

The association between childhood cognitive ability and adult long-term sickness absence in three British birth cohorts: a cohort study

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ABSTRACT

Objectives: The authors aimed to test the relationship between childhood cognitive function and long-term sick leave in adult life and whether any relationship was mediated by educational attainment, adult social class or adult mental ill-health.

Design: Cohort study.

Setting: The authors used data from the 1946, 1958 and 1970 British birth cohorts. Initial study populations included all live births in 1 week in that year. Follow-up arrangements have differed between the cohorts.

Participants: The authors included only those alive, living in the UK and not permanent refusals at the time of the outcome. The authors further restricted analyses to those in employment, full-time education or caring for a family in the sweep immediately prior to the outcome. 2894 (1946), 15 053 (1958) and 14 713 (1970) cohort members were included. Primary and secondary outcome measures: receipt of health-related benefits (eg, incapacity benefit) in 2000 and 2004 for the 1958 and 1970 cohorts, respectively, and individuals identified as 'permanently sick or disabled' in 1999 for 1946 cohort.

Results: After adjusting for sex and parental social class, better cognitive function at age 10/11 was associated with reduced odds of being long-term sick (1946: 0.70 (0.56 to 0.86), $p=0.001$; 1958: 0.69 (0.61 to 0.77), $p<0.001$; 1970: 0.80 (0.66 to 0.97), $p=0.003$). Educational attainment appeared to partly mediate the associations in all cohorts; adult social class appeared to have a mediating role in the 1946 cohort.

Conclusions: Long-term sick leave is a complex outcome with many risk factors beyond health. Cognitive abilities might impact on the way individuals are able to develop strategies to maintain their employment or rapidly find new employment when faced with a range of difficulties. Education should form part of the policy response to long-term sick leave such that young people are better equipped with skills needed in a flexible labour market.

ARTICLE SUMMARY

Article focus

- To what extent does cognitive function in childhood predict long-term sick leave.
- To what extent might any association be mediated through educational attainment, adult social class or adult mental ill-health.

Key messages

- There is a clear dose–response relationship between lower cognitive function in childhood and increased odds of being on long-term sick leave in adulthood.
- This association applies to younger as well as older workers and holds true irrespective of the decade of birth.
- This association is mediated in part by education attainment suggesting improved education, especially for those with lower cognitive abilities, may help inoculate them from the risk of long-term sickness absence.

Strengths and limitations of this study

- Three large birth cohorts with data from across 50 years.
- Cohorts broadly representative of the UK population born in that year.
- Cognitive function assessed using well-recognised tools.
- Different measures of cognitive function, long-term sickness absence and depression used across the cohorts.

BACKGROUND

In the UK, over 2.5 million people are in receipt of health-related benefits (HRBs) including Incapacity Benefit and Employment and Support allowance, most often paid to those off work for more than 6 months due to ill-health.¹ The cost to the economy from reduced tax revenues and payment of benefits is in excess of £50 billion per year.² Reducing long-term sick leave is thus high on the agenda for policy makers.^{3 4} Long-term sick leave increases poverty in the

sick and is associated with premature mortality.^{5–7} At the individual level, long-term sick leave means a loss of income and dignity, and with this, a reduced opportunity for social participation.² Fifty per cent of those in receipt of an HRB have been claiming for more than 5 years, and those claiming for 2 years are more likely to die or retire than get another job.⁸ Long-term sick leave increases and sustains poverty and social disadvantage.

Mental and musculoskeletal disorders are the most common reasons to be awarded an HRB.^{2 9 10} Much of the policy response to sickness absence has focused on reducing occupational risk factors for these disorders. However, there is a disconnect between the increase in incapacity benefit (IB) certifications and the distribution of risk factors in the workplace. Musculoskeletal disorders rose at a time when the physical demands of work decreased,¹¹ and workplaces became increasingly safe.¹² Similarly, the increase in awards of HRBs due to psychiatric disorders was not associated with a concomitant rise in the prevalence of these disorders within the working age population. Further, the relationship between health and occupational function is unclear—while there are 2.5 million people in the UK on IB, over 3 million people with a range of disabilities manage to remain in paid work.¹³

Relatively, few studies have examined individual, as opposed to occupational, risk factors for long-term sick leave.¹⁴ Some difficulties apparent in childhood are associated: data from the Aberdeen Children of the Nineteen Fifties Cohort¹⁵ indicated that emotional or behavioural difficulties were associated with being permanently sick or disabled nearly 40 years later. Similar findings have been shown for adolescent mental disorders in a Swedish cohort.¹⁶ Another early risk factor might be cognitive ability: the Aberdeen study indicated that low cognitive ability independently predicted being permanently sick or disabled in adult life. Work by Gravseth *et al*¹⁷ found that low birth weight and a failure to complete secondary education predicted the award of a disability pension in a cohort of Norwegians born between 1967 and 1976. The same author has shown that lower intellectual performance at age 18 or 19 and educational attainment at age 23 were each independently associated with the award of a disability pension to Norwegian men between the ages of 24 and 36.¹⁸

Low IQ might explain the association of both childhood behavioural problems and poor educational attainment with long-term sickness absence in adult life. If this were the case, a response at policy level which emphasised the attributed health reasons for long-term sick leave and responded by trying to improve the health ‘offer’ to this group may be less than successful. By contrast, one that looked beyond a diagnostic label and emphasised skills and training, especially tailored to the needs of the least cognitively able, might produce better results.

We tested the hypothesis that lower cognitive ability is a risk factor for long-term sickness absence in three British birth cohorts. We further aimed to determine

whether such an association is mediated by educational attainment, adult social class or adult mental health.

METHODS

We used data from the three British birth cohorts whose participants are now working age.¹⁹

The National Survey of Health and Development

The 1946 National Survey of Health and Development obtained information on all singleton births to married women in England, Scotland and Wales in a single week in March 1946.²⁰ An initial sample of 5362 were then followed up, comprising all those with fathers in non-manual and agricultural employment and a 1:4 sample of those with fathers in manual employment. The cohort is described in detail elsewhere.²⁰ In 1999, 3760 of the 5362 were alive, living in the UK and were not permanent refusals. Of these, 3035 (81%) provided data to the study. This group (weighted to adjust for the sampling procedure) is broadly representative of the population born in 1946 in the UK, although there was over-representation among non-responders of the never married and the least advantaged in terms of cognitive ability, educational attainment and social class.²¹

The National Child Development Study

The 1958 National Childhood Development Study included all surviving children born in England, Scotland and Wales in a single week in March 1958. For each follow-up immigrant children who would have been part of the study had they been born in the UK were added. The cohort is described in detail elsewhere.²² In 2000, 16 147 were still eligible to take part and 11 419 (71%) contributed data.

The British Cohort Study

The 1970 British Cohort Study included all live births in 1 week in April 1970 in the whole UK. Children born in Northern Ireland were subsequently dropped from follow-up. The cohort is described in detail elsewhere.²³ In 2004, excluding those who had died, emigrated, been born in Northern Ireland or were permanent refusals, 16 875 were eligible to take part of whom 9656 (59%) contributed data.

Outcome

In all cohorts, data on the outcome, long-term sick leave, were extracted from the most recent data set at the time the present project began: for the 1946 cohort, this was in 1999 when participants were aged 53 years; for the 1958 cohort in 2000, when participants were aged 42 years and in the 1970 cohort in 2004, when participants were aged 34 years.

In both the 1958 and 1970 cohort, participants were asked if they were in receipt of any benefit payments. Individuals reporting receipt of IB or Severe Disablement Allowance were identified as being on long-term sick leave. Typically, these participants were off work for

health reasons for more than 6 months. Data on benefit receipt were not available for the 1946 birth cohort in 1999. Instead, participants were asked (yes/no) if they were in a job. Those who responded 'No' were asked (yes/no) if they were looking for work. Those who also replied 'No' to this question were asked why and asked to select a response from six options, one of which was 'Permanently sick or disabled'. Individuals reporting this option as the reason they were not looking for work were identified as being on long-term sick leave. We have previously described research using this category in the Aberdeen Children of the Nineteen Fifties Cohort.¹⁵

Exposures of interest

The 1946 and 1958 cohorts contain data on participants' cognitive ability at age 11; the 1970 cohort has a measure at age 10. The 1946 cohort members were tested at age 11 on verbal and non-verbal intelligence, arithmetic, word pronunciation and vocabulary. Scores were summed to represent overall cognitive ability. The cognitive ability of the 1958 cohort members was assessed using the General Ability Test.²⁴ The 1970 cohort members completed a modified version of the British Ability Scales.²⁵ A principal components analysis was performed on the four subscales of the British Ability Scale, and the first factor was taken as a general measure of cognitive ability.²⁶ For all cohorts, cognitive ability scores were divided into quartiles and the first (lowest cognitive ability) used as the reference group.

In all three cohorts, occupation of the father at the time of the participants' birth was coded according to the Registrar General's classification. For these analyses, social class was categorised as class I/II, class III (non-manual and manual) and class IV/V. Participants were asked about their current or most recent job, and these were similarly coded. Common mental disorders are the most common reason for sick leave.⁹ All cohorts contained a measure of depression or psychological distress. The 1946 cohort members were administered the Present state Examination²⁷ at age 36, and depression in the preceding year was derived from the CATEGO algorithm.²⁷ The 1958 cohort members were administered the Malaise Inventory²⁸ in 1991. Those scoring 8/24 or more were identified as 'cases' of depression.²⁹ The 1970 cohort members were administered the 12-item General Health Questionnaire³⁰ in 2000. Participants scoring 4 or more were identified as cases of psychological distress/depression.³¹

The highest level of educational qualification was available in the 1946, 1958 and 1970 cohorts at ages 26, 33 and 26, respectively. All three cohorts categorised this information differently, and thus, all data were re-coded into degree, A level or equivalent, O level or equivalent, Certificate of Secondary Education (CSE) grades 2–5 and no qualifications.

Risk set

At any time-point, the workless population is a heterogeneous one, comprising individuals on short-term sick

leave, individuals on long-term sick leave and some individuals who, for whatever reason, have never worked. Many of this latter group will have substantial health difficulties such as severe physical disabilities or learning disabilities. Our outcome was long-term sick leave, and therefore, in all three cohorts, analyses were restricted to those participants who described themselves as either in employment or full-time education or caring for a family in the sweep immediately prior to that from which the outcome was derived (1946 cohort—1989; 1958 cohort—1991; 1970 cohort—2000). This restriction removed 23%, 7% and 13% of the participants, respectively. Post-hoc analysis showed that these participants had very high rates of HRB receipt.

Statistical analyses

All data were analysed using STATA V.9.2.³² As with all longitudinal studies, partial data collection and loss to follow-up meant that there were incomplete data on participants in all three cohorts. To minimise the impact of missing data, multiple imputation using chained equations³³ was carried out.³² All variables were included in the imputation model.³⁴ Ten iterations were completed. The MICOMBINE function was used to calculate average regression estimates across the 10 imputed data sets.

For each cohort, the prevalence of each of the exposures of interest was calculated. The unadjusted association between childhood cognitive ability and long-term sickness absence was estimated and shown as ORs, with 95% CIs, for each of the four quartiles of childhood cognition compared with the reference and for the overall trend. These were then adjusted for the potential confounders of sex and social class at birth. Finally, to examine the potential mediating effects of covariables measured in adult life, adult social class, educational attainment and history of recent depression or psychological distress were each added to the model.

RESULTS

In 1999, 2894 members of the 1946 cohort were eligible for this study, of whom 159 (5.5%) reported themselves as permanently sick or disabled. In 2000, there were 15 053 eligible members of the 1958 cohort, of whom 431 (2.9%) were in receipt of long-term sickness benefits. In 2004, there were 14 713 eligible members of the 1970 cohort, of whom 153 (1.04%) were in receipt of long-term sickness benefits. The distribution of the covariables in each of the birth cohorts is shown in [table 1](#).

The associations between childhood cognitive ability and long-term sickness absence, adjusted for the covariables, are shown in [table 2](#). This shows that for each cohort, there is a strong impact of cognitive ability measured in childhood on the outcome several decades later. The top quartile showed between one quart and one half the odds of long-term sickness absence depending upon the cohort studied. The effect was present after adjusting for sex and paternal social class

Table 1 Distribution of covariables in three British birth cohorts

	1946 cohort		1958 cohort		1970 cohort	
	Whole cohort (n=2894)	Long-term sick leave (n=159)	Whole cohort (n=15 053)	Long-term sick leave (n=431)	Whole cohort (n=14 713)	Long-term sick leave (n=153)
Sex						
Male	1561 (54%)	72 (45%)	7483 (50%)	190 (44%)	7373 (50%)	62 (41%)
Female	1333 (46%)	89 (55%)	7570 (50%)	241 (55%)	7340 (50%)	91 (59%)
Social class at birth						
I/II	656 (23%)	18 (11%)	2611 (17%)	37 (8%)	2431 (17%)	20 (13%)
III	1505 (52%)	76 (47%)	8849 (59%)	274 (64%)	10 359 (70%)	80 (52%)
IV/V	733 (25%)	67 (42%)	3593 (24%)	120 (28%)	1923 (13%)	53 (35%)
Educational attainment						
Degree	30 (1%)	1 (0.5%)	1867 (12%)	21 (5%)	2708 (18%)	16 (10%)
A levels	270 (9%)	3 (2%)	1962 (13%)	86 (20%)	2054 (14%)	14 (9%)
O levels	740 (26%)	17 (10.5%)	5200 (35%)	143 (33%)	6178 (42%)	77 (50%)
CSEs	575 (20%)	25 (16%)	4204 (28%)	79 (18%)	2904 (20%)	34 (22%)
No qualifications	1279 (44%)	115 (71%)	1820 (12%)	103 (24%)	869 (6%)	12 (8%)
Social class at last sweep						
I/II	1284 (45%)	40 (25%)	2675 (18%)	55 (13%)	6037 (41%)	47 (31%)
III	1138 (39%)	60 (37%)	7779 (52%)	220 (51%)	6159 (42%)	64 (42%)
IV/V	472 (16%)	61 (38%)	4599 (30%)	156 (36%)	2517 (17%)	42 (27%)
Previous depression						
Yes	502 (17%)	37 (23%)	946 (6%)	73 (17%)	3387 (23%)	36 (24%)
No	2392 (83%)	124 (77%)	14 107 (94%)	358 (83%)	11 326 (77%)	117 (76%)
Cognitive ability in childhood						
Quartile 1 (least able)	657 (23%)	76 (47%)	3768 (25%)	175 (41%)	3663 (25%)	49 (32%)
Quartile 2	707 (24%)	37 (23%)	3776 (25%)	125 (29%)	3758 (26%)	47 (31%)
Quartile 3	757 (26%)	28 (17%)	3907 (26%)	77 (18%)	3722 (25%)	35 (23%)
Quartile 4 (most able)	773 (27%)	20 (13%)	3602 (24%)	54 (12%)	3570 (24%)	22 (14%)

(model 2). When potential mediating variables were added, effects were reduced. Adding social class in adulthood diminished effect sizes, particularly in the 1946 birth cohort. The overall impact of cognition on the outcome was statistically significant in two of the three cohorts. Adjusting for educational attainment also led to a reduction in effect sizes, with one cohort (the 1958) showing a significant trend of cognition on the outcome, whereas the others showed a marginally statistically significant effect, although the ORs for the lowest quartile were still of the order of two. Adjusting for prior mental disorder had little impact, with all three of the cohorts showing an independent effect of cognition on long-term sickness absence. Finally, a full model controlling for all covariates simultaneously showed a reduction in the effect of cognition on long-term sickness, with one of the three cohorts (1958) remaining significant.

DISCUSSION

Summary of findings

We examined associations between cognitive ability measured in childhood and long-term sickness absence in adult life across three British birth cohorts. In all three cohorts, the effects after adjustment for sex and social class at birth were similar, and all three demon-

strated a clear dose–response effect whereby lower childhood cognitive ability was more strongly associated with long-term sick leave. In each cohort, there was little attenuation when previous history of depression was included. There was some attenuation of the effect when adult social class and, particularly, educational attainment was included, and this attenuation was greater for those of lower cognitive ability. This suggests that some of the effect of lower cognitive ability is mediated by educational attainment. For example, low educational attainment might lead to more insecure jobs or more manual jobs that could be more difficult to sustain in the context of disability. However, educational attainment does not fully explain the association.

Strengths and weaknesses

Strengths of this study include the use of data from three British cohorts across half a century, with outcome data from both early (age 34) and late career (age 53); thus, these results seem to transcend period effects. These cohorts are broadly representative of the population born in UK in the years of their inception. Those relatively disadvantaged were more likely to be under-represented, but we have no reason to believe that this would have altered the pattern of results reported here. Exposures were measured long before the outcomes

Table 2 The association between childhood cognitive ability and long-term sickness absence in three British birth cohorts

	Model 1: unadjusted		Model 2: adjusted for sex, paternal social class		Model 2 + adult social class		Model 2 + educational attainment		Model 2 + depression		Adjusted for all covariables	
	OR (95% CI)	p Value	OR (95% CI)	p Value	OR (95% CI)	p Value	OR (95% CI)	p Value	OR (95% CI)	p Value	OR (95% CI)	p Value
1946 cohort												
Cognition	0.66 (0.53 to 0.82)	<0.001	0.70 (0.56 to 0.86)	0.001	0.78 (0.63 to 0.98)	0.04	0.80 (0.63 to 1.02)	0.07	0.69 (0.56 to 0.86)	0.001	0.84 (0.66 to 1.07)	0.15
Quartile 1 (least able)												
Quartile 2	0.77 (0.46 to 1.29)	0.32	0.84 (0.50 to 1.42)	0.52	0.98 (0.57 to 1.67)	0.93	0.95 (0.56 to 1.61)	0.84	0.84 (0.50 to 1.41)	0.51	1.02 (0.60 to 1.74)	0.94
Quartile 3	0.49 (0.27 to 0.88)	0.02	0.54 (0.30 to 0.96)	0.04	0.65 (0.36 to 1.19)	0.17	0.67 (0.36 to 1.24)	0.21	0.52 (0.29 to 0.95)	0.03	0.71 (0.38 to 1.33)	0.29
Quartile 4 (most able)	0.26 (0.11 to 0.58)	0.001	0.31 (0.13 to 0.70)	0.005	0.44 (0.18 to 1.05)	0.06	0.45 (0.18 to 1.11)	0.09	0.30 (0.13 to 0.69)	0.005	0.53 (0.21 to 1.33)	0.17
1958 cohort												
Cognition	0.68 (0.60 to 0.76)	<0.001	0.69 (0.61 to 0.77)	<0.001	0.69 (0.61 to 0.78)	<0.001	0.77 (0.67 to 0.90)	0.001	0.72 (0.64 to 0.81)	<0.001	0.79 (0.68 to 0.92)	0.002
Quartile 1 (least able)												
Quartile 2	0.67 (0.51 to 0.88)	0.004	0.68 (0.52 to 0.89)	0.005	0.68 (0.52 to 0.90)	0.006	0.78 (0.59 to 1.03)	0.09	0.72 (0.54 to 0.94)	0.02	0.81 (0.61 to 1.07)	0.13
Quartile 3	0.43 (0.31 to 0.59)	<0.001	0.44 (0.32 to 0.61)	<0.001	0.45 (0.33 to 0.62)	<0.001	0.56 (0.40 to 0.79)	0.001	0.49 (0.35 to 0.68)	<0.001	0.60 (0.42 to 0.84)	0.003
Quartile 4 (most able)	0.32 (0.22 to 0.48)	<0.001	0.35 (0.24 to 0.52)	<0.001	0.36 (0.24 to 0.54)	<0.001	0.49 (0.30 to 0.81)	0.005	0.39 (0.26 to 0.59)	<0.001	0.53 (0.32 to 0.86)	0.01
1970 cohort												
Cognition	0.78 (0.64 to 0.94)	0.01	0.80 (0.66 to 0.97)	0.03	0.84 (0.69 to 1.03)	0.1	0.85 (0.70 to 1.02)	0.08	0.80 (0.66 to 0.98)	0.03	0.87 (0.72 to 1.05)	0.15
Quartile 1 (least able)												
Quartile 2	0.94 (0.48 to 1.82)	0.85	0.97 (0.50 to 1.88)	0.93	1.03 (0.52 to 2.03)	0.93	1.0 (0.51 to 1.94)	0.99	0.97 (0.50 to 1.87)	0.92	1.03 (0.52 to 2.02)	0.93
Quartile 3	0.71 (0.44 to 1.15)	0.17	0.74 (0.46 to 1.21)	0.24	0.82 (0.50 to 1.34)	0.43	0.80 (0.50 to 1.29)	0.37	0.75 (0.46 to 1.22)	0.24	0.82 (0.50 to 1.34)	0.43
Quartile 4 (most able)	0.44 (0.21 to 0.90)	0.03	0.48 (0.24 to 0.98)	0.04	0.56 (0.26 to 1.19)	0.13	0.57 (0.28 to 1.12)	0.10	0.48 (0.24 to 0.98)	0.04	0.56 (0.26 to 1.19)	0.13

occurred, and cognitive ability was assessed using well-recognised tools. Exposures in adult life were assessed independently of the research question, limiting the impact of reporting bias.

Weaknesses include the different assessment tools used to measure childhood cognitive ability in each cohort which raises questions of comparability of results between cohorts. Paternal social class at birth was assessed by asking the participant's mother. There is likely to be a degree of misclassification here, most notable in the 1946 cohort as many fathers were just returning from the war. We believe that any such misclassification is likely to be random. Although the associations between lower cognitive ability and long-term sick leave remain after adjustment for parental social class, we are mindful of the possibility of residual confounding from unmeasured variables acting in early life which might influence both childhood cognitive ability and adult ill-health. We used three birth cohorts each of which has included a number of sweeps over at least 30 years. Non-participation in the more recent sweeps of the 1946 cohort is associated with socioeconomic disadvantage and in the 1958 and 1970 cohorts with male sex and lower educational attainment. Although the remaining participants are still broadly representative of their generations and we have used multiple imputation to minimise the impact of loss to follow-up, it is possible that our cohort data are to some degree biased. Any resulting error would, however, tend to underestimate the association between childhood cognitive function and later occupational function.

Depression or psychological distress was measured using different self-report tools in all three cohorts. Only recent difficulties were asked for, and as with all cohort studies, the data are silent as to what happens between 'sweeps'. A more robust measure of depression which identified episodes of illness between sweeps would have been preferable. We included depression in this analysis as it is the leading cause of long-term sick leave. Given the two-way relationship between physical and mental ill-health our results would have been illuminated had we included measures of physical illness in childhood and in adult life.

There is no accepted definition of long-term sick leave. Although available in the 1958 and 1970 cohorts, receipt of IB was not available in the 1946 cohort and this is a limitation. Nonetheless, we believe the population captured under the heading 'permanently sick or disabled' at age 53 to be very similar to those identified as being in receipt of IB. Most IB recipients have been away from work for over 6 months. The median time spent on IB is 5 years, and the advantages of using this outweigh any limitations. The population we have studied represents the persistent and severe long-term absentees. IB receipt is a binary question, asked relatively context free, and this will minimise any recall, reporting or observer bias, and any misclassification is likely to be

random. Furthermore, we believe that our findings have greater salience for policy makers as notwithstanding the introduction of Employment and Support Allowance they can be mapped straight onto the existing UK benefits framework.

The effect sizes we have demonstrated are noticeably similar between the cohorts despite the differences in the cohorts and the methods used to assess cognitive ability. The impact of cognitive ability on later long-term sickness absence attained conventional statistical significance in all three cohorts though most strongly for the 1958 cohort. The higher p value in the 1946 cohorts reflects the smaller size of the cohort (less than half that of the other two). In the 1970 cohort, the outcome was rare (1%), and hence, statistical power was greatest in the 1958 cohort. Our results cannot be accounted for by the people with very low cognitive ability never entering the labour force as we restricted our analyses to only those were either working or fulfilling other social roles (caring for a family or studying) at the previous sweep.

There are no data on the cognitive abilities of people claiming IB. Our study shows that the bottom two quartiles of cognitive ability are responsible for a considerable proportion of the IB recipient population. We present regression models showing the impact of controlling for a number of variables on these associations. It is worth noting that we do not consider these factors predominantly as confounders—in other words, although the association between cognitions is attenuated and becomes non-significant in two of the three cohorts when educational attainment, adult social class and depression are controlled, this does not indicate that the univariate association between cognition and long-term sickness absence is merely a result of confounding. Rather, we consider it probable that these variables are mediators of the association. Thus, the association between lower cognitive status and long-term sickness absence is in part explained by a pathway via educational attainment and adult social class. It should be noted, however, that the relationships between risk factors identified in early life, education, health and employment factors in leading to the receipt of HRBs are not clear and are likely to be complex.

There is an extensive literature on the health implications of low cognitive ability.^{35–37} However, we think it unlikely that the association we have described between low cognitive ability in childhood and adult occupational outcomes is simply because these individuals are more likely to become unwell. First, we have deliberately not attempted to ascertain the clinical labels as to why an individual is in receipt of IB—the effect of cognitive ability is substantial enough to be observed at cohort level. Second, the very limited attenuating effect of depression in all three cohorts suggests that the mechanism behind this association is largely independent of mental health, the most common reason for long-term

sick leave. This is also suggested by the consistency of results across the three cohorts as the health difficulties suffered by those in their 30s are likely to be different to those suffered by people in their 50s. Last, while previous work on the 1946 cohort,³⁸ replicating work on the 1958 and 1970 cohorts,^{26 39–41} has shown an association between cognitive ability and adult chronic physical diseases, these associations were mediated by education and to a lesser degree socioeconomic status, both of which are included in our analyses.

In trying to understand how childhood cognitive function affects adult occupational function, it is important to recognise that long-term sick leave is the result of a process rather than an event.¹⁰ These data are unable to tell us if lower cognitive ability makes it more likely that an individual with a particular disorder or set of symptoms is more likely to go off work sick or less able to get appropriate support when they are ill or find it more difficult to negotiate a successful return to work after a period of ill-health. Cognitive ability might impact on any or all these, possibly in 'soft' ways such as by directing responses to illness or by facilitating the recruitment of support from health professionals, line managers, colleagues and friends. Such a model has previously been proposed for the association between IQ and mortality.⁴² The important role of education, identified in all three cohorts, is consistent with this idea. Low cognitive ability and/or low educational attainment is likely to be associated with a limited ability to transfer skills. So, for example, if an individual with few skills goes off sick from a labouring job, the options with regard to alternative employment are few. The change in last 40 years from a manufacturing economy to a service-based economy makes such a lack of flexibility all the more problematic.

Our findings suggest that health is only one factor in understanding long-term sickness absence. We suggest that education should form part of the policy response to long-term sickness absence: for future generations, equipping children with skills necessary for labour market flexibility may inoculate them from the risk of long-term sickness absence. For the present cohorts of individuals on IBs, it is important to recognise that their cognitive abilities may be below average and that the most fruitful approach to rehabilitation may be to improve skills. More broadly, the devastating outcome of long-term worklessness for those with health problems needs to be seen as having its roots as much in a combination of individual risk factors as in the health and workplace factors which have been the basis for much of the policy response to date.

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Competing interests None.

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement Data from the 1958 and 1970 birth cohorts are publically available. Data from the 1946 birth cohort are available on request (<http://www.nshd.mrc.ac.uk/>).

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RESEARCH CHECKLIST: The association between childhood cognitive ability and long term sickness absence in 3 British birth cohorts

	Item No	Recommendation	Page no. in paper
Title and abstract	1	(a) Indicate the study's design with a commonly used term in the title or the abstract	1
		(b) Provide in the abstract an informative and balanced summary of what was done and what was found	2
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	3-4
Objectives	3	State specific objectives, including any prespecified hypotheses	4
Methods			
Study design	4	Present key elements of study design early in the paper	4
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	4-7
Participants	6	(a) Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up <i>Author note: Our study used data from 3 British birth cohorts, all of which have been extensively investigated and reported on</i>	4-5
		(b) For matched studies, give matching criteria and number of exposed and unexposed	NA
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	
Data sources/measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	5-7
Bias	9	Describe any efforts to address potential sources of bias	9-10
Study size	10	Explain how the study size was arrived at <i>Author note: we used data from 3 well established birth cohorts and have cited detailed descriptions of each</i>	
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	6-7
Statistical methods	12	(a) Describe all statistical methods, including those used to control for confounding	7
		(b) Describe any methods used to examine subgroups and interactions	n/a
		(c) Explain how missing data were addressed	7
		(d) If applicable, explain how loss to follow-up was addressed	7
		(e) Describe any sensitivity analyses	None
Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed (b) Give reasons for non-participation at each stage	8

(c) Consider use of a flow diagram

Author note: we cite “About the cohort” papers for all 3 cohorts in addition to Professor Wadsworth’s book which provides further information. We would happily include further information in the paper if required but with 3 birth cohorts we felt this was too much

Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders (b) Indicate number of participants with missing data for each variable of interest (c) Summarise follow-up time (eg, average and total amount)	13 n/a
Outcome data	15*	Report numbers of outcome events or summary measures over time	8
Main results	16	(a) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included (b) Report category boundaries when continuous variables were categorized (c) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	13 NA NA
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	None
Discussion			
Key results	18	Summarise key results with reference to study objectives	8-9
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	9-10
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	11-12
Generalisability	21	Discuss the generalisability (external validity) of the study results	11-12
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	19

*Give information separately for exposed and unexposed groups.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at <http://www.plosmedicine.org/>, Annals of Internal Medicine at <http://www.annals.org/>, and Epidemiology at <http://www.epidem.com/>). Information on the STROBE Initiative is available at <http://www.strobe-statement.org>.