhood (Ouellet-Morin *et al.* 2011*b*). It is possible that these neuroendocrine abnormalities also contribute to the later risks for high inflammation and obesity observed here (Danese & McEwen, 2012).

Despite these significant effects, not all bullied children had high inflammation or were obese in adult life. Notably, the effect of childhood bullying on midlife BMI was limited to women. This is consistent with reported effects of maltreatment in humans (Danese & Tan, 2014), and early life stress in non-human primates (Conti *et al.* 2012). However, we found that the effect of early life stress could be generalized across genders with regard to abdominal adiposity. The origins and mechanisms of these individual differences need to be further investigated.

Our findings should be interpreted in light of some limitations. First, attrition in the NCDS across nearly five decades of assessment was not negligible, though it is unlikely that this affected the pattern of our findings; drop-out was not associated with bullying victimization and we controlled for other effects of selective attrition by including weights throughout the analyses. Second, the associations we observed could be confounded by a number of unmeasured factors. Reassuringly, findings from our study of a large cohort of human participants are consistent with those of experimental research in non-human primates (Cole *et al.* 2012; Conti *et al.* 2012) that allow greater control over potential confounders. Third, we did not assess age-related diseases *per se*, but rather their established risks. An unknown proportion of participants will not develop age-related disease in later life despite their known risks. For prevention schemes and policies, however, knowing about risks may be useful for deciding targets of effective interventions. Fourth, the public health significance of childhood bullying victimization could be questioned in light of the relatively small effects reported here. This may reflect associations across an extended period over the life course. The high prevalence of bullying victimization, high inflammation and obesity would, however, suggest that preventable effects in the population may still be considerable despite the low effect sizes reported here.

Regardless of these limitations, our results have potential implications for future research, clinical practice and public health. Bullying by peers appears to have a non-negligible effect on risks for age-related disease independent from those of other established risks. This and other forms of childhood adversity are not currently addressed by preventive interventions for age-related disease. In light of these findings, new models of preventive interventions for age-related disease need to consider psychosocial risk factors and life-course trajectories. The main focus of preventive interventions for age-related disease has traditionally been on physical risk factors, such as smoking, physical inactivity and unhealthy diet. These are clearly important, but interventions targeting these established risk factors in adults are challenging and of limited effect (Braunwald, 1997; Ebrahim *et al.* 2006). Some of the root causes of these unhealthy behaviours may be traced back to childhood psychosocial adversity (Anda *et al.* 1999) and these also need to be tackled by health promotion strategies.

Supplementary material

For supplementary material accompanying this paper visit http://dx.doi.org/10.1017/S0033291715000653

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R.T., A.D., B.M. and L.A. are responsible for the study concept and design, interpretation of data, and drafting and revising the manuscript for important intellectual content. R.T. had full access to all the data in the study, takes responsibility for the integrity of the data and the accuracy of the data analysis.

Declaration of Interest

None.

References

Anda RF, Croft JB, Felitti VJ, Nordenberg D, Giles WH, Williamson DF, Giovino GA (1999). Adverse childhood experiences and smoking during adolescence and adulthood. *Journal of the American Medical Association* **282**, 1652–1658.

Arseneault L, Bowes L, Shakoor S (2010). Bullying victimization in youths and mental health problems: 'much ado about nothing'? *Psychological Medicine* **40**, 717–729.

Arseneault L, Cannon M, Fisher HL, Polanczyk G, Moffitt TE, Caspi A (2011). Childhood trauma and children's emerging psychotic symptoms: a genetically sensitive longitudinal cohort study. *American Journal of Psychiatry* **168**, 65–72.

Babor T, Higgins-Biddle J, Saunders J, Monteiro M (2001). *The Alcohol Use Disorders Identification Test, Guidelines for Use in Primary Care*. World Health Organization: Geneva.

Berenson GS, Srinivasan SR, Bao W, Newman WP, III, Tracy RE, Wattigney WA (1998). Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. *New England Journal of Medicine* **338**, 1650–1656.

Bond L, Carlin JB, Thomas L, Rubin K, Patton G (2001). Does bullying cause emotional problems? A prospective study of young teenagers. *BMJ* **323**, 480–484.

Braunwald E (1997). Shattuck lecture – cardiovascular medicine at the turn of the millennium: triumphs, concerns, and opportunities. *New England Journal of Medicine* **337**, 1360–1369.

Brent DA, Silverstein M (2013). Shedding light on the long shadow of childhood adversity. *Journal of the American Medical Association* **309**, 1777–1778.

Christensen K, Doblhammer G, Rau R, Vaupel JW (2009). Ageing populations: the challenges ahead. *Lancet* **374**, 1196–1208.

Clark C, Rodgers B, Caldwell T, Power C, Stansfeld S (2007). Childhood and adulthood psychological ill health as predictors of midlife affective and anxiety disorders: the 1958 British Birth Cohort. *Archives of General Psychiatry* **64**, 668–678.

Cole SW, Conti G, Arevalo JM, Ruggiero AM, Heckman JJ, Suomi SJ (2012). Transcriptional modulation of the developing immune system by early life social adversity. *Proceedings of the National Academy of Sciences of the United States of America* **109**, 20578–20583.

Conti G, Hansman C, Heckman JJ, Novak MF, Ruggiero A, Suomi SJ (2012). Primate evidence on the late health effects of early-life adversity. *Proceedings of the National Academy of Sciences of the United States of America* **109**, 8866–8871.

Copeland WE, Wolke D, Lereya ST, Shanahan L, Worthman C, Costello EJ (2014). Childhood bullying involvement predicts low-grade systemic inflammation into adulthood. *Proceedings of the National Academy of Sciences of the United States of America* **111**, 7570–7575.

Danese A, McEwen BS (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology and Behavior* **106**, 29–39.

Danese A, Pariante CM, Caspi A, Taylor A, Poulton R (2007). Childhood maltreatment predicts adult inflammation in a life-course study. *Proceedings of the National Academy of Sciences of the United States of America* **104**, 1319–1324.

Danese A, Tan M (2014). Childhood maltreatment and obesity: systematic review and meta-analysis. *Molecular Psychiatry* **19**, 544–554.

Douglas JWB (1964). *The Home and the School*. MacGibbon & Kee: London.

Ebrahim S, Beswick A, Burke M, Davey Smith G (2006). Multiple risk factor interventions for primary prevention of coronary heart disease. *Cochrane Database of Systematic Reviews*, Issue 1. Art. no. CD001561. doi:10.1002/14651858.CD001561.pub3.

Fisher HL, Moffitt TE, Houts RM, Belsky DW, Arseneault L, Caspi A (2012). Bullying victimisation and risk of self harm in early adolescence: longitudinal cohort study. *BMJ* **344**, e2683.

Gilbert R, Widom CS, Browne K, Fergusson D, Webb E, Janson S (2009). Burden and consequences of child maltreatment in high-income countries. *Lancet* **373**, 68–81.

Kaptoge S, Di Angelantonio E, Pennells L, Wood AM, White IR, Gao P, Walker M, Thompson A, Sarwar N, Caslake M, Butterworth AS, Amouyel P, Assmann G, Bakker SJ, Barr EL, Barrett-Connor E, Benjamin EJ, Bjorkelund C, Brenner H, Brunner E, Clarke R, Cooper JA, Cremer P, Cushman M, Dagenais GR, D'Agostino Sr, RB, Dankner R, Davey-Smith G, Deeg D, Dekker JM, Engstrom G, Folsom AR, Fowkes FG, Gallacher J, Gaziano JM, Giampaoli S, Gillum RF, Hofman A, Howard BV, Ingelsson E, Iso H, Jorgensen T, Kiechl S, Kitamura A, Kiyohara Y, Koenig W, Kromhout D, Kuller LH, Lawlor DA, Meade TW, Nissinen A, Nordestgaard BG, Onat A, Panagiotakos DB, Psaty BM, Rodriguez B, Rosengren A, Salomaa V, Kauhanen J, Salonen JT, Shaffer JA, Shea S, Ford I, Stehouwer CD, Strandberg TE, Tipping RW, Tosetto A, Wassertheil-Smoller S, Wennberg P, Westendorp RG, Whincup PH, Wilhelmsen L, Woodward M, Lowe GD, Wareham NJ, Khaw KT, Sattar N, Packard CJ, Gudnason V, Ridker PM, Pepys MB, Thompson SG, Danesh J (2012). C-reactive protein, fibrinogen, and cardiovascular disease prediction. *New England Journal of Medicine* **367**, 1310–1320.

Lewis G, Pelosi AJ, Araya R, Dunn G (1992). Measuring psychiatric disorder in the community: a standardized assessment for use by lay interviewers. *Psychological Medicine* **22**, 465–486.

Luppino FS, de Wit LM, Bouvy PF, Stijnen T, Cuijpers P, Penninx BW, Zitman FG (2010). Overweight, obesity, and depression: a systematic review and meta-analysis of longitudinal studies. *Archives of General Psychiatry* **67**, 220–229.

Matthews KA, Chang YF, Thurston RC, Bromberger JT (2014). Child abuse is related to inflammation in mid-life women: role of obesity. *Brain, Behavior, and Immunity* **36**, 29–34.

Midei AJ, Matthews KA (2011). Interpersonal violence in childhood as a risk factor for obesity: a systematic review of the literature and proposed pathways. *Obesity Reviews* **12**, e159–e172.

Murray CJL, Vos T, Lozano R, Naghavi M, Flaxman AD, Michaud C, Ezzati M, Shibuya K, Salomon JA, Abdalla S, Aboyans V, Abraham J, Ackerman I, Aggarwal R, Ahn SY, Ali MK, Alvarado M, Anderson HR, Anderson LM, Andrews KG, Atkinson C, Baddour LM, Bahalim AN, Barker-Collo S, Barrero LH, Bartels DH, Basanez MG, Baxter A, Bell ML, Benjamin EJ, Bennett D, Bernabe E, Bhalla K, Bhandari B, Bikbov B, Bin Abdulhak A, Birbeck G, Black JA, Blencowe H, Blore JD, Blyth F, Bolliger I, Bonaventure A, Boufous SA, Bourne R, Boussinesq M, Braithwaite T, Brayne C, Bridgett L, Brooker S, Brooks P, Brugha TS, Bryan-Hancock C, Bucello C, Buchbinder R, Buckle G, Budke CM, Burch M, Burney P, Burstein R, Calabria B, Campbell B, Canter CE, Carabin H, Carapetis J, Carmona L, Cella C, Charlson F, Chen HL, Cheng ATA, Chou D, Chugh SS, Coffeng LE, Colan SD, Colquhoun S, Colson KE, Condon J, Connor MD, Cooper LT, Corriere M, Cortinovis M, De Vaccaro KC, Couser W, Cowie BC, Criqui MH, Cross M, Dabhadkar KC, Dahiya M, Dahodwala N, Damsere-Derry J, Danaei G, Davis A, De Leo D, Degenhardt L, Dellavalle R, Delossantos A, Denenberg J, Derrett S, Des Jarlais DC, Dharmaratne SD, Dherani M, Diaz-Torne C, Dolk H, Dorsey ER, Driscoll T, Duber H, Ebel B, Edmond K, Elbaz A, Ali SE, Erskine H, Erwin PJ, Espindola P, Ewoigbokhan SE, Farzadfar F, Feigin V, Felson DT, Ferrari A, Ferri CP, Fevre EM, Finucane MM, Flaxman S, Flood L, Foreman K, Forouzanfar MH, Fowkes FGR, Fransen M, Freeman MK, Gabbe BJ, Gabriel SE, Gakidou E, Ganatra HA, Garcia B, Gaspari F, Gillum RF, Gmel G, Gonzalez-Medina D, Gosselin R, Grainger R, Grant B, Groeger J, Guillemin F, Gunnell D, Gupta R, Haagsma J, Hagan H, Halasa YA, Hall W, Haring D, Haro JM, Harrison JE, Havmoeller R, Hay RJ, Higashi H, Hill C, Hoen B, Hoffman H, Hotez PJ, Hoy D, Huang JJ, Ibeanusi SE, Jacobsen KH, James SL, Jarvis D, Jasrasaria R, Jayaraman S, Johns N, Jonas JB, Karthikeyan G, Kassebaum N, Kawakami N, Keren A, Khoo JP, King CH, Knowlton LM, Kobusingye O, Koranteng A, Krishnamurthi R, Laden F, Lalloo R, Laslett LL, Lathlean T, Leasher JL, Lee YY, Leigh J, Levinson D, Lim SS, Limb E, Lin JK, Lipnick M, Lipshultz SE, Liu W, Loane M, Ohno SL, Lyons R, Mabweijano J, MacIntyre MF, Malekzadeh R, Mallinger L, Manivannan S, Marcenes W, March L, Margolis DJ, Marks GB, Marks R, Matsumori A, Matzopoulos R, Mayosi BM, McAnulty JH, McDermott MM, McGill N, McGrath J, Medina-Mora ME, Meltzer M, Mensah GA, Merriman TR, Meyer AC, Miglioli V, Miller M, Miller TR, Mitchell PB, Mock C, Mocumbi AO, Moffitt TE, Mokdad AA, Monasta L, Montico M, Moradi-Lakeh M, Moran A, Morawska L, Mori R, Murdoch ME, Mwaniki MK, Naidoo K, Nair MN, Naldi L, Narayan KMV, Nelson PK, Nelson RG, Nevitt MC, Newton CR, Nolte S, Norman P, Norman R, O'Donnell M, O'Hanlon S, Olives C, Omer SB, Ortblad K, Osborne R, Ozgediz D, Page A, Pahari B, Pandian JD, Rivero AP, Patten SB, Pearce N, Padilla RP, Perez-Ruiz F, Perico N, Pesudovs K, Phillips D, Phillips MR, Pierce K, Pion S, Polanczyk GV, Polinder S, Pope CA, Popova S, Porrini E, Pourmalek F, Prince M, Pullan RL, Ramaiah KD, Ranganathan D, Razavi H, Regan M, Rehm JT, Rein DB, Remuzzi G, Richardson K, Rivara FP, Roberts T, Robinson C, De Leon FR, Ronfani L, Room R, Rosenfeld LC, Rushton L, Sacco RL, Saha S, Sampson U, Sanchez-Riera L, Sanman E, Schwebel DC, Scott JG, Segui-Gomez M, Shahraz S, Shepard DS, Shin H, Shivakoti R, Singh D, Singh GM, Singh JA, Singleton J, Sleet DA, Sliwa K, Smith E, Smith JL, Stapelberg NJC, Steer A, Steiner T, Stolk WA, Stovner LJ, Sudfeld C, Syed S, Tamburlini G, Tavakkoli M, Taylor HR, Taylor JA, Taylor WJ, Thomas B, Thomson WM, Thurston GD, Tleyjeh IM, Tonelli M, Towbin JRA, Truelsen T, Tsilimbaris MK, Ubeda C, Undurraga EA, van der Werf MJ, van Os J, Vavilala MS, Venketasubramanian N, Wang MR, Wang WZ, Watt K, Weatherall DJ, Weinstock MA, Weintraub R, Weisskopf MG, Weissman MM, White RA, Whiteford H, Wiebe N, Wiersma ST, Wilkinson JD, Williams HC, Williams SRM, Witt E, Wolfe F, Woolf AD, Wulf S, Yeh PH, Zaidi AKM, Zheng ZJ, Zonies D, Lopez AD (2012). Disability-adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* **380**, 2197–2223.

Nansel TR, Craig W, Overpeck MD, Saluja G, Ruan WJ (2004). Cross-national consistency in the relationship between bullying behaviors and psychosocial adjustment. *Archives of Pediatrics and Adolescent Medicine* **158**, 730–736.

Nansel TR, Overpeck M, Pilla RS, Ruan WJ, Simons-Morton B, Scheidt P (2001). Bullying behaviors among US youth: prevalence and association with psychosocial adjustment. *Journal of the American Medical Association* **285**, 2094–2100.

Office of Population Censuses and Surveys (1980). *Classification of Occupations*. HMSO: London.

Olweus D (1993). *Bullying at School: What We Know and What We Can Do*. Blackwell Publishers: Oxford.

Ouellet-Morin I, Danese A, Bowes L, Shakoor S, Ambler A, Pariante CM, Papadopoulos AS, Caspi A, Moffitt TE, Arseneault L (2011*a*). A discordant monozygotic twin design shows blunted cortisol reactivity among bullied children. *Journal of the American Academy of Child and Adolescent Psychiatry* **50**, 574–582.e3.

Ouellet-Morin I, Odgers CL, Danese A, Bowes L, Shakoor S, Papadopoulos AS, Caspi A, Moffitt TE, Arseneault L (2011*b*). Blunted cortisol responses to stress signal social and behavioral problems among maltreated/bullied 12-year-old children. *Biological Psychiatry* **70**, 1016–1023.

Ouellet-Morin I, Wong CC, Danese A, Pariante CM, Papadopoulos AS, Mill J, Arseneault L (2013). Increased serotonin transporter gene (SERT) DNA methylation is associated with bullying victimization and blunted cortisol response to stress in childhood: a longitudinal study of discordant monozygotic twins. *Psychological Medicine* **43**, 1813–1823.

Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon RO III, Criqui M, Fadl YY, Fortmann SP, Hong Y, Myers GL, Rifai N, Smith Jr SC Taubert K, Tracy RP, Vinicor F (2003). Markers of inflammation and cardiovascular disease: application to clinical and public

https://doi.org/10.1017/S0033291715000653

health practice: a statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. *Circulation* **107**, 499–511.

Power C, Elliott J (2006). Cohort profile: 1958 British birth cohort (National Child Development Study). *International Journal of Epidemiology* **35**, 34–41.

Power C, Thomas C, Li L, Hertzman C (2012). Childhood psychosocial adversity and adult cortisol patterns. *British Journal of Psychiatry* **201**, 199–206.

Qualter P, Murphy SM, Abbott J, Gardner KJ, Japel C, Vitaro F, Boivin M, Tremblay RE (2015). Developmental associations between victimization and body mass index from 3 to 10 years in a population sample. *Aggressive Behavior*. Published online 14 January 2015. doi:10.1002/AB.21580.

Ronning JA, Sourander A, Kumpulainen K, Tamminen T, Niemela S, Moilanen I, Helenius H, Piha J, Almqvist F (2009). Cross-informant agreement about bullying and victimization among eight-year-olds: whose information best predicts psychiatric caseness 10–15 years later? *Social Psychiatry and Psychiatric Epidemiology* **44**, 15–22.

Rosenman S, Rodgers B (2004). Childhood adversity in an Australian population. *Social Psychiatry and Psychiatric Epidemiology* **39**, 695–702.

Salmon G, James A, Smith DM (1998). Bullying in schools: self reported anxiety, depression, and self esteem in secondary school children. *BMJ* **317**, 924–925.

Schreier A, Wolke D, Thomas K, Horwood J, Hollis C, Gunnell D, Lewis G, Thompson A, Zammit S, Duffy L, Salvi G, Harrison G (2009). Prospective study of peer victimization in childhood and psychotic symptoms in a nonclinical population at age 12 years. *Archives of General Psychiatry* **66**, 527–536.

Seaman SR, White IR (2013). Review of inverse probability weighting for dealing with missing data. *Statistical Methods in Medical Research* **22**, 278–295.

Shakoor S, Jaffee SR, Andreou P, Bowes L, Ambler AP, Caspi A, Moffitt TE, Arseneault L (2011). Mothers and children as informants of bullying victimization: results from an epidemiological cohort of children. *Journal of Abnormal Child Psychology* **39**, 379–387.

Shonkoff JP, Boyce WT, McEwen BS (2009). Neuroscience, molecular biology, and the childhood roots of health disparities: building a new framework for health promotion and disease prevention. *Journal of the American Medical Association* **301**, 2252–2259.

Singh GM, Danaei G, Farzadfar F, Stevens GA, Woodward M, Wormser D, Kaptoge S, Whitlock G, Qiao Q, Lewington S, Di Angelantonio E, Vander Hoorn S, Lawes CM, Ali MK, Mozaffarian D, Ezzati M (2013). The age-specific quantitative effects of metabolic risk factors on cardiovascular diseases and diabetes: a pooled analysis. *PLOS ONE* **8**, e65174.

Stott DH (1969). *The Social Adjustment of Children*. University of London Press: London.

Takizawa R, Maughan B, Arseneault L (2014). Adult health outcomes of childhood bullying victimization: evidence from a five-decade longitudinal British birth cohort. *American Journal of Psychiatry* **171**, 777–784.

van Geel M, Vedder P, Tanilon J (2014). Relationship between peer victimization, cyberbullying, and suicide in children and adolescents: a meta-analysis. *JAMA Pediatrics* **168**, 435–442.

Whitlock G, Lewington S, Sherliker P, Clarke R, Emberson J, Halsey J, Qizilbash N, Collins R, Peto R (2009). Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies. *Lancet* **373**, 1083–1096.

Wienke Totura CM, Green AE, Karver MS, Gesten EL (2009). Multiple informants in the assessment of psychological, behavioral, and academic correlates of bullying and victimization in middle school. *Journal of Adolescence* **32**, 193–211.

Wormser D, Kaptoge S, Di Angelantonio E, Wood AM, Pennells L, Thompson A, Sarwar N, Kizer JR, Lawlor DA, Nordestgaard BG, Ridker P, Salomaa V, Stevens J, Woodward M, Sattar N, Collins R, Thompson SG, Whitlock G, Danesh J (2011). Separate and combined associations of body-mass index and abdominal adiposity with cardiovascular disease: collaborative analysis of 58 prospective studies. *Lancet* **377**, 1085–1095.