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**Neocortical age and fluid ability: Greater accelerated brain aging for thickness,
but smaller for surface area, in high cognitive ability individuals**

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Abstract

Biological (BA) and chronological (CA) age may or may not fit. The available evidence reveals remarkable individual differences in the overlap/mismatch between BA and CA. Increased mismatch can be interpreted as delayed ($BA/CA < 1$) or accelerated biological aging ($BA/CA > 1$). Body and brain health are correlated and both predict aging outcomes associated with physical and mental fitness. Moreover, research has shown that older brain age at midlife correlates negatively with cognitive ability measured in early childhood, which suggests early life predisposition to accelerated aging in adulthood. Under this framework, here we test if increased cognitive ability is associated with delayed brain aging, analyzing structural MRI data of 188 individuals, sixty of whom were recruited from MENSA, an association comprising individuals who obtained cognitive ability scores in the top 2 percent of the population. These high ability individuals (HCA) showed an average advantage of 33 IQ points, on a fluid reasoning test they completed for this research, over those other recruited because of their average cognitive ability (ACA). Next, brain age was computed at the individual level for two distinguishable neocortical features (thickness and surface area) according to models trained in an independent large-scale sample of 2,377 individuals. Results revealed a stronger pattern of accelerated brain aging in HCA compared to ACA individuals for thickness, while the opposite pattern was suggested for surface area. The findings align well with the greater relevance of individual differences in cortical surface area for enhancing our understanding of cognitive differences at the brain level.

Keywords. Brain age, Cognitive ability, Structural MRI, Cortical thickness, Cortical surface area

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INTRODUCTION

At the beginning of the XX century, Alfred Binet designed a standardized method for estimating the mental age of elementary school students in France (Binet & Simon, 1905). The idea behind the method was quite straightforward: (a) choose a set of tests requiring varied cognitive challenges involving high-level perception, memory, and language comprehension, (b) estimate the cognitive complexity that students of a given chronological age can handle successfully, (c) find out which students of a given chronological age fail to pass the complexity level expected from their chronological age or can complete tests designed for older students, and (d) finally, compute the overlap/mismatch between chronological and mental age to know if the student shows standard, delayed or accelerated cognitive development. This led to the estimation of the mental competence at which the individual operates. This first approach was the beginning of the multimillionaire and international psychological testing business (Detterman, 2014; Haier & Colom, 2021; Hunt, 2011; Jensen, 1998; Sternberg, 2020).

Binet's perspective suffered the obvious limitation that cognitive development stops in late adolescence (Steinberg et al., 2009, Steinberg, 2014) whereas chronological age keeps an inexorable upward trend. Therefore, alternative approaches were required for estimating a quotient not limited by the aging issue. IQ scores must reflect individuals' relative standing within their respective reference group, including their age.

Nevertheless, the main idea remained unaltered: people with comparable sociodemographic factors are expected to show a given average cognitive level and they can be ordered according to their distance from this average value. The raw score (P) obtained by a person on a given standardized test based on the number of correct answers to problems of increased cognitive complexity is usually compared with the

mean score (M) obtained by members of the proper comparison group ($P - M$). The result is divided by the standard deviation (SD) of the scores awarded by the complete group for obtaining a z score. This standardized score will be 0 when the person obtains precisely the mean value, greater than 0 if the person obtains higher values, and smaller than 0 if the person obtains lower values. In the final step, IQ scores can be computed using this simple formula: $z * 15 + 100$ (Hunt, 2011; Jensen, 1980).

Life span research studies allow making analyses regarding how chronological, cognitive, and biological development changes across time. In this regard, the meta-analysis by Tucker-Drob et al. (2014) considered fifteen genetically informative longitudinal samples with a wide age range (0.5 to 77 years), although the age distribution was mainly concentrated in childhood and adolescence. These were the key conclusions derived from their analyses: (a) the stability of cognition increases from infancy to late adolescence reaching a top value of 0.80; (b) genetic and nonshared environmental factors account for this increased stability with estimated values of 75% and 20%, respectively, (c) stability values become smaller with increased time gaps between cognitive measurements, (d) not all cognitive abilities show the same stability values; crystallized/cultural abilities show smaller values than fluid/abstract abilities. One important message delivered by this meta-analysis is the “little empirical attention placed on cognitive changes in young and middle adulthood.” (p. 969).

Regarding the later message, the meta-analysis by Tucker-Drob et al. (2019) identified a general factor of aging-related change in adulthood. Analyzing 22 datasets including individuals from age 35.4 to age 85 they found that individual differences in longitudinal changes in a set of varied cognitive abilities are remarkably correlated,

meaning that people showing decline in a given ability also show decline in the other abilities: “cognitive decline may operate along a similar general dimension as does cognitive development.” (p. 21). Age changes in cognitive ability are orchestrated.

The increase and decline of cognitive abilities might be related with brain features (Estrada et al., 2019; Karama et al., 2013; Román et al., 2018). In this regard, Elliott et al.'s (2019) study reported that brain age in midlife is associated with accelerated biological aging and cognitive decline. They defined brain age as the mismatch between chronological age and age predicted from models obtained from brain structural imaging data. After analyzing 869 members from the Dunedin Longitudinal Study at age 45, the findings revealed brain age values showing an amazing range going from 24 to 72 years. Moreover, individuals showing older brain age values at age 45 had poor cognitive ability at this same age and in their childhood, four decades before.

Summarizing the accumulated evidence in this regard, Belsky et al. (2020) wrote: “even by midlife there is evidence of individual differences in the pace of aging, with this difference beginning in the mid-twenties, if not earlier (...) biologically older thirty-eight-year-olds experience greater decline than biologically younger study members in cognitive functioning from age seven to age thirty-eight” (pp. 368-365).

Elliott et al. (2019) considered participants' MRI images for computing cortical thickness and cortical surface area values at the individual level. Using these outputs, brain age scores were estimated using an algorithm trained with 2,354 individuals (age range from 19 to 82 years) (Liem et al., 2017). The estimates were aimed to quantify the degree of deviation from typical development of brain structure according to a model trained on brain imaging data obtained from large samples covering a wide age range.

The resulting model can be applied for estimating the brain age of individuals according simply to their brain-imaging data. Of course, deviation from expectations can be positive (delayed biological aging) or negative (accelerated biological aging).

Although the algorithm developed by Liem et al. (2017) was designed for processing brain anatomy and functional connectivity, Elliott et al. (2019) focused on anatomy, which seems reasonable because functional data were not key for enhancing brain age estimates' accuracy (half a year of prediction accuracy). Also, while Liem et al. (2017) kept cortical thickness and surface area values separated (which allowed identifying greater sensitivity of thickness to aging), Elliott et al. (2019) combined both cortical features in a single score, even when available best evidence supports their remarkable independence (Colom et al., 2013; Escorial et al., 2015; Privado et al., 2017; Vuoksima et al., 2015). Therefore, here we follow the recommendation when doing research aimed at addressing cognitive ability differences and we keep apart these two brain features.

The enumerated findings suggest that individuals showing delayed brain aging might have higher cognitive ability than their peers with accelerated brain aging values.

Here we test if this holds, analyzing one hundred and eighty-eight individuals ranging in chronological age from 18 to 49 years. These individuals completed the same fluid reasoning test and were respectively identified as high cognitive ability (HCA) and average cognitive ability (ACA). We will estimate the brain age of our individuals using an updated version of Liem et al.'s (2017) algorithm. The brain features of interest (cortical thickness and cortical surface area) will be quantified using FreeSurfer, as detailed below. In line with the rationale behind Elliott et al.'s (2019) research, the key prediction is that HCA individuals will show delayed brain aging with respect to ACA

individuals. The rationale suggests that their greater cognitive ability scores reflect, at least in part, better brain integrity and that their higher integrity values would be translated into delayed brain aging.

METHODS

Participants

Sixty high cognitive ability (HCA) individuals were recruited from MENSA, an association that comprises high IQ individuals around the world. Approx. 1,750 of their members live in Spain. Becoming a member requires passing a complex test designed for screening the top 2% of the general population (98th percentile on a standard test of general cognitive ability). Also, (a) one hundred and twenty average cognitive ability (ACA) individuals were carefully selected from the Human Connectome Project (HCP) database (<http://www.humanconnectomeproject.org>) and (b) nine ACA individuals were selected because of their comparable sociodemographic features to those of the recruited HCA individuals –these were scanned with the same MRI scanner. The 189 individuals completed the same general reasoning ability test: the 24-item Penn Matrix Reasoning Test (PMAT-24) (Bilker et al., 2012; Williams & McCord, 2006).

One ACA participant from the HCP database failed to pass the image processing algorithm and, therefore, was removed from the sample. Thus, the finally analyzed sample included 188 individuals (128 ACA and 60 HCA –MENSA—individuals). HCA were older on average (mean age = 34.1; SD = 6.8) than ACA individuals (mean age = 29.9, SD = 3.5) [$t(185) = 4.5; p < .001; d = 0.89$] and they also showed much higher values on the fluid reasoning test [$t(185) = -19.2; p < .001; d = 2.3$]

Fluid reasoning test

All participants completed the Penn Matrix Reasoning Test (PMAT-24) a computerized test included in the Penn Computerized Neurocognitive Battery (Dubois et al., 2018; Moore et al., 2015). This test comprises problems based on spatial and numerical relationships ordered by increased cognitive complexity. These problems are based on patterns made up of 3x3, 2x2, or 1x5 arrangements of squares, of which one is missing. Examinees must select the one, among five alternatives, that best fits the missing square. The items were built using the theoretical framework previously applied for designing widely known tests such as the Raven's Progressive Matrices Test and the Matrix Reasoning subscale from the WAIS-III (Haier & Colom, 2021).

MRI acquisition

Neuroimaging data of the 60 HCA and nine ACA individuals were acquired in a 3T Siemens Magnetom Prisma at Ruber Internacional Hospital (Madrid). MRI images of the remaining 120 ACA individuals were retrieved from the Human Connectome Project (HCP) public database. HCP MRI registrations were done also using a Siemens 3T magnet and the Madrid's registrations strictly adhered to recommendations for adapting the HCP protocol to other scanners. The exact match between every detail of the registration parameters in both sites were carefully checked and approved by technicians from the Siemens company (Santonja et al., 2021).

The MRI images of interest for the present study contained data from the structural session (T1-weighted image and T2-weighted image). The acquisition parameters for the sixty HCA and nine ACA individuals were: T1-weighted images: 0.8 mm isotropic voxel size, a 220 Hz/Px bandwidth, TR = 2400 ms. and TE = 2.22 ms; T2-weighted

images: 0.8 mm isotropic voxel, a 744 Hz/Px bandwidth, TR = 3200 ms. and TE = 563 ms. The acquisition parameters for the 120 ACA individuals from the HCP were: T1-weighted images: 0.7 mm isotropic voxel, 210 Hz/Px bandwidth, TR = 2400 ms. and TE = 2.14 ms; T2-weighted images: 0.7 mm isotropic voxel, 744 Hz/Px bandwidth, TR = 3200 ms. and a TE = 565 ms.

Image processing

Structural images were processed with the Freesurfer software suite (version 5.3) (Desikan et al., 2006; Fischl et al., 2004). The T1-weighted images were segmented into cerebrospinal fluid (CSF), white matter, and gray matter. These segmentations were later used in the creation of a mesh defined by the boundaries between gray matter and white matter (in the case of white surfaces) or by the boundaries between gray matter and CSF (for the pial surfaces). By performing geometrical operations on these surfaces, cortical surface area (CSA) and cortical thickness (CT) values were obtained for each vertex of the pial surface of each participant (Figure 1).

Figure 1 about here

Computation of brain age values

Brain age scores were generated using a publicly available algorithm called BARACUS version 1.1.2 (10.5281/zenodo.1018841), developed by Liem et al. (2017). The software relies on the results of the Freesurfer processing to estimate brain age scores. Native surface models were registered to Freesurfer's fsaverage4 space, and the registered

cortical thickness, cortical surface area and subcortical volume results were used as inputs to the trained model. The model that BARACUS uses was trained on vertex-wise data of cortical data from a two-sample model in which individuals had no objective cognitive impairment, with an age range of 19-82 yrs. and a sample size of $N = 2,377$. The model included a first-level implementation of support vector regression models to predict age from a single morphometric variable, and a second-level random forest model that stacked the first-level predictions, to aggregate information from multiple morphometric variables into one single final prediction. BARACUS was chosen for its optimal performance in dealing with independent samples (Elliott et al., 2019).

The output of BARACUS yields several values per model of predicted age for each individual. These values correspond to (a) aseg, obtained using data from subcortical volumes, (b) thickness, obtained using data from cortical thickness values, (c) area, obtained using data from cortical surface area values, and (d) anatomy, obtained using a stacked vector of subcortical volumes, cortical thickness, and surface area.

Nevertheless, as noted above, here we focus on cortical thickness and surface area values separately. We also note that, when using the BARACUS algorithm, the weights assigned by the model to each morphometric variable, at the vertex level or at the region level, are not given as an output. Therefore, only the brain age predictions obtained from global values are provided in the results of the software and, unfortunately, it is not within our means to obtain regional information regarding brain age prediction.

Statistical analyses

First, descriptive statistics (mean, standard deviation, Kolmogorov-Smirnov test for testing the normality of the distribution, and Levene's test to check the assumption of

equal variances in both groups) were computed regarding brain age estimates of the neocortical features of interest (cortical thickness and cortical surface area). Second, quotients were calculated using these brain age estimates and chronological age values. Brain age values were divided by chronological age values. This quotient is intended to quantify delayed or accelerated aging: 1 means perfect match, values greater than 1 express accelerated aging, and values lower than 1 reflect delayed aging. The resulting quotients will be used for knowing how they are distributed in HCA and ACA individuals applying two approaches: (a) general density distribution of quotient values for both individuals and (b) percent of individuals within each group (HCA versus ACA) across four percentile bands derived from the computed quotients (<25, 25-50, 51-75, >75). The mean differences between groups were explored using Student's t-test using a fixed-effect model; [Cohen's \$d\$ and partial eta squared \(\$\eta^2_p\$ \)](#) values are used as measures of effect size. Finally, we correlated PMAT-24 scores with the obtained quotients at the individual level using the Spearman rank-order correlation (r_s).

RESULTS

The descriptive statistics regarding brain age estimates for cortical thickness and cortical surface area provided by BARACUS are shown in Table 1. t and p values, along with the effect sizes (d and η^2_p) are also included.

[Table 1 about here](#)

Consistent with their average difference in chronological age, HCA and ACA individuals were significantly different in estimated brain age values. Nevertheless, it

must be underscored that the difference was four times smaller for cortical surface area ($d = 0.32$) than for cortical thickness ($d = 1.21$). Also, their average difference was three times smaller for cortical surface area ($d = 0.32$) than for chronological age ($d = 0.89$).

The Spearman correlations among chronological age, brain age CT (cortical thickness) estimates, brain age CSA (cortical surface area) estimates, and PMAT scores are shown in the Appendix (Table A.1 and Figure A.1). PMAT scores were unrelated with the other three variables in both ACA and HCA individuals. Moreover (a) brain age CT and brain age CSA estimates were correlated in the ACA group ($r_s = .321, p < .001$), and (b) chronological age and brain age CT (cortical thickness) estimates were correlated in the HCA group ($r_s = .490, p < .001$).

Table 2 shows the results of main interest: the neocortical age / chronological age quotients for HCA and ACA individuals, obtained from cortical thickness or cortical surface area age values, along with t_{186}, p and d and η^2_p values. Interestingly, the average standardized difference (d) regarding cortical thickness was positive (greater values for HCA individuals, $d = 0.41$) whereas for cortical surface area it was negative (greater values for ACA individuals, $d = -0.21$). The trends are depicted in Figures 2 and 3.

Table 2 about here

Figure 2 displays density plots of these quotients in HCA and ACA individuals, whereas Figure 3 shows the percentage of HCA and ACA individuals within four percentile bands (<25, 25-50, 51-75, >75) with increased quotient values for CT and

CSA. The pattern clearly indicates greater percentages of HCA individuals for thickness, but smaller percentages of HCA individuals for surface area. Therefore, both neocortical features behave remarkably different depending on cognitive status.

Figure 2 about here

Figure 3 about here

Finally, we computed the Spearman rank-order correlation (r_s) between scores obtained in the fluid reasoning ability test (PMAT24) by HCA and ACA individuals, and the obtained quotient values for cortical thickness (CT) and cortical surface area (CSA). Scatterplots with results at the individual level are depicted in Figure 4.

Figure 4 about here

Because HCA individuals show huge restriction of range regarding their PMAT24 scores, correlations were computed separately for ACA and HCA individuals. Correlation values for ACA individuals were $r_s = -0.18$ ($p = 0.04$) [CI = -0.34, -0.01] (cortical thickness) and $r_s = -0.14$ ($p = 0.11$) [CI = -0.31, 0.03] (cortical surface area),

whereas correlation values for HCA individuals were $r_s = -0.03$ ($p = .844$) [CI = -0.28, 0.23] (cortical thickness) and $r_s = -0.02$ ($p = 0.86$) [CI = -0.27, 0.24] (cortical surface area). Therefore, increased fluid ability scores were significantly associated with reduced thickness quotients (delayed brain age) in ACA individuals. Although this same trend was observed for surface area, statistical significance was absent.

DISCUSSION

Based on previous evidence (Elliott et al., 2019), here we made one straightforward prediction: high cognitive ability individuals (HCA) will show delayed brain aging when compared with average cognitive ability individuals (ACA). We inspected this prediction by computing quotient values based on their neocortical and chronological age data. The resulting summary scores were interpreted as delayed (values below 1), expected (value of 1), or accelerated (values above 1) brain aging.

The findings rejected the prediction for cortical thickness but were consistent for cortical surface area. Therefore, these neocortical features behave quite differently depending on the cognitive status of our participants. The average standardized difference (d) separating HCA and ACA individuals for chronological age was 0.89, whereas the d value for the thickness quotient was reduced by half (0.41) and for the surface area quotient was favorable to the high cognitive ability individuals (-0.21). This descriptive pattern was displayed in Figures 2 (density plots) and 3 (percentile bands). There was considerable overlap between the distributions of both individuals, but the percent of individuals showing accelerated brain aging across percentile bands increased for cortical thickness, and decreased for cortical surface area, in high ability individuals. The opposite pattern was true for average cognitive ability individuals.

In the final stage of our analyses, we computed the correlation values relating scores on the administered fluid ability test (PMAT-24) and quotients for thickness and surface area separately for high and average cognitive ability individuals (Figure 4). Because of the large restriction of range in ability for the former individuals, the computed correlation values were null. However, for average cognitive ability individuals, who showed a wide range of PMAT-24 scores, the resulting effect sizes (r) were close to those reported by Elliott et al. (2019). These researchers obtained a value of -0.20 when correlating brain age and cognitive ability assessed at age 45. The value was -0.18 when cognitive ability assessed in childhood was correlated with brain age estimated at age 45. Here, correlation values between cognitive ability and estimated brain aging were $r = -0.18$ for thickness and $r = -0.14$ for surface area. Therefore, higher ability scores were associated with delayed brain aging in average cognitive ability individuals.

Cortical surface area, cortical thickness, and cognitive ability

The role of the two neocortical features considered in the present research has been discussed extensively in the published literature addressing cognitive ability (intelligence) differences. In this regard, Vuoksima et al. (2015) revised the phenotypic and genotypic relationships between neocortical volume and cognitive ability finding greater informative relevance for cortical surface area than for cortical thickness. Neocortical gray matter volume results from the product of these two cortical geometrical features, but their genetic substrate and their development across the life span are remarkably distinguishable (Chen et al., 2013; Estrada et al., 2019; Rakic, 2009; Román et al., 2018). As it is well known, the number of cortical columns defines surface area, while the number of neurons within a given column defines thickness

(Burgaleta et al., 2014, Colom et al., 2013, van der Meer et al., 2020, Vuoksimaa et al., 2015).

After reviewing the available evidence (Burgaleta et al., 2014; Colom et al., 2013; Fjell et al., 2015; Karama et al., 2013), Vuoksimaa et al. (2015) held that individual differences in surface area might be much more important to cognitive ability differences than variations in cortical thickness. Their research considered 534 middle-aged war veterans from the VETSA (Vietnam Era Twin Study of Aging) and their findings revealed that the correlation between cortical volume and cognitive ability was mainly driven by cortical surface area, which was properly seen as consistent with Colom et al. (2013) and Fjell et al. (2015). The contribution of cortical thickness was negligible, a result in tension with previous reports finding a substantial role for thickness only (Karama et al., 2011).

The report by Fjell et al. (2015) demonstrated that brain regions showing correlations with reasoning ability match regions showing higher expansion both during human evolution and human development: “cortical regions involved in higher intellectual functions have expanded the most during development and evolution.” They also showed that surface area was the relevant cortical feature, stating that the radial unit hypothesis (Rakic, 2009) might account for the identified evidence. Surface area expansion during evolution can occur without appreciable thickness increases. As noted by (White et al., 2010) expansion in surface area and greater gyrification might enhance brain connectivity and these processes hardly require increased thickness. After studying 1048 individuals, ranging in age from 8 to 89 years, Fjell et al. (2015) found weak, albeit significant, correlations across brain lobes between cortical surface area

and cognitive ability. The radial unit hypothesis suggests that more cortical columns involve greater numbers of neurons and, therefore, increase computing capacity.

In this latter regard, Goriounova et al. (2018) research found that individuals with higher cognitive ability can keep fast action potentials for longer periods of time. These individuals show larger and more complex neurons. Moreover, they display faster action potentials and efficient synaptic communications. These researchers attribute a key role to pyramidal cells devoted to multimodal integration: “cells have increasingly larger dendrites in regions involved in higher-order cortical processing (...) human neurons of individuals with high cognitive ability are able to translate inputs into action potentials much more efficiently, transfer more information and sustain fast action potential firing compared to individuals with lower cognitive ability.” (Goriounova & Mansvelder, 2019).

In brief, brain computation power might support, at least in part, the cognitive strength reliably estimated by standardized measurements of ability. Individuals showing greater computation capacity, as quantified by cortical surface area values, might also show higher cognitive ability levels. The high cognitive ability individuals considered in the present research are older from a chronological perspective than the analyzed average cognitive ability individuals. However, the brains of the former are younger than expected when their cortical surface area values are considered. This pattern may contribute to support their greater cognitive ability, whereas their worse values for cortical thickness suggest that this cortical feature is much less relevant for cognition.

Why do individuals within the normal range of cognitive scores show delayed brain aging with increased fluid ability scores?

There are potential explanations, raised by differential and cognitive epidemiology, regarding the causes underlying the relationship between cognitive ability differences within the normal range and physical and mental health outcomes, including brain preservation or deterioration with age (Calvin et al., 2017; Caspi & Moffitt, 2018; Deary et al., 2010; Elliott et al., 2019; Gottfredson & Deary, 2004), that might be useful for interpreting our findings. The fact is clear: within the most usual range of cognitive ability scores, the higher the ability, the lower the quotient summarizing the relationship between brain and chronological age. However, the interpretation of this fact is far from clear.

The first explanation suggests that brain age estimates reflect delayed or accelerated aging. Across the lifespan, individuals diverge because of their differences in genetic makeup, environmental circumstances, and lifestyle factors. Aging is associated with progressive degradation across body organs, including the brain. However, there are wide individual differences in these aging effects. Those with higher ability values may age slower than those with lower ability values because they have ‘better’ genomes, inhabit high quality environments, and adhere to healthy lifestyles. Of course, these factors may be correlated: “the stability of heritable influences on age-related outcomes, like cognitive and physical ability, are a consequence, in part, of the individual’s ability to create lifestyles that complement and reinforce their underlying genetically influenced talents.” (McGue et al., 2014).

The second explanation suggests that individuals differ in general health from the very beginning of their lives, even since the time of conception. Brain age and cognitive ability measurements may tap compromised lifelong health. Ability scores might reflect brain efficiency and bodily integrity more broadly. These two explanations are, of course, not mutually exclusive and it is difficult to disentangle the relevant data for supporting or rejecting either one of them.

The findings reported by Elliott et al. (2019) are one perfect example of the latter difficulty. Older brain age was associated with lower cognitive ability, assessed both in adulthood and in childhood, as well as with poor brain health at age 3. But they also found support for accelerated cognitive and biological aging, brain included: “consistent with the common cause hypothesis of aging, the brain is not exempt from the biological aging that causes a generalized deterioration of organ systems across the body.”

Unfortunately, Elliott et al. (2019) did not look at their surface area and thickness results separately. Instead, they analyzed one single value combining these two cortical features. However, we have seen that there are biological reasons to keep them apart and that they tell different stories when related with cognitive ability differences. The pattern is consistent for average cognitive ability individuals, irrespective of the considered neocortical feature (higher ability scores are associated with delayed surface and thickness aging). But the conclusion regarding the comparison between high and average cognitive ability individuals changes a lot for thickness and for surface area.

The hyper brain, hyper body model

In a previous study, we have reported greater resilience of high cognitive ability individuals against simulated attacks to their brain structural networks (Santonja et al., 2021). We speculated that this better resilience might be achieved at some cost, asking the next question: “is there a relationship between heightened cognitive ability and increased negative bodily signs?” High cognitive ability individuals may show greater risk for brain disfunctions of some sort. The suspicion was derived from the provocative model proposed by Karpinski et al. (2018) based on the assumption that high cognitive ability may be a risk factor for overexcitabilities, both at the biological and psychological levels. Their research found supporting evidence regarding physical and mental health, but data at the brain level were unavailable to them: “if these individuals take in their world in an overexcitable manner cognitively (hyper brain), then the potential exists for an intense level of physiological processing as well (hyper body).”

If indeed high functioning brains show wearing signs of some sort, these might be manifested by worst brain age / chronological age quotients. The findings reported here depart from Karpinski et al.'s (2018) prediction when cortical surface area values are considered, but they fit when looking at cortical thickness values. High ability individuals showed remarkably worst brain aging indices than average ability individuals for thickness, but this was not the case for cortical surface area. Importantly, we have seen that the latter neocortical feature is more critical than the former regarding cognitive ability differences. Therefore, this finding reiterates the recommendation of keeping these two brain structural features apart.

Limitations

We can identify at least three limitations in the present research. First, the strong restriction of range in cognitive ability found in the high ability individuals (HCA) precludes the detection of the pattern observed for the average cognitive ability individuals. There are documented reasons to think that high ability individuals are far from homogeneous (Lubinski & Benbow, 2021). Therefore, it would be interesting to check what results from a comparison of individuals showing ability scores within the 130-200 IQ range (Kell et al., 2013; Makel et al., 2016; McCabe et al., 2020). Secondly, as stated by Liem et al. (2017), studies considering white matter anatomy might provide increased refinement of brain aging estimates. The fact that we already found much better structural connectivity in high cognitive ability individuals (Santonja et al., 2021) supports this presumption and invites further research. Finally, it is always preferable to have varied measurements of cognitive ability (Gignac & Bates, 2017; Haier & Colom, 2021; Haier et al., 2009). Here we only had one measure of fluid reasoning ability, but we already know associations between cognitive ability and brain features become stronger and more reliable with comprehensive measurement batteries of cognition. Future research studies should take this cognitive ability measurement recommendation seriously, even at the expense of increasing testing time. This con will be compensated by the pro of a reduced requirement for recruiting big samples (Gignac & Bates, 2017).

In conclusion, following the long tradition starting with the French psychologist Alfred Binet, we computed quotients based on chronological and brain age values for estimating delayed or accelerated brain aging. Relying on previous research, we did predict that high cognitive ability individuals, scoring on average more than two standard deviations above a group comprised by average cognitive ability individuals,

will show generalized delayed brain aging. The results revealed a pattern of accelerated brain aging that was more pronounced for cortical thickness in high ability than in average ability individuals, while the opposite was observed for cortical surface area. Because available evidence supports greater relevance of variations in surface area than those observed in thickness for cognitive ability differences, these opposite patterns provide further support for the enhanced processing capacity derived from the increased numbers of cortical columns reflected by surface area measurements obtained from a neuroimaging approach (Vuoksima et al., 2015). Last, but not least, the recent large-scale genome-wide association study by van der Meer et al (2020) suggesting that cortical surface area is more heritable (less sensitive to environmental factors) than cortical thickness, may help to explain the differential pattern of results reported here regarding these cortical features in average and high cognitive ability individuals.

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TABLES

Table 1. Descriptive statistics for estimated brain age values in HCA (High Cognitive Ability) and ACA (Average Cognitive Ability) individuals. Chronological age data are also shown. KS = Kolmogorov-Smirnov test.

	HCA (N = 60)		ACA (N = 128)		KS (Z; <i>p</i>)	Levene's tets (<i>F</i> _{1,186} ; <i>p</i>)	<i>t</i> ₁₈₆ (<i>p</i>)	η^2_p	<i>d</i>
	Mean	SD (range)	Mean	SD (range)					
Chronological age	34.1	6.8 (18-49)	29.9	3.5 (22-36)	HCA: Z = 0.63; <i>p</i> = .828 ACA: Z = 1.29; <i>p</i> = .072	32.10 (<i>p</i> < .001)	4.5* (<i>p</i> < .001)	0.14	0.89
Brain age estimated: Thickness	42.83	6.53 (25.3-57.2)	34.83	6.72 (22.4-56.0)	HCA: Z = 0.54; <i>p</i> = .929 ACA: Z = 0.76; <i>p</i> = .611	0.00 (<i>p</i> = .999)	7.68 (<i>p</i> < .001)	0.24	1.21
Brain age estimated: Surface	44.72	7.06 (31.6-60.1)	42.29	8.13 (24.4-61.8)	HCA: Z = 0.63; <i>p</i> = .822 ACA: Z = 0.60; <i>p</i> = .868	1.50 (<i>p</i> = .223)	1.99 (<i>p</i> = .048)	0.02	0.32

* *t*_{74.35} due to non-equal variance data

Table 2. Brain age (BA) / chronological age (CA) quotients for HCA (High Cognitive Ability) and ACA (Average Cognitive Ability) individuals. KS = Kolmogorov-Smirnov test.

Quotient BA/CA	HCA (N = 60)		ACA (N = 128)		KS (Z; <i>p</i>)	Levene's tets (<i>F</i> _{1,186} ; <i>p</i>)	<i>t</i> ₁₈₆ (<i>p</i>)	η^2_p	<i>d</i>
	Mean	SD (range)	Mean	SD (range)					
Thickness	1.28	0.23 (0.92-1.98)	1.18	0.26 (0.71-2.05)	HCA: Z = 0.68; <i>p</i> = .741 ACA: Z = .81; <i>p</i> = .524	0.85 (<i>p</i> = .356)	2.66 (<i>p</i> = .008)	0.04	0.41
Surface	1.36	0.35 (0.72-2.62)	1.43	0.31 (0.74-2.32)	HCA: Z = 1.09; <i>p</i> = .185 ACA: Z = 0.59; <i>p</i> = .874	0.10 (<i>p</i> = .747)	-1.35 (<i>p</i> = .179)	0.01	-0.21

APPENDIX

Table A1. Spearman rank-order correlations (r_s) among chronological age, brain age CT (cortical thickness), brain age CSA (cortical surface area) and PMAT scores. Values for **ACA** (average cognitive ability) individuals (N = 128) above the diagonal and for **HCA** (high cognitive ability) individuals (N = 60) below the diagonal.

	Chronological age	Brain age CT	Brain age CSA	PMAT
Chronological age		.035 ($p = .693$)	.074 ($p = .404$)	.046 ($p = .604$)
Brain age CT	.490 ($p < .001$)		.321 ($p < .001$)	-.164 ($p = .065$)
Brain age CSA	.164 ($p = .212$)	.173 ($p = .186$)		-.105 ($p = .240$)
PMAT	-.115 ($p = .380$)	-.082 ($p = .534$)	-.188 ($p = .151$)	

Figure A1 about here

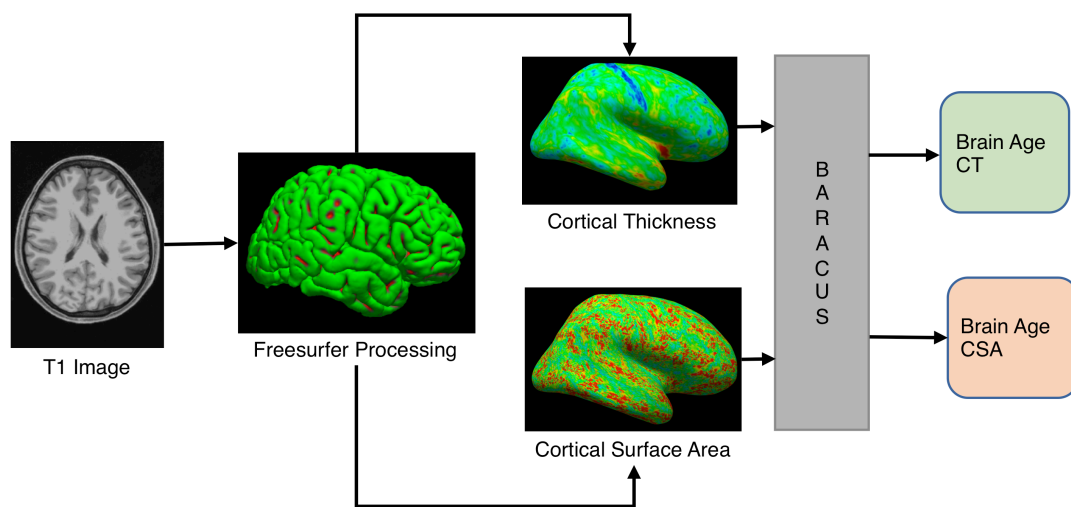


FIGURE 1

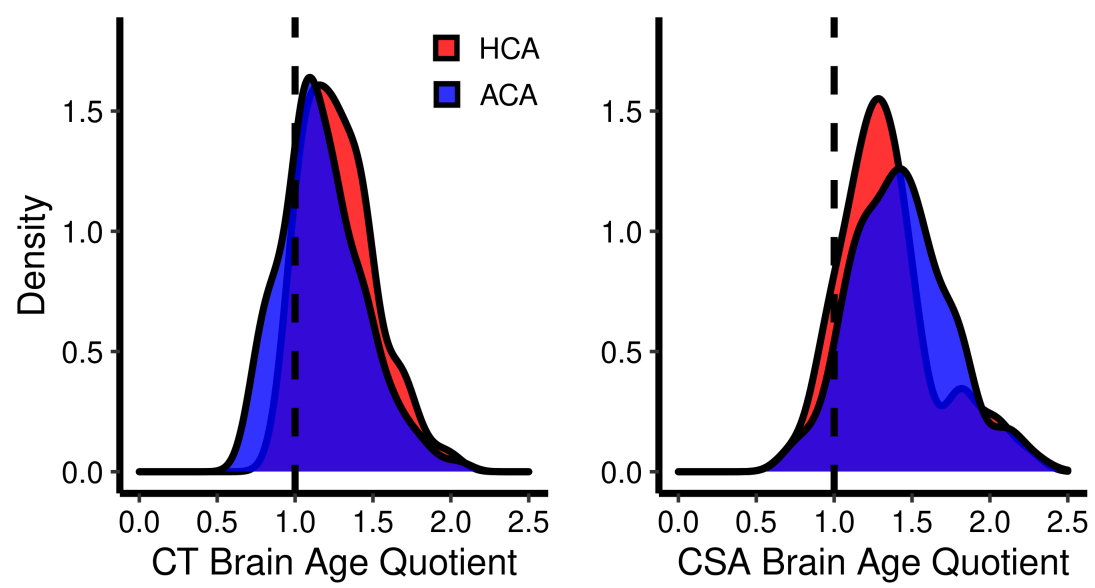


FIGURE 2

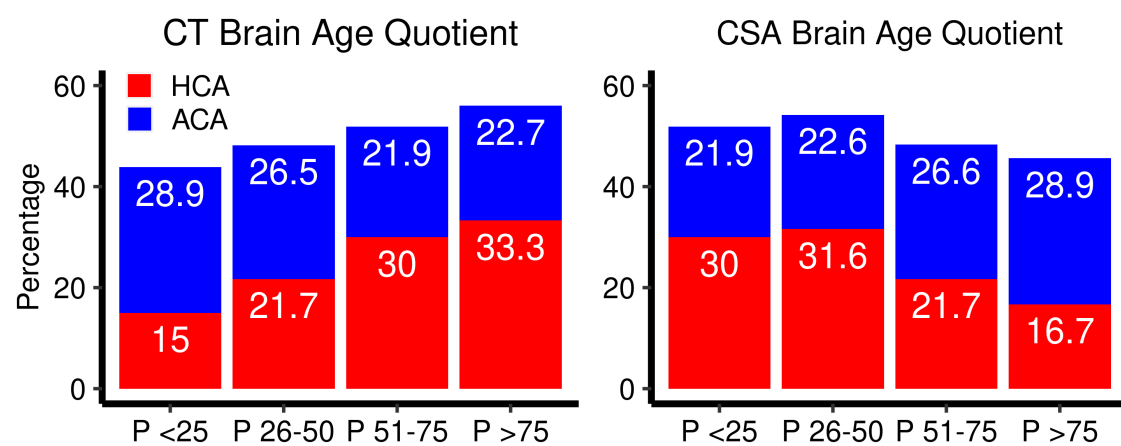


FIGURE 3

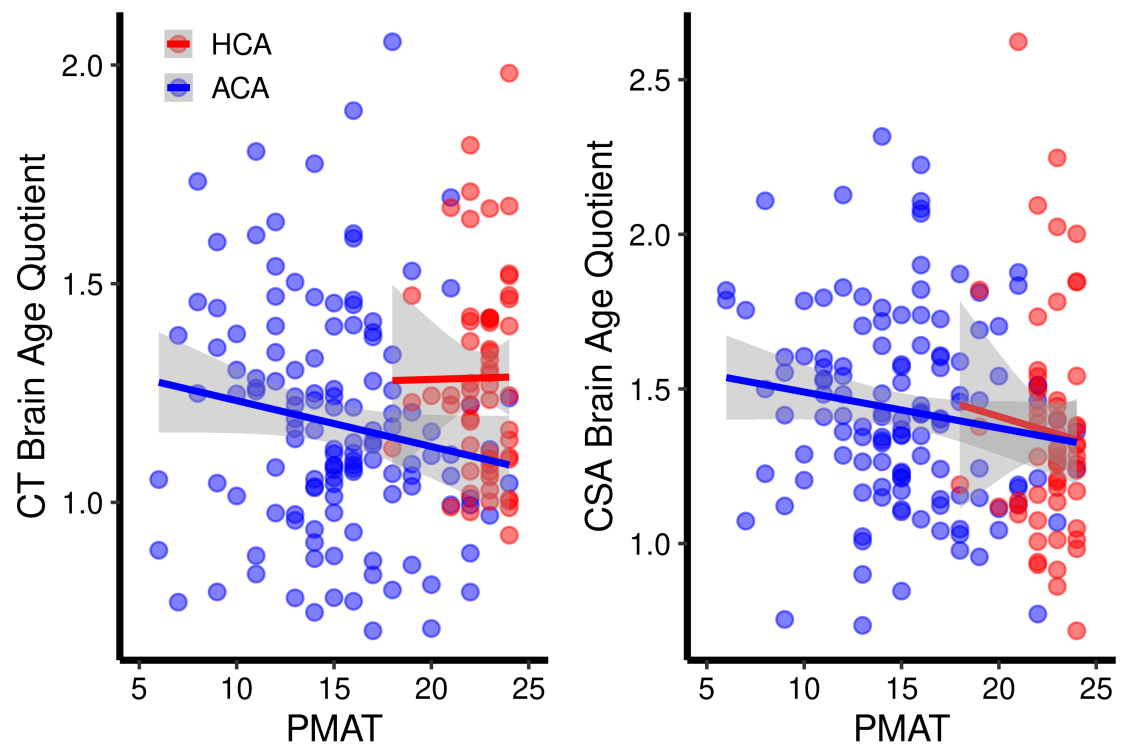


FIGURE 4

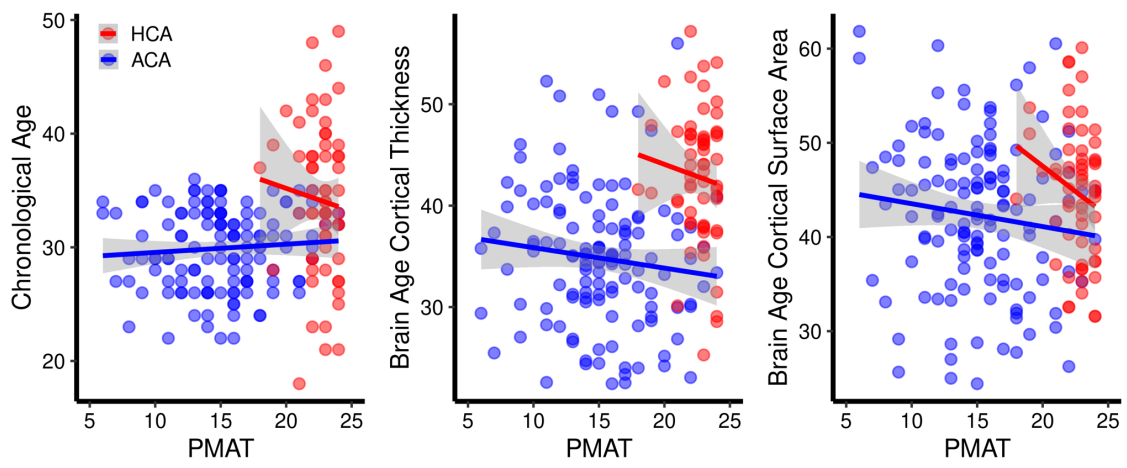


FIGURE A.1