scientific reports

Check for updates

OPEN

Associations between perceived excessive maternal control in childhood, well-being, and dorsal striatum volume in older adults

Delia A. Gheorghe[1,2]✉, Morgane Künzi[2], Nicola Filippini[3], Ligiana M. Petre[4] & Sarah Bauermeister[2]

Maternal bonding has been linked to the functioning of the dorsal striatum. Our aim was to examine how midlife perceptions of maternal parenting relate to late-life brain volume using the "Insight 46" neuroimaging sub-study of the MRC National Survey of Health and Development (NSHD) 1946 birth cohort. In a cross-sectional design, we investigated associations between retrospective maternal bonding and dorsal striatal volumes (bilateral caudate and putamen) in 452 cognitively healthy older adults with similar socio-economic backgrounds. Maternal care, control, and autonomy up to age 16 were reported at age 43, and T1-weighted brain images were acquired at age 70. K-means clustering categorized participants into three groups, with additional analyses on the 10th and 90th percentiles. Results showed that extreme maternal control (10th vs. 90th percentiles) was associated with smaller right caudate and left putamen volumes. No significant volume differences were found for maternal care or autonomy when comparing groups across the full sample or when comparing extreme groups. Greater perceived care and autonomy, alongside lower control, were associated with higher well-being. Our study showed that in a large population sample, extreme perceived maternal control was associated with smaller dorsal striatal volumes and lower well-being at 70 years of age. While there are important limitations related to retrospective and cross-sectional characteristics, our results speak to the potential endurance of effects throughout the lifespan, at a population level.

Keywords Maternal control, Maternal bonding, Putamen, Caudate, Well-being, Population study, Older adults

Abbreviations

CFA	Confirmatory factor analysis
CFI	Comparative fit index
FAST	FMRIB's Automated Segmentation Tool
FIRST	FMRIB Integrated Registration and Segmentation Tool
FLAIR	Fluid-attenuated inversion recovery
GM	Grey matter
MMSE	Mini-Mental State Examination
MRI	Magnetic resonance imaging
MPRAGE	Magnetization prepared rapid gradient echo
MRC	Medical Research Council
NSHD	National Survey of Health and Development
PET	Positron emission tomography
PBI	Parental Bonding Instrument
QC	Quality control
RMSEA	Root mean square error of approximation

[1]Department of Experimental and Theoretical Neuroscience, Transylvanian Institute of Neuroscience, Pta. Timotei Cipariu 9/20, Cluj-Napoca 400191, Romania. [2]Dementias Platform UK, Department of Psychiatry, University of Oxford, Oxford, UK. [3]IRCCS San Camillo Hospital, Venice, Italy. [4]Department of Applied Psychology and Psychotherapy, Faculty of Psychology and Educational Sciences, University of Bucharest, Bucharest, Romania. ✉email: delia.gheorghe@tins.ro

SD	Standard deviation
SE	Standard error
SRMR	Standardized root-mean residual
TR	Repetition time
TE	Echo time
TI	Inversion time
TLI	Tucker Lewis index
WM	White matter

Decades of scientific research have shown that childhood adversity of varying degrees of severity and chronicity is associated with changes in brain volume[1–3]. However, generalising from existing studies is challenging due to (1) the wide variability in how adversity is defined; (2) limited evidence on cumulative effects and co-occurrence[2,4,5]; and (3) challenges in replicability across large cohorts. Despite these challenges, converging evidence suggests that many forms of adversity share common pathways[6]. Indeed, across adversities, there may be shared aspects related to the biology of stress, and the dysregulation of the stress hormone system has been widely investigated[7–9]. Another shared aspect across many childhood adversities emphasizes the role of parenting and the broader family context[10]. Here we contribute to the literature on the associations between adversity and brain structure, by focusing on maternal bonding as one crucial aspect of development with demonstrated impact on stress resilience and early neurodevelopmental pathways[11–13]. While it is a challenge to disentangle the maternal relationship from broader adverse aspects of the home environment and beyond, we argue that given its early developmental timeline, maternal bonding may leave individual signatures on the developing brain.

Maternal bonding refers to the emotional tie that a mother develops toward her infant[14]. One way to assess early maternal bonding is through the Parental Bonding Instrument (PBI)[15]. The PBI retrospectively measures perceived parenting before age 16 and can be administered after age 16, with demonstrated long-term stability[16]. Two continuous dimensions are primarily assessed in the PBI (1) maternal care (empathic, sensitive, responsive parenting) and (2) maternal control (intrusive, infantilizing, demanding parenting). The opposite pole of maternal control is captured by PBI factor maternal autonomy, which reflects encouragement of independence, including support for the child's freedom and decision-making latitude. These dimensions of parenting may support understanding of broader, population-representative developmental mechanisms[17].

Certainly, a caregiver's affection and care are crucial sources of reward-giving during development[18,19]. By seeking closeness to their mothers, infants carry out goal-directed behaviours to achieve physical and emotional security, which, in turn, rewards them with affection and reassurance[20–22]. Through these early interactions, infants learn and internalize the relationship between rewarding stimuli and appropriate behaviours. A large body of evidence suggests that the dorsal striatum (caudate and putamen) plays an important role in such reward-related learning behaviours[23,24]. For example, the dorsal striatum is thought to maintain information linking rewards to the appropriate actions[25] and causal evidence shows that stimulation of the caudate nucleus directly strengthens reward-related learning[26]. Striatal associative learning is thought to be optimal during reward anticipation, reinforcing behaviours that have the potential to determine a reward[27,28], and supporting habitual and goal-directed behaviours[29,30].

Against the background of this literature, several studies support the association between maternal bonding behaviours and the striatum. In a longitudinal cohort study, early maternal care (characterized by affectionate, engaging, and responsive interactions at 3 months) has been shown to shape reward-related brain function in children and may buffer familial risk for psychopathology[18]. Low maternal care on the PBI has been linked to greater dopamine release following acute stress[31] and altered behavioural responsivity to reward after methylphenidate (a dopamine agonist) administration[32]. Similarly, maternal separation was shown to affect the responsiveness of dopamine neurons in the striatum[33]. Furthermore, maternal emotional neglect or cold parenting during childhood was shown to affect reward learning[21,34] and reward anticipation[35] in adolescence or adulthood. At a familial level, healthy collaborative co-parenting was associated with efficient top-down regulation through caudate-prefrontal connectivity[36]. Ample evidence also links the striatum to other forms of adverse childhood experiences. For example, volume differences in the dorsal striatum (caudate) have been associated with adversity reports identified in childhood trauma questionnaires in adults and adolescents[37–40]. Furthermore, reports of harsh parental discipline have been linked to caudate reductions in children[41] and increased T2 relaxation time in the right caudate and putamen in young adults[42].

In later life, negative mental and cognitive outcomes may reflect lasting effects of early stress on neural systems, driven by chronic glucocorticoid exposure[43–46]. Brain regions with prolonged developmental trajectories may be especially vulnerable; notably, the dorsal striatum undergoes substantial changes from childhood into adulthood[47,48]. Adversity-related alterations, particularly in affective and reward networks, may disrupt typical development and persist long after exposure, potentially remaining observable in later life, with implications for increased dementia risk[43,45,48–50]. These changes in the dorsal striatum may also have effects on psychological well-being. Indeed, high maternal control and low care have been linked to later-life mental health outcomes[51–53], which in turn relate to alterations in dorsal striatal function and volume in older individuals[54,55]. Together, this evidence lends support to the idea that dimensions of maternal bonding may impact the structural integrity of the dorsal striatum, leaving lasting signatures in older age. To the best of our knowledge, studies have not yet investigated this association in an older population sample.

In this study, we used a cross-sectional, region-of-interest design to examine associations between perceived maternal care, autonomy, and control (as measured by the PBI) and dorsal striatal volumes (bilateral caudate and putamen) in an older birth cohort. If early maternal interaction shapes reward-related brain development, evidence of its effects in later life may underscore the enduring impact of early caregiving across the lifespan.

Methods

Study sample

Data from the "Insight 46" neuroimaging sub-study of the MRC National Survey of Health and Development (NSHD) 1946 birth cohort was used for this analysis. The NSHD cohort recruited 5362 individuals born during the same week, in March 1946 in England, Scotland, and Wales with follow-up assessments since birth. Members were singleton births from married parents[56–58]. The neuroimaging sub-study was designed to investigate factors impacting late-life brain health, recruiting 502 NSHD members who were selected at random following a study visit at age 60–64 and for whom relevant life course data was available[59]. This cohort allows examination of potential long-term associations between early life experiences and brain structure in older age. Ethical approval for the NSHD study was provided by Research Ethics Committees in England and Scotland. The neuroimaging sub-study obtained ethical approval by the National Research Ethics Service (NRES) Committee London (REC reference 14/LO/1173). Participants provided written informed consent for their participation[56,57,59]. The study was conducted in accordance with the Declaration of Helsinki.

Of the 468 participants' neuroimaging data that was available for this analysis, sixteen were removed due to reported history of neurological events (before the age of 63), existing outlier values in the imaging phenotypes of interest and following QC of the brain extraction result on the T1-weighted images. The final sample included 452 participants and the degree of missingness within each variable of interest is reported in Table 1. Analyses were conducted using listwise deletion, therefore, the total number of participants for each main statistical test is reported below. MRI scanning for the selected participants occurred between mid-2015–early-2018 when participants were 69 to 71 years old (M = 70.17; SD = 0.74; Table 1).

Maternal bonding

The Parental Bonding Instrument – PBI[15] was used to assess perceived maternal attitudes in NSHD responders up to the age of 16 years, based on 24 items and Likert responses on a 1–4 scale: very like-very unlike. The instrument is designed for both mothers and fathers. The current study is concerned with the maternal role in child development and therefore only the maternal scale was investigated.

Psychometric studies of the PBI show good to excellent internal consistency[15,60]. While the original study proposed two factors for the PBI, i.e., maternal care and control[15], many subsequent large-scale studies supported a three-factor solution that separate between maternal care (e.g.: "*Could make me feel better when I was upset*"), control (also called "overprotection", e.g.: "*Tried to control everything I did*") and autonomy (also called "non-engagement" or "authoritarianism", e.g.: "*Gave me as much freedom as I wanted*")[60–64]. In the NSHD sample, factor analyses of PBI data also support the three-factor solution with reference to the following scales: maternal care, control and non-engagement/autonomy[61,64]. The current analysis was conducted on summed item scores pertaining to these scales following confirmation of previously reported dimensionality within our (smaller) sample (Table S1). "Not applicable" and "Unknown" responses were set to missing and sum scores for each scale were calculated only where all item responses were present. Items were adjusted for consistent directionality within scales, and therefore higher scores suggest that an individual is higher on the characteristic.

The PBI was administered to participants in 1989, when they were 43 years old. Given the retrospective nature of this assessment, recall bias and potential false negative reports cannot be ruled out[65,66]. However, the PBI was demonstrated to have long-term stability over 20 years in a non-clinical sample, with limited influence of life experiences, depression history and state mood on perceived parenting[16]. Furthermore, adverse mental health outcomes, have been consistently associated with maternal parenting experiences in adults based on retrospective data from the PBI, suggesting that this questionnaire identifies meaningful early experiences[60,62,67].

Adult well-being

Well-being was measured using the Warwick-Edinburgh Mental Well-being Scale (WEMHS)[68] via a postal questionnaire sent to participants in 2014 at the age of 68 years, therefore close to the time when scanning occurred[57]. The scale includes 14 items measuring positive affect experienced during the 2 weeks prior to completion. Likert responses were coded 1–5 (none of the time – all of the time, e.g.: "*I've been feeling optimistic about the future*"), and higher scores suggest greater well-being. The measure has a one-factor dimension, and it showed excellent internal consistency in the NSHD sample when calculated on WEHMS data also collected from participants four-eight years prior[53]. The computed score made available to researchers as a derived variable was used in this analysis. Here, where more than 4 items were missing, the total score was not calculated.

MRI protocol and structural analysis

Data acquisition is described elsewhere[59]. In brief: brain imaging was performed using a Biograph mMR 3 Tesla PET/MRI scanner (Siemens Healthcare) to simultaneously acquire both dynamic amyloid PET and MRI data, including high resolution 3D T1-weighted, T2-weighted and FLAIR volumetric scans, among other MRI sequences such as resting state functional MRI. The T1-weighted images used here were obtained using a Magnetization Prepared Rapid Gradient Echo Imaging (MPRAGE) sequence with the following parameters: (MPRAGE): Repetition Time (TR) = 2000 ms; Echo Time (TE) = 2.92 ms; Inversion Time (TI) = 870 ms; flip angle = 8°; voxel size resolution = 1.1 mm^3 isotropic. Images underwent manual QC, including issues related to motion, coverage, blurring, contrast.

Data analysis was carried out using FSL tools[69]. Pre-processing for structural images included the following steps: (i) re-orienting images to the standard (MNI) template, (ii) bias field correction, (iii) brain extraction and (iv) brain tissues segmentation using FMRIB's Automated Segmentation Tool (FAST) that allows extracting global measures of total grey matter (GM), white matter (WM) and cerebrospinal fluid (CSF)[70]. Automated segmentation of the left and right caudate and putamen was performed using the FMRIB Integrated Registration

	Maternal care					Maternal autonomy					Maternal control				
	Low	**Medium**	**High**	**Statistic**	***N***	**Low**	**Medium**	**High**	**Statistic**	***N***	**Low**	**Medium**	**High**	**Statistic**	***N***
N	81	178	142		401	86	159	169		414	104	183	126		413
PBI score range	14–34	35–42	43–48			9–17	18–21	22–28			5–6	7–10	11–20		
Parental Bonding Instrument – Mother questionnaire															
Care	29.72 (4.23)	38.89 (2.15)	45.42 (1.76)	$\chi^2(2) = 344.9$, ***p*<.001**	401	34.75 (7.06)	38.46 (5.05)	42.58 (4.88)	$\chi^2(2) = 95.7$, ***p*<.001**	395	42.48 (5.25)	39.32 (5.65)	36.8 (6.72)	$\chi^2(2) = 49.7$, ***p*<.001**	393
Autonomy	17.6 (3.63)	20.18 (3.1)	22.57 (3.09)	$F(2,398) = 63.5$, ***p*<.001**	401	15.35 (1.93)	19.64 (1.05)	24.09 (1.80)	$\chi^2(2) = 360.3$, ***p*<.001**	414	22.8 (3.24)	20.44 (3.31)	18.37 (3.55)	$F(2,410) = 49.2$, ***p*<.001**	413
Control	10.85 (3.46)	9.11 (2.97)	8.05 (2.95)	$\chi^2(2) = 37.9$, ***p*<.001**	393	11.27 (3.58)	9.52 (2.93)	7.57 (2.51)	$\chi^2(2) = 75.5$, ***p*<.001**	402	5.41 (0.49)	8.35 (1.06)	13.19 (2.05)	$\chi^2(2) = 361.9$, ***p*<.001**	413
Participant demographics and relevant adulthood characteristics															
Gender (% female)	49.38	42.7	52.11	$\chi^2(2) = 3$, $p = .226$	452	60.47	40.88	50.89	$\chi^2(2) = 9$, ***p*=.011**	452	45.19	43.17	57.94	$\chi^2(2) = 7$, ***p*=.030**	452
Age	70.09 (0.71)	70.21 (0.72)	70.09 (0.8)	$F(2,398) = 1.2$, $p = .290$	452	70.12 (0.69)	70.18 (0.77)	70.14 (0.74)	$F(2,411) = 0.2$, $p = .822$	452	70.11 (0.76)	70.09 (0.77)	70.26 (0.69)	$F(2,410) = 2.3$, $p = .107$	452
Townsend	− 0.60 (3.18)	−1.23 (2.73)	− 0.79 (2.71)	$\chi^2(2) = 4.9$, $p = .088$	391	−1.08 (2.59)	−1.04 (2.78)	− 0.71 (2.79)	$\chi^2(2) = 1.8$, $p = .414$	403	− 0.88 (2.29)	− 0.94 (2.91)	− 0.8 (3.12)	$\chi^2(2) = 1.2$, $p = .539$	402
APOE (% ϵ4 carrier)	25.71	27.22	25	$\chi^2(2) = 0.2$, $p = .912$	352	26.76	27.14	24.34	$\chi^2(2) = 0.3$, $p = .847$	363	26.09	30.12	20.75	$\chi^2(2) = 2.9$, $p = .231$	364
BMI	27.7 (4.44)	27.41 (4.54)	27.97 (4.80)	$\chi^2(2) = 1.6$, $p = .448$	396	27.11 (4.78)	28.12 (4.71)	27.29 (4.32)	$\chi^2(2) = 4.9$, $p = .088$	409	26.94 (3.97)	27.71 (4.47)	28.01 (5.07)	$\chi^2(2) = 2.3$, $p = .318$	408
GM	38.14 (1.59)	38.46 (1.59)	38.28 (1.95)	$F(2,398) = 1.1$, $p = .340$	401	38.52 (1.73)	38.33 (1.59)	38.31 (1.93)	$F(2,411) = 0.5$, $p = .633$	414	38.30 (1.82)	38.25 (1.73)	38.42 (1.68)	$F(2,410) = 0.4$, $p = .701$	413
WM	36.67 (1.51)	36.76 (1.41)	36.68 (1.46)	$F(2,398) = 0.2$, $p = .849$	401	36.52 (1.60)	36.72 (1.36)	36.82 (1.45)	$F(2,411) = 1.3$, $p = .280$	414	36.71 (1.43)	36.84 (1.41)	36.59 (1.53)	$F(2,410) = 1.2$, $p = .316$	413
CSF	25.19 (2.15)	24.78 (2.15)	25.04 (2.55)	$F(2,398) = 1.1$, $p = .347$	401	24.96 (2.44)	24.94 (2.07)	24.87 (2.56)	$F(2,411) = 0.1$, $p = .941$	414	24.98 (2.46)	24.91 (2.37)	24.99 (2.23)	$F(2,410) = 0.1$, $p = .942$	413
Early life factors															
Birthweight (kg)	3.35 (0.48)	3.45 (0.47)	3.46 (0.52)	$F(2,397) = 1.5$, $p = .235$	400	3.38 (0.51)	3.43 (0.52)	3.46 (0.45)	$F(2,410) = 0.7$, $p = .494$	413	3.44 (0.42)	3.48 (0.48)	3.34 (0.53)	$\chi^2(2) = 5.1$, $p = .077$	412
Pubertal growth	− 0.06 (0.22)	− 0.04 (0.26)	− 0.003(0.26)	$F(2,398) = 1.6$, $p = .205$	401	− 0.08 (0.21)	− 0.03 (0.27)	− 0.02 (0.25)	$F(2,411) = 1.4$, $p = .237$	414	− 0.06 (0.26)	− 0.01 (0.26)	− 0.06 (0.24)	$F(2,410) = 2.1$, $p = .125$	413
Social class of father	56.06	65.43	63.2	$\chi^2(2) = 1.8$, $p = .412$	353	57.89	63.24	64.43	$\chi^2(2) = 0.9$, $p = .620$	361	60.67	64.02	60.55	$\chi^2(2) = 0.4$, $p = .800$	362
Social class at 22yo	98.68	91.98	92.54	$\chi^2(2) = 4.2$, $p = .122$	372	97.47	91.84	93.04	$\chi^2(2) = 2.8$, $p = .251$	384	92.93	94.05	93.91	$\chi^2(2) = 0.1$, $p = .931$	382
Mental Health															
Well-being	52.65 (7.4)	55.45 (7.15)	56.21 (7.21)	$F(2,381) = 6.3$, ***p*=.002**	384	53.41 (6.84)	54.57 (7.65)	56.6 (6.87)	$F(2,393) = 6.2$, ***p*=.002**	396	57.46 (7.21)	54.85 (7.15)	53.69 (7.03)	$F(2,392) = 7.9$, ***p*<.001**	395
Cognition															

Continued

	Maternal care					Maternal autonomy					Maternal control				
	Low	**Medium**	**High**	**Statistic**	***N***	**Low**	**Medium**	**High**	**Statistic**	***N***	**Low**	**Medium**	**High**	**Statistic**	***N***
MMSE	29.31 (0.77) [27–30]	29.26 (1.09) [22–30]	29.25 (0.99) [23–30]	$\chi^2(2) = 0.3$, $p = .853$	401	29.43 (0.68) [27–30]	29.12 (1.22) [22–30]	29.33 (0.86) [26–30]	$\chi^2(2) = 2.8$, $p = .243$	414	29.12 (1.11) [23–30]	29.23 (1.04) [22–30]	29.41 (0.85) [25–30]	$\chi^2(2) = 5.1$, $p = .077$	413
Word-list memory test	24.05 (5.29)	23.9 (5.96)	23.57 (5.94)	$F(2,389) = 0.2$, $p = .818$	392	24.32 (5.94)	23.42 (5.86)	23.88 (5.68)	$F(2,402) = 0.7$, $p = .503$	405	23.82 (6.47)	23.82 (5.49)	23.99 (5.58)	$F(2,401) = 0.04$, $p = .962$	404

Table 1. Sample characteristics. Note. Unless otherwise specified, descriptive statistics refer to means and standard deviations: M(SD). For clarity of interpretation, table refers to brain volumes adjusted for total brain volume only (brain structure/total brain volume*100). Statistics refer to simple group differences (one-way ANOVAs, Kruskall-Wallis, chi^2 tests as applicable) conducted list-wise (results in bold are significant); Age refers to age at the time of scanning; Townsend Index refers to economic status at the age of 53 years old; BMI = Body Mass Index; BMI refers to measurements done when participants were 69 years old; GM = Grey Matter; WM = White Matter; CSF = Cerebrospinal fluid; Pubertal growth refers to velocity of growth and is estimated for ages up to 15 years old using a growth curve analysis (Super-Imposition by Translation And Rotation) that relates pubertal growth to bone health in later life[82]; Social class of father when child was 15 years old corresponds to class of occupations in 1970 (% skilled non manual: professional, intermediate, skilled non-manual); Social class when participants were 22 years old was used as a proxy for education (% skilled non-manual: foreman, professional, middle non-manual, middle – rest incl. officers, skilled – forces, ranks); Well-being was measured using the WEMHS[68], which was self-administered when participants were 68 years old[57]; MMSE was assessed at the time of MRI scanning and descriptives above include M(SD) and [range]; The word-list memory test was assessed at the age of 69 years old using a typical recall task (remembering as many words as possible from a previously presented set); the variable was available as a derived score representing the sum of three word-list memory test trials, where each trial was scored for the total number of different correct words recalled (and therefore higher scores represent better memory)[59,83].

and Segmentation Tool (FIRST)[71]. Derived masks were visually inspected. Resulting tissue-type and subcortical segmentations were normalized to the whole brain volume (brain structure/total brain volume*100).

Statistical analysis

All analyses were conducted on the Dementias Platform UK Data Portal[72]. The statistical analysis was carried out using Stata Version 18.0 SE (Appendix S1).

First, we determined the number of PBI factors within our analysis sample using Horn's parallel analysis and the point of inflection in scree plots[73], followed by a confirmatory factor analysis (CFA) with a latent variable organization in a structural equation model to determine the factor loadings for each item. The Satorra-Bentler maximum likelihood estimator is considered robust for ordinal-level data, and therefore it was applied here[74]. Goodness-of-fit was assessed based on the following indices: Root Mean Square Error of Approximation (RMSEA), Comparative Fit Index (CFI), Tucker Lewis Index (TLI), Standardized Root-Mean Residual (SRMR). Values ≥ 0.95 for TLI and CFI, < 0.07 for RMSEA, and < 0.08 for SRMR are considered to show a good model fit[75,76]. Cronbach's alpha assessed internal consistency and average inter-item correlations (AIC). Cronbach's alpha > 0.70 indicates good internal consistency and AIC is generally expected to be moderate, ranging between 0.15 and 0.50[77].

Second, using the resulting factors, sample characteristics were examined by comparing participants grouped by levels of maternal care, control, and autonomy across multiple relevant variables. As per previous approaches[31,78], k-means clustering with a set seed was used to group the scores on each maternal sub-scale into three groups: low, medium and high care/autonomy/control, which allowed comparison across demographic and biological variables, and exploration of potential threshold effects. Clusters covered between 20 and 44% of the participant scores (Table 1). Additionally, extreme cases of maternal practices were investigated (10th vs. 90th percentiles), considering that any potential differences if present, would persist after a long follow-up. To ensure robustness, we also ran supplementary regressions using continuous PBI scores. Group differences (k-means clusters) on multiple variables were assessed using one-way ANOVAs, Kruskall-Wallis or chi^2 tests, where applicable. There were no significant group differences on age, adult socio-economic status, social class during childhood and early adulthood, birthweight, pubertal growth, genetic risk for Alzheimer's dementia (ApoE4 carrier status), overall brain volumes, body mass index, and cognition around the time of scanning (Table 1). Sex was significantly different across groups, and together with scores on the Mini Mental State Examination (MMSE), these variables were regressed out of the outcome variables and the resulting standardized residuals were included in the subsequent analyses. De-confounding for MMSE was conducted given the participants' age as well as evidence that the rate of cognitive decline is larger after 65 years in the general population[79]. Supplementary analyses controlling for additional confounds are also reported. Furthermore, analyses also explored potential sex differences, given reported sex-specific associations between maternal factors and subcortical structures[8]. For readability, means and standard deviations are reported for unadjusted dependent variables (brain volumes adjusted for total volume only). Finally, the absolute deviation around the median with a 3.5 threshold was used for outlier exclusion on the brain volume measures prior to de-confounding[80,81].

Finally, differences in the bilateral caudate and putamen volumes, as well as well-being, were simultaneously assessed in separate MANOVA's with each of the three maternal sub-scales as the predictors, using the Wilk's λ result. The significance level was set at 0.017 (α = 0.05/3). The same threshold was considered for the univariate tests following a significant MANOVA, and any subsequent pairwise comparisons were also Bonferroni corrected within each test. The assumption of equality of covariance matrices was met for all scales (Box's test: $p > .05$). Multivariate analyses were followed up by univariate tests (*mvreg*) and subsequently by Bonferroni corrected pairwise comparisons on the resulting coefficients (*pwcompare*), as well as by bootstrapped comparisons of means with 1000 replications. Homogeneity of variance was assessed using Levene's test prior to all univariate analyses (all $p > .05$). Partial eta-squared (η_p^2) was reported to describe effect sizes for both multivariate and univariate tests, and SPSS (IBM, Armonk, NY, USA) was used for this purpose. All analyses were conducted list-wise by modelling the full information datasets (Table 1).

Results

Parental bonding instrument

Horn's parallel analysis ran 690 iterations and yielded a three-factor solution with adjusted eigenvalues larger than 1 (eigenvalues 1–3: 7.50, 2.64, 1.48) (Table S1; Figure S1). This result was consistent with previous reports[61,64] and therefore a CFA was conducted using the same item groupings to assess individual loadings (N = 388 without missing values). Standardized factor loadings ranged from small (0.35: "*Let me do the things I liked doing*") to large (0.85: "*Let me decide things for myself*") with medium-large average loadings for maternal care, autonomy, and control: 0.67, 0.55, and 0.68, respectively. Goodness-of-fit was adequate: RMSEA = 0.06, CFI = 0.91, TLI = 0.89, SRMR = 0.06, and errors were allowed to correlate for two items ("*Gave me as much freedom as I wanted*" and "*Let me go out as often as I wanted*"). Internal consistency was high for care (α = 0.91; AIC range: 0.45-0.50), autonomy (α = 0.80; AIC range: 0.34-0.43), and control (α =. 81; AIC range: 0.44-0.48) (Table S1). As expected, maternal care correlated positively with autonomy (r =.511) and negatively with control (r=-.319). Control also correlated negatively with autonomy (r = −.493).

Study sample characteristics

Table 1 shows relevant sample characteristics, including simple group differences across the three scales (also see Table S2 for overall sample characteristics). Significant group differences between care, autonomy and control groups across all three maternal bonding scales showed that group separation was successful (all $p < .001$). Well-being was also significantly different across groups and its contribution was assessed inferentially in the subsequent analyses (all $p < .0023$). Across all other characteristics, there were no significant group differences

(all $p > .077$), except for gender where more females were present in the low autonomy and the high control groups ($p < .030$).

Multivariate statistical analyses

The left and right caudate and putamen volumes as well as well-being scores were included simultaneously in three separate one-way multivariate analyses to determine the effects of maternal care, autonomy and control on $N = 384$, 396, and 395, respectively. There was a significant effect of control: $F(2,392) = 2.66$, $p = .003$, $\eta_p^2 = 0.033$. There were no significant effects of care ($F(2,381) = 1.78$, $p = .061$, $\eta_p^2 = 0.023$) or autonomy ($F(2,393) = 1.78$, $p = .061$, $\eta_p^2 = 0.022$).

Follow-up univariate tests on the maternal control scale were therefore conducted. Results were significant for well-being ($F(2,392) = 7.52$, $p < .001$, $\eta_p^2 = 0.037$) and marginally for the right caudate ($F(2,392) = 3.90$, $p = .021$, $\eta_p^2 = 0.019$). First, comparisons of the estimated margins showed that well-being was higher in the low control group ($M = 57.46$, $SD = 7.21$) compared to medium ($M = 54.85$, $SD = 7.15$) and high ($M = 53.69$, $SD = 7.03$), (medium vs. low: $p = .011$; high vs. low: $p = .001$) (Tables S3, S4). Robustness of differences was further assessed using bootstrapped comparisons of means, which mirrored these results (and the direction of effects), revealing a significant difference between low and medium ($p = .001$, 95% CI: 0.12, 0.49) and between high and low control groups ($p = .019$, 95% CI: −0.36, −0.03) on well-being scores (note that CI sign reflects contrast order, directionality remained consistent). Second, for the right caudate, follow-up comparisons were also conducted given the marginal effect, as well as the assumption that any long-term effects in an older population might be small, if detectable. Pairwise differences of the estimated marginal means showed that right caudate volumes were larger in the low ($M = 0.25$, $SD = 0.03$) compared to the high ($M = 0.24$, $SD = 0.02$) control groups; however, the result remained marginal ($p = .023$) (Fig. 1). Similarly, simple bootstrapped comparisons confirmed a significant difference between the high and low groups on the right caudate volumes, $p = .009$, 95% CI [−0.37, −0.05]. For comprehensiveness, univariate tests and follow-up pairwise comparisons for maternal care and autonomy are also reported in supplementary materials. One-way ANOVAs showed significant differences for well-being only (Table S4; $p = .002$).

We also conducted supplementary regression analyses using the continuous PBI scores. Results were largely consistent, with maternal control showing negative associations with the right caudate (β=−0.13, $p = .007$), left and right putamen (β=−0.12, $p = .013$; β=−0.13, $p = .010$) and well-being (β=−0.2, $p < .001$). Maternal care and

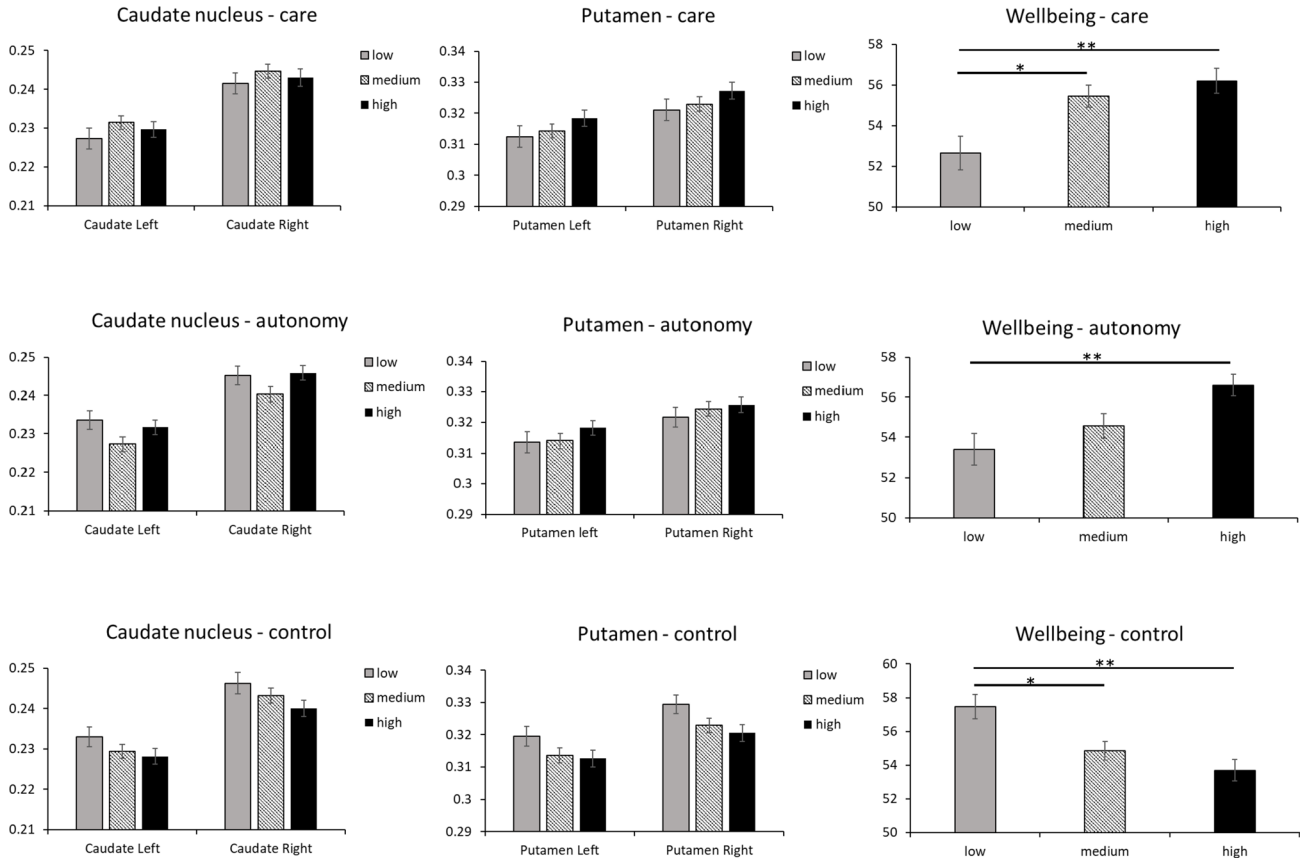

Fig. 1. Subcortical brain volumes and well-being scores across three maternal control groups. **Note.** Table shows subcortical brain volumes adjusted for total brain volume only; Well-being scores are unadjusted. Error bars show standard errors. *$p < .02$, **$p < .01$.

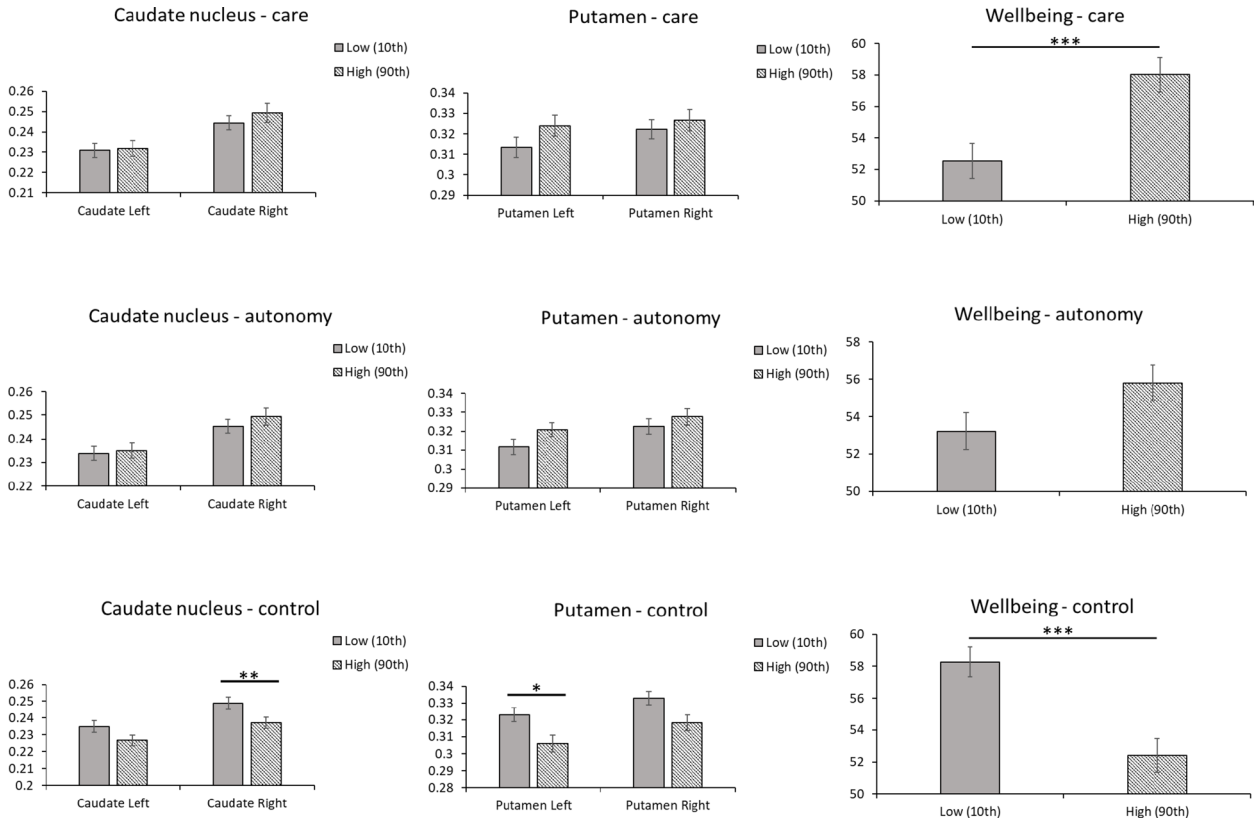

Fig. 2. Subcortical brain volumes and well-being scores across two extreme maternal control groups. **Note.** Table shows subcortical brain volumes adjusted for total brain volume only; Well-being scores are unadjusted. Error bars show standard errors. *p <.02, **p <.01, ***p <.001.

autonomy were not associated with striatal volumes (all p >.113), but were positively associated with well-being (both p <.006) (Table S5).

Furthermore, moderation was additionally assessed using multiple MANOVAs and specifying the interaction between each maternal scale separately and well-being (also grouped here using k-means clustering). All interaction terms were non-significant (all p >.873), suggesting that marginal brain volume differences in the right caudate were present independently of well-being status. In addition, no significant correlations were observed between well-being and dorsal striatal volumes (all r <.08) (Table S6). Finally, to assess sex-specific effects, multivariate models were re-run, including interaction terms between sex and each maternal bonding scale (care, autonomy, and control), adjusting for MMSE. None of the interactions reached significance at the adjusted alpha (all p >.031). For completeness, sex-stratified follow-up tests were also conducted, which revealed no consistent sex-specific differences in the effects of maternal bonding on brain volumes. Notably, with reduced power, the previously marginal effect on right caudate volume was non-significant (Tables S7 – S9).

Stratified analyses

Separate MANOVAs assessed bilateral caudate and putamen volumes as well as well-being simultaneously, comparing the 10th and 90th percentile scores on each of the three PBI scales. We found a significant effect of maternal control, N = 104, F(1,102) = 5.25, p <.001, η_p^2 = 0.211 and maternal care, N = 91, F(1,89) = 3.02, p =.015, η_p^2 = 0.151. There was a non-significant effect of maternal autonomy, N = 111, F(1,109) = 1.39, p =.235, η_p^2 = 0.062. Univariate tests showed that participants scored higher on well-being in the high versus low maternal care groups (high: M = 58.02, SD = 7.21; low: M = 52.53, SD = 7.61; F(1,89) = 12.66, p <.001, η_p^2 = 0.125). There were no significant differences in brain volumes between maternal care extreme groups (p <.244). Differences in maternal control were found for the right caudate (low: M = 0.25, SD = 0.03; high: M = 0.24, SD = 0.02; F(1,102) = 8.79, p =.004, η_p^2 = 0.079), left putamen (low: M = 0.32, SD = 0.03; high: M = 0.31, SD = 0.03; F(1,102) = 6.26, p =.014, η_p^2 = 0.058), well-being (low: M = 58.27, SD = 7.18; high: M = 52.41, SD = 6.96; F(1,102) = 16.95, p <.001, η_p^2 = 0.142), and marginally for the left caudate (low: M = 0.235, SD = 0.03; high: M = 0.227, SD = 0.02; F(1,102) = 5.79, p =.018, η_p^2 = 0.054) and the right putamen (low: M = 0.33, SD = 0.03; high: M = 0.32, SD = 0.03; F(1,102) = 5.39, p =.022, η_p^2 = 0.050). Direction of differences mirrored previous results with smaller brain volumes and lower well-being scores in the high control group (Fig. 2). Separate moderation tests also showed that there were no interactions between the extreme PBI groups and clustered well-being (all p >.228). Full statistics for all scales are presented in supplementary materials (Tables S10, S11).

Furthermore, additional analyses on outcomes corrected for all relevant variables in Table 1 yielded largely consistent results: no effects for care or autonomy, with persistent maternal control effects for the right caudate (p =.004), while all well-being associations remained significant (see Supplementary Tables S12-S13 for further information and analyses on both k-means and extreme groupings). Similarly, models including sex × (extreme) group interaction terms yielded no significant interactions (all p >.525). However, sex-stratified analyses indicated that females reporting high maternal control had marginally smaller right caudate volumes compared to those with low control (p =.018), with no corresponding effect in males (again, noting small subgroup sizes) (Tables S7-S9).

Discussion

Our results showed that in an older population sample of cognitively healthy men and women, retrospective extreme maternal control was associated with smaller right caudate and left putamen volumes, with marginal associations for the left caudate and right putamen. No significant associations were found between dorsal striatal volumes and maternal care or autonomy, either across the full sample or when comparing extreme groups. Perceived maternal bonding characterised by high care and autonomy alongside low control was associated with higher self-reported well-being both across the full sample and when comparing extreme groups. Supplementary analyses using continuous scores, as well as analyses controlling for additional confounds, yielded largely consistent results (Tables S5, S12-S13).

The maternal control scale of the PBI measures the extent to which a mother was intrusive, infantilizing, overprotective, monitoring, denying of psychological and behavioural autonomy[17]. Affection from caregivers is a key developmental reward, and early interactions shape children's motivation to seek proximity, comfort, and security through goal-directed behaviours[20–22]. When such rewards are conditional, inconsistent, or ambiguous, children may be less motivated to pursue proximity and attachment. Disruptions in these early motivational patterns may contribute to alterations in reward-related neurodevelopmental pathways, including structural differences in the caudate and putamen[20,21,36,84]. This interpretation aligns with neuroimaging evidence linking cold/hostile maternal behaviour to increased caudate activation during reward processing[21], and with findings suggesting intergenerational similarities in putamen response to reward[85]. In line with these findings, maternal control has also been implicated in mental health difficulties, especially when combined with low maternal care. Parenting characterised by high control and low care is associated with increased symptoms of anxiety and depression in both children and adults[67,86,87]. However, maternal control appears to be a more robust predictor than maternal care for outcomes such as anxiety[88,89], mid-life well-being[61], eating disorders[90], and externalizing disorders[62,91–94]. Broader family dynamics, such as coparenting style, have also been shown to influence child externalizing behaviours. For instance, collaborative coparenting predicts lower externalising symptoms in children, whereas undermining coparenting has been linked to increased salivary vasopressin, a hormone implicated in aggression[36]. Authoritarian parents who prioritise compliance and control[17], are more likely to use punitive discipline[95], which in turn increases the likelihood of child externalizing behaviours[96].

Our results did not identify significant associations between maternal care or autonomy and dorsal striatal volumes. Care and control (the inverse of autonomy on the PBI) are conceptually distinct parenting dimensions with potentially different effects on development[97–99]. Low care has been linked to depression, and control more consistently to anxiety[86–88]. Furthermore, studies on stress regulation suggest distinct physiological profiles related to hypothalamic-pituitary-adrenal (HPA) axis functioning relative to this dichotomy[98,99]. Whether these physiological differences translate to dorsal striatal structure remains unclear, although cortisol may influence dopamine release[31,100]. Care fosters exploration and goal-directed behaviour, while low care neglects these behaviours; in contrast, control may promote avoidance and limit stress-adaptation strategies[86,98]. Finally, the lack of observed associations may reflect the long follow-up interval or involvement of brain regions outside our region-of-interest.

While sex × maternal bonding interactions were not significant, exploratory sex-stratified analyses revealed a marginal association between high maternal control and smaller right caudate volume in females within the extreme groups. Although inconclusive, this warrants further investigation in larger samples. Females may exhibit greater dopamine release to low-intensity stimuli[101], and maternal control may predict anxiety more strongly in females[88]. Notably, striatal changes following early adversity may be more pronounced in males[102,103]. If present, such sex-dependent differences were likely underpowered in our sample.

Finally, across all PBI dimensions, our findings revealed associations with later-life well-being, aligning with the broader literature linking early parental bonding with psychological adjustment across the lifesp an[15,51,52,88,89]. These results underscore the potential long-term influence of early caregiving experiences on both neurobiological structure and subjective well-being in older adulthood, at a population level, emphasizing the importance of considering early life experiences when assessing later life well-being.

Taken together, our findings suggest that perceived excessive maternal control, reflecting overprotective and autonomy-limiting parenting, may contribute to enduring alterations in reward-related neurodevelopment and well-being across the lifespan. High maternal control may chronically limit children's opportunities for independent reward-based learning and exploration[17]. Such chronic stress may disrupt brain development and increase vulnerability to internalizing psychopathology, such as anxiety and depression[86,87]. Clinically, these findings highlight the importance of autonomy-supportive caregiving that balances structure and guidance with autonomy, consistent with evidence-based interventions targeting parental overcontrol, to reduce child anxiety and build resilience[104]. Given the observed long-term associations, further research should clarify their developmental trajectories and evaluate whether interventions during critical developmental windows could reduce associated psychological risks.

There are several important limitations to our study. First, while our analysis was conducted on a longitudinal population sample, our outcome variables were measured cross-sectionally, and the parental bonding information

was acquired retrospectively. This implies that causality cannot be inferred and recall bias cannot be excluded[66,105]. Second, although our study matched groups across multiple confounding variables and additionally controlled for other relevant variables, nonetheless confounder bias cannot be ruled out, including bias associated with variables that were unavailable to us (e.g., striatal size at birth)[106]. Third, while the PBI was administered at age 43, it is important to recognise that the mother-child relationship may evolve dramatically over time, and such changes were not captured in our study. In addition, the 26-28-year gap between the completion of the PBI and the brain measurements introduces the influence of other life factors on brain morphology, such as a variety of health issues or other socio-economic dynamics. These shifts could impact well-being and brain structure in older age, potentially introducing variability in the results that was not accounted for by the PBI measure administered at a single time point. We also acknowledge the potential for healthy survivor bias, as participants imaged in later life may represent a healthier, more socioeconomically advantaged, or resilient subset of the original cohort. This may limit generalisability to the broader aging population, and it is particularly relevant given evidence of accelerated dorsal striatal shrinkage with advancing age[107]. Finally, analyses using listwise deletion excluded cases and may have introduced bias if data were not missing completely at random.

Conclusion

Our study showed that in a large population sample, extreme retrospective perceptions of maternal control were associated with smaller dorsal striatal volumes and lower well-being at 70 years old. The absence of significant associations between dorsal striatal volumes and maternal care or autonomy highlights the complex relationships between early life experiences and later brain structure[17]. Clinical guidelines that address internalized parental control patterns, promote emotional autonomy, and consider children's perceived control may inform mechanistically-tailored psychological treatments.

Data availability

Data used in this publication are available to bona fide researchers upon request to the NSHD Data Sharing Committee via a standard application procedure. Further details can be found at http://www.nshd.mrc.ac.uk/data. doi: https://doi.org/10.5522/NSHD/Q101; doi: https://doi.org/10.5522/NSHD/Q102; doi: https://doi.org/10.5522/NSHD/Q103.

Received: 9 May 2025; Accepted: 15 September 2025
Published online: 17 October 2025

References

1. De Bellis, M. D. et al. Developmental traumatology. Part II: brain development. *Biol. Psychiatry*. **45**, 1271–1284 (1999).
2. Vaidya, N., Marquand, A. F., Nees, F., Siehl, S. & Schumann, G. The impact of psychosocial adversity on brain and behaviour: an overview of existing knowledge and directions for future research. *Mol. Psychiatry*. https://doi.org/10.1038/s41380-024-02556-y (2024).
3. Lupien, S. J., Juster, R. P., Raymond, C. & Marin, M. F. The effects of chronic stress on the human brain: from neurotoxicity, to vulnerability, to opportunity. *Front. Neuroendocrinol.* **49**, 91–105 (2018).
4. Künzi, M., Gheorghe, D. A., Kliegel, M., Ballhausen, N. & Gallacher, J. Cumulative life course adversity, mental health, and cognition in the UK biobank. *Sci. Rep.* **12**, 14700 (2022).
5. Nelson, C. A. et al. Adversity in childhood is linked to mental and physical health throughout life. *BMJ* **371**, 1–9 (2020).
6. McEwen, B. S. & Gianaros, P. J. Central role of the brain in stress and adaptation: links to socioeconomic status, health, and disease. *Ann. N Y Acad. Sci.* **1186**, 190–222 (2010).
7. Danese, A. & McEwen, B. S. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiol. Behav.* **106**, 29–39 (2012).
8. Hashempour, N. et al. Prenatal maternal depressive symptoms are associated with neonatal left amygdala microstructure in a sex-dependent way. *Eur. J. Neurosci.* **57**, 1671–1688 (2023).
9. Wade, M. et al. Blunted stress reactivity as a mechanism linking early psychosocial deprivation to psychopathology during adolescence. *Nat. Ment Heal.* **2**, 703–711 (2024).
10. Kessler, R. C. et al. Childhood adversities and adult psychopathology in the WHO world mental health surveys. *Br. J. Psychiatry*. **197**, 378–385 (2010).
11. Champagne, F. & Meaney, M. J. Like mother, like daughter: evidence for non-genomic transmission of parental behavior and stress responsivity. *Prog Brain Res.* **133**, 287–302 (2001).
12. Cicchetti, D. Resilience under conditions of extreme stress: A multilevel perspective. *World Psychiatry*. **9**, 145–154 (2010).
13. Luecken, L. J. Childhood attachment and loss experiences affect adult cardiovascular and cortisol function. *Psychosom. Med.* **60**, 765–772 (1998).
14. Kinsey, C. B. & Hupcey, J. E. State of the science of maternal-infant bonding: A principle-based concept analysis. *Midwifery* **29**, 1–14 (2013).
15. Parker, G., Tupling, H. & Brown, L. B. A parental bonding instrument. *Br. J. Med. Psychol.* **52**, 1–10 (1979).
16. Wilhelm, K., Niven, H., Parker, G. & Hadzi-Pavlovic, D. The stability of the parental bonding instrument over a 20-year period. *Psychol. Med.* **35**, 387–393 (2005).
17. Farber, M. J., Gee, D. G. & Hariri, A. R. Normative range parenting and the developing brain: A scoping review and recommendations for future research. *Eur. J. Neurosci.* **55**, 2341–2358 (2022).
18. Holz, N. E. et al. Early maternal care May counteract Familial liability for psychopathology in the reward circuitry. *Soc. Cogn. Affect. Neurosci.* **13**, 1191–1201 (2018).
19. Noriuchi, M., Kikuchi, Y. & Senoo, A. The functional neuroanatomy of maternal love: mother's response to infant's attachment behaviors. *Biol. Psychiatry*. **63**, 415–423 (2008).
20. Choi, E. J. et al. Attachment security and striatal functional connectivity in typically developing children. *Dev. Cogn. Neurosci.* **48**, 100914 (2021).
21. Schneider, S. et al. Maternal interpersonal affiliation is associated with adolescents' brain structure and reward processing. *Transl Psychiatry*. **2**, e182 (2012).
22. Der-Avakian, A. & Markou, A. The neurobiology of anhedonia and other reward-related deficits. *Trends Neurosci.* **35**, 68–77 (2012).
23. Delgado, M. R. Reward-related responses in the human striatum. *Ann. N Y Acad. Sci.* **1104**, 70–88 (2007).

24. Haruno, M. & Kawato, M. Different neural correlates of reward expectation and reward expectation error in the putamen and caudate nucleus during stimulus-action-reward association learning. *J. Neurophysiol.* **95**, 948–959 (2006).
25. O'Doherty, J. et al. Dissociable roles of ventral and dorsal striatum in instrumental conditioning. *Science* **304**, 452–454 (2004).
26. Bick, S. K. et al. Caudate stimulation enhances learning. *Brain* **142**, 2930–2937 (2019).
27. Tricomi, E. M., Delgado, M. R. & Fiez, J. A. Modulation of caudate activity by action contingency. *Neuron* **41**, 281–292 (2004).
28. Calabro, F. J. et al. Striatal dopamine supports reward expectation and learning: A simultaneous PET/fMRI study. *Neuroimage* **267**, 119831 (2023).
29. Kupferschmidt, D. A., Juczewski, K., Cui, G., Johnson, K. A. & Lovinger, D. M. Parallel, but dissociable, processing in discrete corticostriatal inputs encodes skill learning. *Neuron* **96**, 476–489e5 (2017).
30. Robbins, T. W., Costa, R. M. & Habits *Curr. Biol.* **27**, R1200–R1206 (2017).
31. Pruessner, J. C., Champagne, F., Meaney, M. J. & Dagher, A. Dopamine release in response to a psychological stress in humans and its relationship to early life maternal care: a positron emission tomography study using [11 C]raclopride. *J. Neurosci.* **24**, 2825–2831 (2004).
32. Engert, V., Joober, R., Meaney, M. J., Hellhammer, D. H. & Pruessner, J. C. Behavioral response to methylphenidate challenge: influence of early life parental care. *Dev. Psychobiol.* **51**, 408–416 (2009).
33. Brake, W. G., Zhang, T. Y., Diorio, J., Meaney, M. J. & Gratton, A. Influence of early postnatal rearing conditions on mesocorticolimbic dopamine and behavioural responses to psychostimulants and stressors in adult rats. *Eur. J. Neurosci.* **19**, 1863–1874 (2004).
34. Pryce, C. R., Dettling, A. C., Spengler, M., Schnell, C. R. & Feldon, J. Deprivation of parenting disrupts development of homeostatic and reward systems in marmoset monkey offspring. *Biol. Psychiatry*. **56**, 72–79 (2004).
35. Mehta, M. A. et al. Hyporesponsive reward anticipation in the basal ganglia following severe institutional deprivation early in life. *J. Cogn. Neurosci.* **22**, 2316–2325 (2010).
36. Abraham, E. et al. The human Coparental bond implicates distinct corticostriatal pathways: longitudinal impact on family formation and child well-being. *Neuropsychopharmacology* **42**, 2301–2313 (2017).
37. Cohen, R. A. et al. Early life stress and morphometry of the adult anterior cingulate cortex and caudate nuclei. *Biol. Psychiatry*. **59**, 975–982 (2006).
38. Dannlowski, U. et al. Limbic scars: Long-term consequences of childhood maltreatment revealed by functional and structural magnetic resonance imaging. *Biol. Psychiatry*. **71**, 286–293 (2012).
39. Edmiston, E. E. et al. Corticostriatal-limbic Gray matter morphology in adolescents with self-reported exposure to childhood maltreatment. *Arch. Pediatr. Adolesc. Med.* **165**, 1069–1077 (2011).
40. Frodl, T. et al. Childhood adversity impacts on brain subcortical structures relevant to depression. *J. Psychiatr Res.* **86**, 58–65 (2017).
41. Merz, E. C., Maskus, E. A., Melvin, S. A., He, X. & Noble, K. G. Parental punitive discipline and children's depressive symptoms: associations with striatal volume. *Dev. Psychobiol.* **61**, 953–961 (2019).
42. Sheu, Y. S., Polcari, A., Anderson, C. M. & Teicher, M. H. Harsh corporal punishment is associated with increased T2 relaxation time in dopamine-rich regions. *Neuroimage* **53**, 412–419 (2010).
43. Huang, Z., Jordan, J. D. & Zhang, Q. Early life adversity as a risk factor for cognitive impairment and alzheimer's disease. *Transl Neurodegener*. **12**, 1–18 (2023).
44. Lupien, S. J. et al. Cortisol levels during human aging predict hippocampal atrophy and memory deficits. *Nat. Neurosci.* **1**, 69–73 (1998).
45. Lupien, S. J., McEwen, B. S., Gunnar, M. R. & Heim, C. Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nat. Rev. Neurosci.* **10**, 434–445 (2009).
46. McEwen, B. S. Physiology and neurobiology of stress and adaptation: central role of the brain. *Physiol. Rev.* **87**, 873–904 (2007).
47. Choi, E. J., Vandewouw, M. M., de Villa, K., Inoue, T. & Taylor, M. J. The development of functional connectivity within the dorsal striatum from early childhood to adulthood. *Dev. Cogn. Neurosci.* **61**, 101258 (2023).
48. Pechtel, P. & Pizzagalli, D. A. Effects of early life stress on cognitive and affective function: an integrated review of human literature. *Psychopharmacol. (Berl)*. **214**, 55–70 (2011).
49. Gheorghe, D. A., Li, C., Gallacher, J. & Bauermeister, S. Associations of perceived adverse lifetime experiences with brain structure in UK biobank participants. *J. Child. Psychol. Psychiatry*. **62**, 822–830 (2021).
50. Teicher, M. H., Samson, J. A., Anderson, C. M. & Ohashi, K. The effects of childhood maltreatment on brain structure, function and connectivity. *Nat. Rev. Neurosci.* **17**, 652–666 (2016).
51. Burns, R. A., Loh, V., Byles, J. E. & Kendig, H. L. The impact of childhood parental quality on mental health outcomes in older adults. *Aging Ment Health*. **7863**, 0–7 (2017).
52. Stafford, M. et al. Parent–child relationships and offspring's positive mental wellbeing from adolescence to early older age. *J. Posit. Psychol.* **11**, 326–337 (2016).
53. Wood, N., Hardy, R., Bann, D., Gale, C. & Stafford, M. Childhood correlates of adult positive mental well-being in three British longitudinal studies. *J. Epidemiol. Community Health*. **75**, 177–184 (2021).
54. Alexopoulos, G. S. Frontostriatal and limbic dysfunction in late-life depression. *Am. J. Geriatr. Psychiatry*. **10**, 687–695 (2002).
55. Steffens, D. C., Wang, L., Manning, K. J. & Pearlson, G. D. Negative affectivity, aging, and depression: results from the neurobiology of Late-life depression (NBOLD) study. *Am. J. Geriatr. Psychiatry*. **25**, 1135–1149 (2017).
56. Kuh, D. et al. Cohort profile: updating the cohort profile for the MRC National survey of health and development: A new clinic-based data collection for ageing research. *Int J. Epidemiol* **40**, e1–e9 (2011).
57. Kuh, D. et al. The MRC National survey of health and development reaches age 70: maintaining participation at older ages in a birth cohort study. *Eur. J. Epidemiol.* **31**, 1135–1147 (2016).
58. Wadsworth, M., Kuh, D., Richards, M. & Hardy, R. Cohort profile: the 1946 National birth cohort (MRC National survey of health and Development). *Int. J. Epidemiol.* **35**, 49–54 (2006).
59. Lane, C. A. et al. Study protocol: insight 46 - a neuroscience sub-study of the MRC National survey of health and development. *BMC Neurol.* **17**, 1–25 (2017).
60. Kullberg, M. L., Maciejewski, D., van Schie, C. C., Penninx, B. W. J. H. & Elzinga, B. M. Parental bonding: psychometric properties and association with lifetime depression and anxiety disorders. *Psychol. Assess.* **32**, 780–795 (2020).
61. Huppert, F. A., Abbott, R. A., Ploubidis, G. B., Richards, M. & Kuh, D. Parental practices predict psychological well-being in midlife: Life-course associations among women in the 1946 British birth cohort. *Psychol. Med.* **40**, 1507–1518 (2010).
62. Enns, M. W., Cox, B. J. & Clara, I. Parental bonding and adult psychopathology: results from the US National comorbidity survey. *Psychol. Med.* **32**, 997–1008 (2002).
63. Heider, D. et al. Empirical evidence for an invariant three-factor structure of the parental bonding instrument in six European countries. *Psychiatry Res.* **135**, 237–247 (2005).
64. Xu, M. K., Morin, A. J. S., Marsh, H. W., Richards, M. & Jones, P. B. Psychometric validation of the parental bonding instrument in a UK population–based sample: role of gender and association with mental health in mid-late life. *Assessment* **25**, 716–728 (2018).
65. Fergusson, D. M., Horwood, L. J. & Woodward, L. J. The stability of child abuse reports: A longitudinal study of the reporting behaviour of young adults. *Psychol. Med.* **30**, 529–544 (2000).
66. Newbury, J. B. et al. Measuring childhood maltreatment to predict early-adult psychopathology: comparison of prospective informant-reports and retrospective self-reports. *J. Psychiatr Res.* **96**, 57–64 (2018).

67. Heider, D., Matschinger, H., Bernert, S., Alonso, J. & Angermeyer, M. C. Relationship between parental bonding and mood disorder in six European countries. *Psychiatry Res.* **143**, 89–98 (2006).
68. Tennant, R. et al. The Warwick-Edinburgh mental well-being scale (WEMWBS): development and UK validation. *Health Qual. Life Outcomes*. **5**, 1–13 (2007).
69. Smith, S. M. et al. Advances in functional and structural MR image analysis and implementation as FSL. *Neuroimage* **23**, S208–S219 (2004).
70. Zhang, Y., Brady, M. & Smith, S. Segmentation of brain MR images through a hidden Markov random field model and the expectation-maximization algorithm. *IEEE Trans. Med. Imaging*. **20**, 45–57 (2001).
71. Patenaude, B., Smith, S. M., Kennedy, D. N. & Jenkinson, M. A bayesian model of shape and appearance for subcortical brain segmentation. *Neuroimage* **56**, 907–922 (2011).
72. Bauermeister, S. et al. The dementias platform UK (DPUK) data portal. *Eur. J. Epidemiol.* **35**, 601–611 (2020).
73. Dinno, A. Exploring the sensitivity of horn's parallel analysis to the distributional form of random data. *Multivar. Behav. Res.* **44**, 362–388 (2009).
74. Satorra, A. & Bentler, P. M. A scaled difference chi-square test statistic for moment structure analysis. *Psychometrik* **66**, 507–514 (2001).
75. Hooper, D., Coughlan, J. & Mullen, M. R. Structural equation modelling: guidelines for determining model fit. *Electron. J. Bus. Res. Methods*. **6**, 53–60 (2008).
76. Hu, L. T. & Bentler, P. M. Cutoff criteria for fit indexes in covariance structure analysis: conventional criteria versus new alternatives. *Struct. Equ Model.* **6**, 1–55 (1999).
77. Clark, L. A. & Watson, D. Tripartite model of anxiety and depression psychometric evidence. *J. Abnorm. Psychol.* **100**, 316–336 (1991).
78. Engert, V. et al. Perceived early-life maternal care and the cortisol response to repeated psychosocial stress. *J. Psychiatry Neurosci.* **35**, 370–377 (2010).
79. Cornelis, M. C. et al. Age and cognitive decline in the UK biobank. *PLoS One*. **14**, 1–16 (2019).
80. Leys, C., Ley, C., Klein, O., Bernard, P. & Licata, L. Detecting outliers: do not use standard deviation around the mean, use absolute deviation around the median. *J. Exp. Soc. Psychol.* **49**, 764–766 (2013).
81. Iglewicz, B. & Hoaglin, D. C. *How To Detect and Handle Outliers*vol. 16 (Statistics Division, 1993). American Society for Quality Control.
82. Cole, T. J. et al. Using Super-Imposition by translation and rotation (SITAR) to relate pubertal growth to bone health in later life: the medical research Council (MRC) National survey of health and development. *Int. J. Epidemiol.* **45**, 1125–1134 (2016).
83. Silverwood, R. J. et al. Cognitive and kidney function: results from a British birth cohort reaching retirement age. *PLoS One* **9**, e86743 (2014).
84. Curley, J. P. & Champagne, F. A. Influence of maternal care on the developing brain: Mechanisms, Temporal dynamics and sensitive periods. *Front. Neuroendocrinol.* **40**, 52–66 (2016).
85. Colich, N. L. et al. Like mother like daughter: putamen activation as amechanism underlying intergenerational risk for depression. *Soc. Cogn. Affect. Neurosci.* **12**, 1480–1489 (2017).
86. Yap, M. B. H. & Jorm, A. F. Parental factors associated with childhood anxiety, depression, and internalizing problems: A systematic review and meta-analysis. *J. Affect. Disord.* **175**, 424–440 (2015).
87. Kidd, K. N., Prasad, D., Cunningham, J. E. A., de Azevedo Cardoso, T. & Frey, B. N. The relationship between parental bonding and mood, anxiety and related disorders in adulthood: A systematic review and meta-analysis. *J. Affect. Disord.* **307**, 221–236 (2022).
88. Carollo, A., De Marzo, S. & Esposito, G. Parental care and overprotection predict worry and anxiety symptoms in emerging adult students. *Acta Psychol. (Amst).* **248**, 104398 (2024).
89. Kimbrel, N. A., Nelson-Gray, R. O. & Mitchell, J. T. Reinforcement sensitivity and maternal style as predictors of psychopathology. *Pers. Individ Dif.* **42**, 1139–1149 (2007).
90. Monteleone, A. M. et al. Parental bonding, childhood maltreatment and eating disorder psychopathology: an investigation of their interactions. *Eat. Weight Disord.* **25**, 577–589 (2020).
91. Campbell, S. B., Pierce, E. W., Moore, G., Marakovitz, S. & Newby, K. Boys' externalizing problems at elementary school age: pathways from early behavior problems, maternal control, and family stress. *Dev. Psychopathol.* **8**, 701–719 (1996).
92. Savage, J. The association between attachment, parental bonds and physically aggressive and violent behavior: A comprehensive review. *Aggress. Violent Behav.* **19**, 164–178 (2014).
93. Segura-García, C. et al. Parental bonding in substance and alcohol abusers. *Heroin Addict. Relat. Clin. Probl.* **18**, 13–20 (2016).
94. Reti, I. M. et al. Adult antisocial personality traits are associated with experiences of low parental care and maternal overprotection. *Acta Psychiatr Scand.* **106**, 126–133 (2002).
95. Fletcher, A. C., Walls, J. K., Cook, E. C., Madison, K. J. & Bridges, T. H. Parenting style as a moderator of associations between maternal disciplinary strategies and child well-being. *J. Fam Issues*. **29**, 1724–1744 (2008).
96. Button, T. M. M., Lau, J. Y. F., Maughan, B. & Eley, T. Parental punitive discipline, negative life events and gene- environment interplay in the development of externalizing. *Psychol. Med.* **38**, 29–39 (2008).
97. Barber, B., Stolz, H. E., Olsen, J. A., Collins, W. A. & Burchinal, M. Parental support, psychological control, and behavioral control: assessing relevance across time, culture, and method. *Monogr. Soc. Res. Child. Dev.* **70**, 1–147 (2005).
98. Chen, L., Du, H., Zilioli, S., Zhao, S. & Chi, P. Effects of parental care and overprotection on adolescents' diurnal cortisol profiles. *Horm. Behav.* **140**, 105121 (2022).
99. Vergara-Lopez, C., Chaudoir, S., Bublitz, M., O'Reilly Treter, M. & Stroud, L. The influence of maternal care and overprotection on youth adrenocortical stress response: A multiphase growth curve analysis. *Stress* **19**, 567–575 (2016).
100. Lopez, S. A. & Flagel, S. B. A proposed role for glucocorticoids in mediating dopamine-dependent cue-reward learning. *Stress* **24**, 154–167 (2021).
101. Zachry, J. E. et al. Sex differences in dopamine release regulation in the striatum. *Neuropsychopharmacology* **46**, 491–499 (2021).
102. de Carvalho, G., Khoja, S., Haile, M. T. & Chen, L. Y. Early life adversity impaired dorsal striatal synaptic transmission and behavioral adaptability to appropriate action selection in a sex-dependent manner. *Front. Synaptic Neurosci.* **15**, 128640 (2023).
103. Helpman, L. et al. Sex differences in trauma-related psychopathology: A critical review of neuroimaging literature (2014–2017). *Curr. Psychiatry Rep.* **19**, 104 (2017).
104. Allen, K. B. et al. Parental autonomy granting and child perceived control: effects on the everyday emotional experience of anxious youth. *J. Child. Psychol. Psychiatry*. **57**, 835–842 (2017).
105. Baldwin, J. R., Reuben, A., Newbury, J. B. & Danese, A. Agreement between prospective and retrospective measures of childhood maltreatment: A systematic review and meta-analysis. *JAMA Psychiatry*. **76**, 584–593 (2019).
106. Susser, E. & Widom, C. S. Still searching for lost truths about the bitter sorrows of childhood. *Schizophr Bull.* **38**, 672–675 (2012).
107. Raz, N. et al. Regional brain changes in aging healthy adults: general trends, individual differences and modifiers. *Cereb. Cortex*. **15**, 1676–1689 (2005).

Author contributions

DAG, MK, and SB conceptualised the idea. DAG analysed the data and wrote the initial draft of the manuscript.

NF assisted with the neuroimaging analysis. LP advised on the clinical interpretation of results. All authors interpreted the analyses, edited, and proofread the manuscript. All authors approved the final manuscript.

Funding
All analyses were conducted on the Dementias Platform (DPUK) Data Portal, project 0144 (NSHD approval). The Medical Research Council supports DPUK through grant MR/T0333771. DAG is supported by a fellowship from the European Research Executive Agency, through HORIZON-WIDERA-2022-TALENTS-02 (Project number: 101090316). MK is supported by the Swiss National Science Foundation (grant number: P500PS_210853). The contribution from NF to this work is supported by the Italian Ministry of Health (Ricerca Corrente). SB is supported by DPUK.

Declarations

Competing interests
The authors declare no competing interests.

Ethics approval and consent to participate
The present analysis was conducted on secondary data for which ethical approval was obtained by the original cohort study. Ethical approval for the NSHD study was provided by Research Ethics Committees in England and Scotland. The neuroimaging sub-study obtained ethical approval by the National Research Ethics Service (NRES) Committee London (REC reference 14/LO/1173). Participants provided written informed consent for their participation (Kuh et al., 2011, 2016; Lane et al., 2017).

Additional information
Supplementary Information The online version contains supplementary material available at https://doi.org/10.1038/s41598-025-20526-4.

Correspondence and requests for materials should be addressed to D.A.G.

Reprints and permissions information is available at www.nature.com/reprints.

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.