Symmetric faces are a sign of successful cognitive aging
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Abstract

It has been proposed that a common cause underlies individual differences in bodily and cognitive decline in old age. No good marker for this common cause has been identified to date. Here, fluctuating asymmetry (FA), an indicator of developmental stability that relates to intelligence differences in young adults, was measured from facial photographs of 216 surviving members of the Lothian Birth Cohort 1921 at age 83 and related to their intelligence at ages 11, 79 and 83 years. FA at age 83 was unrelated to intelligence at ages 11 and 79 and to cognitive change between 11 and 79 years. It was, however, associated with intelligence and information processing efficiency at age 83 and with cognitive change between 79 and 83 years. Significant results were limited to men, a result predicted by sex differences in life history tradeoffs and life expectancy. Results were stronger when directional asymmetries were corrected in facial FA measures. Thus, FA is a candidate marker for the common cause of differential senescence.

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1. Introduction

The threat of cognitive decline is the most feared aspect of human aging for many people, but the reasons why some people show more cognitive decline than others remain largely unknown. The observation that cognitive functions often decline in concert both with each other (Wilson et al., 2002) and with sensory, organic, and muscular functionality has made the “common cause” hypothesis an influential account for individual differences in cognitive aging (Christensen, Mackinnon, Korten, & Jorm, 2001; Lindenberger & Baltes, 1994). This hypothesis suggests that cognitive decline is in part an aspect of body-wide senescence in what we shall call system integrity. It is assumed that no single one mechanism underlies declining system integrity. Instead, the proximate causes of this decline may be accumulated environmental stresses, recurrent failures to adapt to stressors (i.e., allostatic load), diminishing cellular repair efficiency, and genetic and epigenetic chance events (Batty, Deary, & Gottfredson, 2007; Finch & Kirkwood, 2000; McEwen, 2006; Seeman, McEwen, Rowe, & Singer, 2001). The loss of system integrity with senescence can be considered to be the result of a life-history trade-off in which reduced investment in somatic maintenance and repair (or “own embodied capital”) frees more energy for reproductive effort (Kaplan, Mueller, Gangestad, & Lancaster, 2003; Kirkwood, 2008).

A ready marker summarizing all these small, diverse, detrimental processes that impede system integrity in old age would be invaluable, and since the formulation of the common cause hypothesis, several such markers have been proposed. For example, the length of telomeres (nucleoprotein complexes that protect the ends of chromosomes and that shrink with every cell division), was thought to be a promising candidate, but supportive empirical evidence is limited so far (Harris et al., 2006). Alternative markers such as mitochondrial DNA integrity remain active research targets (Krishnan, Greaves, Reeve, & Turnbull, 2007).
One marker of high system integrity, including successful cognitive aging, might be facial symmetry. This is suggested by a very similar common cause hypothesis that has been proposed to explain individual differences in general cognitive ability (i.e., intelligence) at any age (Furlow, Armijo-Prewitt, Gangestad, & Thornhill, 1997; Penke, Denissen, & Miller, 2007). According to this hypothesis, developmental instability (DI; Polak, 2003) is the latent causal factor that links neurophysiological and bodily condition. DI results from imprecision in the expression of developmental design due to imperfect buffering against genetic and environmental perturbations. The most established indicator for DI is fluctuating asymmetry (FA), the degree to which the size of bilateral body parts deviates from the population mean, usually aggregated across several traits (Palmer & Strobeck, 2003). Research on predominantly young people has revealed significant associations between higher general intelligence and lower FA in nine samples (Bates, 2007; Furlow et al., 1997; Luxen & Buunk, 2006; Prokosch, Yeo, & Miller, 2005; Rahman, Wilson, & Abrahams, 2004; Thoma et al., 2005, 2006), while the one study reporting a null finding used marginal in Luxen & Buunk, 2006), Johnson et al. (2007) and the two studies in Furlow et al. (1997) found no difference, and Bates (2007, study 2) found the opposite sex difference.

However, greater male investment in reproductive effort (including fitness indicators) is assumed to come at the cost of lower investment in somatic maintenance, and these costs should become especially obvious when they have accumulated in old age. Indeed, this life-history trade-off has been used to explain the shorter life expectancy of males compared to females (Finch, 2007), which is still about 4 years in Western countries, including the current study populations (Trovato & Heyen, 2006; www.mortality.org). Thus, it can be expected that the relationship between cognitive ability and DI will be especially strong in elderly men. Note that this sex–specific prediction can be derived from a combination of life history theory and parental investment theory regardless of whether Miller’s (2000) hypothesis of intelligence as a fitness indicator is correct or not. However, if intelligence was indeed a condition-dependent fitness indicator, the predicted sex difference in the FA-intelligence link in old age should be especially pronounced.

Here, for the first time, we examine FA in relation to cognitive functioning and cognitive decline in old age. The sample used is a narrow age cohort tested on cognitive ability at ages 11, 79 and 83 years. Assessments included both standard cognitive ability tests and reaction time (RT) measures. RT tasks are considered to be indicators of the efficiency of information processing and are especially prone to cognitive aging (Der & Deary, 2006; Jensen, 2006). They are also candidate markers for the common cause of cognitive aging (Deary & Der, 2005). We predicted that increased FA (and thus increased DI) would be associated with cognitive decline and RT deterioration from age 79 to 83 years, especially in men.

2. Material and methods

2.1. Subjects

The sample consisted of surviving participants of the Scottish Mental Survey 1932 (SMS1932) (Scottish Council for Research in Education, 1933), all born in 1921. They took part, as members of the Lothian Birth Cohort 1921 (LBC1921), in a follow-up study between 1999 and 2001 at a mean age of 79 years (Deary, Whiteman, Starr, Whalley, & Fox, 2004). At that time, all were generally healthy and living independently in the community. Further cognitive testing took place at a mean age of 83 years (Gow et al., 2008).

2.1.1. Facial photographs

Photos of 314 participants of the Lothian Birth Cohort 1921 were taken at age 83 with a Nikon E5700 Digital
Camera. Participants were instructed to maintain a neutral expression. Four to six images were taken of each participant under standardized lighting conditions, distance to the camera, and camera zoom. The images of 91 participants (29.0%) were excluded a priori and without knowledge of the participants’ cognitive test scores because they were unsuitable for FA measurements due to facial hair that obscured landmarks, no image with a neutral expression, or no image with a planar orientation of the face. The remaining images were rotated in Adobe Photoshop 10.0 so that both pupils and the line where the lips met were horizontally aligned (see Grammer & Thornhill, 1994).

2.1.2. Symmetry measurements

Scion Image 4.0.3.2 (www.scioncorp.com) was used to place the 15 landmarks on every facial picture (Fig. 1). Corresponding landmarks were positioned on the inside and outside corners of the eyes, cheekbones (widest horizontal part of the face below the eyes), widest points at the sides of the nostrils, corners of the mouth, the jaw (widest horizontal part of the cheeks at the mouth), and the corners of the chin. One point was placed on the lip vertex. These standard landmarks are well identifiable and have been shown to result in highly reliable symmetry measurements (Grammer & Thornhill, 1994; Simmons, Rhodes, Peters, & Koehler, 2004) that converge with subjective symmetry ratings (Penton-Voak et al., 2001; Simmons et al., 2004). Results were analyzed separately for men and women because faces are sexually dimorphic.

Two different indices of FA were calculated. The first was horizontal fluctuating asymmetry (HFA; Grammer & Thornhill, 1994), arguably the most established measure of facial symmetry. In this method, a vertical midline is defined as the average midpoint of all bilateral pairs of landmarks and the horizontal distance of each landmark from midline is measured. The absolute left-right difference of these distances, aggregated across all seven pairs of landmarks, is the HFA index. Simmons et al. (2004) introduced a second, more comprehensive index of facial fluctuating asymmetry, which will be abbreviated as CFA (comprehensive fluctuating asymmetry index). This index includes not only the distances of each landmark from a vertical midline (as in HFA), but also all possible distances between all landmarks on the same half of the face, plus the distances between all landmarks and P13 (see Fig. 1). This yields a total of 35 bilateral pairs of distances, whose absolute differences are averaged after standardization for trait size to the form the CFA index. Thus, the CFA index includes the HFA index, and extends it with 28 additional measures.

Simmons et al. (2004) reported significant directional asymmetry (DA) for human facial traits. DA occurs when the population mean of the absolute left-right difference of a bilateral trait deviates from zero, indicating a systematic bias towards greater trait expression on one side. Unlike FA, DA is assumed to be uninformative of DI (Palmer & Strobeck, 2003). To control for this potential confound, we tested for DA in each of the 35 left-right differences. This revealed significant DA in all 35 cases for both sexes (ps<.001). Graham, Emlen, Freeman, Leamy, and Kieser (1998) suggested a principal components method to control for DA in FA analyses. Briefly, when each bilateral pair of distances is submitted to a (sex-specific) principal components analysis, the first unrotated component represents the DA and the second component represents the FA of this trait. Following Simmons et al. (2004), we aggregated the unsigned factor scores on the second component for all 35 traits to receive an index of the informative FA around (or about) the uninformative DA (CFA about DA). Similarly, we aggregated the unsigned factor scores on the second component of the seven traits that contribute to the HFA index to receive an index of HFA about DA. Thus, CFA about DA and HFA about DA are the purest measures of FA in this study, with the former being the more comprehensive one. Table 1 presents internal consistencies for CFA about DA and HFA about DA based on the unsigned second component factor scores, which indicate that all FA indices had acceptable reliabilities. Internal consistencies were somewhat higher for CFA about DA (based on 35 measures) than for HFA about DA (based on 7 measures), which nicely illustrates the power of aggregation in studies of FA (Gangestad, Bennett, & Thornhill, 2001). The mean intercorrelations of the unsigned second component factors

![Fig. 1. Average face of 50 men from the LBC1921 sample with the 15 landmarks used for symmetry measurements.](image.png)
scores, as well as their ranges, are also shown in Table 1. The mean intercorrelations are somewhat higher than the one reported for the FAs of male body traits (mean $r$=.04), with some individual correlations being very high. However, this can be expected for FA measures taken from a single trait (the face) instead of multiple different body traits, since the developmental processes are likely much more interdependent in the former than in the latter case.

As suggested by Palmer and Strobeck (2003), all four indices of measured symmetry were natural log-transformed to normality. In addition, participants’ age in days was statistically controlled in these indices. All four asymmetry indices showed strong intercorrelations in men ($rs=.69–.93$) and women ($rs=.60–.90$).

### 2.1.3. Psychometric cognitive tests

The Moray House Test (Deary et al., 2004; Scottish Council for Research in Education 1933) is a group-administered test with a 45-min time limit and a maximum score of 76. It contains many verbal reasoning items and also some numerical, spatial, and abstract reasoning items. Subjects completed this test on June 1, 1932, at about age 11 years, and a second time at a mean age of 79 years. After statistically controlling for age in days, it was converted, separately for both measurement points, to an IQ-type score ($M=100$; S.D.$=15$).

Three psychometric cognitive ability tests were administered to all subjects during both follow-ups at mean ages of 79 and 83 years, respectively (Deary et al., 2004; Gow et al., 2008). Verbal Fluency (Lezak, 1995) is used to assess executive function. It involves stating as many words as possible in 1 min that begin with a given letter. The letters used were C, F, and L. The 60-item Raven’s Standard Progressive Matrices (Raven, Court, & Raven, 1977) is used to assess nonverbal reasoning. It was applied with a 20-min time limit. The test involves inspecting logically constructed abstract designs, each with a piece missing. The task is to select the correct missing piece from a number of answer options. Logical Memory is from the Wechsler Memory Scale–Revised (Wechsler, 1998) and is used to assess immediate and delayed verbal declarative memory. Participants are read aloud a short story (A) containing 25 memory items. Immediately after this, the participant recalls as much of the story as possible. The process is repeated with a second story (B). After a minimum of 30-min delay, participants recall as much as they can about each story. Because the immediate and delayed recall scores were highly correlated ($r=.79$ and $.81$ for stories A and B), they were summed to form a single memory score. At each measurement point, age in days was partialled out from test scores on all three tests, and they were submitted to a principal components analysis. The first unrotated principal component explained 49.7% and 56.1% of the variance at age 79 and 83 years, respectively. Factor loadings were .75 and .77 for Raven’s Matrices, .57 and .71 for verbal fluency, and .77 and .76 for Logical Memory, with the first loading being on the $g$ factor at age 79 and the second on the $g$ factor at age 83 years. Factor scores on this factor were used as an indicator of the subjects’ general cognitive ability ($g$; Jensen, 1998).

### 2.1.4. Chronometric cognitive ability tests

Information processing efficiency was assessed at age 83 years using a self-contained reaction time device (Deary, Der, & Ford, 2001), measuring simple reaction time mean and S.D., and four-choice reaction time mean and S.D.. There were 20 and 40 test trials for the simple and choice reaction time tests, respectively, with eight practice items each. The interstimulus interval varied randomly between 1 and 3 s. Since choice reaction times usually show stronger relationships with general cognitive ability than simple reaction times (Jensen, 1998, 2006), we expect them to show stronger links with facial FA, too.

### 2.1.5. Dementia screening

The Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975) is used as a brief screening test for dementia. It has a maximum score of 30, and dementia is thought probable with scores below 24. In addition, participants were asked to self-report any history of dementia. Four men and three women were excluded because of a history of dementia or MMSE scores below 24.

### 2.1.6. Physical health and fitness measures

To control for possible confounding effects of general health, participants’ histories for diabetes, cardiovascular disease, high blood pressure, and other vascular disease were taken at age 79. Furthermore, an index of physical fitness was derived from three measures taken at age 79 (see Deary, Whalley, Batt, & Starr, 2006, for details): the time taken to walk a measured length of 6 meters at a normal pace, the best out of three trials of grip strength measurement with a Jamar Hydraulic Hand Dynamometer (using the dominant hand), and the best out of three trials of forced expiratory volume from the lungs in 1 s, assessed with a microspirometer. Body height was partialled from all three measures, and they were submitted to principal components analyses. Scores on the first unrotated principal component, which accounted for 50.97% and 53.47% of the variance in men women, respectively, were used as an index of physical fitness.
2.2. Comparison sample

To substantiate the claim that FA increases with age, we compared the CFA scores of the current sample with FA scores from an independent general population sample of 142 young adults from the city of Berlin, Germany (70 men, 72 women, age 20–30 years, $M=24.2, S.D.=2.8$). Details on the sample (of which this was a subsample) and the way the facial pictures were taken can be found in Penke and Asendorpf (2008, Study 2). Picture standardization, symmetry measurement, and CFA score calculation were identical to the procedure described above. Table 1 shows that the internal consistencies for the CFA index was also good in this sample.

3. Results

After excluding participants with unsuitable photos or signs of dementia, 216 subjects (95 men, 121 women) remained in the main LBC1921 sample for the following analyses.

The raw frequency distributions of the CFA facial asymmetry scores of both the elderly main sample and the younger comparison sample are shown in Fig. 2. As expected, CFA showed both a significantly higher mean [$t(364)=9.68, p<.001, d=1.01$] and increased variance [$F(1, 364)=12.39, p<.001$] in the older sample. The mean difference was 3.41% (95% CI=2.72–4.10), and the coefficients of variation (CVs) showed a difference of 3.35%. Neither sex nor the interaction of sex and sample had significant effects ($p$s $>$ .49).

Facial asymmetry indices at age 83 were largely unrelated to intelligence differences, whether assessed at age 11, 79, or 83 (Table 2). However, after controlling for DA, both the HFA and the CFA index showed trends toward significant relationships with concurrently assessed $g$ factor scores at age 83 in men, with effect sizes ($r$ $=$ $-$.18/$-.17$) comparable to the average effect size found in earlier studies ($r$ $=$ $-.20$).

Age 83 facial asymmetry was not related to cognitive change from age 11 to age 79 years (i.e., the residual score of age 79 IQ scores regressed on age 11 IQ scores) (Table 2). However, cognitive change on the $g$ factor between age 79 and 83 (also based on regression residuals) in men was significantly related to all symmetry indices ($r$s $=$ $-$.24 to $-$.35), indicating that men with lower facial FA at age 83 had experienced less cognitive decline in the preceding 4 years. This effect was not found in women, and the sex difference was significant when controlling for DA ($p$s $<$ .01). Exemplary scatter plots for men and women using the CFA about DA score as the symmetry index are shown in Fig. 3.

![Figure 2](image-url)

**Fig. 2.** Frequency distributions of fluctuating asymmetry (CFA) in the elderly adults from the LBC1921 compared to a sample of young German adults.

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Pearson correlations between symmetry indices, cognitive abilities, and age-related cognitive change</th>
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<tr>
<td></td>
<td>Men</td>
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<tr>
<td></td>
<td>HFA</td>
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<td>IQ at age 11 (MHT)</td>
<td>.04</td>
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<td>IQ at age 79 (MHT)</td>
<td>.08</td>
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<td>g at age 79</td>
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<td>g at age 83</td>
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<td>IQ change age 11–79</td>
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<tr>
<td>g change age 79–83</td>
<td>$-$ .24*</td>
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<tr>
<td>Simple RTs (means)</td>
<td>.14</td>
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<tr>
<td>Simple RTs (S.D.s)</td>
<td>.10</td>
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<tr>
<td>4-choice RTs (means)</td>
<td>.27**</td>
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<tr>
<td>4-choice RTs (S.D.s)</td>
<td>.26*</td>
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</tbody>
</table>

Correlations printed in italics indicate significant ($p$ $<$ .05, two-sided) sex differences in correlation size. MHT, Moray House Test; $g$: general intelligence factor based on Raven’s Matrices, verbal fluency, and logical memory.

* $p$ $<$ .10.
** $p$ $<$ .05.
*** $p$ $<$ .01.
constituting the \( g \) factor showed similar trends for Logical Memory, Raven’s Matrices, and Verbal Fluency (in descending order of effect sizes, with only the Logical Memory effects reaching significance).

Finally, male facial symmetry was related to reaction-time based measures of information processing speed; men with more symmetric faces had faster reaction times and showed a tendency towards less variable RTs (Table 2). These effects were stronger and more consistent for four-choice \((r_s=0.14-0.30)\) than for simple reaction times \((r_s=0.05-0.17)\) and somewhat more pronounced for means than for standard deviations. Again, no consistent effects were found for women. Indeed, some correlations in women showed a trend toward the opposite direction, and many relationships were significantly stronger in men than in women.

Neither sex showed significant relationships between any of the symmetry indices and the physical fitness factor \((r_s<0.11, ps>0.30)\), even though it has previously been shown in the same sample that a lower score on this physical fitness factor was related to more cognitive decline from age 11 to 79 years (Deary et al., 2006). All results remain virtually unchanged when excluding participants with diabetes and statistically controlling for physical fitness and histories of cardiovascular disease, high blood pressure, and other vascular diseases.

4. Discussion

In this first-ever study on developmental instability in relation to intelligence and cognitive aging in the elderly, we found that, in men, greater facial FA was significantly associated with cognitive decline between 79 and 83 years, as well as with slower and more variable reaction times that were concurrently assessed at age 83, and marginally with concurrently assessed general intelligence. There were no significant relationships between FA and earlier-age intelligence or midlife cognitive change, and all significant relationships were exclusive to men. Cross-sectional comparisons of our sample in their eighties with a sample in their twenties from a close-by country with similar ecology revealed substantially higher FA means and variances in the older sample, tentatively indicating an increase of FA means and variability increase in old age. Further support comes from the convergence of this result with that of a previous study based on a single population (Kobyliansky & Livshits, 1989). Of course, longitudinal studies would be valuable to examine potentially confounding cohort effects.

Our results also show that childhood intelligence does not predict old-age FA. This suggests that the life-long trait of cognitive ability (Deary et al., 2004) does not cause FA. In addition, there is no reason to believe that facial symmetry has a straightforward influence on intelligence differences or change. Thus, a common cause explanation is more plausible than any alternative explanation based on direct causation.

The common cause hypothesis proposes an age-related relationship between bodily condition and cognitive functioning (Lindenberger & Baltes, 1994). Whereas the developmental stability model, on the other hand, is usually used to explain individual differences in phenotypic condition (Polak, 2003), the explicit developmental rationale underlying developmental stability makes it as much a model of development and change as it is a model of individual differences at any given time: While developmental stability is often equated with genetic fitness in younger samples, it does in fact reflect both genetic fitness and successful somatic maintenance, with the latter being a life history trait in which individuals invest to varying degrees. Over the lifetime, an allostatic load of pathogenic and environmental insults, as well as accumulated stochastic events accumulate and continuously challenge the maintenance of developmental stability by somatic repair processes (Finch & Kirkwood, 2000; McEwen, 2006; Seeman et al., 2001), which highlights individual differences in investments to somatic maintenance in old age. As a consequence, DI in old age should reflect investment in maintenance much more
than genetic fitness, especially when compared to younger samples. FA, information processing efficiency, and old-age cognitive decline might all be indicators of this common cause — terminal deterioration of developmental stability and general bodily system integrity (Deary & Der, 2005; Wilson, Beckett, Bienias, Evans, & Bennett, 2003). Initial support for this idea comes from the cohort-level observation of increased levels of FA in older populations that was observed by us and by Kobyliansky and Livshits (1989), and from the individual-level relationship of FA to cognitive decline also shown in the present report.

While we found a link between old-age FA and both concurrent general intelligence and cognitive decline, FA at age 83 years was unrelated to intelligence at earlier ages. Links between FA and intelligence in young age are usually interpreted as being caused by an underlying genetic fitness factor of which both are indicators (Miller, 2000; Penke et al., 2007). Our failure to find a link between FA in old age and intelligence at any age suggests that this explanation no longer holds later in life. Given that DI reflects both genetic fitness and somatic maintenance investment, with the latter becoming more important in old age, our results might mean that the FA-intelligence link that is caused by genetic fitness in younger samples is masked by superimposed somatic maintenance effects that affects DI in the elderly. The result that cognitive decline, but not absolute intelligence levels, are associated with FA in old age is in line with this interpretation, since individuals’ cognitive functions decline from different “baselines,” their “cognitive reserves” (which basically reflect their early-life intelligence, see Whalley, Deary, Appleton, & Starr, 2004). If our interpretation is correct, then the cognitive reserve (childhood IQ) reflects mostly genetic fitness; old-age cognitive decline and old-age FA reflect mostly investment in somatic maintenance; and absolute old-age intelligence levels reflect a mixture of both. Of course, FA also starts from a young-age baseline that is supposed to reflect mostly genetic fitness, but unlike young-age individual differences in intelligence, young-age differences in FA tend to be small (probably as a result of the life conditions in highly developed Western societies) so that these baseline differences in FA might be easily overwitten be the larger FA differences that occur in old age due to different investment in somatic maintenance. This interpretation is supported by the greater mean and variance of facial FA in the older compared to the younger sample. Taken together, our results suggest that mechanisms governing developmental stability and system integrity in the later phase of life might not be completely identical with those that support developmental stability during early growth. Unfortunately, too few empirical studies have addressed the lifespan development of DI and FA to allow for a strong conclusion.

All of the effects found in this study were limited to men. Indeed, some of the chronometric cognitive measures showed weak (and difficult to explain, thus probably spurious) effects in the opposite direction for women. Stronger results for men are in line with sex differences in the tradeoff between reproductive effort and somatic maintenance, but two additional effects might have contributed to them. First of all, cognitive decline has been shown to accelerate markedly in the last 3 to 6 years before death (Wilson et al., 2003). Since men die earlier than women, terminal cognitive decline has probably started to affect more men than women in our very age-homogeneous sample, causing the associations we found. Secondly, mitochondrial DNA (mtDNA), which plays a prominent role in the regulation of cell metabolism and which has been associated with aging and neurodegenerative diseases (Krishnan et al., 2007), is maternally inherited. This implies that mtDNA has evolved to serve the metabolic requirements of female bodies. However, the sexes differ in their basal metabolic rates, which could make mtDNA a causal factor in the faster senescence of male brain and body functions (Zeh & Zeh, 2005).

The links between FA and cognitive functioning in the current study were not mediated by health conditions or physical fitness. This might be surprising, since a link between facial symmetry and health conditions could be theoretically predicted and is found in some studies (e.g., Thornhill & Gangestad, 2006). The overall evidence, however, is rather inconsistent (reviewed in Rhodes & Simmons, 2007). FA, reaction times, and cognitive decline might be more sensitive to system integrity senescence in old age than are diagnosed major health issues or physical robustness. FA is thus an interesting novel risk factor for cognitive decline, and potentially also for death. Future studies should aim to evaluate its potential in this regard.

Among the strengths of this study are the narrow age range of the sample, which removes age as a confounding factor (Hofer & Šliwinski, 2001), and the assessment of cognitive ability at three times over a period of more than 70 years, which allowed us to compare relationships of FA with lifetime and within-old-age cognitive change. Certainly the biggest limitation of our study is that FA was only assessed once, which allowed us to provide only limited insight into the causal relationships and correlated changes between FA and cognitive ability (but see the causal considerations above). Another limitation is that individuals who survived to age 83 are already pre-selected in terms of overall phenotypic condition. However, this limited variance made the relationships we looked for statistically harder to detect, thus working against our hypothesis. Similarly, the cognitive change we observed between age 79 and 83 is likely of limited extent due to the relatively brief test-retest interval. Thus, the associations with FA that we report here might well be understimates.

The current study is the first to use facial measures of FA in relation to cognitive functioning. While it is generally assumed that facial and bodily FA tap into the same latent construct of DI, using facial symmetry could be an advantage over earlier studies: the intimate link between the face and the brain during development (Hammond et al., 2008)
renders facial symmetry an especially good candidate for predicting cognitive functioning. However, the pattern of our results, which were consistently stronger after symmetry measures were controlled for DA, support Simmons et al.’s (2004) conclusion that controlling for DA is important in studies of facial FA. In addition, landmark-based facial symmetry measures from 2D photographs are a somewhat crude and error-prone technique. Novel 3D photogrammetric techniques will likely be able to capture facial symmetry much more precisely than the techniques that have been used so far (Hammond et al., 2008), as has recently been demonstrated for body FA in a study using a full-body 3D scanner (Brown et al., 2008). Replications of our results with these new techniques for facial and body FA assessments would be desirable.

Facial FA was a significant predictor of cognitive decline and information processing efficiency in old age in men. These results support the common cause hypothesis of cognitive aging and link it to the theoretically richer developmental stability model (Polak, 2003) and more broadly to life-history theories of longevity and senescence (Kaplan et al., 2003; Kirkwood, 2008), which could turn out to be fruitful theoretical addition to gerontology. They also suggest FA as a candidate marker for this common cause, and as a new risk factor for cognitive aging and possibly death.

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