Childhood cognitive performance and risk of generalized anxiety disorder

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Background Perception of control over one’s environment, particularly when faced with an ambiguous situation, has been identified as a critical cognitive process involved in worry and generalized anxiety disorder (GAD). Similarly, it is thought that individuals with lower cognitive skills feel less in control, and do not cope as well as individuals with higher cognitive skills. This study tests the hypothesis that individuals with higher cognitive skills are less likely to develop a lifetime diagnosis of GAD, and considers onset in three developmental periods: childhood, adolescence and adulthood.

Methods Survival analysis and multivariate regression models were used to evaluate the relationship between cognitive performance at age seven, and DSM-IV diagnosis of GAD. Study participants were 689 individuals in their mid-30s, who had been followed since birth as part of the National Collaborative Perinatal Project in Providence, RI, USA.

Results A 15-point (1 SD) advantage in childhood cognitive performance was significantly associated with a 50% reduced risk of lifetime GAD and an 89 and 57% reduction in risk of GAD in childhood and adolescence, respectively, after adjusting for relevant covariates including socio-economic status and parent history of mental health problems. These results were not affected by behavioural inhibition or learning disabilities in childhood.

Conclusions Childhood cognitive performance is associated with a diagnosis of GAD in childhood and adolescence. Further research on the association between childhood cognitive performance and GAD is warranted.

Keywords Anxiety disorders, intelligence, children, adolescents, longitudinal studies, mental health

Introduction Generalized anxiety disorder (GAD) is one of a constellation of anxiety disorders that affects a substantial proportion of the general population, leading to increased medical costs, days of work lost and disability.1,2 Epidemiological research to date has improved our understanding of the treatment and long-term prognosis for GAD, but less attention has been focused on the aetiology of GAD and risk factors associated with its onset.

Perception of control over one’s environment, particularly when faced with an ambiguous situation,3,4 has been identified as a critical cognitive process involved in worry and GAD. Research has also shown that individuals with GAD have significantly less tolerance for uncertainty than comparison subjects,3,5,6 underestimate their ability to cope with difficult or ambiguous circumstances and overestimate the likelihood of negative consequences resulting from those circumstances.7 Supporting these findings are studies showing that individuals who have successfully navigated ambiguous or unpredictable situations are less likely to develop GAD compared with those
who have not had similar successes.\textsuperscript{6–10} It is thought that early experiences of uncontrollability\textsuperscript{9} and unpredictable negative events\textsuperscript{11} may contribute to the development of GAD particularly when those negative events occur in the presence of a biological or genetic vulnerability to the disorder.\textsuperscript{9,12}

Similarly, it has been hypothesized that individuals with a lower level of cognitive performance feel less in control, and do not cope as well as individuals with a high level of cognitive performance, particularly when faced with negative events.\textsuperscript{13–16} This hypothesis is supported, in part, by research on resilience in childhood and adolescence. Individuals with a higher level of cognitive skills are consistently found to be more resilient to adversity compared with individuals with fewer cognitive resources\textsuperscript{17–21} and skills such as memory, problem solving and communication may contribute to an individual’s sense of control and ability to successfully cope with unpredictable or negative events. Taken together, these findings suggest that individuals with higher cognitive skills may be less likely to develop GAD.

Consistent with the above theory, research has found that individuals with a lower level of cognitive performance are more likely to develop other psychiatric disorders\textsuperscript{22} including anti-social behaviour,\textsuperscript{18} dementia,\textsuperscript{23} schizophrenia\textsuperscript{24,25} and post-traumatic stress disorder.\textsuperscript{17–21}

Using longitudinal data from a community sample, the present study was conducted to evaluate whether higher cognitive performance, measured by IQ at age seven, is associated with the development of GAD across the life course. This study tests the hypothesis that individuals with higher cognitive skills are less likely to develop a lifetime diagnosis of GAD, and also considers onset in three developmental periods: childhood, adolescence and adulthood.

**Methods**

**Study sample**

Subjects were offspring of mothers enrolled in the Providence, RI site of the National Collaborative Perinatal Project (NCPP).\textsuperscript{29} The NCPP was a multi-site cohort study that involved the prospective observation and examination of over 50,000 pregnancies through the first seven years of life in 12 cities across the US. In the Providence site, 4140 pregnancies were enrolled between 1959 and 1966.

In 1996, when subjects were entering middle adulthood, 1062 individuals in the Providence cohort with a Full Scale IQ (FSIQ) ≥ 80 at age seven were selected for a follow-up study, for the purposes of evaluating long-term outcomes of individuals with and without learning disabilities (LDs) as children.\textsuperscript{30,31} The study sample was comprised of all individuals in the Providence cohort whose academic tests indicated that they were in the bottom 10% in reading, spelling or arithmetic from age seven. Of 1062 subjects selected for follow-up, 47 had died or were otherwise ineligible. Overall, 720 (70.9%) of the surviving 1015 subjects selected for follow-up were successfully located and interviewed.\textsuperscript{31,32} Thirty-one individuals were missing information on key covariates, including socio-economic status (SES) in childhood and/or family history of mental illness, resulting in a final analytic sample of 689. There were no appreciable differences on any variables of interest between those excluded due to missing data ($n = 31$) and those included in the analyses ($n = 689$).

**GAD**

Diagnoses of GAD and age of onset were obtained using the National Institute of Mental Health Diagnostic Interview Schedule (DIS) for the DSM-IV. The DIS was administered in adulthood by specially trained study personnel. Age of onset was determined by the subject’s report of the age at which he or she first had a period of six months or longer of feeling worried and anxious most of the time. Retrospective reports of age of onset are commonly used in large-scale psychiatric studies such as the National Comorbidity Study,\textsuperscript{33} and a comparison of time-related information to well-documented clinical records found good validity for subject retrospective reports of age of onset.\textsuperscript{34} An individual was considered to have an onset in childhood if they met diagnostic criteria for GAD and reported having their first episode when they were age 12 or younger, an onset in adolescence if their first episode occurred between ages 13 and 18 and an onset in adulthood if their first episode occurred after age 18.

**Cognitive performance**

Children enrolled in the NCPP were evaluated throughout the first 7 years of life on a wide range of neurological, cognitive, developmental and behavioural measures. Cognitive performance at age seven was assessed using the Wechsler Intelligence Scale for Children (WISC),\textsuperscript{35} which provided an estimate of each individual’s FSIQ. The mean FSIQ in this sample was 98.8 with a SD of 12.4. In the general population, the mean FSIQ is 100 with an SD of 15.

**Covariates**

SES at age seven was measured by a composite index adapted from the United States Bureau of the Census that averaged percentiles derived from the education and occupation of the head of the household, as well as family income.\textsuperscript{36} SES at age seven was a continuous measure ranging from 0.3 to 9.3, with 9.3 indicating the highest SES.

A history of parental psychopathology was obtained from parent interviews at study enrolment, and at 7 years of age. An individual was considered to have a family history of mental illness if it was reported that either their mother or father had ever been hospitalized or received out-patient care for a mental illness, or had problems with alcoholism or a drug addiction.

**Learning disability**

Individuals were classified as having a LD at age seven if their achievement on standardized tests of reading, spelling or arithmetic was much lower than would be expected given their overall level of general intelligence. Within each of four strata of cognitive performance (FSIQ 80–89, 90–99, 100–109, 110+), individuals were identified as having an LD if their performance on the reading, spelling or arithmetic subtest of the Wide Range Achievement Test (WRAT)\textsuperscript{37} was in the bottom 6.7% (1.5 SD).\textsuperscript{31} LD was defined in this manner because this
method shows considerable overlap with alternative approaches to defining LD, and has strong diagnostic validity.

Analysis

We used Cox proportional hazards models and multiple logistic regression models to evaluate the relationship between cognitive performance at age seven and GAD using the SAS system. For ease of interpretation, coefficients of childhood cognitive performance in the logistic regression models were multiplied by 15 and then exponentiated to obtain odds ratios (OR) that correspond to a 15-point (1 SD in the general population) advantage in childhood cognitive performance. All multivariate models were controlled for sex, race (white/non-white), SES at age seven, parental history of mental health treatment and LD at age seven.

We present the results for the study population overall, and in addition, present this relationship by developmental period. Thus, for each developmental period, the analytic sample excluded individuals whose age of onset occurred prior to the developmental period of interest; for childhood onset, the full sample was used; for adolescence, we excluded individuals who had an onset of GAD in childhood; and in adulthood, excluded individuals who had an onset of GAD in childhood or adolescence.

Finally, we performed a series of sensitivity analyses to examine whether the results of our study were influenced by (i) the large proportion of individuals with LDs in the study sample; (ii) GAD onset prior to age seven; (iii) behavioural inhibition in childhood; and (iv) exposure to stressful life events prior to age seven.

Results

Prevalence of GAD

At the time of follow-up, subjects were between the ages of 30 and 39, with a mean age of 33.6 (SD = 1.8). Overall, 39% of the analytic sample was female, 23% non-white, 42% had a LD in childhood and about 5% had a family history of treatment for mental disorder. Seventy-five individuals (10.9%) met DSM-IV criteria for GAD with an average age of onset of 18.7 years. Eighteen individuals (32%) had their onset in childhood, 16 (21.3%) had an onset in adolescence and 41 (54.7%) had an onset in adulthood. Females were significantly more likely to meet criteria for GAD. Childhood cognitive performance was also significantly associated with GAD, with 17.8% of individuals in the lowest cognitive performance group (FSIQ 80–89) meeting diagnostic criteria for GAD, followed by 9.8% of individuals with a FSIQ between 90 and 99, 8.5% of individuals with a FSIQ between 100 and 109, and 6.1% of individuals with a FSIQ over 110 (Table 1). Overall, the mean FSIQ for individuals with GAD (94.6) was about five points lower than that of individuals without GAD (99.3).

Table 1 Characteristics of study participants by diagnosis of GAD

<table>
<thead>
<tr>
<th></th>
<th>GAD diagnosis</th>
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<tbody>
<tr>
<td></td>
<td>n (%) (n = 689)</td>
<td>Yes n (%) (n = 75)</td>
<td>No n (%) (n = 614)</td>
<td>Test of No association P-value (χ²)</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>421 (61.1)</td>
<td>35 (46.7)</td>
<td>386 (62.9)</td>
<td>0.006 (7.38)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>268 (38.9)</td>
<td>40 (53.3)</td>
<td>228 (37.1)</td>
<td></td>
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</tr>
<tr>
<td>Race</td>
<td></td>
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<tr>
<td>White</td>
<td>528 (76.6)</td>
<td>63 (84.0)</td>
<td>465 (75.7)</td>
<td>0.11 (2.55)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>161 (23.4)</td>
<td>12 (16.0)</td>
<td>149 (24.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Socio-economic Status</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Low (0.3–3.0)</td>
<td>215 (31.2)</td>
<td>22 (29.3)</td>
<td>193 (31.4)</td>
<td>0.90 (0.19)</td>
<td></td>
</tr>
<tr>
<td>Middle (3.1–4.7)</td>
<td>252 (36.6)</td>
<td>29 (38.7)</td>
<td>223 (36.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High (4.8–9.3)</td>
<td>222 (32.2)</td>
<td>32 (42.0)</td>
<td>198 (32.2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Childhood LD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>291 (42.2)</td>
<td>40 (53.3)</td>
<td>251 (40.9)</td>
<td>0.03 (4.24)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>398 (57.8)</td>
<td>35 (46.8)</td>
<td>363 (59.1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Childhood IQ</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>80–89</td>
<td>174 (25.3)</td>
<td>31 (41.3)</td>
<td>143 (23.3)</td>
<td>0.006 (12.6)</td>
<td></td>
</tr>
<tr>
<td>90–99</td>
<td>224 (32.5)</td>
<td>22 (29.3)</td>
<td>202 (32.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>100–109</td>
<td>176 (25.5)</td>
<td>15 (20.0)</td>
<td>161 (26.2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥110</td>
<td>115 (16.7)</td>
<td>7 (9.3)</td>
<td>108 (17.6)</td>
<td></td>
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<tr>
<td>Parental psychopathologya</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Yes</td>
<td>36 (5.2)</td>
<td>4 (5.3)</td>
<td>32 (5.2)</td>
<td>0.96 (0.002)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>653 (94.8)</td>
<td>71 (94.7)</td>
<td>582 (94.8)</td>
<td></td>
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</tr>
</tbody>
</table>

a Parental psychopathology was based on parental report of any hospitalization or out-patient care for a mental illness, or reports of problems with alcoholism or a drug addiction at the time of the subject's birth or when the subject was age 7.
Relation between childhood cognitive performance and GAD

A plot of survival curves for the four categories of childhood cognitive performance suggests that, after adjustment for relevant background factors, cognitive performance was negatively associated with the development of lifetime GAD (Figure 1). Individuals in the lowest group (FSIQ 80–89) were at increased risk for GAD over the 30-year study period compared with those with a FSIQ of \( \geq 110 \), [Hazard ratio (HR) = 4.40; 95% confidence interval (CI) 1.77–10.97]. Individuals with a FSIQ between 90 and 99 and between 100 and 109 were also at higher risk with HRs of 2.31 (\( P = 0.07 \)) and 1.66 (\( P = 0.29 \)), respectively.

Results from the categorical analysis suggest a dose–response relationship between childhood IQ and lifetime risk of GAD. We examined this relationship in greater detail treating childhood cognitive performance as a continuous variable. Based on multiple logistic regression, results in Table 2 indicate that a 1 SD increase in childhood cognitive performance was associated with a 50% decreased risk of lifetime GAD (OR = 0.50; 95% CI 0.35–0.72) after adjustment for covariates. Table 2 also presents a clear gradient in the association between childhood cognitive performance and GAD across developmental periods. The strongest association was for childhood onset, where, after adjustment for covariates, a 1 SD increase in childhood cognitive performance was associated with an 89% reduced risk of GAD (OR = 0.11; 95% CI 0.04–0.33). In adolescence, childhood cognitive performance continues to protect against GAD (OR = 0.43; 95% CI 0.20–0.95), while in adulthood, the association between childhood cognitive performance and GAD is weaker (OR = 0.81; 95% CI 0.52–1.27).

Sensitivity analyses

We performed additional analyses to assess the extent to which (i) the prevalence of LD in our sample, (ii) retrospective reporting of age of onset, (iii) high behavioural inhibition in childhood and (iv) exposure to stressful life events in childhood, may have influenced the association between cognitive performance and GAD.

Learning disability

Individuals with a childhood LD were almost twice as likely to meet lifetime criteria for GAD compared with those without a LD (Table 2). For individuals with a LD, a 15-point advantage in childhood cognitive performance was associated with a 58% reduced risk of lifetime GAD (OR = 0.42; 95% CI 0.26–0.70), compared with a 36% reduced risk of GAD in individuals without a LD (OR = 0.63; 95% CI 0.36–1.12). When examined by developmental period, the association between cognitive performance and GAD onset in childhood/adolescence was similar for individuals with (OR = 0.20; 95% CI 0.09–0.47) and without a LD (OR = 0.27; 95% CI 0.10–0.74). For adult onset

![Figure 1](http://ije.oxfordjournals.org/)

**Figure 1** Time to onset of GAD by IQ group. Controlling for gender, socio-economic status at age seven, learning disability at age seven, and parental psychopathology.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Lifetime GAD diagnosis OR (95% CI) n = 689</th>
<th>Child onset OR (95% CI) n = 689</th>
<th>Adolescent onset OR (95% CI) n = 671</th>
<th>Adult onset OR (95% CI) n = 655</th>
</tr>
</thead>
<tbody>
<tr>
<td>FSIQ(^c)</td>
<td>0.50 (0.35–0.72)(^{**})</td>
<td>0.11 (0.04–0.33)(^{***})</td>
<td>0.43 (0.20–0.95)(^{\dagger})</td>
<td>0.81 (0.52–1.27)</td>
</tr>
<tr>
<td>Female</td>
<td>1.95 (1.19–3.20)(^*)</td>
<td>2.54 (0.92–6.98)</td>
<td>4.84 (1.53–15.3)(^*)</td>
<td>1.22 (0.64–2.34)</td>
</tr>
<tr>
<td>Socio-economic Status</td>
<td>1.06 (0.92–1.22)</td>
<td>1.33 (0.97–1.83)</td>
<td>1.08 (0.80–1.47)</td>
<td>0.97 (0.81–1.16)</td>
</tr>
<tr>
<td>Learning Disability</td>
<td>1.98 (1.20–3.26)</td>
<td>3.89 (1.35–11.2)(^*)</td>
<td>1.40 (0.50–3.91)</td>
<td>1.61 (0.84–3.07)</td>
</tr>
<tr>
<td>Caucasian</td>
<td>2.41 (1.23–4.71)</td>
<td>1.98 (0.61–6.40)</td>
<td>3.31 (0.72–15.2)</td>
<td>2.22 (0.89–5.57)</td>
</tr>
<tr>
<td>Parental Psychopathology</td>
<td>1.06 (0.36–3.14)</td>
<td>1.62 (0.19–14.0)</td>
<td>1.23 (0.15–10.0)</td>
<td>0.91 (0.21–3.96)</td>
</tr>
</tbody>
</table>

\(^a\) Controlling for sex, socio-economic status at age seven, LD at age seven, race and parental psychopathology.

\(^b\) Number of GAD cases: lifetime = 75, child onset = 18, adolescent onset = 16, adult onset = 41.

\(^c\) Corresponds to a 15-point, 1 SD increase in FSIQ at age seven.

\(^* P < 0.05, ^{**} P < 0.001, ^{***} P < 0.0001.\)
GAD, there was no relationship between cognitive performance and GAD onset in those with ($P = 0.19$) or without LD ($P = 0.94$).

**Age of onset**

Thirteen of 18 individuals reported age of childhood onset at age seven or younger. Therefore, we examined the association between FSIQ and childhood onset GAD limiting the analytic sample to the five individuals whose reported GAD onset occurred after the assessment of cognitive performance. Despite loss of statistical power, results were largely consistent with findings presented above. A 15-point increase in cognitive performance at age seven was associated with a 74% reduction in child onset GAD ($OR = 0.26$, 95% CI 0.05–1.25, $P = 0.09$).

**Behavioural inhibition**

Using derived measures of behavioural inhibition at age seven, defined as shy, withdrawn and having difficulty communicating, we examined whether inhibition may have influenced our findings by examining the association of cognitive performance and lifetime GAD by high and moderate/low behavioural inhibition.

Effect sizes were similar among those who scored high on a measure of behavioural inhibition ($n = 108$; $OR = 0.40$, 95% CI 0.10–1.55) and those who did not ($n = 581$; $OR = 0.48$, 95% CI 0.33–0.71). The addition of an interaction term in the non-stratified model suggested that there was no systematic difference in the association between cognitive performance and GAD among those who were highly behaviourally inhibited in childhood and those who were not.

**Stressful life events**

Another potential explanation for the association between FSIQ and GAD is that experiencing stressful life events in childhood may contribute to both lower performance on standardized tests of intelligence as well as the development of GAD. We examined this possibility by including an index of stressful life events prior to age seven (coded as 0, 1, 2+) in the analyses. While this index, which included parental divorce, moving more than three times, sibling death and living away from home for more than three months, was not available for our full analytic sample due to missing data, analyses suggested that the addition of the stressful life index to the model did not confound the association between FSIQ and lifetime GAD ($OR = 0.55$, 95% CI 0.37–0.80), or onset in specific developmental periods: childhood ($OR = 0.14$, 95% CI 0.04–0.45), adolescence ($OR = 0.43$, 95% CI 0.19–0.97) or adulthood ($OR = 0.84$, 95% CI 0.53–1.34).

**Discussion**

This is the first study to report an association between childhood cognitive performance and GAD onset. A 15-point (1 SD) advantage in IQ was associated with a 50% reduction in risk of lifetime GAD after controlling for relevant background characteristics. When examined by developmental period, the association between childhood IQ and GAD appears stronger for earlier onset, as a 1 SD advantage in FSIQ was associated with an 89% ($P < 0.001$) and 67% ($P = 0.04$) reduction in risk of GAD in childhood (age 12 or younger) and adolescence (ages 13–18), respectively, yet only a 19% ($P = 0.37$) reduced risk of GAD onset in adulthood.

This study has several strengths and limitations. First, we were able to partially account for parental mental health status. While it is unknown whether parents were diagnosed with GAD specifically, this study includes information on the treatment of mental health problems of both the study participant’s mother and father. While our measure of parental mental health may be underestimated as it relied on reports of treatment seeking, rather than a diagnosis, no studies to our knowledge have found that offspring of parents with GAD have lower levels of cognitive performance, suggesting that residual confounding by parental GAD may be minimal. While this study accounted for a host of theoretically important sociodemographic background factors, there may be other unmeasured confounders, such as other childhood psychiatric disorders, which may be biasing our results.

Another strength of this study is its longitudinal design; childhood cognitive performance was obtained prior to the reporting of GAD, and in general, prior to GAD onset. However, it is important to note that GAD was assessed at a single point in time, when individuals were in their 30s, and that age of onset was retrospectively reported. While recall of age of onset of GAD over a shorter, 4-year period has been found to be acceptable, it is not clear whether similar results to this study would be found if GAD onset were prospectively collected. While our sensitivity analyses examining the association between cognitive performance and childhood onset GAD between those who reported onset after age seven and those who reported earlier onset showed comparable effect sizes, there may be some misclassification by developmental period.

Results of this study may also have been confounded by the presence of early behavioural inhibition or stressful life events, which may contribute to both later GAD onset as well as poorer performance on the WISC. However, a stratified examination of those with and without high levels of behavioural inhibition and stressful life events showed similar effect sizes for the parameters of interest. A sensitivity analysis examining the influence of LD on our association of interest also suggested that the higher prevalence of LD in our sample did not explain these results.

While the analyses controlled for SES in childhood, this measure was limited both in terms of its range, in that it utilized information from the head of the household, and assessed SES at a single point in time. Use of additional measures of childhood SES in the analyses did not alter the results; however, there remains the possibility of residual confounding by SES. In addition, there may be unmeasured confounders, such as additional stressful life events, which may be influencing the results of the study.

Although the lifetime prevalence of GAD in our study sample of 10.9% is higher than the 4–7% estimated in the general population, this is not unexpected given that a large proportion of our study population had a childhood LD, and individuals with a LD are at increased risk for anxiety disorders. Further, the prevalence of GAD among our non-LD population (1.5% for childhood onset and 2.3% for
adolescent onset) is consistent with that found in community samples of children and adolescents, which report rates of GAD between 0.8% and 4.3%.43

Results of this study suggest that childhood cognitive performance may be an important early risk factor for the development in GAD. Further research on the association between cognitive performance and GAD is warranted and may ultimately contribute to the earlier identification of individuals, when the likelihood of GAD onset is greatest.

**KEY MESSAGES**

- Childhood cognitive performance is associated with GAD net of relevant background factors.
- The association is strongest for childhood and adolescent onset.
- Further research should examine the association between cognitive performance and GAD, particularly early in life when the risk of onset is greatest.

**References**


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**Conflict of interest:** None declared.


Martin L, Buka S, Fitzmaurice G, Kindlon D. Cognitive Performance in Childhood and Early Adult Illness: A Prospective Cohort Study. *J Epidemiol Community Health* 2004;58:674–79.

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