Minor physical anomalies, intelligence, and cognitive decline

Citation for published version:

Digital Object Identifier (DOI):
10.1080/0361073X.2012.672126

Link:
Link to publication record in Edinburgh Research Explorer

Document Version:
Publisher's PDF, also known as Version of record

Published In:
Aging clinical and experimental research

General rights
Copyright for the publications made accessible via the Edinburgh Research Explorer is retained by the author(s) and / or other copyright owners and it is a condition of accessing these publications that users recognise and abide by the legal requirements associated with these rights.

Take down policy
The University of Edinburgh has made every reasonable effort to ensure that Edinburgh Research Explorer content complies with UK legislation. If you believe that the public display of this file breaches copyright please contact openaccess@ed.ac.uk providing details, and we will remove access to the work immediately and investigate your claim.
Minor Physical Anomalies, Intelligence, and Cognitive Decline

David Hope a, Timothy Bates a, Alan J. Gow a, John M. Starr b c & Ian J. Deary a

a Centre for Cognitive Ageing and Cognitive Epidemiology and Department of Psychology, University of Edinburgh, Edinburgh, UK

b Centre for Cognitive Ageing and Cognitive Epidemiology, University of Edinburgh, Edinburgh, UK

c Geriatric Medicine Unit, Royal Victoria Hospital, Edinburgh, UK

Published online: 27 Apr 2012.


To link to this article: http://dx.doi.org/10.1080/0361073X.2012.672126

PLEASE SCROLL DOWN FOR ARTICLE

Taylor & Francis makes every effort to ensure the accuracy of all the information (the “Content”) contained in the publications on our platform.
MINOR PHYSICAL ANOMALIES, INTELLIGENCE, AND COGNITIVE DECLINE

David Hope
Timothy Bates
Alan J. Gow

Centre for Cognitive Ageing and Cognitive Epidemiology and Department of Psychology, University of Edinburgh, Edinburgh, UK

John M. Starr

Centre for Cognitive Ageing and Cognitive Epidemiology, University of Edinburgh, Edinburgh, UK and Geriatric Medicine Unit, Royal Victoria Hospital, Edinburgh, UK

Ian J. Deary

Centre for Cognitive Ageing and Cognitive Epidemiology and Department of Psychology, University of Edinburgh, Edinburgh, UK

Received 5 July 2010; accepted 14 March 2011.

This study was supported by grant No. CZB/4/505 to T.C.B., A.J.G., J.M.S., and I.J.D. from the Scottish Government’s Chief Scientist Office. A.J.G. is funded as part of the Help The Aged–funded Disconnected Mind research program. The work was undertaken by The University of Edinburgh Centre for Cognitive Ageing and Cognitive Epidemiology, part of the cross council Lifelong Health and Wellbeing Initiative (G0700704/84698). Funding from the Biotechnology and Biological Sciences Research Council (BBSRC), Engineering and Physical Sciences Research Council (EPSRC), Economic and Social Research Council (ESRC), and Medical Research Council (MRC) is gratefully acknowledged.

Address correspondence to Professor Timothy C. Bates, Centre for Cognitive Ageing and Cognitive Epidemiology, Department of Psychology, University of Edinburgh, 7 George Square, Edinburgh EH8 9JZ, Scotland, United Kingdom. E-mail: tim.bates@ed.ac.uk
Background/Study Context: Minor physical anomalies are thought to be markers of development and increased frequency of such anomalies has been linked to lower levels of intelligence. Here the authors examine a finger curvature anomaly, and evaluate its potential as a marker of the causes of cognitive aging.

Methods: Participants were members of the Lothian Birth Cohort 1921 (LBC 1921). Intelligence was assessed at ages 11, 79, and 87. In wave 3, at age 87, 192 participants had both hands scanned with a high-resolution flatbed scanner and the curvature of the fifth digit was measured with image editing software. Multiple regression analyses were conducted to examine the proportion of unique variance in cognitive decline that could be explained by the finger curvature anomaly.

Results: Finger curvature was significantly associated with cognitive decline across the life span ($\beta = -0.19, p = .02$). Curvature was not associated with intelligence at age 11 or with decline during the period age 79 to age 87.

Conclusion: Continuously varying minor physical anomalies may accumulate to provide a marker of factors impacting life span cognitive change. Curvature anomalies may reflect the common causes underlying cognitive and physical decline.

Minor physical anomalies (MPAs) are small, distinctive bodily features that do not impair everyday functioning, but that may be indicative of congenital disorder (Compton & Walker, 2009) or of stress and inflammation accumulated over the life span (Flatt, 2005). Here we test the association of a well-established MPA with cognitive ability and cognitive decline.

Such anomalies are diverse in nature and can occur anywhere on the body. For example, humans typically exhibit only a single hair whorl (sometimes referred to as a crown), and the presence of two (or more) hair whorls is considered anomalous. Similarly, a flat and narrow roof of the mouth is unusual, as are malformed ears or large gaps between digits (Waldrop, Pedersen, & Bell, 1968). Such traits are usually categorized as anomalous only where they are distinctively different from what would be found in the general population (Waldrop et al., 1968).

MPAs are positively correlated with a number of disorders, including Down syndrome (Waldrop et al., 1968), greater emotionality and extraversion (Paulhus & Martin, 1986), and schizophrenia (Compton & Walker, 2009). Meta-analyses of the link to schizophrenia (Weinberg, Jenkins, Marazita, & Maher, 2007) and autism (Ozgen, Hop, Hox, Beemer, & van Engeland, 2010) suggest large effect sizes (Cohen’s $d = 1.13$ and 0.84, respectively) irrespective of the site of the
MPA. The fact that MPAs linked to cognitive impairment cognition are not restricted to particular body regions suggests that MPAs may reflect problems affecting the entire system. This relationship is as predicted from the “common cause hypothesis” of aging, suggesting that cognitive and noncognitive declines in old age share one or a small number of “common” causes (Christensen, Mackinnon, Korten, & Jorm, 2001). This shared factor has been conceptualized as system integrity or general fitness (Deary, 2008; Prokosch, Yeo, & Miller, 2005). Common cause models share features with the concept of developmental stability described by Waddington (1957) as an organism’s ability to develop normally despite the presence of perturbations. These perturbations include malnutrition, pathogens, environmental toxins, and illness, among others. Greater developmental instability (DI) would be expected to lead to more severe MPAs.

Although DI is usually considered in terms of early development—indicating a system that was initially poorly put together—DI may also emerge over the life span as a consequence of poor buffering against environmental perturbations such as high allostatic load (McEwen & Stellar, 1993). Because both early (Barker, 1995, 2007; Marmot, 2010) and accumulated (Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010) stress have been proposed as causes of physical, sensory, and cognitive decline (Christensen et al., 2001), MPAs may form a valuable marker of health and decline among older people. Here we test the association of a well-established MPA—finger curvature—with cognitive ability and cognitive decline. Of course MPAs would not be expected to cause age-related decline, but rather to function as a readily measured indicator of the causes of cognitive decline.

Previous research relating MPAs to cognition has been restricted to younger samples, and sample sizes involved are typically small or recruited on the basis of conditions such as schizophrenia that might independently influence scores. Rosenberg and Weller (1973) reported a negative association of MPAs with verbal (but not spatial) intelligence, a finding replicated by Pine, Shaffer, Schonfeld, and Davies (1997) in 118 male participants. Gally, Kantola-Sorsa, and Granström (1988) found higher numbers of MPAs were associated with lower Wechsler Preschool and Primary Scale of Intelligence scores in 108 children of epileptic mothers and 100 control children (a nonverbal intelligence measure, the Leiter International Performance Scale, did not associate significantly with MPAs) in either group. Dimambro et al. (2008) found that in a sample of people with schizophrenia, MPA count was associated with lower intelligence.
Importantly, MPAs were also associated with a greater decrease in intelligence over time in this sample. One study (Marcus, Hans, Byhouwer, & Norem, 1985) identified no association between intelligence and MPAs, though they suggested low sample size might have been a factor (the total sample size was 100, of which 27 exhibited no anomalies and only 7 exhibited three anomalies). In summary, most studies, but not all (cf. Marcus et al., 1985), have reported significant negative associations between MPA count and intelligence, particularly verbal intelligence.

Whereas many MPAs appear early in development and are stable across time (e.g., syndactyly, where digits are at least partially fused together), biological variation believed to underpin other MPAs accumulates during aging (Lloyd, Doody, Brewin, Park, & Jones, 2003). Hence, the frequency and severity of some MPAs increases with age. As with cognitive aging itself, the causes of MPAs accumulated during adult life (rather than early in development) are not well understood. It is possible that an inability to maintain basic bodily processes might lead to a failure to maintain outward systems so that age-related MPAs exhibit more clearly initial underlying stress. MPAs that accumulate or even appear de novo in old age may be consequences of factors occurring early in development. If this is the case, MPAs in old age should predict early cognition, as well as life span cognitive decline. Alternatively, novel or increasing anomalies such as curvature may reflect accumulating new stresses such as damage and inflammation that accumulate independently of initial developmental state. In this case, we expect MPAs to relate to differences in rates of cognitive decline, and that this would not be accounted for by initial cognitive status.

MPAs, then, are promising aging biomarkers that are readily recorded, and have potentially tractable biological pathways. However, MPAs have typically been scored categorically—for example, large gaps between digits might be categorized as abnormal rather than actually measured. Moving to a continuous scale has been suggested to be more sensitive and useful (Sivkov & Akabaliev, 2003). For this reason, here we focused on a quantitative rather than categorical assessment of the common MPA of curvature of the fifth (little) finger. This marker has several advantages. It is known to be particularly common among older people. It has also been suggested as an indicator of accumulated stressors, including familial factors, trauma (Flatt, 2005), and a wide range of inflammatory processes (Sokka et al., 2009). As a potential biomarker of aging, we predict that greater fifth finger curvature will be associated with relatively greater cognitive decline. Moreover, as we propose that finger MPA
is a specific biomarker for aging, we also predict that curvature does not solely arise as a result of specific age-associated pathologies such as arthritis. We therefore predict that the association of MPA and cognitive decline should persist even after accounting for the use of anti-inflammatory drugs and levels of C-reactive protein (CRP), indicators of specific illnesses (such as arthritis) that might influence curvature.

**METHODS**

**Participants**

Participants were members of the Lothian Birth Cohort 1921 (LBC 1921). The initial recruitment and testing of this 550-strong sample has been described elsewhere (Deary, Whiteman, Starr, Whalley, & Fox, 2004). Participants were all born in 1921 and took part in the Scottish Mental Survey 1932 (SMS) at an average age of 11 years. They were subsequently recruited for further cognitive and medical tests beginning at age 79, in 1999–2001. Measures analyzed here were taken at testing waves where the mean age of the participants was about 79 and 87 years.

Of the initial wave of 550 (234 men and 316 women), 454 were contacted for the second wave at age 83 (335 agreed, and 321 were tested, of which 145 were men and 176 were women). Excluding those who had died or withdrawn, 268 were contacted for wave 3 at age 87 and 207 completed all the measures (Starr et al., 2010). The analyses here use data from childhood, and waves 1 and 3. Testing at waves 1 and 3 was conducted at a clinical research facility.

**Cognitive Ability Test**

Cognitive ability at mean ages of 11, 79, and 87 years was assessed using the Moray House Test No. 12 (Deary et al., 2004; Scottish Council for Research in Education, 1933). This is a 45-min, time-limited test of verbal (principally), numerical, spatial, and abstract reasoning with a maximum possible score of 76. Scores were controlled for age in days at time of testing and then converted to a standardized IQ-type scale (with $M = 100$ and $SD = 15$).

Participants completed the Mini-Mental State Examination (MMSE) at age 87, a screening tool for cognitive impairment (Folstein, Folstein, & McHugh, 1975). The mean score was 27.80 ($SD = 2.22$) out of a total score of 30. In order to evaluate the level
of dementia in the sample, we recoded this variable with scores above 23 indicating normal or near-normal functioning \( (n = 182) \) and scores at 23 or below indicating impairment \( (n = 10) \).

**Parental Education**

Between ages 80 and 81, participants answered questions on their family history, including number of years of parental education. Father’s education was \( M = 10.10 \) \( (SD = 3.00) \), and mother’s education was \( M = 9.68 \) \( (SD = 2.36) \).

**Minor Physical Anomalies**

At mean age 87 in wave 3, participants undertook a variety of physical and mental tests, including having both hands scanned individually using a high-resolution flatbed scanner. In total, 192 participants (90 male, 102 female) completed the hand scan and provided usable images.

Curvature of the fifth digit (the little finger) was assessed using GIMP image editing software (available at www.gimp.org) to record digitally the length and angle of each segment of the little finger. The change in angle between each segment was summed and averaged bilaterally providing a continuous MPA severity variable. To our knowledge, all other studies investigating curvature of the fifth digits have simply categorized the digit as being either very curved, slightly curved, or not curved. The present study is the first that we are aware of in this research area to use a continuous measurement of curvature. To test reliability, we measured fifth digit curvature for three participants drawn from another sample (with no relation to the participants in the present study). Each participant provided two different images of both hands. We calculated the intraclass correlation coefficient for each of the three pairs by comparing the curvature of the fifth digits between the paired images. Results were \( r = .89, .85, \) and \( .90 \) for the three pairs, indicating very high reliability.

**Inflammatory Markers**

At age 79, use of anti-inflammatory drugs (NSAIDs) was recorded categorically as either use or nonuse according to self-report. This class of drug is most frequently used in older adults to treat pain caused by inflammation. Fourteen participants reported use of NSAIDs and were removed from the analysis. At mean age 87 in wave 3, levels of CRP were measured via blood test. CRP levels
increase significantly during inflammation (from, among other factors, arthritis). Mean CRP level was 3.96 mg/L, $SD = 8.61$ mg/L (median 1.92 mg/L).

**RESULTS**

The sample had a mean curvature of $3.83^\circ$ ($SD = 2.26^\circ$). On average, females had around 25% greater curvature ($n = 102, M = 4.24^\circ, SD = 2.20^\circ$) than males ($n = 90, M = 3.37^\circ, SD = 2.24^\circ$). This sex difference was significant and of medium effect size ($t(190) = -2.72, p = .007, d = 0.39$). Curvature in excess of $8^\circ$ is considered abnormal (Smith, 1970) but few subjects exhibited these levels of curvature. For the whole sample, mean IQ scores were 102.17, 103.40, and 99.09 at ages 11, 79, and 87, respectively. Figure 1 displays an image of, from left to right, low, moderate, and high curvature. When the sample was ranked from lowest to highest, the images were representative of the 25th, 50th, and 75th percentiles for curvature, respectively. The correlation matrix of all variables is given in Table 1. Note that curvature does not correlate with intelligence measures at age 11, 79, or 87.

Three models were tested using multiple linear regressions to assess the association between finger curvature and cognitive change: between age 11 and age 79, between age 11 and age 87, and between age 79 and age 87. We initially included father’s and mother’s education, MMSE score, and C-reactive protein level as covariates. C-reactive protein level did not influence the models and so was

![Figure 1. Low, medium, and high curvature of the fifth digit. Images are representative of the 25th, 50th, and 75th percentiles when participants are ranked according to curvature score from lowest to highest.](image-url)
Table 1. Correlation matrix of intelligence, curvature, Mini-Mental State Examination score, and markers of inflammation

<table>
<thead>
<tr>
<th></th>
<th>Age 11 IQ</th>
<th>Age 79 IQ</th>
<th>Age 87 IQ</th>
<th>Father’s education</th>
<th>Mother’s education</th>
<th>Curvature (right fifth digit)</th>
<th>Curvature (left fifth digit)</th>
<th>Curvature (average of both digits)</th>
<th>Use of anti-inflammatory drugs</th>
<th>C-reactive protein level (age 87)</th>
<th>Mini-Mental State Examination age 87</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age 11 IQ</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>.659**</td>
<td>.513**</td>
<td>.139*</td>
<td>.122*</td>
<td>.031</td>
<td>.111</td>
<td>.089</td>
<td>.051</td>
<td>.116</td>
<td></td>
<td>.327**</td>
<td></td>
</tr>
<tr>
<td>(485)</td>
<td>(178)</td>
<td>(338)</td>
<td>(336)</td>
<td>(168)</td>
<td>(168)</td>
<td>(168)</td>
<td>(460)</td>
<td>(161)</td>
<td></td>
<td>(183)</td>
<td></td>
</tr>
<tr>
<td>Age 79 IQ</td>
<td>.707**</td>
<td>.154**</td>
<td>.118*</td>
<td>-.073</td>
<td>-.039</td>
<td>-.070</td>
<td>.028</td>
<td>.078</td>
<td>.460**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(200)</td>
<td>(377)</td>
<td>(375)</td>
<td>(189)</td>
<td>(189)</td>
<td>(189)</td>
<td>(189)</td>
<td>(460)</td>
<td>(182)</td>
<td></td>
<td>(204)</td>
<td></td>
</tr>
<tr>
<td>Age 87 IQ</td>
<td>.093</td>
<td>.082</td>
<td>-.051</td>
<td>.020</td>
<td>-.019</td>
<td>.028</td>
<td>.018</td>
<td>.568**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(166)</td>
<td>(166)</td>
<td>(186)</td>
<td>(186)</td>
<td>(186)</td>
<td>(173)</td>
<td>(178)</td>
<td>(200)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Father’s education</td>
<td>.744**</td>
<td>-.038</td>
<td>.216**</td>
<td>.103</td>
<td>-.018</td>
<td>-.063</td>
<td>.146</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(374)</td>
<td>(159)</td>
<td>(159)</td>
<td>(159)</td>
<td>(322)</td>
<td>(151)</td>
<td>(169)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother’s education</td>
<td>.052</td>
<td>.032</td>
<td>.053</td>
<td>.026</td>
<td>-.118</td>
<td>.168*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(160)</td>
<td>(160)</td>
<td>(160)</td>
<td>(160)</td>
<td>(320)</td>
<td>(152)</td>
<td>(169)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Curvature (right</td>
<td>.289**</td>
<td>.803**</td>
<td>-.043</td>
<td>-.068</td>
<td>.020</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>fifth digit)</td>
<td>(192)</td>
<td>(192)</td>
<td>(162)</td>
<td>(180)</td>
<td>(192)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Curvature (left</td>
<td>.803**</td>
<td>.049</td>
<td>.004</td>
<td>.015</td>
<td>.015</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>fifth digit)</td>
<td>(192)</td>
<td>(192)</td>
<td>(162)</td>
<td>(180)</td>
<td>(192)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Curvature (average</td>
<td>.005</td>
<td>-.040</td>
<td>.022</td>
<td>.070</td>
<td>.088</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>of both digits)</td>
<td>(162)</td>
<td>(162)</td>
<td>(180)</td>
<td>(180)</td>
<td>(192)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Use of anti-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>inflammatory</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>drugs</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C-reactive protein</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>level (age 87)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.167*</td>
</tr>
<tr>
<td>(157)</td>
<td>(184)</td>
<td>(177)</td>
<td>(177)</td>
<td>(177)</td>
<td>(177)</td>
<td>(177)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. Values are correlation coefficient (r) with sample size in parentheses. *p < .05; **p < .01.
dropped. MMSE score did not influence the significance or magnitude of curvature scores and so was dropped. Father’s and mother’s education, which correlated highly with age 11 and age 79 IQs and for some analyses approached significance, were retained. We also initially excluded users of anti-inflammatory drugs, but model fits and significance of the predictors did not vary according to whether or not they were excluded, so we opted to retain them in the final models. Results for all three models are presented in Table 2.

Model 1 examined predictors of IQ change between age 11 and age 79. It used age 79 IQ as the outcome variable, predicted by finger curvature, and with age 11 IQ and father and mother’s education as covariates. Including age 11 IQ effectively tested the contribution of finger curvature to the change in IQ between age 11 and age 79. As expected, age 11 IQ made a large and significant contribution to IQ at age 79, so that those with greater IQ at age 11 had greater IQ at age 79. Parental education did not contribute significantly to the model, whereas finger curvature did ($\beta = -.18$, $p = .01$): greater finger curvature was associated significantly with greater relative IQ decline between age 11 and age 79. When the sexes were analyzed separately, the contribution of finger curvature was significant in women ($\beta = -.28$, $p = .004$), but not in men (though the direction of effect was the same as for women: $\beta = -.09$, $p = .37$).

We next examined predictors of IQ change between age 11 and age 87 (Model 2). Age 87 IQ was the outcome variable, with the same covariates as in Model 1. As in Model 1, age 11 IQ contributed significantly to IQ at age 87 so that those with higher IQ at age 11 had relatively higher IQ at age 87. Finger curvature also accounted for a significant amount of variance in age 87 cognitive decline ($\beta = -.19$, $p = .02$). Sex-specific analyses found finger curvature significant in women ($\beta = -.30$, $p = .004$), but not in men ($\beta = -.15$, $p = .25$): in all cases greater curvature was associated with relatively greater cognitive decline.

Model 3 examined change in IQ during old age, from 79 to 87. Age 87 IQ was used as the outcome variable with the same covariates used in Model 2, with the addition of age 79 IQ. After accounting for the effect of age 11 IQ, age 79 IQ made a large and significant contribution to IQ at age 87, with higher initial IQ predicting higher IQ at age 87. Finger curvature was not significantly associated with change either in the combined ($\beta = -.08$, $p = .24$) or sex-specific (male $\beta = -.10$, $p = .34$; female $\beta = -.13$, $p = .14$) samples. In women but not men, there was a significant effect of mother’s education on cognitive change ($\beta = .26$, $p = .033$).
### Table 2. Linear regression models of Moray House Test IQ at ages 79 and 87

<table>
<thead>
<tr>
<th></th>
<th>Model 1: Age 79 IQ</th>
<th></th>
<th>Model 2: Age 87 IQ</th>
<th></th>
<th>Model 3: Cognitive Decline Ages 79–87</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All subjects</td>
<td>Males</td>
<td>Females</td>
<td>All subjects</td>
<td>Males</td>
<td>Females</td>
</tr>
<tr>
<td>Age 11 IQ</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>.56 (.06)</td>
<td>.56 (.08)</td>
<td>.57 (.09)</td>
<td>.53 (.09)</td>
<td>.52 (.14)</td>
<td>.58 (.11)</td>
</tr>
<tr>
<td></td>
<td><strong>.65</strong></td>
<td><strong>.68</strong></td>
<td><strong>.62</strong></td>
<td><strong>.50</strong></td>
<td><strong>.49</strong></td>
<td><strong>.54</strong></td>
</tr>
<tr>
<td>Age 79 IQ</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>.65 (.38)</td>
<td>−.06 (.60)</td>
<td>.48 (.51)</td>
<td>.04 (.56)</td>
<td>.41 (1.02)</td>
<td>−.36 (.64)</td>
</tr>
<tr>
<td></td>
<td>.07</td>
<td></td>
<td></td>
<td>.01</td>
<td>.08</td>
<td>−.08</td>
</tr>
<tr>
<td>Father’s education</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>.10 (.45)</td>
<td>.41 (.74)</td>
<td>−.08 (.62)</td>
<td>.20 (.69)</td>
<td>−1.09 (1.28)</td>
<td>1.36 (.78)</td>
</tr>
<tr>
<td></td>
<td>−.18</td>
<td>.09</td>
<td>−.02</td>
<td>.03</td>
<td>−.18</td>
<td>.25</td>
</tr>
<tr>
<td>Mother’s education</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>−1.00 (.36)</td>
<td>−.48 (.53)</td>
<td>−1.62 (.54)</td>
<td>−1.28 (.54)</td>
<td>−1.04 (.89)</td>
<td>−2.03 (.68)</td>
</tr>
<tr>
<td></td>
<td>−.18**</td>
<td>−.09</td>
<td>−.28**</td>
<td>−.19*</td>
<td>−.15</td>
<td>−.30**</td>
</tr>
<tr>
<td>Finger curvature</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>.46</td>
<td>.48</td>
<td>.42</td>
<td>.26</td>
<td>.20</td>
<td>.33</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td></td>
<td></td>
<td></td>
<td>.47</td>
<td>.44</td>
<td>.54</td>
</tr>
</tbody>
</table>

**Note.** Effects are given as B (SE), with standardized β below.

* $p < .05$; ** $p < .01$; *** $p < .001$. Significant values are indicated in bold.
DISCUSSION

We found a significant association between fifth finger curvature and lifetime change in cognitive function after accounting for parental education. The inclusion or exclusion of MMSE scores and signs of inflammation did not influence the findings. Those with higher levels of curvature experienced relatively greater general cognitive decline, scoring lower at both age 79 and age 87 than predicted based on their age 11 performances. When the sexes were analyzed separately, the results were significant in women but not in men. As such, the present study is the first to report an association between a minor physical anomaly and cognitive decline in old age.

Significantly, there was no association between fifth finger curvature and intelligence at age 11. This finding mirrors the results from measures of facial fluctuating asymmetry and cognitive aging (Penke et al., 2009), which also revealed links between accumulated asymmetry and cognitive decline, with no relationship to initial levels. This supports the interpretation that greater frequency and severity of minor physical anomalies function as a marker of enhanced cognitive decline, rather than higher initial ability.

The finger curvature MPA has previously been associated with inflammatory processes and stress (Flatt, 2005). The association of this marker with cognitive decline implies that, at least in women, such inflammatory processes may be linked to an increased rate of decline in general cognitive function, relative to baseline levels measured early in life. The present study provides further support for the idea of a common cause, or set of common causes, as a foundation for the shared variance between cognitive and physical decline (Christensen et al., 2001).

The present study also demonstrates further that MPAs are not only indicative of problems in severe cases (e.g., clinically diagnosable conditions such as autism or schizophrenia) but also can provide information on behavioral and cognitive variables on a normal continuum. In children, higher levels of MPAs, even when not manifesting in severe developmental disorders, can still predict increased levels of behavioral issues and conduct disorder (Waldrop et al., 1968). This also seems to be true in male adults, as those men with higher incidences of MPAs demonstrate differences in personality such as emotionality, extraversion, and Type A personality (Paulhus & Martin, 1986). Just as some MPAs are associated with differences in personality traits, they may predict differences in cognitive change over time.
Among several positive attributes of the present study, it is, to our knowledge, the first to adopt a continuous measure of MPA, allowing a continuous rather than a categorical assessment. The advantage of this is demonstrated by very few of the participants having what would be considered abnormal curvature (Smith, 1970)—using a categorical system only a small number of participants would have demonstrated unusual curvature. Furthermore, in this sample, IQ at age 11 was tested rather than estimated, ensuring that cognitive change was reliably measured. Similarly, homogeneity of age was a major strength of the present study, largely eliminating the effects of chronological age. The sample was made up of Caucasian Scots, and although they were of diverse socioeconomic origins, they are representative of their population. It is unknown how such findings might generalize to other ethnicities and nationalities. However, these factors ensured that subject differences reflected relative differences in biological aging.

Although this study is novel in the use of a quantitative measure of an MPA, and draws on an extremely useful longitudinal sample, it is important to note the use of a single MPA is a limitation. Replication and expansion are necessary to better understand the relationship of MPAs to cognitive aging, preferably with additional MPA markers in larger samples to compare the predictive value of multiple morphological traits. Particular attention should be paid to the sex specificity of MPA effects. In this sample, surviving men showed significantly less curvature, and the cognitive decline–curvature association was significant in women alone, though the direction of the effect was the same in men. Given sex-specific mortality rates, in the present sample males with higher levels of MPA may have incurred increased rates of mortality, reducing the mean level of observed MPA in the remaining population and thus reducing the association with cognitive decline, creating the appearance of sex specificity. Alternatively, the effect may be genuine in both sexes, but not identified here in men due to the relatively smaller sample size. It remains possible that specific MPAs may be more indicative of developmental instability in one sex or the other. Notably, Sokka et al. (2009) found evidence that rheumatoid arthritis was more severe in women as opposed to men. If women are more vulnerable to inflammatory conditions such as arthritis, reduced fifth digit curvature may be indicative of greater developmental stability in women as compared with men. This is uncertain, however, and repeating the study with a broader range of MPAs would be the most effective means of addressing this issue.
REFERENCES


