Educational attainment does not influence brain aging

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Education has been related to various advantageous lifetime outcomes. Here, using longitudinal structural MRI data (4,422 observations), we tested the influential hypothesis that higher education translates into slower rates of brain aging. Cross-sectionally, education was modestly associated with regional cortical volume. However, despite marked mean atrophy in the cortex and hippocampus, education did not influence rates of change. The results were replicated across two independent samples. Our findings challenge the view that higher education slows brain aging.

Do higher levels of education attained in childhood and early adulthood slow the rate of brain and cognitive decline in later adulthood and old age? Prominent accounts of heterogeneity in neural and behavioral aging positulate that this is the case, arguing that education acts as a modifiable protective factor (1) or cognitive reserve (2, 3) of human neurocognitive aging. However, findings from cross-sectional studies provide only inconclusive support for an association between education and neurocognitive aging (4–7). Hippocampus atrophy in aging is well documented, and we found a marked age-related reduction in hippocampus volume with increasing age regardless of whether ICV was included as covariate (Fig. 1C). Crucially, rates of hippocampus volume change were not influenced by level of education (F = 1.51, P = 0.22; Fig. 1D).

Longitudinal analyses in LB (1,844 scans), revealed no significant relationship between education and vertex-wise volume change across the cortex. Similarly, when restricting the analysis to regions where volume loss was significantly larger with higher age (Fig. 1B), we found no support that higher education was related to less volume loss (Fig. 1C). This open access article is distributed under Creative Commons Attribution-NonCommercial-NoDerivatives License 4.0 (CC BY-NC-ND).


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We used hypothesis testing with Bayes factors (BF) to quantify the evidence in favor of the null hypothesis of no relation of education with longitudinal brain aging. Given similar patterns of results for the cortex and hippocampus in both samples, the Bayesian analysis was restricted to the computationally less demanding hippocampal region in LB where education was coded as a continuous variable. We used mean-zero Gaussian priors with an uninformative prior followed by a sensitivity analysis with an informative prior. Based on previous studies of the effect of education on brain aging (5, 10), the informed prior stated that hippocampal volume loss (about 50 mm³ per y, or 1%) would be slowed by around 0.5 mm³/y for each year of education. The uninformative prior’s SD was set at 10 times the main effect (SD = 500 mm³/y), and yielded a BF corresponding to very strong evidence in favor of the null (BF₀₁ = 1,170). The informative prior assigned a very large prior probability that the effect of interest was close to zero (SD = 0.5 mm³/y). The obtained BF₀₁ = 1.29 implies that the posterior probability is even more concentrated around zero than the informative prior, thus favoring the null hypothesis.

Finally, in both LB and UKB, cross-sectional analyses revealed modest associations of education with regional cortical volume around left central sulcus (Fig. 2D; LB: cluster extension, 5,298 mm²; cluster P values < 0.019–0.0002; UKB: 30,800 mm², P < 0.013–0.0002). Even in these cortical regions where education was related to intercept no relation was seen for slope (Fig. 2E). A large-scale (n = 19,646) cross-sectional UKB study reported a very small positive education–hippocampus volume association (12), but in the present smaller samples no significant cross-sectional associations were observed in LB or UKB between education and hippocampus volume.

Discussion

Taken together, the results from two large-scale datasets totaling almost 4,500 observations and over 2,000 individuals provided no support for the hypothesis that higher education translates into slower rates of brain aging. Instead, parallel rates of change were seen in cortical regions and in the hippocampus. It remains an open question whether other measures of brain aging than structural MRI are related to education levels.

Cross-sectionally, in both LB and UKB, education was modestly related to regional cortical volume, but even in the cortical regions where education was related to volume no relation was seen for rate of change. Thus, our brain-aging findings mimic those previously demonstrated for cognitive aging (8) by showing that education to some degree is related to level (intercept) but not rate of change (slope). A positive association between level of education and level of neurocognitive functioning has been reported in some past studies (5, 8, 12) (but see ref. 6), and is consistent with the notion of a “passive” reserve (13), which posits that individuals with higher education have an initial advantage over individuals with lower education that they may carry through their adult lives. It is this advantage, not attenuated longitudinal change, that reduces the risk among more educated individuals to be diagnosed with dementia and delays them reaching a threshold below which independent living is no longer possible. There is evidence for a genetic association of educational attainment with cortical surface area (14) and cognitive functions (15), indicating that shared genetic influences may account for cross-sectional relations of education with neurocognitive levels.

In conclusion, education is linked to many advantageous outcomes, but the present findings challenge theoretical and empirical claims that higher education slows brain aging.

Materials and Methods

MRIs were processed using FreeSurfer, version 7.1. All participants gave informed consent, subprojects were approved by the relevant ethical review board, and the Lifebrain project was approved by Regional Committees for Medical Research Ethics–South East Norway. Screening criteria were not identical across studies, but participants were recruited to be cognitively normal, and all were free from known neurodegenerative disorders.
healthy and did not suffer from neurological conditions known to affect brain function, such as dementia. All samples consisted of community-dwelling participants, some were convenience samples, whereas others were contacted on the basis of population registry information. Details on samples, MRI acquisition and processing, statistical analyses, and data and code availability are presented in SI Appendix.

Data Availability. The LB data supporting the results of the current study are available from the Principal Investigator of each substudy on request, given appropriate ethical and data protection approvals. UK Biobank data requests can be submitted to http://www.ukbiobank.ac.uk.

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