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Does gender moderate the association between intellectual ability and accidental injuries? Evidence from the 1953 Stockholm Birth Cohort study

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ABSTRACT

In this paper, we test for gender differences in the effects of intellectual ability on accidental injury risks using longitudinal data from the 1953 Stockholm Birth Cohort study (n = 14,294). Intellectual ability was measured using IQ tests issued during a school survey at age ~13, and outcome and covariate data was collected via record linkage to population and health registers, following the cohort from childhood to 55 years of age. We used ICD codes to identify accidental injuries resulting in hospital admissions and deaths, and shared frailty models to quantify the effects of IQ, while allowing for within-individual dependencies and recurrent events. The models included tests for the moderating effects of gender, as well as childhood family variables (parental socioeconomic status), and cohort member mediators (highest achieved education, socioeconomic status and income at the time of the event). The results indicate an inverse association between childhood IQ and subsequent accidental injury events, where 1 SD decrease in IQ implies a 17.8% increase in injury risk. We also found evidence that gender moderates this relationship, where the effect size was twice as large for men than for women (21.8% vs 9.3% per 1 SD decrease). Adult socioeconomic status can explain roughly half of the observed association. Potential explanations for these results are discussed.

1. Introduction

Accidental injuries are a global public health problem, resulting in large societal and individual losses in terms of morbidity, disability and premature mortality each year (Murray et al., 2012). There are many individual, social and environmental risk factors for injury, which can affect injury risks in general (e.g. age, socioeconomic status and alcohol consumption (Carpenter and Dobkin, 2009; Laflamme and Diderichsen, 2000)), or increase the risk for specific external causes of injury (e.g. poor road quality, building standards, or occupational safety regulations). In this paper, we examine the relationship between cognitive ability and overall accidental injury risks in a Swedish birth cohort. We pay extra attention to the potential moderating effects of gender, and the impact of intermediary socioeconomic and educational mechanisms, on this relationship.

There is a plethora of evidence that cognitive ability is inversely associated with all-cause mortality (Batty et al., 2007a; Calvin et al., 2011) and other adverse health outcomes (see e.g. Der et al., 2009; Lundin et al., 2015). However, few studies have examined the association between childhood cognitive ability and subsequent accidental injuries, especially with respect to gender differences. In fact, most have used male only samples, which directly inhibit such comparisons. For instance, a study by Whitley et al. (2010) found evidence of an increased risk of dying in traffic accidents, fires and poisonings with lower scores on an intelligence test issued to Swedish male conscripts. Similarly, Osler et al. (2007) found an inverse relationship between intelligence and accidental injuries, specifically falls and poisoning, among Danish men. In a follow-up to the Swedish conscript study that linked the draft data to offspring data, Jelenkovicet al. (2014) found an association between lower paternal intelligence scores and increased risk of offspring mortality and injuries by external causes.

1.1. Gender differences in the effect of intelligence

The current literature on the effects of intelligence on all-cause mortality generally disfavors the gender-intelligence interaction hypothesis (Calvin et al., 2011). However, roughly 90 percent of the all-cause health burden is comprised of diseases rather than injuries (Haagsma et al., 2016), which could mask any differences specific to injury-related mortality and morbidity.

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We argue that the etiology of injuries clearly distinguishes itself from that of diseases by being a result of an acute exposure to energy caused by some adverse event, which in itself is a consequence of an instantaneous and harmful interaction between an individual at risk and their surrounding physical and social environment (Haddon 1980). While the determinants of ill-health often overlap with those of accidents, injuries are more directly connected to activities that may put an individual at risk. They are also highly susceptible to human error in the absence of protective barriers that account for such errors (Reason, 2000). Thus, any variable that causes a systematic variation in the selection into, or the preference for, different activities with varying degrees of danger will likely affect the risk of injury. In addition, factors that (directly or indirectly) cause individual differences in the probability of human error will also play an important role. We argue that gender and intelligence independently serve as two of these variables, and that their interaction may modify the strength of their effects.

1.1.1. Gender differences

Independently, gender affects several factors that may cause individual differences in the exposure to injury risks. For instance, men are less likely to wear seatbelts, and more likely to drive under the influence of alcohol and to speed (Harré et al., 1996), which implies that they are less likely to comply with safety regulations. This is also consistent with research on gender differences in conformity and risk-taking (Byrnes et al., 1999; Charness and Gneezy, 2012; Cooper, 1979). Closely related to risky driving in the sense of being a correlate thereof (Oltedal and Rundmo, 2006), men also tend to score higher on tests of sensation-seeking personality traits (Cross et al., 2013), which implies that they are more likely to perform more risky behaviors and activities in general (Horvath and Zuckerman, 1993). Factors such as gender norms (Badgett and Folbre, 1999), differential preferences and attitudes towards risk and competition (Croson and Gneezy, 2009), and labor market discrimination (Azmat and Petrongolo, 2014) are also assumed to have contributed to a large occupational segregation of men and women (Kreimer, 2004), where most occupations with higher-than-average rates of work-related deaths are typically male-dominated (DeLeire and Levy, 2004). The explanations are likely multifaceted and far more complex than the snapshot of evidence provided here, but no matter what the exact causal factors and intermediate mechanisms of the relationship between gender and accidental injuries are, we can clearly observe a strong correlation between gender and the risk of all-cause injury. In fact, the relative risk is higher among men in almost all external cause categories (Haagsma et al., 2016).

1.1.2. Potential mechanisms behind the intelligence-injury association

While recurrent cognitive failures have been found to increase the risk of accidents, such failures have seldom been directly linked to measures of intelligence (Clarke, 2016). However, other traits that are directly related to intelligence, for instance speed of information-processing (Sheppard and Vernon, 2008), might have a negative effect on injury risks by affecting an individual's ability to successfully counteract the injury process before or during an accident. There is also some evidence that motor function is inversely related to intellectual ability, at least in pediatric samples (Smits-Engelsman and Hill, 2012). It is also likely that other environmental, social and behavioral factors can explain the observed relationship between intelligence and injury risks. Holding gender constant, intellectual ability affects a large set of variables that correlate with injury risks, e.g. personality, interests (Ackerman and Heggestad, 1997), labor market outcomes (Lindqvist and Vestman, 2011), educational achievement (Deary et al., 2007) and other socioeconomic variables (Strenze, 2007). These variables are likely to create inequalities in the exposure to hazardous environments, behaviors and safety information (Laflamme and Diderichsen, 2000; Pampel et al., 2010), and thereby affect injury risks as an indirect causal effect of intellectual ability. Thus, there is most likely a social gradient to the relationship between intelligence test scores and injury mortality and morbidity (and all-cause mortality), which is supported by empirical studies that find that socioeconomic status attenuates the association by approximately half (Batty et al., 2009).

1.1.3. An interactive model

If we consider these two components together, we have a male population that is more likely to perform more dangerous activities and less likely to use safety equipment and conform to safety regulations. Assuming that the effects of intellectual ability plays a more important role during these activities by either increasing the probability of an adverse event or the severity of its consequences, it is possible that men with lower cognitive ability will be more affected by injuries than women of equal cognitive ability. In fact, even if the gender differences are non-existent under similar conditions, we assume that, in a general population sample, men will be affected more greatly due to a larger exposure to hazardous environments. In this paper, we aim to test this interaction hypothesis using longitudinal data from a Swedish birth cohort that includes childhood intelligence test scores for both men and women, linked to future injury outcomes.

2. Methods and materials

2.1. Data

We used data from a Swedish birth cohort of 14,294 individuals born in Stockholm 1953, living in Stockholm in 1963 and still alive and residing in Sweden in 1980 and/or 1990 (which is the current definition of the cohort after the most recent follow-up data linkage). Mainly, we utilized data from intelligence tests collected during a school survey in 1966, when the cohort members were approximately 13 years of age. Due to changes in Swedish research ethics regulations during the course of the original cohort study, all individual records were anonymized in 1986, which prohibits direct identification of cohort members in administrative registers. Despite this caveat, 95% of the original cohort has been successfully supplemented with follow-up data by Stenberg et al. (2007), who used probability matching on 13 different variables to link the cohort to administrative registers (including the cohort member's year and month of birth, their parents' years of birth, and other variables describing occupational and housing conditions). This allows us to compare childhood intelligence data to follow-up injury data from the Swedish National Inpatient and Cause of the Death registers. For more information on the cohort itself, see Stenberg et al. (2007). The current study was approved by the Regional Ethics Committee in Uppsala (dnr 2016/125).

2.2. Variables

2.2.1. Outcome

We extracted information on external cause of hospital admissions and deaths from the Swedish hospital discharge and cause of death registers. Accidental injuries were classified according to the International Classification of Diseases (ICD)-8, ICD-9 and ICD-10 codes depending on the year of the event and the classification system used at that time (ICD-8: E800-E929 and E940-E949; ICD-9: E800-E869 and E880-E949; ICD-10: V00-X59 and Y85-Y86). Intentional injuries, injuries with undetermined intent and misadventures during hospital care were excluded (falls and traffic accidents, the two largest categories, account for roughly half of the recorded accidental injuries). Besides external cause codes, the observed injury data also contained information on year of death or hospital discharge. If multiple events were recorded within the same year, with the exact same injury diagnosis and external cause code, they were treated as double registrations and merged into one record (a double registration may e.g. occur if a patient is transferred from one hospital to another). We studied both hospital admissions and deaths as a single outcome variable, although it should be noted that non-fatal injuries heavily outweigh the number of fatalities and are thus likely to drive the majority of the results presented below.

The outcome data is linked to the cohort for the period 1969–2008 which means we follow the cohort from ages 16–55 years. Before analyzing the data, we structured the dataset into recurrent time-to-event data using information on the year of the injury event. Our timescale was defined by the time (in years) from the first year of observable injury data (1969) to the year of death, year of hospital discharge or study exit (in 2008). A practical issue with only observing the year, and not the actual date, of the event is that one individual can have multiple recorded events within the same year, which is problematic from an analytical standpoint (since the time from event 1–2 could be 0). When this occurred, ties were solved by adding a miniscule random uniform number to the time-to-event variable (the results remained invariant to repeating this process several times).

2.2.2. Independent variables

The Härnqvist intelligence test was used to measure intelligence at ~13 years of age in the 1966 school survey (Härnqvist, 1960). The test consisted of three subtests that measured verbal, spatial and numerical ability, which sum to a composite overall test score. The average between-subtest correlation coefficient was $r = 0.95$, and the average subtest-to-composite score correlation was $r = 0.96$, which implies that they measure the same latent intelligence construct. We used the composite score to derive a measure of childhood intelligence quotient (IQ) by standardizing the raw test scores to a variable with a mean 100 and standard deviation (SD) 15. To facilitate a more intuitive interpretation of the coefficients associated with this variable, we analyzed its inverse, which gives the change in risk with each one unit decrease in IQ.

Childhood family variables and cohort member mediators were also included in the model, as described more in detail below. Summary statistics for all covariates are available in Table 1. To measure gender differences, we include an interaction term between individual's biological sex and IQ.

2.2.2.1. Childhood family variables. We included a categorical measure of the father's social class in 1963 (Upper and upper middle class; lower middle class (officials and non-agricultural workers); lower middle class (entrepreneurs); skilled working class; unskilled working class). In addition to this, we also used both the father's and mother's assessed incomes in 1963 (and their interaction effect) as an adjustment for childhood economic status.

2.2.2.2. Cohort member mediators. The cohort member's own income was derived from Swedish National Tax Board records (in 1971, 1974, 1977, 1978, 1979, 1980, 1981 and 1982) and from the Longitudinal integration database for health insurance and labor market studies (annual data from 1990 to 2008). To code a time-varying income variable that is insensitive to changes in data collection methods and inflation, we categorized an individual's relative income quintiles within every decade (1969–1979, 1980–1989, 1990–1999, 2000–2008), using period averages.

We also defined a categorical cohort member socioeconomic status indicator based on occupational census data from 1990 (roughly midway through the study period), with the following categories: white-collar (leading, senior or intermediate positions), white-collar (lower grades), blue-collar (skilled), blue-collar (unskilled), others (self-employed, freelance professionals, farmers and other unclassifiable employees), and unemployed (or otherwise missing).

Data on the cohort member's highest completed education was obtained from the Longitudinal integration database for health insurance and labor market studies. The data is coded according to Swedish education nomenclature (SUN), which is a 7 step ordinal scale from "Pre secondary upper school education, less than 9 years" to "Researcher education". While time-varying data is available according to current definitions from 2000 to 2008, we used the last available year to code a fixed education variable due to low variations within this period (the correlation between the first and last year is $r = 0.98$).

Table 1
Summary of the covariates included in the individual-level dataset.

Variable	Mean	Std. Dev.	Min	Max	Valid obs.
Fathers income [at age 10]	24.63	22.4	0	733	14291
Mothers income [at age 10]	4.48	6.86	0	115	14291
Father's social class [at age 10]	2.93	1.46	1	6	14291
Cohort member's social class [at age 37]	2.88	1.81	1	6	14291
Highest completed education	4.1	1.5	1	9	13976
Income quintile [at event]	3	1.4	1	5	14008
IQ [at ~age 13]	100	15	53.11	139.8	12770
Male	0.51	0.5	0	1	14291
Fatality	0.05	0.21	0	1	14291
Accidental injury event	0.19	0.39	0	1	14291

2.3. Statistical analysis

We analyzed the data using gamma shared frailty models (assuming a Weibull distribution) to account for the repeated-events nature of the dataset. The use of this technique allows for more modelling flexibility than standard Cox regression models, allowing for both repeated events and time-varying covariates (Amorim and Cai, 2015; Ullah et al., 2014). In essence, shared frailty models mimic the classical Anderson-Gill method for recurrent events survival analysis, but relax the common baseline hazards and independent events assumptions by the addition of random-effect terms. The latter property may be especially relevant to our case as the risk of recurrent events may be dependent on the occurrence of previous injuries (e.g., a person may change their behavior after sustaining an injury). Further, it allows for the inclusion of time-varying covariates, such as income level at the time of the event, and provides a more efficient use of the data as compared to 0nly analyzing time to the first event (Amorim and Cai, 2015). Despite this, it should be noted that the results are robust and largely invariant to the removal of the shared frailty parameter, and to the use of standard Cox proportional hazards models. P-values below 0.05 were considered statistically significant, and the analysis was performed using Stata 13.

3. Results

During the observed time period (1969–2008), the cohort members in the analysis sample incurred a total of 3623 injury events that led to hospital admission, 56 of which were fatal (mean age of injury: 36.1 years, SD: 11.1). Accounting for exposure time, this number corresponds to 7.35 accidental injury events per 1000 person-years. We present this data by IQ score (in deciles) and gender in Fig. 1, where we can see that IQ is inversely associated with the risk of accidental injury, but that the response is more strongly pronounced for men than for women.

The unadjusted estimates from the survival analysis suggest that a 1 SD decrease in childhood IQ increases the risk of accidental injury by 17.8 percent (Table 2, Column I), and adjusting for childhood socioeconomic status and gender only has a small effect on the estimates (Table 2, Column II). As expected, the estimate is more affected by the inclusion of the adult socioeconomic mediators. Still, it remains significant at 6.8 percent (per 1 SD decrease), suggesting only partial mediation (Table 2, Column III).

Moving on to the moderation models, we found that men have more than double the effect size (21.8% per 1 SD decrease) compared to women (9.3% per 1 SD decrease) after adjustment for childhood family variables (Table 2, Column IV). The gender-specific mediation model suggests that socioeconomic status can explain the relationship fully for women, but not men, for whom we still observe a marginally

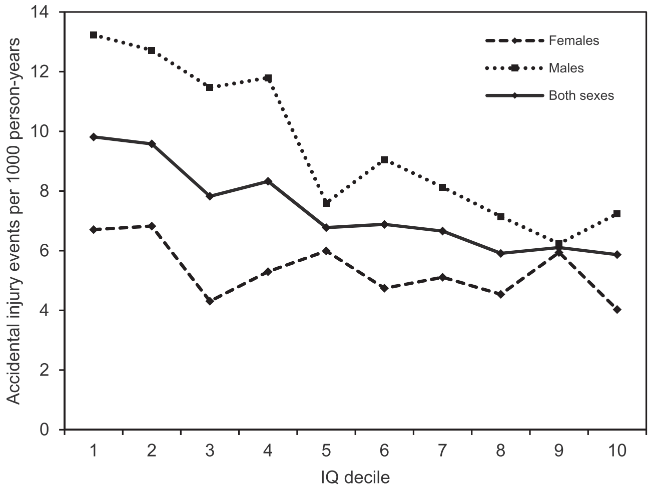

Fig. 1. Accidental injury risk per IQ decile for men and women in the Stockholm Birth Cohort study.

insignificant tendency towards risk increase (Table 2, Column V). Due to the closeness to the significance cutoff for men, we advise caution in interpreting the loss of significance in this model as evidence of full mediation, especially since increase is still significant in the full sample after inclusion of the mediating variables (Table 2, Column III).

Using models IV and V in Table 2, we calculated the predicted, gender-specific injury rates per 1000 person-years across the observed span of the IQ variable, keeping the continuous variables constant at their mean values and categorical variables constant at their most common category. We plot these results in Fig. 2, where we can clearly see that the predicted values after adjustment for childhood socioeconomic variables (Panel A) mimics the raw data presented in Fig. 1 (since they have little impact on the estimates in Table 2). We can also see the impact of adjusting for the adult socioeconomic mediators in Panel B, where the injury risk is constant across the span for women, but not for men, where an inverse effect is still visible.

4. Discussion

The results support previous evidence regarding a link between premorbid cognitive ability and accidental injury events, which is consistent with previous research on the effects of intelligence on adverse health outcomes in general. There are a set of potential intermediary mechanisms that could serve as explanations for the average increase in risk observed in individuals with lower intelligence test scores. For instance, low cognitive ability has been associated with higher alcohol consumption, which in it itself increases the risk of mortality and hospital admissions due to external causes (Carpenter and Dobkin, 2009; Sjölund et al., 2015). Cognitive ability is also associated with a range of mental health conditions, such as depression (Der et al., 2009; Hatch et al., 2007), that may also modify injury risks (Shi et al., 2015). It may also modify an individual's ability to process information regarding health and injury risks (Gottfredson and Deary, 2004). Further, intelligence also predicts educational attainment, socioeconomic success and income levels (Strenze, 2007), which are correlated with injury risks (Cubbin et al., 2000; Laflamme and Diderichsen, 2000). In our analyses, we found evidence of a partial mediation of socioeconomic status on the relationship between childhood intelligence test scores and future injury risks. This is also supported by previous studies on the relationship with all-cause mortality (Hemmingsson et al., 2006). Still, further research is required to fully understand this aspect as we do not observe the many other possible mechanisms, e.g. leisure time activities and other risk-modifying individual pReferences

Table 2
The association between childhood IQ and subsequent accidental injury events, and the moderating effects of gender on this relationship, according to shared frailty models with different covariate specifications.

	Overall models			Moderation models	
	I IQ Only	II Family variables	III Mediators	IV Family variables	V Mediators
	A. Hazard ratios (95% confidence intervals)				
Inverse IQ	1.012***	1.011***	1.005**	1.006**	1.001
	(1.009, 1.015)	(1.008, 1.014)	(1.002, 1.008)	(1.002, 1.011)	(0.996, 1.006)
Male		1.748***	1.567***	3.924***	2.750***
		(1.589, 1.901)	(1.426, 1.724)	(2.175, 7.080)	(1.522, 4.968)
Male * Inverse IQ				1.008**	1.006
				(1.002, 1.015)	(0.999, 1.013)
	B. Transformed coefficients (1 SD decrease, relative risk)				
Inverse IQ	17.8	16.7	6.8		
Inverse IQ (Women)				9.3	1.8
Inverse IQ (Men)				21.8	10.4
	C. Model details and diagnostics				
Childhood family variables					
Father's income		Yes	Yes	Yes	Yes
Mother's income		Yes	Yes	Yes	Yes
Income interaction		Yes	Yes	Yes	Yes
Father's social class		Yes	Yes	Yes	Yes
Cohort member variables					
Highest completed education			Yes		Yes
Income quintile			Yes		Yes
Social class			Yes		Yes
n subjects	12770	12770	12419	12270	12419
n injury events	3623	3623	3526	3623	3526
Person-years at risk	492615	492615	481114	492615	481114
Log-likelihood	−10689	−10603	−10240	−10600	−10239
ρ (Weibull ancillary parameter)	1.05**	1.05**	1.07***	1.05**	1.07***
θ (frailty parameter)	2.97***	2.73***	2.41***	2.72***	2.40***

Notes: ***$p < 0.001$,**$p < 0.01$,*$p < 0.05$.The transformed coefficients for men were derived using $(\beta*\gamma-1)*15*100$, where$\beta$is the hazard ratio for inverse IQ (for women, since they are coded as 0) and γ is the interaction effect.

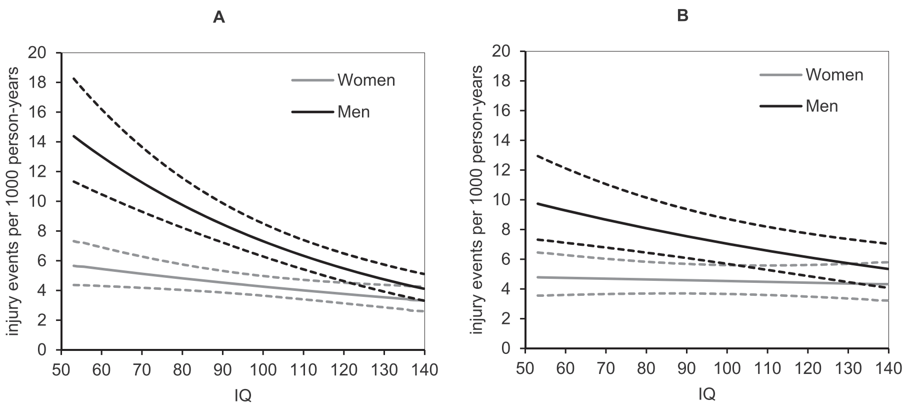

Fig. 2. Predicted gender-specific accidental injury rate per 1000 person-years across the IQ span after adjustment for childhood socioeconomic status (Panel A) and after inclusion of socioeconomic mediators in adulthood (Panel B). Dashed lines are 95% confidence intervals.

Moving beyond individual-level risk factors, we also assume that structural social inequalities may play an important role. Identifying and understanding these mechanisms more clearly may aid in the search for possible preventative measures. For instance, the range of job options available for persons with low cognitive ability and low educational attainment may expose such individuals to more dangerous physical work environments (Deary et al., 2007). Further, the negative effects of cognitive ability on economic prospects may also, e.g., inhibit the ability of this group to maintain a safe home environment, or select the most secure transport options (Laflamme and Diderichsen, 2000). Unfortunately, we cannot answer these questions in the current study, but we believe that determining these aspects may be the most fruitful avenue for future research, especially given that interventions that target risk factors at higher structural levels seem to be more likely to have an impact on population-level injury rates (Lund and Aarø, 2004; Reason, 2000).

Turning to our results regarding the moderating effects of gender on the observed relationship, it should be noted that they differ slightly from the limited literature on the topic (Batty et al., 2007b; Lawlor et al., 2007). Some of the differences could potentially be explained by the use of different methodologies and samples. Nonetheless, Lawlor et al. (2007) report not having found any statistically significant moderating effect of gender despite using similar data and methods to our study. Still, most previous studies on the subject, especially those from a Nordic setting, have been limited to male-only conscription samples. This aspect directly inhibits the study of gender differences, which makes re-testing this hypothesis more important. Although we advise some caution in extrapolating this result to other settings without further replication, the interaction effect holds in the studied cohort, even after adjustment for observable covariates. Given that we are willing to assume that cognitive ability truly has a stronger impact on injury risks among men, our results could serve as another piece of the puzzle in understanding how and why lower scores on intelligence tests predict future adverse injury events. To the best of our knowledge, the current state of the literature does not fully explain why these gender differences may occur. As noted above, we speculate that a gender-intelligence risk-taking interaction effect might exist, or that social norms, inequalities and preferences cause men with lower cognitive ability to be selected into more hazardous environments. Still, further research is likely required to understand and evaluate the likelihood of these claims.

4.1. Strengths and limitations

The main strength of this study lies in the prospective cohort design, which ensures that the temporality condition for causality is met. Nonetheless, as with any observational study that does not explicitly exploit natural experiments or other exogenous sources of variation as grounds for causal interpretation, we cannot rule out the possibility that the measured IQ scores are simply some proxy for unobserved confounders.

An additional strength lies in the high data quality inherent in Swedish health registers, especially those that we derive our outcome data from (Ludvigsson et al., 2011). However, as noted above, the outcome data is linked to cohort members via probability matching. While Stenberg et al. (2007) were able to match 95 percent of the original birth cohort (n = 15,177) to follow-up data using 13 variables that should provide a secure match, matching errors cannot be ruled out completely. However, if there are any such errors, we have no reason to suspect that they would be systematically correlated with the variables under study here, and should therefore not pose any strong threat to the validity of the presented results.

Another issue is that we do not observe the full range of intermediary variables necessary to fully explain the observed relationship; more detailed data on e.g. leisure time activities and job type may have enabled us to formally test some of the hypotheses presented above. However, these questions will have to be answered by future studies. Furthermore, while we observe a large sample of individuals, hospitalization due to accidental injury is fairly uncommon, especially if broken down into cause-specific categories. Attempts to do this for the most common categories (falls and traffic accidents) were made, but the results were similar (albeit less precise). While Batty et al. (2009) found no striking differences in the effects of cognitive ability on different injury event types in a study of Swedish men, we were unable to sufficiently answer if the interaction effect varies by event type. These limitations prevent us from answering a range of interesting questions, such as whether gender modifies the effect of IQ on driving or work-related injuries, which future studies with larger samples should consider examining.

A potential weakness pertaining to the external validity of the results is that we only observe data from a metropolitan sample, and may not be representative for rural populations with e.g. different labor force compositions. Further, the data covers an historical period under which some of the societal conditions may not be representative of today's society. While our results regarding the effects of IQ in general are supported by evidence collected elsewhere (and for other health conditions), replication in future cohorts may be warranted.

5. Conclusion

Childhood cognitive ability is inversely associated with subsequent accidental injury events in the 1953 Stockholm Birth Cohort. Gender may partly moderate this effect, as we find evidence of a significantly stronger association for men than for women. Since we were only able to partly explain this link as a function of the intermediary effects of socioeconomic status, further studies are required to fully understand the causal pathways underlying this relationship.

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