#### ARTICLE

# Pre-morbid intelligence, the metabolic syndrome and mortality: the Vietnam Experience Study

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#### Abstract

*Aims/hypothesis* We examined the relationship between pre-morbid intelligence quotient (IQ) and the metabolic syndrome, and assessed the role of the metabolic syndrome as a mediating factor in the association of IQ with total and cardiovascular disease (CVD) mortality.

*Methods* In this cohort study, 4,157 men with IQ test results from late adolescence or early adulthood [mean age

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M. J. Shipley Department of Epidemiology and Public Health, University College London, London, UK (range) 20.4 (16–30) years] attended a clinical examination in middle-age [38.3 (31–46) years] at which the components of the metabolic syndrome were measured. They were then followed for 15 years to assess mortality.

*Results* In age-adjusted analyses, IQ was significantly inversely related to four of the five individual components comprising the metabolic syndrome: hypertension, high BMI, high triglycerides and high blood glucose, but not low HDL-cholesterol. After controlling for a range of covariates that included socioeconomic position, higher IQ scores were associated with a reduced prevalence of the metabolic syndrome itself (odds ratio<sub>1 SD increase in IQ</sub> 0.87, 95% CI 0.78–0.98). Structural equation modelling revealed that education was not a mediator of the relationship between IQ and the metabolic syndrome. The metabolic syndrome partially mediated the relationship between IQ and CVD but not that between IQ and total mortality.

*Conclusions/interpretation* In this cohort, higher scores on a pre-morbid IQ test were associated with a lower prevalence of the metabolic syndrome and most of its components. The metabolic syndrome was a mediating variable in the IQ–CVD relationship.

Keywords Cardiovascular disease  $\cdot$  CVD  $\cdot$ Intelligence quotient  $\cdot$  IQ  $\cdot$  Metabolic syndrome  $\cdot$ Mortality  $\cdot$  Socioeconomic position

# Abbreviations

- CVD cardiovascular disease
- HR hazard ratio
- IQ intelligence quotient
- OR odds ratio
- PCA principal components analysis

### Introduction

Empirical research in the field of cognition (i.e. intelligence quotient, IQ), has a long tradition in the social sciences. It has recently been suggested that the skills captured by IO tests, such as comprehension and reasoning, may have a role in an individual's interpretation of health promotion advice and hence their choice of behaviours [1]. Thus, in comparison to their lower-performing counterparts, higher IQ-scoring individuals have more favourable levels of cigarette smoking [2–5] (uptake and cessation), physical activity [6] and dietary characteristics [6, 7]. Although there are fewer studies exploring the link between IQ and physiological variables, it has been suggested that higher IO scores are associated with lower levels of blood pressure or hypertension [3, 8, 9] and obesity [10-12]. The scant reports of the relationship between IQ and later measurement of serum cholesterol and high blood glucose or diabetes [13] reveal null results, although sample sizes are modest. While some of these risk indices represent the cluster of factors comprising the metabolic syndrome, to our knowledge, the relationship between IO and this constellation of risk indices has not been examined.

Data from the Vietnam Experience Study, a large cohort of former US army personnel who had their IQ assessed at entry to the service in early adulthood and then took part in a telephone interview and medical examination some 20 years later [14–16], provide a novel opportunity to examine the IQ-metabolic syndrome relationship. As the vital status of these men post-medical examination is also known, an additional novel objective of the present analyses is to examine the mediating role, if any, of the metabolic syndrome in the association between IQ and both total and cardiovascular disease (CVD) mortality.

## Methods

Data collection in late adolescence or early adulthood Study participants were identified retrospectively using existing military records, a process that has been described in detail elsewhere [14–16]. In brief, information pertaining to place of service, military rank, ethnicity and IQ were extracted from military archives for 18,313 former military personnel.

On enlistment into military service, the men routinely completed a general aptitude (IQ) test: the Army General Technical Test. This consists of two subtests, verbal and arithmetic reasoning. We validated this test by comparing scores from it with those from components of the Wechsler Adult Intelligence Scale, a comprehensive and widely used test of cognitive ability that study participants (n=4,411) completed during the medical examination in middle-age (described below). Total Army General Technical Test results were strongly correlated with information subtest scores (r=0.74, 95% CI 0.73–0.76), block design scores (r=0.51, 95% CI 0.49–0.55) and overall results (r=0.74, 95% CI 0.73–0.76) from the Wechsler Adult Intelligence Scale. Mean age at army entry when IQ was assessed was 20.4 years (range 16–30).

Data collection in middle-age—telephone survey A total of 15,288 (85.6% of target population) of 17,867 men found to be alive on 31 December 1983 participated in the 1985 telephone survey, during which enquiries were made about the study participants' medical history, health behaviours and socioeconomic characteristics. Socioeconomic position was measured using household income, an index of occupational prestige [17, 18] and years of completed education.

Data collection in middle-age-medical examination In 1986, a random sample of telephone interview respondents (n=6,443) was invited to attend a 3 day medical examination; 4,462 attended (69.3% of those invited; mean age 38.3 years, range 31-46). All men were requested to fast from 19:00 hours on the evening before medical testing. Blood was drawn the following morning and levels of triglycerides and cholesterol fractions ascertained using an autoanalyser (Ektachem 700; Kodak, Rochester, NY, USA) [19, 20]. Serum glucose level was determined with a standard adaptation of the glucose oxidase-peroxidasechromogen-coupled system for glucose determination in biological fluids [19, 20]. Blood pressure, height and weight were assessed using standard protocols. Body mass index was computed using the usual formula (weight [kg]/ height  $[m]^2$ ). Subsequent to completion of the medical examination, attempts were made to match study participants against mortality databases for a 15 year period.

Definition of the metabolic syndrome We used two methods to define the metabolic syndrome. In the first method, we defined the metabolic syndrome and its components using a modified version of the Adult Treatment Panel III recommended diagnostic criteria [21] (here, termed the conventionally derived index). According to this definition, participants were classified as having the metabolic syndrome if any three of the following were present: BMI  $>30 \text{ kg/m}^2$  (in the absence of data on waist circumference, BMI at this threshold is regarded by the WHO as an acceptable substitute in defining the metabolic syndrome [22]); fasting plasma glucose  $\geq 6.1 \text{ mmol/l}$  (110 mg/dl) or medication for diabetes (as reported at the medical examination); triglycerides  $\geq 1.7 \text{ mmol/l} (150 \text{ mg/dl});$ HDL-cholesterol level <1.036 mmol/l (40 mg/dl); and blood pressure ≥130/85 mmHg and/or use of antihypertensive medication. A second approach was based on evidence that a single latent factor may underlie the core components of the metabolic syndrome [23]. We therefore carried out a principal components analysis (PCA) of BMI, triglycerides, HDL, blood glucose, systolic and diastolic blood pressure, and use of antihypertensive medication. To ascertain whether a single underlying component was sufficient to describe the associations among these variables, we used the scree slope criterion [24]. The scree slope showed just one substantial component above the 'scree' of trivial components and we therefore extracted a single score based on the first unrotated principal component (here, termed the PCA-derived index).

Statistical analysis The present analyses are based on a sample of men with complete information on IQ, covariates obtained at entry into the army, telephone interview and medical examination, and mortality (n=4,157). This group represents 23.3% of persons originally enrolled in the study. Although this analytical sample is based on the recruitment of a random sample of surviving men, concerns about selection bias are nonetheless possible; that is, if the reported results differ markedly between persons included in the analyses and those not. In Table 1 we present the early adult characteristics of men in the analytical cohort with those of men excluded from the sample. Differences were very small: in comparison to the excluded group, men

 
 Table 1
 Comparison of early adult characteristics of men included in the analytical sample with those excluded

	Included $(n=4,157)$	Excluded ( <i>n</i> =11,131)	p value
Army income			
(US\$ per week)			
83-119	513 (12.3)	1,395 (12.5)	0.94
120–144	2,059 (49.5)	5,486 (49.3)	
145	1,585 (38.1)	4,250 (38.2)	
Place of service			
Ever served in Vietnam	2,299 (55.3)	5,625 (50.5)	< 0.001
Other overseas posting	1,069 (25.7)	3,179 (28.6)	
Served in USA only	789 (19.0)	2,327 (20.9)	
Ethnic group			
White	3,405 (81.9)	9,228 (82.9)	0.21
Black	492 (11.8)	1,203 (10.8)	
Other <sup>a</sup>	261 (6.3)	700 (6.3)	
IQ at enlistment, mean (SD)	101.3 (15.2)	100.4 (14.9)	0.001
Age at enlistment (years), mean (SD)	19.9 (1.72)	19.9 (1.70)	0.67

Values are n (%), unless otherwise indicated

<sup>a</sup> Comprises Hispanics, Asians, Pacific Islanders, American Indians and Alaskan Natives

in the analytical sample had higher IQ test scores and a greater proportion with service experience in Vietnam. The fact that these marginal differences reached statistical significance can be ascribed to the large sample size.

We assessed the relationship between IO and the metabolic syndrome in three ways that provided complementary information. In the first analyses, we used logistic regression to examine the relationship between IQ and both each individual component of the metabolic syndrome and the conventionally derived metabolic syndrome. In these analyses, outcomes were dichotomised and adjustments were made for a range of covariates (age, rank, ethnicity, education, income and social prestige). In the second analyses, by computing Pearson partial correlation coefficients, we related IO to the naturally coded (continuous) versions of the components of the metabolic syndrome and the PCA-derived index described above, which represents a score for the metabolic syndrome. This preserves all the information in the variables and provides effect sizes for associations. Third, we carried out an analysis using structural equation modelling with the EQS program, version 6.1 [25]. In the hypothetical model, a latent trait of general cognitive ability was formed from the verbal and arithmetic ability test scores. The following indicators were used to form a latent trait of metabolic syndrome: systolic blood pressure, BMI, triglycerides, HDL-cholesterol, glucose and hypertensive medications. The explicit hypothesis tested was that latent general mental ability influenced the latent metabolic syndrome trait and that some of that effect was mediated via education, a close correlate of IO [26]. The measurement models (for the latent traits) and the structural model (the path part of the model) are given in Fig. 1. The fit of the model was tested comprehensively, as described in the results section.

Finally, to examine the mediating role of the metabolic syndrome, if any, between IQ and mortality from all causes and CVD we computed hazard ratios (HRs) and accompanying 95% CIs using Cox proportional hazards regression model with age as the underlying time scale [27]. IQ was used as a continuous measure with effect estimates reported for a 1 SD increase. Follow-up time was taken from the medical examination until censoring, death or 31 December 2000, whichever came first (mean follow-up time 15.1 years).

The Vietnam Experience Study was authorised by the US Congress. The protocol for the study was reviewed by the US Office for Technology Assessment, the Department of Health and Human Sciences Advisory Committee, the Agent Orange Working Group Science Panel and a panel from the US Centers for Disease Control. On the first day of the medical examination, participants attended an orientation session and signed a consent form.

## Results

In Table 2 we present the relationships between IQ and study covariates. IQ was associated with each of the indicators of socioeconomic position during service (army income) and in middle-age (income, occupational prestige and education), such that there was a lower level of disadvantage in the groups with higher IQ scores. Higher IQ scores were also apparent in veterans who were white and in those whose place of service did not include Vietnam.

Table 3 depicts the relationship between IO, components of the metabolic syndrome and the metabolic syndrome itself. In age-adjusted analyses, with the exception of HDLcholesterol, all components of the metabolic syndrome in middle-age were inversely related to IQ in early adulthood at conventional levels of statistical significance. Controlling for ethnicity led to partial attenuation of the inverse associations of IO with hypertension and blood glucose. and accentuated the triglycerides association, but had little effect on the other metabolic components. With the exception of triglycerides as an endpoint, separate adjustment for adult indices of socioeconomic position, i.e. income, social prestige and education, had little impact on the odds ratios (ORs) for individual components. Following collective adjustment for the aforementioned covariates, a significant inverse relationship between IQ and high BMI, high triglycerides and low HDL-cholesterol (emerging for the first time) was apparent. When the metabolic syndrome was the endpoint of interest, higher IO scores were associated with a reduced prevalence of this condition (OR1 SD increase in IQ 0.86, 95% CI 0.80-0.99). Individual control for study covariates, including education, weakened this gradient very little. After full adjustment for all covariates, the OR was essentially the same as the analysis adjusted only for age and statistical significance was retained. When we related IQ to naturally coded (continuous) versions of each component that comprises the metabolic syndrome and to a PCA-derived index based on each of the components, the pattern of association of IQ with individual components was similar to that in Table 2 when each of the components was dichotomised. Thus, ageadjusted correlation coefficients were: -0.067 (p<0.001) for systolic blood pressure; -0.077 (p<0.001) for diastolic blood pressure; -0.071 (p<0.001) for BMI; -0.038 (p< 0.05) for triglycerides; -0.021 (p > 0.05) for HDL-cholesterol; -0.092 (p<0.001) for blood glucose; and -0.102 (p< 0.001) for the factor-derived metabolic syndrome index (results for other adjustments available on request).

Figure 1 shows the results of the structural equation model used to explore the possible mediating effect of educational attainment between IQ and the metabolic syndrome. We present a number of different recommended fit indices so that the adequacy of the model can be assessed comprehensively [28, 29]. The measurement parts of the model were successful; the two indicators of mental ability loaded very highly on the latent trait of cognitive ability, and all indicators of the metabolic syndrome loaded significantly on the latent trait. The overall fit of the model was good. The average of the off-diagonal absolute standardised residuals was 0.03. This indicates that the model accounted for most of the covariance among the measured variables. The root mean square error of approximation was 0.06 (95% CI 0.055–0.066), with good fit indicated by an upper bound of the 95% CI of less than 0.1. The  $\chi^2$  test value was large (388.1, df=23) and significant (p<0.0001), but this is common in large samples. The Bentler-Bonett normed and non-normed and comparative fit indices were all good (>0.9) at 0.95, 0.92 and 0.95, respectively. These

**Table 2** IQ and study characteristics in the Vietnam Experience Study (n=4,157)

	n (%)	IQ score <sup>b</sup> , mean (SD)
Data recorded at baseline		
Age group (years)		
≤18	584 (14.0)	98.1 (13.1)
19–22	3,192 (76.8)	101.3 (15.1)
≥23	381 (9.2)	106.3 (17.6)
Army income (US\$ per week)		
83–119	513 (12.3)	93.6 (13.8)
120–144	2,059 (49.5)	99.1 (15.1)
145	1,585 (38.1)	106.7 (13.9)
Place of service		
Ever served in Vietnam	2,299 (55.3)	100.4 (14.8)
Other overseas posting	1,069 (25.7)	101.6 (15.7)
Served in USA only	789 (19.0)	103.8 (15.5)
Ethnic group		
White	3,405 (81.9)	103.9 (14.2)
Black	492 (11.8)	87.7 (13.2)
Other <sup>a</sup>	261 (6.3)	93.6 (14.5)
Data recorded at follow-up		
Educational attainment		
Grade ≤11	502 (12.1)	86.6 (12.1)
Grade 12	1,515 (36.4)	97.0 (12.8)
Grade ≥13	2,140 (51.5)	107.9 (13.9)
Family income (US\$ per year)		
≤20,000	1,195 (28.7)	94.1 (14.7)
20,001-40,000	2,065 (49.7)	101.7 (14.0)
>40,000	897 (21.6)	110.2 (13.4)
Occupational prestige		
≤25th percentile	1,132 (27.2)	94.1 (14.4)
26th-50th percentile	961 (23.1)	97.2 (13.8)
51st-75th percentile	1,313 (31.6)	104.8 (13.7)
>75th percentile	751 (18.1)	111.5 (12.8)

<sup>a</sup> Comprises Hispanics, Asians, Pacific Islanders, American Indians and Alaskan Natives

 $^{b}p < 0.001$  for all comparisons

	Hypertension	High BMI	High triglycerides	Low HDL-cholesterol	High blood glucose	Metabolic syndrome
With condition, $n (\%)$	2,212 (53.21)	537 (12.92)	807 (19.41)	1,544 (37.14)	220 (5.29)	649 (15.61)
Age-adjusted	0.90(0.84 - 0.95)	0.82 (0.75–0.89)	$0.91 \ (0.84 - 0.98)$	$0.97 \ (0.92 - 1.03)$	$0.75 \ (0.66 - 0.86)$	0.86(0.80-0.99)
Age- + rank-adjusted	0.90(0.84 - 0.96)	0.81 (0.74–0.89)	$0.91 \ (0.84 - 0.98)$	0.96(0.91 - 1.03)	$0.75 \ (0.65 - 0.86)$	0.86(0.79-0.94)
Age- + ethnicity-adjusted	$0.95\ (0.89-1.01)$	$0.83 \ (0.75 - 0.91)$	0.83 (0.77 - 0.90)	0.95(0.89 - 1.02)	0.85(0.74-0.98)	$0.84 \ (0.77 - 0.92)$
Age- + place of service-adjusted	0.90(0.84 - 0.95)	0.81 (0.74–0.89)	$0.91 \ (0.85 - 0.98)$	0.97 (0.91 - 1.03)	0.76(0.67 - 0.87)	0.86(0.80-0.94)
Age- + education-adjusted	0.88(0.81 - 0.94)	$0.84 \ (0.75 - 0.93)$	$0.96\ (0.87{-}1.02)$	1.01(0.94 - 1.09)	0.76(0.65 - 0.88)	$0.89 \ (0.81 - 0.98)$
Age- + social prestige-adjusted	0.89(0.83 - 0.95)	$0.82 \ (0.74 - 0.90)$	$0.94 \ (0.86 - 1.02)$	0.95(0.88 - 1.02)	$0.79 \ (0.68 - 0.91)$	0.88(0.80-0.96)
Age- + income-adjusted	0.88(0.83 - 0.94)	$0.83 \ (0.75 - 0.91)$	$0.93 \ (0.85 - 1.03)$	0.97 (0.91 - 1.04)	$0.78 \ (0.67 - 0.89)$	$0.87 \ (0.80 - 0.95)$
Age- + socioeconomic position <sup>a</sup>	0.88(0.81 - 0.94)	$0.85 \ (0.76 - 0.95)$	$0.97 \ (0.88 - 1.06)$	0.99(0.92 - 1.07)	0.80(0.69 - 0.93)	0.88 (0.80–0.97)
Age- + fully adjusted	$0.96\ (0.89{-}1.05)$	0.87 (0.77–0.98)	0.87 (0.79–0.97)	0.89 (0.82–0.97)	$0.92 \ (0.78 - 1.08)$	0.87 ( $0.78-0.98$ )
Fig. 1 Pathway model linking IQ in early adulthood with the metabolic syndrome in middle- age in the Vietnam Experience Study ( $n=4,379$ ). Numbers adjacent to the arrows represent path coefficients. <sup>a</sup> With significance set at $p<0.05$ , all coefficients were significant except this one	Arithmetic	0.80 General mental 0.80 ability	0.13 0.61 Education	0.03 <sup>a</sup> Metabolic syndrome	0.50 Systolic blood 0.71 BMI 0.41 Triglycerides	blood ides
					0.28 Hypertensive medications	lsive

indices compare the stipulated model with a baseline model (in which the variables are uncorrelated) and have a maximum value of 1. The path weight of the association between general mental ability and the metabolic syndrome latent trait was 0.13 (p<0.01). These path weights may be considered as being similar to standardised partial beta weights in a regression model; if squared, they give the variance shared by adjacent variables in the model. As anticipated, general mental ability was highly related to education (path weight 0.61). In the path model, there was only a weak and statistically non-significant association between education and the metabolic syndrome latent trait (path weight 0.03, p>0.05); therefore, education did not act as a significant mediator of the association between early adult mental ability and the metabolic syndrome.

Finally, as shown in Table 4, we examined the impact of controlling for the metabolic syndrome on the relationship between IQ and both total and CVD mortality. Some mediating impact was anticipated, given the previously described relationship between IQ and the metabolic syndrome, and the finding that the metabolic syndrome, as expected, was associated with all-cause mortality in this cohort. Thus in age-adjusted analyses, both the conventionally derived metabolic syndrome (HR<sub> $\geq 3$ </sub> components vs fewer 1.98, 95% CI 1.48–2.65) and the PCA-derived version (HR<sub>1 SD increase</sub> 1.35, 95% CI 1.21–1.51) predicted total mortality risk. However, the age-adjusted relationship between high IQ and total mortality (HR<sub>1 SD increase in IQ</sub> 0.70, 95% CI 0.61–0.79) was little changed when the convention-

tionally derived index (HR1 SD increase in IQ 0.72, 95% CI 0.63-0.81) and the PCA-derived index (HR<sub>1 SD increase in IO</sub> 0.73, 95% CI 0.64-0.82) were separately added to the multivariable model. The change in comparison to ageadjusted HRs was <10%. Controlling for markers of socioeconomic position and education, particularly in middle-age, yielded by far the greatest attenuation in the strength of the IQ-mortality relationship (70%). When CVD was the outcome of interest, adding the conventionally derived metabolic syndrome (HR<sub>1 SD increase in IO</sub> 0.78, 95% CI 0.61-1.00) and, particularly, the PCA-derived index (HR<sub>1 SD increase in IO</sub> 0.83, 95% CI 0.65-1.06) to the multivariable model had a larger impact on its relationship with IQ than in the analyses in which total mortality was the endpoint; statistical significance was lost at conventional levels. Again, controlling for later life socioeconomic position led to the greatest attenuation (92%) of any of the covariates (HR<sub>1 SD increase in IQ</sub> 0.98, 95% CI 0.72-1.32).

## Discussion

To our knowledge, this is the first study to examine the link between IQ and the metabolic syndrome, and to assess the impact, if any, of controlling for the metabolic syndrome on the IQ–mortality gradient. We found that men with higher IQ test scores in early adulthood experienced a reduced prevalence of the metabolic syndrome in middle-age. This gradient was not explained by control for markers of

 Table 4
 HRs (95% CI) for the relationship of a 1 SD increase in IQ with total and cardiovascular disease mortality in the Vietnam Experience Study

	Total mortality <sup>a</sup>		CVD mortality <sup>a,b</sup>	
	HR (95% CI)	Per cent reduction in HR <sup>d</sup>	HR (95% CI)	Per cent reduction in HR <sup>d</sup>
Age-adjusted	0.70 (0.61-0.79)	_	0.75 (0.59-0.96)	_
Age- + ethnicity-, place of service-, army income-adjusted	0.83 (0.81–1.09)	43.3	0.86 (0.65–1.13)	44.0
Age- + systolic blood pressure-adjusted	0.71 (0.63-0.81)	3.3	0.77 (0.61-0.98)	8.0
Age- + BMI-adjusted	0.71 (0.63-0.81)	3.3	0.79 (0.62-1.01)	16.0
Age- + blood glucose-adjusted	0.73 (0.64-0.83)	10.0	0.81 (0.63-1.03)	24.0
Age- + HDL-cholesterol-adjusted	0.71 (0.62-0.81)	3.3	0.76 (0.60-0.98)	4.0
Age- + triglycerides-adjusted	0.71 (0.62-0.81)	3.3	0.78 (0.61-0.99)	12.0
Age- + adult socioeconomic position <sup>c</sup> -adjusted Metabolic syndrome:	0.91 (0.78–1.06)	70.0	0.98 (0.72–1.32)	92.0
Age- + conventionally derived-adjusted	0.72 (0.63-0.81)	6.7	0.78 (0.61-1.00)	12.0
Age- + PCA-derived-adjusted	0.73 (0.64–0.82)	10.0	0.83 (0.65-1.06)	32.0

<sup>a</sup> Total mortality: n=231 deaths; CVD mortality: n=63 deaths (both among 4,157 men)

<sup>b</sup> Deaths for CVD were coded according to International Classification of Diseases codes 390-434 and 436-445 in version 9, and I00-I99 in version 10

<sup>c</sup> Based on income, education and social prestige

<sup>d</sup> Percentage effect of controlling for a given covariate was calculated as follows:  $[(HR_{age-adjusted} - 1) - (HR_{covariate-adjusted} - 1)/(HR_{age-adjusted} - 1)] \times 100$  [36]

socioeconomic circumstances either at the time of IQ testing (army income) or during measurement of the components of the metabolic syndrome (income, occupation, education). We did not find evidence that the IQ-metabolic syndrome association is mediated by education. Controlling for the metabolic syndrome had little impact on the IQ-total mortality relationship, with greater attenuation seen for IQ-CVD. This might be ascribed to CVD being more strongly linked with the metabolic syndrome than all-cause mortality [30], the latter comprising a range of outcomes that will dilute the strength of the association.

*Plausible mechanisms* As described, early life IQ is associated with more favourable levels of smoking [2–5], physical exertion [6], dietary characteristics [6, 7] and heavy alcohol consumption [31], although the latter is not a universal finding [32–34]. This suggests that the skills captured by IQ tests, such as verbal comprehension and reasoning, may be important in the successful management of a person's health behaviours. As such, it is perhaps unsurprising that IQ is also associated with several physiological correlates of these behaviours, e.g. blood pressure, adiposity, blood glucose and cholesterol, which collectively represent the metabolic syndrome.

Study strengths and limitations This study has a number of strengths, not least its prospective design and the temporal measurement of IQ, which preceded that of the metabolic syndrome and, in turn, mortality. Moreover, we used an appropriate combination of epidemiological and structural equation models to fully understand the associations and possible confounding and mediating factors. Having IQ test scores from late adolescence or early adulthood, rather than from older ages, is also crucial in examining the link between cognitive ability and the metabolic syndrome. Where mental test data are available from earlier in life, any apparent protective effect of high IQ scores is unlikely to be explained by reverse causality, in which components of the metabolic syndrome themselves could actually lead to a reduction in cognitive function [35].

The study is limited in that women were not examined. It is uncertain to what extent these results might be applicable to women. Second, the range of IQ scores in the present cohort may be narrower than in general population groups, not only due to the occupational nature of the population studied (the so called 'healthy worker' effect), but also because, from the outset, personnel with a rank corresponding to above sergeant were excluded from the study sample. However, any reduced variance would lead to an underestimation of the true effect of IQ on the metabolic syndrome. Third, to our knowledge, the original IQ tests were in the English language, which may have handicapped ethnic minorities. In conclusion, in this first study to examine the link between IQ and the metabolic syndrome, we found an inverse relationship, which was not mediated by education. The metabolic syndrome did not, however, appear to substantially mediate the relationship between high IQ scores and lower mortality rates, although some attenuation was apparent when CVD death was the endpoint of interest.

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