Associations between IQ and cigarette smoking among Swedish male twins☆

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A B S T R A C T

It has been suggested that certain health behaviours, such as smoking, may operate as mediators of the well-established inverse association between IQ and mortality risk. Previous research may be afflicted by unadjusted confounding by socioeconomic or psychosocial factors. Twin designs offer a unique possibility to take genetic and shared environmental factors into account. The aim of the present national twin study was to determine the interrelations between IQ at age 18, childhood and attained social factors and smoking status in young adulthood and mid-life. We studied the association between IQ at age 18 and smoking in later life in a population of 11 589 male Swedish twins. IQ was measured at military conscription, and data on smoking and zygosity was obtained from the Swedish Twin Register. Information on social factors was extracted from censuses. Data on smoking was self-reported by the twins at the age of 22–47 years. Logistic regression models estimated with generalised estimating equations were used to explore possible associations between IQ and smoking among the twins as individuals as well as between-and within twin-pairs.

A strong inverse association between IQ and smoking status emerged in unmatched analyses over the entire range of IQ distribution. In within-pair and between-pair analyses it transpired that shared environmental factors explained most of the inverse IQ–smoking relationship. In addition, these analyses indicated that non-shared and genetic factors contributed only slightly (and non-significantly) to the IQ–smoking association. Analysis of twin pairs discordant for IQ and smoking status displayed no evidence that non-shared factors contribute substantially to the association. The question of which shared environmental factors might explain the IQ–smoking association is an intriguing one for future research.

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Introduction

Several studies have been conducted exploring the association between cognitive ability, IQ in childhood or early adulthood and mortality in later life (Batty, Deary, & Gottfredson, 2007; Hart et al., 2005; Hemmingsson, Melin, Allebeck, & Lundberg, 2006; Modig-Wennerstad et al., 2008; Silventoinen, Modig-Wennerstad, Tyne-lius, & Rasmussen, 2007). Different psychometric tests have been used and different populations have been studied and most studies have found inverse associations, suggesting that a higher IQ decreases the risk of morbidity and mortality. However, the studies exploring the potential mechanisms behind this association are few in number. Different mechanisms have been proposed to underlie the IQ–mortality association. One explanation is a bodily system integrity effect, meaning that an optimally working brain is associated with an optimal somatic system in other organs, such as the cardiovascular system. The association could also be related to perception and understanding of symptoms and self-management of disease. A third possible explanation is that behavioural risk factors might mediate the association, and that individuals with higher IQ score may display more favourable health behaviours, such as being smokers to a lesser degree or quitting smoking earlier.

The association between IQ and smoking has been explored in some studies and all of them have found inverse associations,
meaning that a lower IQ score increases the risk of smoking (Batty, Deary, Schoon, & Gale, 2007; Batty et al., 2008; Hemmingsson, Kröbel, Melin, Allebeck, & Lundberg, 2008; Kubicka, Matejecck, Drytrch, & Roth, 2001; Taylor et al., 2003, 2005). In some of the studies the associations were attenuated or disappeared after adjustment for indicators of social class (Batty, Deary, Schoon, et al., 2007; Batty et al., 2008).

Two studies have explored the association of IQ with mortality and controlled for smoking. Kuh, Richards, Hardy, Butterworth, and Wadsworth (2004) found no evidence of smoking being a mediator of the IQ–mortality association (Kuh et al., 2004) whereas Batty et al. (2008) found that adjustment for smoking marginally attenuated the IQ–mortality association (Batty et al., 2008).

The conclusion from previous studies is that there seems to be an inverse association between IQ and smoking, but whether this can be explained by socioeconomic or other factors, and if so, to which extent, remains unclear. With better understanding of the underlying mechanisms or pathways linking IQ to smoking habits, guided by results from well-designed studies which take into account confounding and mediating mechanisms, it seems likely that the emerging body of knowledge will have the necessary qualities to become useful in the development of health promotion plans and/or health education programmes.

It has been hypothesized that any association between IQ and health behaviour is related to difficulties in understanding health-prevention messages and acting in accordance with these, either directly or through an inability to draw conclusions about how different behaviours may affect one’s own health in the long run. An alternative hypothesis is that the same set of underlying factors, either genetic or socioeconomic, affects both IQ and the risk of becoming a smoker. We have investigated the association between IQ and smoking status in two large cohorts of Swedish male twins. Besides analysing the association among the twins as individuals, we have used the co-twin control design and also investigated between- and within-pair differences. It is conceivable that previously reported associations between IQ and smoking may be partially explained by residual confounding from socioeconomic or psychosocial factors in childhood, or genetic factors. Using twin pairs enabled us to partially or completely control for genetic factors and socioeconomic and psychosocial circumstances in childhood.

Methods

Material

Target cohort and record-linkage of registers

Our target population consisted of all Swedish male twins born 1951–1984 who were identified in the Multi-Generation Register (MGR) (Statistics Sweden, 2005) and in the Military Service Conscription Register (MSCR); altogether 29,524 male twins. The MSCR provides information on all Swedish men eligible for conscription examination. During the years covered by this study, military conscription (1969–1994) was compulsory for all Swedish men except only those with severe disabilities. Information about zygosity and smoking habits was obtained from the Swedish Twin Register (STR). Determination of zygosity by a few classical questions has been shown to have high validity in studies using DNA analysis of multiple markers as the golden standard (Lichtenstein et al., 2006; Silventoinen, Magnusson, Tyrellius, Kaprio, & Rasmussen, 2008).

Measures of IQ

IQ was measured at military conscription at a mean age of 18.3 years (SD 0.55 years). During the study period two different IQ tests have been in use to obtain a global IQ score. The first test was in use 1969–1994 and has been described in detail elsewhere (Carlstedt, 2000; Carlstedt & Mardberg, 1993). It consists of four sub-tests: a logical test (capacity to understand written instructions and apply them to the task of solving a problem), a verbal test (knowledge of synonyms, ability to determine which out of four alternatives is the synonym of a given word), a spatial test (identifying the correct three-dimensional object from a series of two-dimensional drawings), and a technical test (mathematical/physics problems, measuring a component of general knowledge). All tests were presented in succession to the subjects in the form of written questionnaires. The second test, which was used after 1994, is a computer-based test and is described in Mardberg & Carlstedt, 1998. The two tests were similar and the global IQ score for both tests was standardised against the entire population to follow a Gaussian distribution between 1 and 9, with a mean of 5 and an SD of 2. A higher value indicates greater cognitive ability. Our study population had a mean global IQ of 5.2 and an SD of 1.9.

Measures of smoking

Information about smoking status and smoking history was collected through the STR and was based on two large twin surveys called SALT and STAGE, which have been described elsewhere (Lichtenstein et al., 2002, 2006), including questions about representativeness (Furberg, Lichtenstein, Pedersen, Bulik, & Sullivan, 2006; Furberg et al., 2008). All data was self-reported, either through a telephone interview or through a web questionnaire. SALT ended in 2002 and the individuals in that cohort were between the ages of 44 and 51 when they responded to the questionnaire. STAGE ended in 2006 and the individuals in that cohort were between the ages of 22 and 47 when they responded to the questionnaire. Smoking was reported in terms of having smoked regularly, currently or in the past, having smoked occasionally (now and then or at parties, currently or in the past), never smoked or only experimented. Participants also reported the number of cigarettes smoked, age when they started smoking, etc.

Measures of socioeconomic position

Information about socioeconomic position in childhood was based on parental occupation. It was assessed through Statistics Sweden’s socioeconomic index and was extracted from the Population and Housing Censuses when the participants were 5–10 years of age. Parental occupation was classified as: (6) higher level non-manual, (5) middle level non-manual, (4) lower level non-manual, (3) skilled workers, (2) unskilled workers, and (1) others including farmers, students, homemakers, and those with disability pensions. Own attained level of education was extracted from the Longitudinal Database of Education, Income and Occupation (from 1990 to 2004). Level of education was classified into four categories: (4) PhD education, (3) higher education 13–15 years, (2) full secondary education 11–12 years, and (1) up to 10 years of compulsory school.

Outcome variables and confounding factors

Smoking

Smoking was analysed both as dichotomous variable, where those who had smoked regularly were coded as 1 and those who had never smoked regularly were coded as 0, and as polynomeous variable for those who had never been smokers (never smoked or only tried), occasional/party smokers, past regular smokers and current regular smokers. We also examined the number of cigarettes smoked per day and mean age of onset over the whole range of stanine IQ scores (from 1 to 9).
Socioeconomic variables

We used the highest of the two parents’ occupational socioeconomic position to adjust for childhood socioeconomic status and own attained education as a measure of adult socioeconomic position. Both measures were used as categorical variables in the regression models.

Final study population

From the target population of 29 524 men, 2975 were excluded since they did not have information on IQ. In the analyses of smoking habits, another 14 961 men were excluded because they did not participate in any of the twin surveys on smoking habits (SALT and STAGE). The analyses were therefore conducted on a study population consisting of 40% of the target population (exact numbers are given in the tables).

Statistical analyses

Firstly we analysed the association between IQ and smoking status among all twins, treated as individuals but with adjustment for correlations within-pairs, using logistic regression models estimated with generalised estimating equations (GEE). The analyses were performed with the Genmod procedure in SAS. We also performed polytomous logistic regression, where we used a categorised outcome variable with four classes. This analysis was performed with the mllogit command in STATA (version 9.0) in order to get robust standard errors due to within-pair correlations. This regression model allowed us to compare different smoking categories over the IQ span.

Secondly, we analysed only the complete twin pairs and used logistic regression models to estimate effects on smoking between- and within-pairs per unit increase in IQ. This was analysed with the Genmod procedure, also using GEE. In this model, the between-pair effect was parameterised as the mean IQ of the brother pairs and the within-pair effect as the twins’ differences from the pair mean. The within-pair effect is then by design matched for all common environmental and genetic factors (for MZ pairs, 100% and for DZ pairs on average 50% of the segregating genes) i.e. any within-pair effect represents an association that is free from confounding due to factors which are shared by the two twins in a pair. Since the between-pair effect lacks this matching any association found may be confounded by common factors (Carlin, Gurrin, Sterne, Morley, & Hopper, 2006). We also used conditional logistic regression in which twin brothers from pairs discordant for smoking were compared with respect to IQ. The odds ratios for being the twin who smoked compared to his non-smoking co-twin were estimated for MZ and DZ pairs. If any association is observed for MZ pairs of twins, it should be attributable to non-shared environmental factors as the model controls for common environmental and genetic factors (in MZ pairs).

In both analyses we only adjusted for own education since childhood socioeconomic factors are shared by the brothers and therefore controlled for by design. All analyses were also adjusted for birth year as a continuous variable.

The Ethics Committee in Stockholm, Sweden, has approved this study (2007/1566-32).

Results

The overall response rates for the SALT and STAGE questionnaires were 74% and 53% respectively. Comparison of descriptive statistics of the two cohorts for variables such as IQ distribution, smoking prevalence and socioeconomic variables with national statistics, (Swedish Surveys of Living Conditions) revealed that participants in SALT were representative for the general population in the same age span. In the STAGE cohort, the participants had higher IQ, were from higher socioeconomic groups and were smokers to a lesser degree than the general population in the same age span (approximately 30% in national statistical data were ever regular smokers against only 9% in our data). This is in line with other comparisons of participants and non-participants in STAGE (Furberg et al., 2008) Participants in STAGE were also younger than the participants in SALT when responding to the questionnaires. The mean age of the SALT participants was 47.5 years (SD 2.3) and that of the STAGE participants was 34.9 years (SD 7.4). Due to these differences we have stratified the analyses according to cohort. Most analyses are therefore presented separately for the SALT cohort, born 1951–1958, and the STAGE cohort, born 1959–1984.

Table 1 presents descriptive information about IQ and smoking for the two cohorts. Even though the proportion of smokers was much higher in SALT than in STAGE, the pattern between IQ and

<table>
<thead>
<tr>
<th>IQ score</th>
<th>Ever regular smokers</th>
<th>Mean number of cigarettes per day (SD)</th>
<th>Mean age at start of smoking (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SALT</td>
<td>STAGE</td>
<td>SALT</td>
</tr>
<tr>
<td>1</td>
<td>69.5%</td>
<td>24.6%</td>
<td>15.3 (9.1)</td>
</tr>
<tr>
<td>2</td>
<td>66.8%</td>
<td>16.7%</td>
<td>14.3 (8.2)</td>
</tr>
<tr>
<td>3</td>
<td>59.7%</td>
<td>13.3%</td>
<td>15.0 (7.0)</td>
</tr>
<tr>
<td>4</td>
<td>55.0%</td>
<td>13.0%</td>
<td>14.7 (7.6)</td>
</tr>
<tr>
<td>5</td>
<td>53.3%</td>
<td>8.0%</td>
<td>14.8 (7.7)</td>
</tr>
<tr>
<td>6</td>
<td>47.0%</td>
<td>6.5%</td>
<td>15.0 (8.6)</td>
</tr>
<tr>
<td>7</td>
<td>44.1%</td>
<td>5.1%</td>
<td>13.9 (7.7)</td>
</tr>
<tr>
<td>8</td>
<td>38.0%</td>
<td>3.5%</td>
<td>15.4 (10.12)</td>
</tr>
<tr>
<td>9</td>
<td>28.1%</td>
<td>1.8%</td>
<td>15.4 (9.57)</td>
</tr>
<tr>
<td>Tot</td>
<td>52.0%</td>
<td></td>
<td>14.7 (8.1) N – 2172</td>
</tr>
<tr>
<td>Corr*</td>
<td>–0.17</td>
<td>–0.14</td>
<td>–0.01**</td>
</tr>
</tbody>
</table>

*p – 0.596, all other p < 0.001.

* Spearman correlation.
smoking was similar in both cohorts, with the smoking prevalence decreasing as IQ increases. There was also a trend towards a higher mean age of onset of smoking with higher IQ. The only trend that seemed to be different between the two cohorts was the mean number of cigarettes among smokers. In SALT there was no clear trend over the IQ span, whereas in STAGE there was a downward trend of number of cigarettes per day as IQ increased. However, the question about the number of cigarettes was phrased differently in the two cohorts. In SALT the question referred to how many cigarettes the participant usually smoked per day, whereas in STAGE the question referred to the period when the participant smoked the most.

Table 2 presents mean IQ over four smoking status categories. In both cohorts, mean IQ was lower among current/regular smokers than among those who had never smoked. Even though the mean IQ in general was higher in the STAGE cohort, the mean IQ of regular smokers was similar in the two cohorts. The only estimate which differed was the mean IQ of occasional smokers. Within the group of regular smokers, current regular smokers had lower mean IQ than past regular smokers.

Table 3 shows crude and adjusted odds ratios (OR) for regular smoking, ever and current versus non regular smoking. The twins were analysed as individuals with control for the correlation within brother pairs. In both cohorts there was a strong inverse association between IQ and regular smoking. In the crude analysis, the association was stronger for STAGE, which might reflect the previously mentioned selection bias in that cohort. After adjustment for socioeconomic variables in childhood and adulthood, the difference was attenuated and the two cohorts showed very similar results. Since the associations between IQ and smoking have been shown to differ in the two cohorts after adjustments for socioeconomic factors, and taking into consideration the fact that the discordant pairs were quite few, we chose to analyse the two cohorts together in order to increase statistical power.

Table 6 presents the between- and within-pair effects among DZ and MZ twins separately. The between-pair effect shows the association between IQ and smoking (ever having smoked regularly) between-pair means of IQ. The within-pair effect is based on the differences from the mean within twin pairs. Both the between- and the within-pair effects were similar in DZ and MZ twins. The between-pair effect was strong and significant (DZ OR = 0.84 95% CI 0.78–0.91 and MZ OR = 0.83 95% CI 0.77–0.90). The within-pair effect, taking common environmental factors and genetics into account, however, was weak and not statistically significant (DZ OR = 0.95 95% CI 0.84–1.07 and MZ OR = 0.97 95% CI 0.80–1.17). This suggests that factors in the shared environment underlie the association, and the results showed no clear evidence of a contribution from genetic or non-shared factors. We performed the same analysis for current regular smokers and the results were similar (data not shown).

We also performed conditional logistic regression for the complete twin pairs and they showed no statistically significant association, as the ORs adjusted for own education were 0.89 (0.77, 1.04) among DZ pairs, and 0.92 (0.72, 1.19) among MZ pairs, which gives no statistical evidence that non-shared factors were involved.

**Discussion**

The overall purpose was to study any association between IQ in early adulthood and smoking status in later life. The twin data allowed us to control for shared environmental factors such as socioeconomic position in childhood and genetics. National register data also allowed us to take own socioeconomic position in

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**Table 2**

<table>
<thead>
<tr>
<th>Smoking status</th>
<th>Mean IQ (SD) All</th>
<th>Mean IQ (SD) SALT</th>
<th>Mean IQ (SD) STAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never</td>
<td>5.4 (1.9) N = 6939</td>
<td>5.4 (1.9) N = 1311</td>
<td>5.4 (1.9) N = 5628</td>
</tr>
<tr>
<td>Occasional</td>
<td>5.2 (1.9) N = 1676</td>
<td>5.0 (2.0) N = 633</td>
<td>5.4 (1.8) N = 1043</td>
</tr>
<tr>
<td>Regular past</td>
<td>4.8 (1.9) N = 1861</td>
<td>4.8 (1.9) N = 1344</td>
<td>4.8 (1.9) N = 517</td>
</tr>
<tr>
<td>Regular current</td>
<td>4.3 (1.9) N = 1113</td>
<td>4.3 (2.0) N = 759</td>
<td>4.3 (1.8) N = 354</td>
</tr>
</tbody>
</table>

---

**Table 3**

<table>
<thead>
<tr>
<th></th>
<th>Crude*</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SALT N = 4025</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regular smoker</td>
<td>0.84 (0.81, 0.87)</td>
<td>0.84 (0.81, 0.87)</td>
<td>0.89 (0.86, 0.92)</td>
<td>0.89 (0.85, 0.92)</td>
</tr>
<tr>
<td>Regular smoker</td>
<td>0.81 (0.78, 0.85)</td>
<td>0.81 (0.78, 0.85)</td>
<td>0.86 (0.82, 0.91)</td>
<td>0.86 (0.82, 0.91)</td>
</tr>
<tr>
<td><strong>STAGE N = 7534</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regular smoker</td>
<td>0.79 (0.77, 0.83)</td>
<td>0.81 (0.77, 0.84)</td>
<td>0.88 (0.84, 0.92)</td>
<td>0.88 (0.84, 0.92)</td>
</tr>
<tr>
<td>Regular smoker</td>
<td>0.74 (0.70, 0.78)</td>
<td>0.77 (0.72, 0.81)</td>
<td>0.85 (0.79, 0.90)</td>
<td>0.86 (0.81, 0.92)</td>
</tr>
</tbody>
</table>

Model 1 adjusted for birth year and paternal occupation.
Model 2 adjusted for birth year, and own education.
Model 3 adjusted for birth year, parental occupation, and own education.* adjusted for birth year.
adulthood into account. We found a strong inverse association between IQ and regular smoking (currently and in the past), both in the SALT cohort and in the younger and less representative STAGE cohort. These results are in line with other studies (Batty, Deary, & Gottfredson, 2007; Batt, Deary, Schoon, et al., 2007; Hemmingsson et al., 2008). The association between IQ and occasional smoking was not as clear as that between IQ and regular smoking. This association was present in the SALT, but not the STAGE cohort. The two cohorts differed with regards to socioeconomic position, IQ, and smoking status. Smoking prevalence was much lower in STAGE, both due to a cohort effect and to the fact that highly educated individuals were over-represented in STAGE. Even so, the probability of being a regular smoker given a low IQ was similar in the two cohorts after adjustments for socioeconomic factors. In the polytomous regression, where the effect was shown over five categories of IQ score, the same type of linear trend was present in both cohorts. The presence of similar results in two different cohorts makes the results less likely to be the product of chance or selection bias.

Besides a high correlation for IQ within the twin pairs, the twin pairs were very similar in their smoking status, which is why the number of pairs discordant for IQ and smoking was low. This, coupled with the fact that the vast majority of smokers start smoking in adolescence, indicates that the effects of shared environmental factors and/or genetics might be strong. This was confirmed in the analyses of complete twin pairs, where the relatively strong association between IQ and smoking found between-pairs vanished when the analyses were adjusted, by design, for shared environment and genetics within-pairs. Some studies have shown a considerable genetic contribution to smoking initiation (Hamilton et al., 2006; Li, Cheng, Ma, & Swan, 2003) and other studies have shown that parental and siblings’ smoking status are important predictors (Kempainen et al., 2008). Peer influence has also been shown to be associated with smoking initiation (Kempainen et al., 2008). However, to which extent the IQ–smoking association can be explained by these factors is not known.

We are not aware of any study which has been able to control for shared environmental factors when investigating the IQ–smoking association. Previous studies have tried to control for socioeconomic factors in childhood, and some have shown attenuated estimates after such adjustments. Others have not had suitable variables. In our analyses of the twins as individuals, adjustment for parental occupation did not have any significant effect on the IQ–smoking association. In the within-pair analyses, however, the IQ–smoking association vanished both among MZ and DZ pairs after controlling for shared factors. This suggests that parental occupation does not capture the factors in the shared environment which affect smoking. Our results can therefore be interpreted in several ways. Any association between IQ and smoking may be confounded by childhood socioeconomic position, which previously used variables did not capture, or other factors in the shared environment may explain the association. Those factors might for example be parental smoking status, attitudes within families and psychosocial factors shared within families. Consequently, our results do not support a causal relationship between IQ and smoking. These results are in line with another previously published paper which suggests that shared environmental factors are important in the IQ–smoking association (Johnson, Hicks, McGue, & Iacono, 2009).

The mean age at which onset of smoking occurred was 15 and 16 years respectively in the two cohorts. The fact that the analyses were adjusted for own education, attained later in life, might be questioned, together with the fact that IQ and education are highly correlated. However, the participants were asked about their smoking status at a mean age of 35 and 47 years, and especially the odds of being a current smoker can be considered dependent on one’s own socioeconomic group, since several studies have shown that smoking cessation is associated with socioeconomic status (Broms, Silventoinen, Labhelma, Koskenluu, & Kaprio, 2004). Furthermore, achieved education is likely to be dependent on the participant’s school performance in adolescence. It can therefore be seen as a proxy of previous school performance, which might be considered an influencing factor on the risk of becoming a smoker, as it has been shown to influence the risk of alcohol abuse (Osler, Nordentoft, & Andersen, 2006). Based on this we chose to adjust the estimates for highest attained education, but results adjusted for childhood socioeconomic position and adult socioeconomic position were presented separately as well as combined.

Table 4
Odds ratios from polytomous logistic regression with 95% confidence intervals for smoking status over five groups of IQ in the SALT cohort, never smoker is the outcome reference.

<table>
<thead>
<tr>
<th></th>
<th>IQ 1–2</th>
<th>IQ 3–4</th>
<th>IQ 5</th>
<th>IQ 6–7</th>
<th>IQ 8–9</th>
</tr>
</thead>
<tbody>
<tr>
<td>SALT&lt;sup&gt;a&lt;/sup&gt; N = 4047</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Occasional</td>
<td>0.76 (0.53,1.09)</td>
<td>0.54 (0.36,0.79)</td>
<td>0.50 (0.35,0.71)</td>
<td>0.45 (0.30,0.69)</td>
<td></td>
</tr>
<tr>
<td>Regular past</td>
<td>0.69 (0.51,0.94)</td>
<td>0.58 (0.42,0.80)</td>
<td>0.41 (0.30,0.56)</td>
<td>0.29 (0.20,0.42)</td>
<td></td>
</tr>
<tr>
<td>Regular current</td>
<td>0.43 (0.31,0.59)</td>
<td>0.29 (0.20,0.41)</td>
<td>0.21 (0.15,0.29)</td>
<td>0.10 (0.07,0.16)</td>
<td></td>
</tr>
<tr>
<td>Occasional</td>
<td>0.77 (0.54,1.01)</td>
<td>0.25 (0.37,0.82)</td>
<td>0.51 (0.35,0.75)</td>
<td>0.48 (0.31,0.73)</td>
<td></td>
</tr>
<tr>
<td>Regular past</td>
<td>0.74 (0.55,1.00)</td>
<td>0.66 (0.48,0.91)</td>
<td>0.52 (0.38,0.71)</td>
<td>0.41 (0.28,0.59)</td>
<td></td>
</tr>
<tr>
<td>Regular current</td>
<td>0.48 (0.35,0.65)</td>
<td>0.36 (0.25,0.51)</td>
<td>0.31 (0.22,0.43)</td>
<td>0.20 (0.13,0.32)</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> Adjusted for birth year and correlation within brother pairs.

Table 5
Odds ratios from polytomous logistic regression with 95% confidence intervals for smoking status over five groups of IQ in the STAGE cohort, never smoker is the outcome reference.

<table>
<thead>
<tr>
<th></th>
<th>IQ 1–2</th>
<th>IQ 3–4</th>
<th>IQ 5</th>
<th>IQ 6–7</th>
<th>IQ 8–9</th>
</tr>
</thead>
<tbody>
<tr>
<td>STAGE&lt;sup&gt;b&lt;/sup&gt; N = 7541</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Occasional</td>
<td>0.90 (0.69,1.17)</td>
<td>0.93 (0.72,1.21)</td>
<td>0.88 (0.69,1.14)</td>
<td>0.84 (0.63,1.12)</td>
<td></td>
</tr>
<tr>
<td>Regular past</td>
<td>0.63 (0.44,0.90)</td>
<td>0.38 (0.26,0.56)</td>
<td>0.28 (0.19,0.41)</td>
<td>0.19 (0.11,0.33)</td>
<td></td>
</tr>
<tr>
<td>Regular current</td>
<td>0.64 (0.46,0.90)</td>
<td>0.37 (0.26,0.53)</td>
<td>0.26 (0.18,0.37)</td>
<td>0.08 (0.04,0.16)</td>
<td></td>
</tr>
<tr>
<td>Occasional</td>
<td>0.92 (0.71,1.20)</td>
<td>0.98 (0.75,1.28)</td>
<td>0.96 (0.74,1.26)</td>
<td>0.94 (0.69,1.29)</td>
<td></td>
</tr>
<tr>
<td>Regular past</td>
<td>0.70 (0.49,1.00)</td>
<td>0.48 (0.32,0.72)</td>
<td>0.42 (0.28,0.64)</td>
<td>0.35 (0.19,0.66)</td>
<td></td>
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<tr>
<td>Regular current</td>
<td>0.81 (0.58,1.14)</td>
<td>0.59 (0.41,0.85)</td>
<td>0.53 (0.36,0.77)</td>
<td>0.22 (0.11,0.46)</td>
<td></td>
</tr>
</tbody>
</table>

<sup>b</sup> Adjusted for birth year, correlation within brother pairs and own education.
The idea that health behaviours such as smoking might be a plausible mediator of the IQ–mortality association inspired us to conduct this study. The strong association between IQ and smoking status found in our analyses of single subjects and the fact that smoking is a strong risk factor for cardiovascular disease speaks in favour of smoking being a mediator of the IQ–mortality association. However, our results from the twin pair analyses suggest that smoking appears to be a confounder rather than a mediating factor of the IQ–mortality association. The fact that the IQ–smoking association disappeared after adjustments for shared environment and genetics suggests that the IQ–smoking association found in the analyses of single subjects was confounded by unmeasured social and psychosocial factors or by genetic factors, and that smoking appears to be associated with, but not the result of, IQ. Since the association within-pairs disappeared in both MZ and DZ twins, it is likely that factors in the shared environment, rather than genetics, confound the IQ–smoking association. Therefore, our results suggest that the hypothesis that individuals with a low IQ score do not understand health messages and start to smoke due to their low IQ is not supported, but rather that IQ correlates with shared environmental factors which in turn affect smoking status.

When separating the regular smokers into past regular smokers and current regular smokers, we found a somewhat stronger association of IQ with current smokers, suggesting that among smokers, IQ is positively related to quitting smoking. In order to see whether IQ is related to smoking to a greater or lesser extent (among smokers), a combined measure of number of years and number of cigarettes smoked would have been needed. In our data we divided the participants into never smokers, past smokers and current smokers which give some idea of the degree of smoking. We also analyse the number of cigarettes smoked per day. However, none of these outcomes are good measures of cumulative smoking.

Our study does not support a causal relation between IQ and smoking. Further research is needed in order to understand and identify the underlying environmental factors which are shared by siblings and which may account for the IQ–smoking association.

**Strengths and limitations**

This study had several strengths, such as a large twin sample, detailed information on smoking and register-based data on socioeconomic variables. However, the main strength of this study was its ability, through the twin analyses, to control for shared environmental factors and genetics when investigating the inverse IQ–smoking association. This added valuable information to previous findings on the association between IQ and smoking. A limitation of our study was the low response rate in the STAGE cohort, around 50%, and also the fact that the respondents in STAGE were from higher socioeconomic groups than the non-responders. This resulted in a higher mean IQ and a lower smoking prevalence than what was seen in SALT and in the general population.

However, after adjustments for socioeconomic factors, the results in the STAGE cohort were very similar to the results from the more representative SALT cohort. This, together with the strength of the association, makes it unlikely that the associations found could be explained by selection bias or chance. Another limitation of our study was the low power in the analyses of twin pairs. This might have led us to overlook a possible association in the within-pair analyses, however we believe that any such effect would have been relatively small. Furthermore, information on covariates such as parental smoking status and other familial factors known to be predictors of smoking initiation would have been valuable, together with a good measure of cumulative smoking. Information on externalising behaviours among the twins, which could act as confounders of the IQ–smoking association, would also have been useful.

**Conclusion**

A strong inverse association was found between IQ and regular smoking within the cohort of twins. The analyses within twin pairs indicated that shared socioeconomic and psychosocial factors partially explain the inverse IQ–smoking relationship. Non-shared and genetic factors indicated weak and non-significant contributions to the association. The method used in this study allowed us to separate shared environmental factors and genetics from individual factors, but it did not allow us to estimate the size of the shared environmental effect and the size of the genetic effect. The question of which shared environmental factors might explain the IQ–smoking association is an intriguing one for future research.

**References**


**Table 6**

<table>
<thead>
<tr>
<th>Type and number of pairs</th>
<th>Between-pair odds ratios with 95% confidence interval from GEE logistic regression forever regular smoking.</th>
<th>Within-pair difference</th>
<th>Odds ratios with 95% confidence interval from GEE logistic regression forever regular smoking.</th>
</tr>
</thead>
<tbody>
<tr>
<td>In all twins N = 2241</td>
<td>0.78 (0.75, 0.82) 0.93 (0.84, 1.02)</td>
<td></td>
<td>0.78 (0.75, 0.82) 0.93 (0.84, 1.02)</td>
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<tr>
<td>DZ N = 1053</td>
<td>0.84 (0.79, 0.88) 0.96 (0.87, 1.06)</td>
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<td>0.84 (0.79, 0.88) 0.96 (0.87, 1.06)</td>
</tr>
<tr>
<td>MZ N = 1188</td>
<td>0.78 (0.73, 0.83) 0.95 (0.78, 1.14)</td>
<td></td>
<td>0.78 (0.73, 0.83) 0.95 (0.78, 1.14)</td>
</tr>
</tbody>
</table>

*Adjusted for birth year.*

*Additional adjusted for own education.*


