

Providing social support to others is associated with younger brain age in mid-to-late adulthood

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Abstract

Social connectedness is a robust predictor of health and longevity. While previous research has mainly focused on the benefits of receiving social support, the neuroprotective potential of providing social support to others remains underexplored. Using data from the Midlife in the United States (MIDUS) study, we tested whether total monthly hours spent providing instrumental and emotional support prospectively predict a brain age estimation gap (BrainAGE), a transdiagnostic biomarker of neurodegenerative disorders, psychiatric conditions, and mortality. BrainAGE was estimated with a machine-learning algorithm trained to predict chronological age from structural magnetic resonance imaging (MRI) scans. Combining hierarchical regression with bootstrapping and data-driven variable selection, we found that greater support provision was significantly associated with a younger-appearing brain for a given chronological age. This association persisted after controlling for a comprehensive set of covariates, including demographic characteristics, health conditions, lifestyle factors, and broader psychosocial contexts, including social support received. Notably, the hourly measure of support provision explained incremental variance in BrainAGE beyond role-based indices such as caregiving status or grandparenthood. Exploratory analyses further indicated that emotional support was more strongly associated with lower BrainAGE than instrumental assistance. Our findings highlight everyday prosocial behavior as a potential neuroprotective pathway in mid-to-late adulthood, with implications for intervention and policies for healthy aging.

Introduction

Decades of research have shown that individuals embedded in supportive social networks experience lower rates of chronic illness, improved stress regulation, and reduced mortality [1–3]. Yet, what specific behaviors within affiliative relationships account for these benefits? Most prior work has focused on the benefits of receiving support from others [4]. By contrast, the consequences of providing support have received far less empirical attention and are often presumed to be burdensome [5, 6].

While support provision can be demanding, nascent evidence suggests it may not be uniformly costly. The “Healthy Caregiver Hypothesis” posits that activities inherent in helping others can be conducive to psychological and cognitive functioning among caregivers [7, 8]. Consistent with this view, involvement in personal caregiving has been shown to correlate with greater well-being [9, 10], slower age-related cognitive decline [11], and a reduced mortality risk compared with non-caregivers, albeit with varying effect sizes [12–15]. Similar benefits have been

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identified outside caregiving contexts, with a range of “small acts of kindness” having modest yet significant positive effects on mood, sense of purpose, and mental health in young and older adults [16–20].

Do the benefits of helping extend beyond psychological well-being to influence brain health? If prosocial engagement is a key building block of healthy aging, its effects may also be reflected in aging-related changes in the brain. Aging in the human brain is accompanied by well-characterized reductions in gray matter volume and white matter integrity [21]. Brain age estimates, derived from machine-learning algorithms trained to predict chronological age from these neuroanatomical features, can provide a powerful window into whether individuals are on normative aging trajectories at the neural level [22, 23]. Critically, an older brain age relative to chronological age, or greater brain age estimation gap (BrainAGE), has emerged as a transdiagnostic biomarker of vulnerability across multiple neurological and psychiatric conditions: BrainAGE is associated with elevated risk for dementia, depression, and schizophrenia, as well as cardiometabolic dysregulation and mortality [24–28]. Establishing whether prosocial behaviors coincide with or lead to a lower BrainAGE can therefore provide unique insights into how helping behaviors get “under the skull” and may point to a modifiable, biologically grounded target for promoting healthy aging, with reduced vulnerability to mental and physical health decline [29, 30].

Despite this promise, evidence linking prosociality to brain age remains scarce. Several recent studies have suggested that parenting may have protective effects on brain structure and function [31], with the number of offspring associated with a younger brain age in both sexes [32]. Similarly, grandmothers actively involved in childcare or family caregivers for people living with dementia show lower BrainAGE than demographically matched controls [33]. These pioneering studies, however, relied on indirect proxies for prosocial engagement, such as parity counts or caregiver status. This approach obscures variation in the intensity and types of helping and provides only partial insight into how prosocial behaviors occurring outside caregiving or parenting relate to brain age. In addition, most prior work did not adequately account for factors known to influence both helping and biological aging, including socioeconomic background, health conditions, lifestyle, the size and quality of the relational network, and, importantly, social support received from others [34–38]. These limitations restrict the generalizability of existing findings and call for approaches that quantify the actual time and effort invested in support provision and incorporate systematic adjustments for potential confounding factors.

The goal of this study is to address these gaps and rigorously test whether everyday prosocial behaviors are associated with brain age across adulthood. We leveraged the Midlife in the United States (MIDUS) study [39], one of the largest studies on aging and well-being in the United States. MIDUS includes rich psychosocial, behavioral, and neuroimaging data from nationally representative samples, making it uniquely suited to our aim. Specifically, we examined whether individual differences in unpaid instrumental assistance and emotional support to others are associated with younger brain age, controlling for a wide array of covariates. We hypothesized that greater support provision would be associated with a younger estimated brain age for a given chronological age. Successful validation of this hypothesis would strengthen the case for a link between prosocial behavior and helpers’ brain health, paving the way for future studies with a mechanistic and translational focus.

Methods

Sample characteristics

We analyzed data from participants in the MIDUS 3 and MIDUS Refresher 1 cohorts [40, 41]. MIDUS 3 (M3) is the second longitudinal follow-up of the original MIDUS cohort, collected in 2013–2022, and includes adults aged approximately 40–95 years at the time of MRI scanning. MIDUS Refresher 1 (MR) is a cross-sectional cohort recruited in 2011–2016 to replenish the younger and middle-aged adults (aged 25–74). Data collection procedures were closely harmonized across the MR and M3 neuroscience projects. This provides analytic consistency not possible with the earlier MIDUS cohort, which employed different MRI acquisition protocols [42]. We focused on participants who completed the core surveys, biomarker assessment, and MRI scan. Only those without missing data

were selected, resulting in a final sample of 194 adults (mean age = 55.9 ± 13.4 ; 105 females; 148 white, 32 black, 14 others). This sample size is adequate, as determined by a priori power analysis (Supporting Information SI1). To characterize potential selection bias, we compared participants across inclusion stages, from the eligible core survey sample to the Neuroscience Project subsample and the final complete-case analytic sample used here (SI2). Notably, all non-MRI data, including measures of social support provision, were collected prior to the MRI session (Mean lag = 3.7 ± 2.6 years), providing a temporal separation between predictors and neural outcomes. All MIDUS study procedures and materials were approved by the University of Wisconsin-Madison Institutional Review Board (IRB) (Core survey: 2016-1051; Biomarker: 2014-0813; MRI: 2016-0054), and all participants provided written informed consent.

Key variables

Social support provision

We operationalized social support provision as the total monthly hours of unpaid assistance and emotional support given to spouses, children, relatives, friends, and anyone else (“On average, about how many hours per month do you spend providing unpaid assistance/emotional support to each of the following people?”). Participants were prompted to consider a broad range of daily activities, such as assisting with household tasks, transportation, or childcare; comforting others; listening to problems; or offering advice. In this way, we were able to capture everyday prosocial behaviors beyond formal caregiving roles and traditional parenting. Raw values for each measure were aggregated and standardized into a global index of support provision. To explore dissociable effects of unpaid instrumental assistance and emotional support, we also calculated aggregate scores for each type of help separately (See Analysis). Descriptive statistics on social support provision by recipient type are summarized in SI3.

Covariates

We controlled for a comprehensive set of variables known to be associated with biological aging, longevity, and prosocial engagement. These include demographic characteristics (i.e., age at MRI scan, sex) [43], health conditions (i.e., body mass index, total number of chronic conditions, depressive symptoms) [33, 44], lifestyle (i.e., frequency of exercise, drinking, smoking, and cognitively engaging activities) [34, 45, 46], and broader psychosocial contexts (i.e., income, education level, social network size) [47–49]. Education and income levels were standardized and combined into a composite SES score. Exploratory analyses assessing the effect of these variables separately are reported in SI4. Importantly, we also directly controlled for total weekly hours of unpaid assistance and emotional support *received* from others, perceived relationship quality with others (i.e., Positive Relationship with Others subscale of the Ryff Scale of Psychological Well-Being) [50], and subjective stress (i.e., Perceived Stress Scale) [51]. When needed, data were recoded so that higher values corresponded to greater magnitude. The list of all covariates and the rationale for their inclusion are described in SI5. We note that some covariates, such as depressive symptoms or perceived stress [19, 52], may lie within the mechanistic pathway linking support provision and BrainAGE, rather than acting solely as confounders. Since the present sample size is not optimal for estimating such indirect effects [53, 54], we treated them as covariates in the focal behavior-brain relationship rather than as mediators. Three additional nuisance variables related to sample characteristics and data collection procedures (i.e., race, MIDUS cohort, and the time lag between the core survey and the MRI scan) were tested and found to have negligible effects on model estimates (SI5–SI8).

Structural MRI acquisition and pre-processing

High-resolution T1-weighted anatomical images (1 mm^3 isotropic) were acquired using a 3 T scanner (MR750, GE Healthcare, Waukesha, WI) with repetition time = 8.2 milliseconds (ms), echo time = 3.2 ms, flip angle = 12° ,

Table 1 Descriptive statistics of the key variables.

Variable	Mean	SD	Min	Max
Age	55.91	13.41	27	95
Predicted Brain Age	51.67	12.97	22.03	79.59
BrainAGE	−4.24	7.23	−21.04	21.37
Support provision	69.73	71.09	0	311
Total # of chronic conditions	3.15	2.51	0	13
Body Mass Index	28.87	5.39	18.29	43.6
Smoking (Year)	7.27	12.37	0	45
Drinking (Year)	7.08	10.43	0	60
Exercise frequency (Weekly)	5.01	4.78	0	22
Cognitive activities	17.73	8.17	0	31
Depression	8.09	7.06	0	36
Social network index	16.9	12.0	3	66
Education level	7.84	2.41	3	12
Pre-tax annual income	58,625	57,127	0	300,000
Relationship quality	40.04	7.01	14	49
Perceived stress	19.68	4.57	10	37
Support received	36.07	49.11	0	272

matrix = 256×256 , field of view = 256 mm, slices = 160, slice thickness = 1 mm, and inversion time = 450 ms. Raw T1-weighted images were segmented using SPM12 (University College London, London, UK) and normalized to MNI152 space using Diffeomorphic Anatomical Registration through Exponentiated Line Algebra (DARTEL) [55].

Brain age estimation

Brain age was computed using BrainageR v2.0 [56] (<https://github.com/james-cole/brainageR/releases/tag/2.0>), which predicts chronological age from structural MRI by projecting tissue-segmented gray and white matter maps into a principal component space. The algorithm uses a Gaussian Process Regression (GPR) model trained on a sample of 3,337 healthy individuals (mean age = 36.9 ± 18.1) and validated on independent datasets [57]. GPR has been shown to yield predictive accuracy (mean absolute error, MAE = 4.67) comparable to that of more complex deep learning-based algorithms (e.g., Convolutional Neural Networks, MAE = 4.66) [58], while mitigating the risk of overfitting and estimation biases due to the quality of input data [59, 60]. Such generalization capacity is crucial for analyzing multiple MIDUS datasets. A similar GPR-based approach was previously used to investigate the link between caregiving and brain age [33].

Brain age estimation gap calculation

BrainAGE was calculated for each subject as the difference between the algorithm-predicted brain age and the subject's chronological age at the time of the MRI scan. Negative values indicate younger-appearing brains for a given age, and thus healthier neurobiological aging profiles [29]. Raw BrainAGE estimates tend to retain a residual dependence on chronological age (i.e., systematic overprediction in younger and underprediction in older individuals). This “regression-to-the-mean” pattern is thought to be a statistical artifact not attributable to biological or demographic characteristics of the training data, study samples, and is observed generally across BrainAGE algorithms [61]. We included chronological age as a covariate in all models to account for this bias in downstream analyses, following recommendations in the literature [62, 63] and by the developer of the algorithm used for the current study (SI5–2). We also report results from age-residualized gap scores (BrainAGER) as an alternative

Table 2 Hierarchical Multiple Regression Model Structure.

Model #	Predictors
Model 1: Baseline Model	age at MRI scan, sex, support provision
Model 2: Health/Lifestyle covariates	Model 1 + BMI, chronic conditions, years of drinking, years of smoking, exercise frequency, cognitive activities, depressive symptoms
Model 3: Psychosocial covariates	Model 2 + socioeconomic status (SES) ^a , perceived relationship quality, social network ^b , perceived stress, and support received

^aAnnual income and education level were standardized and averaged into a SES score (See SI4 for analyses examining income and education separately).

^bThe total number of children, friends, and relatives, marriage/cohabitation status, and connections to the religious community were combined into a Social Network Index (See SI5-1, 2).

outcome [64] (SI19). The distributions of estimated and chronological age are shown in SI10. Descriptive statistics for social support provision, all covariates, and predicted brain age are summarized in Table 1.

Statistical analysis

All analyses were conducted in IBM SPSS (version 29) and R (version 4.5.0) with $\alpha = 0.05$ (Two-tailed). We employed hierarchical multiple regression to assess whether the composite index of support provision explains variance in brain age and whether this association is robust to the inclusion of progressively more comprehensive control variables. Predictors were entered into models in three sequential blocks (Table 2). For the fully adjusted model, we computed 95% bias-corrected accelerated (BCa) bootstrap confidence intervals (CIs) with 10,000 iterations to assess robustness (“boot” package in R) [65]. BCa accounts for both bias and skewness in the bootstrap distribution, yielding more reliable CIs when the sample size is moderate [66].

We conducted two exploratory analyses. First, we tested whether unpaid instrumental assistance and emotional support provision were differentially associated with BrainAGE. Our approach was motivated by prior evidence that instrumental and emotional support are dissociable domains of social support [67] and may affect helpers’ health and mortality differently [68, 69]. We decomposed the total support provision into unpaid instrumental assistance and emotional support given to others, and support received into parallel indices for each support type. These new measures of providing or receiving instrumental and emotional support were moderately to strongly correlated ($\rho = 0.29$ – 0.78 , all P s < 0.001). Including both in a single model increased multicollinearity. Accordingly, we report and interpret only the results from these domain-specific models separately. Second, to assess potential estimation biases imposed by complex, theory-driven covariate structures, we conducted data-driven variable selection using bootstrapped elastic net regression, which applies regularization penalties that automatically eliminate weak or redundant predictors [70]. Multiple comparisons were not corrected for the exploratory analyses. This study was not preregistered.

Results

All three hierarchical models significantly predicted BrainAGE, and the fully adjusted model (Model 3) accounted for approximately 25% of total variance (SI11). Regression diagnostics indicated no violations of model assumptions, no influential cases, and no problematic multicollinearity (SI12).

Social support provision consistently emerged as a significant predictor of BrainAGE (Table 3). In Model 1, greater support provision was associated with a younger BrainAGE ($\beta = -0.139$, $p = 0.044$). This association persisted and increased in magnitude in Model 2 ($\beta = -0.194$, $p = 0.005$) and Model 3 ($\beta = -0.198$, $p = 0.023$; Fig. 1). For context, individuals providing high levels of social support (+1 SD; approximately 71 hours/month) had BrainAGE values 1.4 years lower than those providing average levels of support. In terms of unique variance explained, support provision accounted for approximately 2.2% of the variance in BrainAGE in the fully adjusted

Table 3 Coefficients of Hierarchical Regression Models.

Model	Variable	<i>B</i>	<i>SE</i>	β	<i>t</i>	<i>sr</i>	<i>p</i>
Model 1	(Constant)	4.209	2.605		1.616		0.108
	Sex	1.222	0.988	0.084	1.238	0.084	0.217
	Age at MRI Scan***	−0.185	0.037	−0.342	−5.036	−0.341	<0.001
	Support Provision**	−1.004	0.496	−0.139	−2.024	−0.137	0.044
Model 2	(Constant)	9.302	3.811		2.44		0.016
	Sex	1.385	0.985	0.096	1.406	0.092	0.161
	Age at MRI Scan***	−0.214	0.043	−0.397	−4.928	−0.322	<0.001
	Support Provision**	−1.403	0.494	−0.194	−2.839	−0.185	0.005
	Body Mass Index	−0.151	0.094	−0.112	−1.61	−0.105	0.109
	Years of Smoking*	0.114	0.041	0.194	2.76	0.180	0.006
	# of Chronic Conditions	0.386	0.225	0.134	1.719	0.112	0.087
	Years of Drinking	−0.056	0.048	−0.081	−1.175	−0.077	0.242
	Exercise Frequency	−0.074	0.106	−0.049	−0.699	−0.046	0.485
	Cognitive Activities	−0.071	0.061	−0.08	−1.163	−0.076	0.246
	Depression	0.077	0.074	0.076	1.045	0.068	0.297
Model 3	(Constant)	15.851	5.901		2.686		0.008
	Sex	0.894	1.033	0.062	0.865	0.056	0.388
	Age at MRI Scan***	−0.238	0.046	−0.44	−5.161	−0.335	<0.001
	Support Provision*	−1.435	0.627	−0.198	−2.289	−0.148	0.023
	Body Mass Index	−0.168	0.095	−0.125	−1.775	−0.115	0.087
	Years of Smoking*	0.096	0.042	0.164	2.304	0.149	0.022
	# of Chronic Conditions	0.421	0.230	0.146	1.827	0.118	0.069
	Years of Drinking	−0.066	0.048	−0.095	−1.37	−0.089	0.172
	Exercise Frequency	−0.059	0.106	−0.039	−0.557	−0.036	0.578
	Cognitive Activities	−0.068	0.062	−0.076	−1.09	−0.071	0.277
	Depression	0.066	0.081	0.064	0.807	0.052	0.421
	Social Network Size	−0.187	0.298	−0.042	−0.628	−0.041	0.531
	Perceived Stress	−0.151	0.117	−0.095	−1.284	−0.083	0.201
	Relationship Quality	−0.023	0.078	−0.022	−0.298	−0.019	0.766
	SES*	−1.372	0.600	−0.161	−2.285	−0.148	0.023
	Support Received*	−0.238	0.624	−0.033	−0.381	−0.025	0.704

N = 194; Dependent variable = BrainAGE; Model 1 $R^2 = 0.131$, Model 2 $R^2 = 0.221$, Model 3 $R^2 = 0.251$; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. *sr* = semi-partial correlation.

model ($sr = -0.148$; Table 3). Bootstrapping confirmed the stability of this effect, with CIs for global support provision not crossing zero (BCa 95% CI = -2.68 to -0.03) (SI13). Besides social support provision, years of smoking ($\beta = 0.164$, $p = 0.022$) and SES ($\beta = -0.161$, $p = 0.023$) remained significant after full model adjustment, with education levels driving the observed SES effect (SI14). Chronological age was also negatively associated with BrainAGE (SI14), following the well-documented regression-to-the-mean bias in brain age prediction [61]. A sensitivity analysis using the age-residualized scores (BrainAGER) as the outcome yielded results that substantively converged with those of the original model (SI9). Zero-order correlations and age-corrected partial correlations among focal variables are reported in SI15.

MIDUS includes several commonly used proxies for prosocial engagement (e.g., caregiving status, grandparenthood, and the number of living children) and discretionary time available for support provision (e.g., retirement status) [11, 32, 71, 72]. In direct comparison, monthly hours spent providing support explained significant incremental variance in BrainAGE beyond each proxy, whereas none of the proxies remained significant (SI16–SI19) nor added significant variance once support-provision hours were accounted for (SI20).

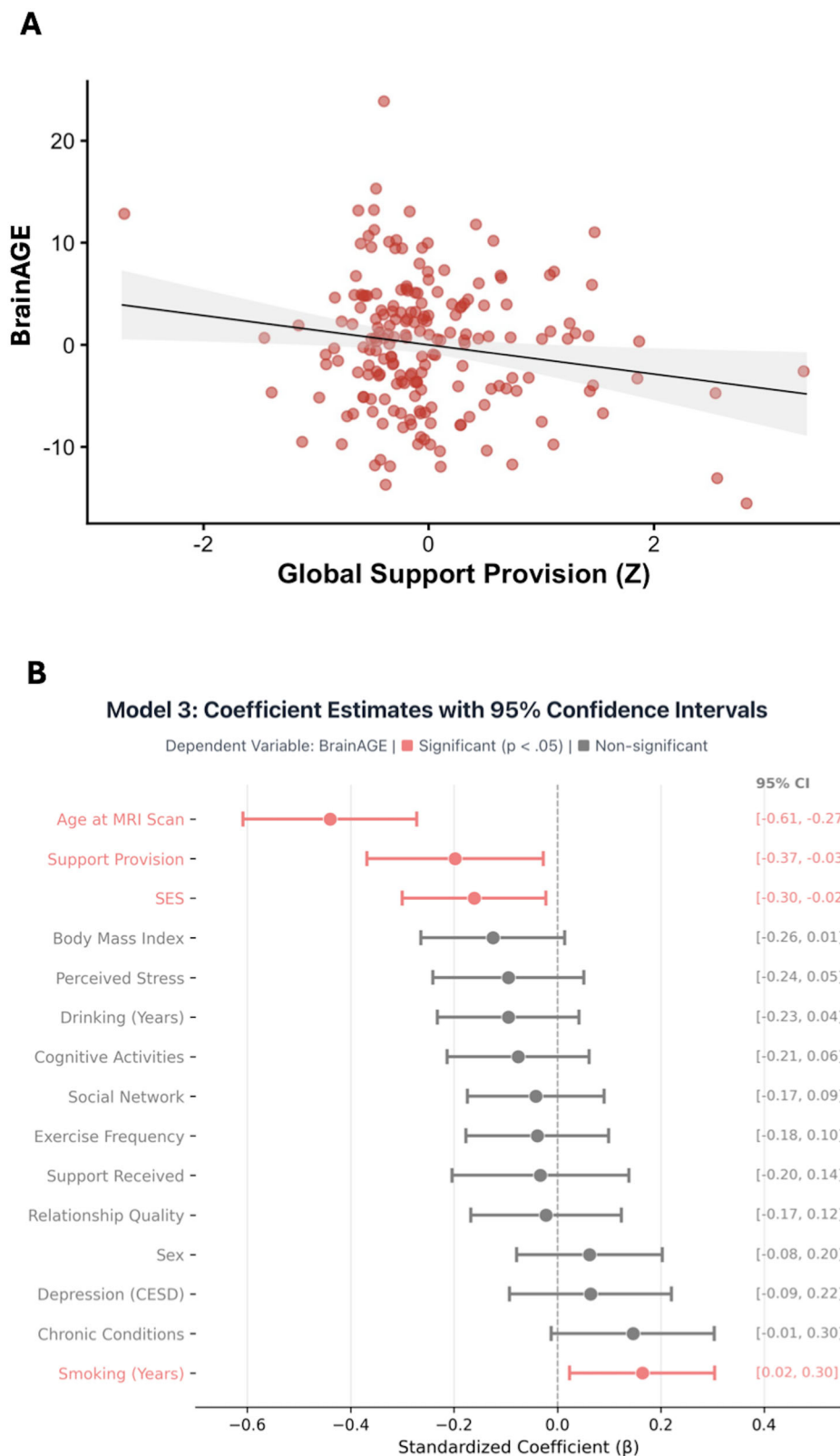


Fig. 1 Partial regression plot of the association between social support provision and BrainAGE (A), and standardized coefficient estimates with 95% confidence intervals for all predictors (B). Standardized coefficients (β) are plotted to enable comparison of effect sizes. Support provision was associated with a younger estimated brain age relative to chronological age after adjusting for all covariates in Model 3. Unstandardized coefficients and 95% CIs are visualized in SI23.

Our exploratory analyses revealed differential associations between BrainAGE and the provision of unpaid assistance and emotional support. In Model 3, total monthly hours of emotional support provision were significantly associated with lower BrainAGE ($\beta = -0.26$, $p = 0.007$). In contrast, providing unpaid assistance did not show an effect ($\beta = -0.09$, $p = 0.167$). Bootstrapping yielded convergent results (SI21). Social support provision also emerged as a reliably selected predictor in the data-driven variable selection analysis, suggesting that the association is robust to model specification (SI22).

Discussion

Can helping be neuroprotective? Although prior evidence suggests that prosocial engagement may confer neurobiological benefits [30, 33], empirical support for this association has been limited. Our results and analytical strategy demonstrate this hypothesized relationship with greater rigor. By modeling actual hours of social support provision, we captured granularity in prosocial behaviors that explained unique incremental variance in BrainAGE beyond commonly used role-based proxies [32, 33, 73]. Comprehensive covariate control and bootstrapping further strengthen the robustness of our observation. While several covariates predicted BrainAGE in isolation, replicating previously reported patterns [33, 34], most associations attenuated in the fully adjusted model except for smoking and SES. By contrast, the association with support provision emerged consistently and increased in magnitude, suggesting that prosocial engagement may operate as a distinct behavioral pathway to healthier brain aging.

The unique and robust association between social support provision and BrainAGE raises an important question: through which psychological and neurobiological pathways might prosocial behaviors confer resilience to the aging brain? While there is no consensus on the mechanism, prior work on the psychological and physiological effects of prosocial behavior offers insights. Helping often elicits hedonic and eudaimonic well-being [16, 74], which can dampen physiological arousal in response to acute stressors [75, 76] and buffer against chronic adversity [77–79]. This may disrupt stress-related pathways that affect brain structures and functions [80–83]. Affiliative social interactions also release endogenous oxytocin [84], which exerts anxiolytic effects by dampening amygdala activity [85]. Oxytocin can also suppress pro-inflammatory cascades and oxidative damage, preventing cellular aging in the brain [86–89]. These interconnected biopsychosocial processes may contribute to the downstream health effects of prosocial engagement [83, 90].

Elucidating these intermediate pathways may help clarify whether different forms of helping are equally associated with BrainAGE. We found that giving emotional support to others was more strongly associated with lower BrainAGE than giving unpaid assistance. This asymmetry may reflect the distinct psychological and behavioral demands of each support type. Emotional support in close relationships often involves empathic responding [91], which fosters bonding [92], a sense of purpose [93], and self-worth [94]. By contrast, instrumental assistance may be routinized and lack the same degree of dyadic attunement linked to well-being [95]. Prior work indeed suggests that the psychological benefits of instrumental aid for providers emerge only when emotional engagement is present [68]. We caution against a strict dichotomy, however, as different types of helping can co-occur and may influence health in mutually non-exclusive ways: instrumental assistance through physical and cognitive activities [96] and emotional support through affective processes [4, 97]. Moreover, the effect of support provision on health may be context dependent. For instance, emotional support sustained in a high-stress environment can lead to empathic distress [98], curtailing the neuroprotective potential observed in our data.

Due to the modest sample size, we are unable to test these mechanisms and their interactions directly in the current study. The psychological benefits of prosocial behavior tend to be modest and heterogeneous [16]. A similar pattern holds for peripheral indices of stress and inflammation, whose associations with prosocial behavior and BrainAGE are likewise modest and often contingent on moderators, even in large cohorts [99, 100]. While such markers (e.g., IL-6 and C-reactive protein) are available in MIDUS, the small effect sizes, together with limited data availability in our imaging subcohort, constrain our ability to adequately test these potential indirect pathways. Embedding BrainAGE measurement in community volunteering programs for older adults [101], care-facility trials [102], or prosocial interventions [103] may allow us to examine whether and through which mechanisms the provision of social support,

especially emotional support, influences brain aging trajectories and related health outcomes. Importantly, although the unique variance in BrainAGE attributable to support provision was modest, it was comparable in magnitude to that of SES, a widely recognized structural determinant of health [104]. If our findings are substantiated, support provision could emerge as a modifiable behavioral target for healthy brain aging. Unlike pharmacological or lifestyle interventions, which often require significant resources or clinical infrastructure, prosociality can be cultivated through everyday activities and community programs [16, 105]. Such approaches may offer an actionable and cost-effective strategy for supporting brain health at scale, an urgent priority in aging societies [106].

Limitations and future directions

The current study has several limitations that warrant future inquiry. First, our observational design precludes causal inference. Although behavioral assessment temporally preceded MRI acquisition, we cannot rule out reverse causality, in which individuals with greater neurobiological reserve are better able to sustain support provision. Larger longitudinal studies that track changes in both prosocial behaviors and BrainAGE will be necessary to test this possibility, including the possibility of a positive feedback loop between prosociality and neuroprotection [107].

Such replication is also necessary to resolve selection bias in our current sample. 76% of our participants were white. We also included only those who completed multi-year, multimodal data collection, which limits the generalizability. Our sample-selection analyses showed that individuals who entered the Neuroscience Project were younger and reported a lower health burden than eligible participants who did not participate in the Neuroscience Project. Those retained in the final BrainAGE analysis were again younger than Neuroscience Project participants excluded due to missing or incomplete data. This pattern of age- and health-related selection is consistent with prior MIDUS reports of patterned longitudinal retention and suggests that our findings may best characterize support provision among relatively young and healthy adults [108]. Individuals under intense caregiving burdens or high levels of chronic stress, groups who may show accelerated biological aging [109, 110], were likely underrepresented in our sample.

Second, our assessment of prosociality has specific boundary conditions. We used retrospective self-reports of time spent supporting close others. While focusing on objective dimensions of helping reduces desirability bias [16], it may still be susceptible to recall bias (e.g., overemphasizing more tangible instrumental support) and fails to capture prosocial behaviors not reflected in hours (e.g., charitable giving).

Finally, the predictive validity of the BrainAGE framework has been supported in some contexts but questioned in others [62]. Its associations with clinical outcomes appear to vary with the population studied, the health variables being predicted [111], the quality of the input data, and the algorithms used [112]. This underscores the need for cross-population validation and benchmarking before full clinical translation (See: [113]). Relatedly, because BrainAGE is a global summary metric derived from a high-dimensional combination of structural features [29], it may not map onto specific conventional metrics such as regional gray matter volume across functionally dissociable neural systems. More fine-grained, region-specific approaches (e.g., Regional BrainAGE) [114], along with further examination of which neural features drive estimation, will be needed to enhance the interpretability and clinical utility of BrainAGE.

Conclusion

Taken together, we present evidence that higher levels of prosocial behavior are prospectively associated with a younger-looking brain at a given chronological age. These associations appear distinct from other established predictors of aging and may be more closely tied to the emotional dimensions of helping. Although longitudinal and mechanistic studies are needed to clarify causal pathways, our findings lay a foundation for more precise models of how prosociality may protect against the neural deterioration underlying age-related physical and mental health decline, as well as for designing intervention trials to promote healthy brain aging at scale.

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Data availability MIDUS data, instruments, protocols, and codebooks are available through the Inter-university Consortium for Political and Social Research (ICPSR). Restricted-use data from the MIDUS project can be obtained via ICPSR, and raw imaging data are available from the MIDUS Neuroscience Repository (<https://midus.wisc.edu/midus-neuroscience-repository/>) upon application and approval. Detailed descriptions of the variables used are fully provided in the manuscript. Scripts used for analyses can be obtained upon request.

Competing interests The author declare no competing of interest.

Ethics declaration The authors confirm that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Declaration of Helsinki. All MIDUS study procedures and materials were approved by the University of Wisconsin-Madison Institutional Review Board (IRB) (Core survey: 2016-1051; Biomarker: 2014-0813; MRI: 2016-0054), and all participants provided written informed consent.

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References

1. Asante S, Karikari G. Social relationships and the health of older adults: an examination of social connectedness and perceived social support. *J Ageing Longev.* 2022;2:49–62.
2. Lyyra T-M, Heikkinen R-L. Perceived social support and mortality in older people. *J Gerontol B Psychol Sci Soc Sci.* 2006;61:S147–S152.
3. Holt-Lunstad J, Smith TB, Layton JB. Social relationships and mortality risk: a meta-analytic review. *PLoS Med.* 2010;7:e1000316.
4. Vila J. Social support and longevity: Meta-analysis-based evidence and psychobiological mechanisms. *Front Psychol.* 2021;12:717164.
5. Lovell B, Wetherell MA. The cost of caregiving: endocrine and immune implications in elderly and non elderly caregivers. *Neurosci Biobehav Rev.* 2011;35:1342–1352.
6. Scheffer JA, Cameron CD, Inzlicht M. Caring is costly: People avoid the cognitive work of compassion. *J Exp Psychol: Gen.* 2022;151:172.
7. Bertrand RM, Saczynski JS, Mezzacappa C, Hulse M, Ensrud K, Fredman L. Caregiving and cognitive function in older women: Evidence for the healthy caregiver hypothesis. *J Aging Health.* 2012;24:48–66.
8. Li L, Wister AV, Mitchell BA. Examination of the healthy caregiver effect among older adults: Findings from the Canadian Longitudinal Study on Aging. *Gerontology.* 2023;69:289–300.
9. Poulin MJ, Brown SL, Ubel PA, Smith DM, Jankovic A, Langa KM. Does a helping hand mean a heavy heart? Helping behavior and well-being among spouse caregivers. *Psychol Aging.* 2010;25:108.

10. Marino VR, Haley WE, Roth DL. Beyond hedonia: A theoretical reframing of caregiver well-being. *Transl Issues Psychol Sci.* 2017;3:400.
11. Bhattacharyya KK, Liu Y, Das Gupta D, Molinari V, Fauth EB. The healthy caregiver? A positive impact of informal caregiving status on cognitive functions over time from the midlife in the United States study. *J Aging Health.* 2024;36:631–641.
12. Poulin MJ, Brown SL, Dillard AJ, Smith DM. Giving to others and the association between stress and mortality. *Am J Public Health.* 2013;103:1649–1655.
13. Fredman L, Lyons JG, Cauley JA, Hochberg M, Applebaum KM. The relationship between caregiving and mortality after accounting for time-varying caregiver status and addressing the healthy caregiver hypothesis. *J Gerontol Series A: Biomed Sci Med Sci.* 2015;70:1163–1168.
14. Mehri N, Kinney J, Brown S, Rajabi Rostami M. Informal caregiving and all-cause mortality: A meta-analysis of longitudinal population-based studies. *J Appl Gerontol.* 2021;40:781–791.
15. Brown SL, Nesse RM, Vinokur AD, Smith DM. Providing social support may be more beneficial than receiving it: Results from a prospective study of mortality. *Psychol Sci.* 2003;14:320–327.
16. Hui BP, Ng JC, Berzaghi E, Cunningham-Amos LA, Kogan A. Rewards of kindness? A meta-analysis of the link between prosociality and well-being. *Psychol Bull.* 2020;146:1084.
17. Fryburg DA. Kindness as a stress reduction–health promotion intervention: a review of the psychobiology of caring. *Am J Lifestyle Med.* 2022;16:89–100.
18. Mongrain M, Barnes C, Barnhart R, Zalan LB. Acts of kindness reduce depression in individuals low on agreeableness. *Transl Issues Psychol Sci.* 2018;4:323.
19. Byrne M, Tan RKJ, Wu D, Marley G, Hlatshwako TG, Tao Y, et al. Prosocial interventions and health outcomes: a systematic review and meta-analysis. *JAMA Network Open.* 2023;6:e2346789–e2346789.
20. Curry OS, Rowland LA, Van Lissa CJ, Zlotowitz S, McAlaney J, Whitehouse H. Happy to help? A systematic review and meta-analysis of the effects of performing acts of kindness on the well-being of the actor. *J Exp Soc Psychol.* 2018;76:320–329.
21. Lockhart SN, DeCarli C. Structural imaging measures of brain aging. *Neuropsychol Rev.* 2014;24:271–289.
22. Kumari LS, Sundararajan R. A review on brain age prediction models. *Brain Res.* 2024;1823:148668.
23. Cole JH, Franke K. Predicting age using neuroimaging: innovative brain ageing biomarkers. *Trends Neurosci.* 2017;40:681–690.
24. Liem F, Varoquaux G, Kynast J, Beyer F, Masouleh SK, Huntenburg JM, et al. Predicting brain-age from multimodal imaging data captures cognitive impairment. *Neuroimage.* 2017;148:179–188.
25. Beck D, de Lange AMG, Pedersen ML, Alnæs D, Maximov II, Voldsbekk I, et al. Cardiometabolic risk factors associated with brain age and accelerated brain ageing. *Hum Brain Mapp.* 2022;43:700–720.
26. Cole JH, Ritchie SJ, Bastin ME, Valdés Hernández M, Muñoz Maniega S, Royle N, et al. Brain age predicts mortality. *Mol Psychiatry.* 2018;23:1385–1392.
27. Hua JP, Abram SV, Loewy RL, Stuart B, Fryer SL, Vinogradov S, et al. Brain age gap in early illness schizophrenia and the clinical high-risk syndrome: associations with experiential negative symptoms and conversion to psychosis. *Schizophr Bull.* 2024;50:1159–1170.
28. Biondo F, Jewell A, Pritchard M, Aarsland D, Steves CJ, Mueller C, et al. Brain-age is associated with progression to dementia in memory clinic patients. *NeuroImage: Clinical.* 2022;36:103175.
29. Franke K, Gaser C. Ten years of BrainAGE as a neuroimaging biomarker of brain aging: what insights have we gained? *Front Neurol.* 2019;10:789.
30. Ibanez A, Matallana D, Miller B. Can prosocial values improve brain health? *Front Neurol.* 2023;14:1202173.
31. Orchard ER, Chopra S, Ooi LQ, Chen P, An L, Jamadar SD, et al. Protective role of parenthood on age-related brain function in mid-to late-life. *Proc Natl Acad Sci.* 2025;122:e2411245122.
32. Ning K, Zhao L, Franklin M, Matloff W, Batta I, Arzouni N, et al. Parity is associated with cognitive function and brain age in both females and males. *Sci Rep.* 2020;10:6100.
33. Rilling JK, Lee M, Zhou C, Hepburn K, Perkins MM, Gaser C. Caregiving is associated with lower brain age in humans. *Soc Cogn Affect Neurosci.* 2025;20:nsaf013.
34. Behrenbruch N, Schwarck S, Schumann-Werner B, Molloy EN, Garcia-Garcia B, Hochkeppeler A et al. A physically and mentally active lifestyle relates to younger brain and cognitive age. *GeroScience* 2025: 1-21.
35. Lövdén M, Bäckman L, Lindenberger U The link of intellectual engagement to cognitive and brain aging. *Cogn Neurosci Aging* 2017: 461-484.
36. Selitser M, Dietze LM, McWhinney SR, Hajek T. Cardiometabolic risk factors and brain age: a meta-analysis to quantify brain structural differences related to diabetes, hypertension, and obesity. *J Psychiatry Neurosci.* 2025;50:E102–E111.
37. Cohen JW, Ramphal B, DeSerisy M, Zhao Y, Pagliaccio D, Colcombe S, et al. Relative brain age is associated with socioeconomic status and anxiety/depression problems in youth. *Dev Psychol.* 2024;60:199.
38. Elliott AF, Burgio LD, DeCoster J. Enhancing caregiver health: findings from the resources for enhancing Alzheimer’s caregiver health II intervention. *J Am Geriatr Soc.* 2010;58:30–37.
39. Barry TR. The Midlife in the United States (MIDUS) series: A national longitudinal study of health and well-being. *Open Health Data.* 2014;2:e3.

40. Ryff CD, Davidson RJ Midlife in the United States (MIDUS Refresher 1): Neuroscience Project, 2012-2016. Inter-university Consortium for Political and Social Research [distributor] 2025.
41. Ryff C, Almeida D, Ayanian J, Binkley N, Carr DS, Coe C et al. Midlife in the United States (MIDUS 3), 2013-2014. (*No Title*) 2015.
42. Urban-Wojcik EJ, Lee S, Grupe DW, Quinlan L, Gresham L, Hammond A, et al. Diversity of daily activities is associated with greater hippocampal volume. *Cogn Affect Behav Neurosci*. 2022;22:75–87.
43. Austad SN, Bartke A. Sex differences in longevity and in responses to anti-aging interventions: a mini-review. *Gerontology*. 2015;62:40–46.
44. Ronan L, Alexander-Bloch AF, Wagstyl K, Farooqi S, Brayne C, Tyler LK, et al. Obesity associated with increased brain age from midlife. *Neurobiol Aging*. 2016;47:63–70.
45. Rolland Y, van Kan GA, Vellas B. Healthy brain aging: role of exercise and physical activity. *Clin Geriatr Med*. 2010;26:75–87.
46. Guggenmos M, Schmack K, Sekutowicz M, Garbusow M, Sebold M, Sommer C, et al. Quantitative neurobiological evidence for accelerated brain aging in alcohol dependence. *Transl Psychiatry*. 2017;7:1279.
47. McNeil CJ, Habota T, Sandu A-L, Waiter G, Whalley H, Murray AD. The influence of birth weight, socio-economic status, and adult health on brain volumes during ageing. *Neuroepidemiology*. 2025;59:489–497.
48. Ashida S, Schafer EJ Social networks, social relationships, and their effects on the aging mind and brain. *Wiley Handb Aging Mind Brain* 2018: 17-36.
49. Ning K, Zhao L, Matloff W, Sun F, Toga AW. Association of relative brain age with tobacco smoking, alcohol consumption, and genetic variants. *Sci Rep*. 2020;10:10.
50. Ryff CD, Keyes CLM. The structure of psychological well-being revisited. *J Pers Soc Psychol*. 1995;69:719.
51. Cohen S, Kamarck T, Mermelstein R. Perceived stress scale. *Meas Stress: A Guide Health Soc Sci*. 1994;10:1–2.
52. Dintica CS, Habes M, Erus G, Simone T, Schreiner P, Yaffe K. Long-term depressive symptoms and midlife brain age. *J Affect Disord*. 2023;320:436–441.
53. Schoemann AM, Boulton AJ, Short SD. Determining power and sample size for simple and complex mediation models. *Soc Psychol Personal Sci*. 2017;8:379–386.
54. Fritz MS, MacKinnon DP. Required sample size to detect the mediated effect. *Psychol Sci*. 2007;18:233–239.
55. Ashburner J. A fast diffeomorphic image registration algorithm. *Neuroimage*. 2007;38:95–113.
56. Cole JH. Multimodality neuroimaging brain-age in UK biobank: relationship to biomedical, lifestyle, and cognitive factors. *Neurobiol Aging*. 2020;92:34–42.
57. Taylor JR, Williams N, Cusack R, Auer T, Shafto MA, Dixon M, et al. The Cambridge Centre for Ageing and Neuroscience (Cam-CAN) data repository: Structural and functional MRI, MEG, and cognitive data from a cross-sectional adult lifespan sample. *Neuroimage*. 2017;144:262–269.
58. Cole JH, Poudel RP, Tsagkrasoulis D, Caan MW, Steves C, Spector TD, et al. Predicting brain age with deep learning from raw imaging data results in a reliable and heritable biomarker. *Neuroimage*. 2017;163:115–124.
59. Rajabli R, Soltaninejad M, Fonov VS, Bzdok D, Collins DL. Brain age prediction: Deep models need a hand to generalize. *Hum Brain Mapp*. 2025;46:e70254.
60. Williams CK, Rasmussen CE. Gaussian processes for machine learning. 2. Cambridge, MA: MIT press; 2006.
61. Liang H, Zhang F, Niu X Investigating systematic bias in brain age estimation with application to post-traumatic stress disorders: Wiley Online Library; 2019. Report no. 1065-9471.
62. Gaser C, Kalc P, Cole JH. A perspective on brain-age estimation and its clinical promise. *Nat Comput Sci*. 2024;4:744–751.
63. Zhang F, Chang H, Schaefer SM, Gou J. Biological age and brain age in midlife: relationship to multimorbidity and mental health. *Neurobiol Aging*. 2023;132:145–153.
64. Le TT, Kuplicki RT, McKinney BA, Yeh H-W, Thompson WK, Paulus MP, et al. A nonlinear simulation framework supports adjusting for age when analyzing BrainAGE. *Front Aging Neurosci*. 2018;10:317.
65. Cauty AJ. Resampling methods in R: the boot package. *The Newsletter of the R Project Volume*. 2002;2:2–7.
66. Hall P Theoretical comparison of bootstrap confidence intervals. *Ann Stat* 1988: 927-953.
67. Abraham E, Feldman R The many faces of human caregiving. *Routledge Int Handb Soc Neuroendocrinol* 2018.
68. Morelli SA, Lee IA, Arnn ME, Zaki J. Emotional and instrumental support provision interact to predict well-being. *Emotion*. 2015;15:484.
69. Chen E, Lam PH, Finegood ED, Turiano NA, Mroczek DK, Miller GE. The balance of giving versus receiving social support and all-cause mortality in a US national sample. *Proc Natl Acad Sci*. 2021;118:e2024770118.
70. Meinshausen N, Bühlmann P. Stability selection. *J R Stat Soc Series B: Stat Methodol*. 2010;72:417–473.
71. Bhat AC, Trexberg KM, Apsley AT, Almeida DM, Shalev I. Grandparent caregiving and epigenetic aging among midlife and older adults in the United States. *J Gerontol, Series B: Psychol Sci Soc Sci*. 2026;81:gbaf255.
72. Tang F. Retirement patterns and their relationship to volunteering. *Nonprofit Volunt Sect Q*. 2016;45:910–930.
73. Orchard ER, Ward PG, Sforazzini F, Storey E, Egan GF, Jamadar SD. Relationship between parenthood and cortical thickness in late adulthood. *PLoS ONE*. 2020;15:e0236031.
74. Meneghini AM, Colledani D. “Doing well by doing good”: When and how volunteering fosters hedonic and eudaimonic well-being. *Humanist Psychol*. 2024;52:102.

75. DuPont CM, Weis TM, Manuck SB, Marsland AL, Matthews KA, Gianaros PJ. Does well-being associate with stress physiology? A systematic review and meta-analysis. *Health Psychol.* 2020;39:879.
76. Jurkiewicz O, Oveis C Helping you helps me: Beneficial effects of regulating others' emotions on well-being and physiological stress. *Emotion* 2024.
77. Morozink JA, Friedman EM, Coe CL, Ryff CD. Socioeconomic and psychosocial predictors of interleukin-6 in the MIDUS national sample. *Health Psychol.* 2010;29:626.
78. Ryff CD, Radler BT, Friedman EM. Persistent psychological well-being predicts improved self-rated health over 9–10 years: Longitudinal evidence from MIDUS. *Health Psychol Open.* 2015;2:2055102915601582.
79. Friedman EM, Ryff CD. Living well with medical comorbidities: A biopsychosocial perspective. *J Gerontol Series B: Psychol Sci Soc Sci.* 2012;67:535–544.
80. Urry HL, Nitschke JB, Dolski I, Jackson DC, Dalton KM, Mueller CJ, et al. Making a life worth living: Neural correlates of well-being. *Psychol Sci.* 2004;15:367–372.
81. Kaufmann E, Rojczyk P, Sydnor VJ, Guenette JP, Tripodis Y, Kaufmann D, et al. Association of war zone-related stress with alterations in limbic gray matter microstructure. *JAMA Network Open.* 2022;5:e2231891–e2231891.
82. Hölzel BK, Carmody J, Evans KC, Hoge EA, Dusek JA, Morgan L, et al. Stress reduction correlates with structural changes in the amygdala. *Soc Cogn Affect Neurosci.* 2010;5:11–17.
83. Inagaki TK. Neural mechanisms of the link between giving social support and health. *Ann N Y Acad Sci.* 2018;1428:33–50.
84. Crockford C, Deschner T, Ziegler TE, Wittig RM. Endogenous peripheral oxytocin measures can give insight into the dynamics of social relationships: a review. *Front Behav Neurosci.* 2014;8:68.
85. László K, Kovács A, Zagoracz O, Ollmann T, Péczely L, Kertes E, et al. Positive reinforcing effect of oxytocin micro-injection in the rat central nucleus of amygdala. *Behav Brain Res.* 2016;296:279–285.
86. Lee M, Rilling JK. Oxytocin and the pace of life history strategies: from evolutionary trade-offs to translational pathways. *Neurosci Biobehav Rev.* 2025;180:106457.
87. Kamrani-Sharif R, Hayes AW, Gholami M, Salehirad M, Allahverdikhani M, Motaghinejad M, et al. Oxytocin as neuro-hormone and neuro-regulator exert neuroprotective properties: a mechanistic graphical review. *Neuropeptides.* 2023;101:102352.
88. Panaro MA, Benameur T, Porro C. Hypothalamic neuropeptide brain protection: focus on oxytocin. *J Clin Med.* 2020;9:1534.
89. Stevenson JR, McMahon EK, Boner W, Haussmann MF. Oxytocin administration prevents cellular aging caused by social isolation. *Psychoneuroendocrinology.* 2019;103:52–60.
90. Brown SL, Brown RM. Connecting prosocial behavior to improved physical health: Contributions from the neurobiology of parenting. *Neurosci Biobehav Rev.* 2015;55:1–17.
91. O'Brien TB, DeLongis A, Pomaki G, Puterman E, Zwicker A. Couples coping with stress: The role of empathic responding. *Eur Psychol.* 2009;14:18–28.
92. Gerace A, Oster C, O'Kane D, Hayman CL, Muir-Cochrane E. Empathic processes during nurse–consumer conflict situations in psychiatric inpatient units: A qualitative study. *Int J Ment Health Nurs.* 2018;27:92–105.
93. Czyżowska D, Gurba E, Czyżowska N, Kalus A. Intimate relationship and its significance for eudaimonic well-being in young adults. *Health Psychol Rep.* 2020;8:155–166.
94. Weilenmann S, Schnyder U, Keller N, Corda C, Spiller TR, Brugger F, et al. Self-worth and bonding emotions are related to well-being in health-care providers: a cross-sectional study. *BMC Med Educ.* 2021;21:290.
95. Chambliss DF *Beyond caring: Hospitals, nurses, and the social organization of ethics.* University of Chicago Press 1996.
96. O'Sullivan M, Brennan S, Lawlor BA, Hannigan C, Robertson IH, Perlt MM. Cognitive functioning among cognitively intact dementia caregivers compared to matched self-selected and population controls. *Aging Ment Health.* 2019;23:566–573.
97. Heinrichs M, Baumgartner T, Kirschbaum C, Ehlert U. Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. *Biol Psychiatry.* 2003;54:1389–1398.
98. Brown CL, Grimm KJ, Wells JL, Hua AY, Levenson RW. Empathic accuracy and shared depressive symptoms in close relationships. *Clin Psychol Sci.* 2023;11:509–525.
99. Bell MJ, Ferraro KF, Sauerteig-Rolston MR. Volunteer engagement and systemic inflammation: Does helping others benefit oneself? *Gerontologist.* 2022;62:1477–1485.
100. Dunk MM, Huang H, Wang J, Dove A, Sakakibara S, Guo J, et al. The association between a pro-inflammatory diet and brain age in middle-aged and older adults. *Eur J Epidemiol.* 2026;41:39–50.
101. Guiney H, Machado L. Volunteering in the community: Potential benefits for cognitive aging. *J Gerontol: Series B.* 2018;73:399–408.
102. Sellon AM, Chapin RK, Leedahl SN. Engaging nursing home residents in formal volunteer activities: a focus on strengths. *Ageing Int.* 2017;42:93–114.
103. Naclerio M, Lazar L, Hornstein EA, Eisenberger NI Exploring the effects of prosocial and self-kindness interventions on mental health outcomes. *Emotion* 2025.
104. Fiorito G, McCrory C, Robinson O, Carmeli C, Rosales CO, Zhang Y, et al. Socioeconomic position, lifestyle habits and biomarkers of epigenetic aging: a multi-cohort analysis. *Aging.* 2019;11:2045.

105. Miles A, Andiappan M, Upenieks L, Orfanidis C. Using prosocial behavior to safeguard mental health and foster emotional well-being during the COVID-19 pandemic: A registered report of a randomized trial. *PLoS ONE*. 2022;17:e0272152.
106. Kubzansky LD, Epel ES, Davidson RJ. Prosociality should be a public health priority. *Nat Hum Behav*. 2023;7:2051–2053.
107. Layous K, Nelson SK, Kurtz JL, Lyubomirsky S. What triggers prosocial effort? A positive feedback loop between positive activities, kindness, and well-being. *J Posit Psychol*. 2017;12:385–398.
108. Song J, Radler BT, Lachman ME, Mailick MR, Si Y, Ryff CD. Who returns? Understanding varieties of longitudinal participation in MIDUS. *J Aging Health*. 2021;33:896–907.
109. Palma-Gudiel H, Fañanás L, Horvath S, Zannas AS. Psychosocial stress and epigenetic aging. *Int Rev Neurobiol*. 2020;150:107–128.
110. Roth DL, Sheehan OC, Haley WE, Jenny NS, Cushman M, Walston JD. Is family caregiving associated with inflammation or compromised immunity? A meta-analysis. *Gerontologist*. 2019;59:e521–e534.
111. Dörfel RP, Ozenne B, Ganz M, Svensson JE, Plavén-Sigra P. Prediction of brain age using structural magnetic resonance imaging: a comparison of clinical utility of publicly available software packages. *EBioMedicine*. 2026;123:106094.
112. Han J, Kim SY, Lee J, Lee WH. Brain age prediction: A comparison between machine learning models using brain morphometric data. *Sensors*. 2022;22:8077.
113. Legaz A, Moguilner S, Barttfeld P, Cuadros Castro J, Galván Rial DS, Migeot J et al. The exposome of brain aging across 34 countries. *Nat Med* 2026: 1-14.
114. Chen Y-S, Kuo C-Y, Lu C-H, Wang Y-W, Chou K-H, Lin W-C. Multiscale brain age prediction reveals region-specific accelerated brain aging in Parkinson's disease. *Neurobiol Aging*. 2024;140:122–129.

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