Research Articles: Behavioral/Cognitive

Age-related enhancements in positive emotionality across the lifespan: structural equation modelling of brain and behaviour

<https://doi.org/10.1523/JNEUROSCI.1453-21.2022>

Cite as: J. Neurosci 2022; 10.1523/JNEUROSCI.1453-21.2022

Received: 15 July 2021

Revised: 8 February 2022

Accepted: 11 February 2022

This Early Release article has been peer-reviewed and accepted, but has not been through the composition and copyediting processes. The final version may differ slightly in style or formatting and will contain links to any extended data.

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Title: Age-related enhancements in positive emotionality across the lifespan: structural equation modelling of brain and behaviour

Abbreviated Title: Positivity across the life-span

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Number of Pages: 39

Number of Figures: 6

Number of Tables: 3

Number of Words;

Abstract; 240

Introduction; 928

Discussion; 702

Acknowledgements: The Cambridge Centre for Ageing and Neuroscience (Cam-CAN) research was supported by the Biotechnology and Biological Sciences Research Council (BB/H008217/1); TD, JS and SS are additionally supported by the UK Medical Research Council (MC_A060_5PQ60). TD is supported by the National Institute for Health Research Cambridge Biomedical Research Centre. We are grateful to the Cam-CAN respondents and their primary care teams in Cambridge for their participation in this study. We also thank colleagues at the MRC Cognition and Brain Sciences Unit MEG and MRI facilities for their assistance. The Cam-CAN corporate author consists of the project principal personnel: Lorraine K Tyler, Carol Brayne, Ed Bullmore, Andrew Calder, Rhodri Cusack, Tim Dalgleish, John Duncan, Rik Henson, Fiona Matthews, William Marslen-Wilson, James Rowe, Meredith Shafto; Research Associates: Karen Campbell, Teresa Cheung, Simon Davis, Linda Geerligs, Rogier Kievit, Anna McCarrey, Jason Taylor, Kamen Tsvetanov, Nintin Williams; Research Assistants: Lauren Bates, Tina Emery, Sharon Erzinçlioglu, Sofia Gerbase, Stanimira Georgieva, Claire Hanley, Beth Parkin, David Troy; Research Interviewers: Jodie Allen, Gillian Amery, Liana Amunts, Anne Barcroft, Amanda Castle, Cheryl Dias, Jonathan Dowrick, Melissa Fair, Hayley Fisher, Anna Goulding, Adarsh Grewal, Geoff Hale, Andrew Hilton, Frances Johnson, Patricia Johnston, Thea Kavanagh-Williamson, Magdalena Kwasniewska, Alison McMinn, Kim Norman, Jessica Penrose, Fiona Roby, Diane Rowland, John Sargeant, Maggie Squire, Beth Stevens, Aldabra Stoddart, Cheryl Stone, Tracy Thompson, Ozlem Yazlik; and administrative staff: Dan Barnes, Marie Dixon, Jaya Hillman, Joanne Mitchell, Laura Willis.

50 **Author contributions:** TD and SS conceived the study. JS analysed the data. TD, JS and SS
51 wrote the paper.

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53 **Conflict of Interest Statement;**

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55 The authors declare no competing financial interests

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62 **Abstract**

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64 Aging is associated with a bias in attention and memories towards positive and away from
 65 negative emotional content. In addition, emotion regulation appears to improve with age,
 66 despite concomitant widespread cognitive decline coupled with gray matter volume loss in
 67 cortical and subcortical regions thought to sub-serve emotion regulation. Here, we address
 68 this emotion-aging paradox using the behavioural data of an emotion regulation task from a
 69 population derived, male and female, human sample (CamCAN) and utilise Structural
 70 Equation Modelling together with multivariate analysis of structural MRI images of the same
 71 sample to investigate brain-behaviour relationships. In a series of measurement models, we
 72 show the relationship between age and emotionality is best explained by a four-factor model,
 73 compared to single and hierarchical factor models. These four latent factors are interpreted as
 74 Basal Negative Affect, Positive Reactivity, Negative Reactivity and Positive Regulation
 75 (upregulating positive emotion to negative content). Increasing age uniquely contributes to
 76 increased Basal Negative Affect, Positive Reactivity and Positive Regulation, but not
 77 Negative Reactivity. Furthermore, we show gray-matter volumes, namely in the bilateral
 78 frontal operculum, medial frontal gyrus, bilateral hippocampal complex, bilateral middle
 79 temporal gyri and bilateral angular gyrus, are distinctly related to these four latent factors.
 80 Finally, we show that a subset of these brain-behaviour relationships remain significant when
 81 accounting for age and demographic data. Our results support the notion of an age-related
 82 increase in positivity and are interpreted in the context of the Socioemotional Selectivity
 83 Theory of improved emotion regulation in older age.

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93 **Significance Statement**

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95 Aging is associated with a paradoxical increase in well-being and improved emotion
96 regulation despite widespread cognitive decline and gray-matter volume loss in neural
97 regions which underlie emotion regulation. Using a population-derived sample we test the
98 theories behind this emotion-aging paradox with an emotion-regulation task and structural
99 MRI data. We report robust age-related increases in positivity across the lifespan and show
100 structural neural integrity influences this relationship with increasing age. Several brain-
101 behaviour relationships remained unaffected by age and may represent empirically-derived
102 neural markers to explore the paradox of increased well-being in old age. The results support
103 the predictions of Socioemotional Selectivity Theory of improved emotion regulation in older
104 age and challenge the amygdala-focused neural predictions of the Aging Brain Model.

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131 **Introduction**

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133 Current reported levels of personal well-being in the United Kingdom are at their highest
134 after the age of 65 (ONS, 2016) . While ratings do fall from age 75 onwards, epidemiological
135 surveys of self-reported life satisfaction and happiness in the over 90's remains higher than
136 that of middle-aged individuals (ONS, 2016). This is supported by laboratory studies that
137 reveal that healthy older adults report enhanced positive affect and greater emotional stability
138 than their younger counterparts (Carstensen et al., 2000; Carstensen et al., 2011). Given the
139 ubiquity of physical and cognitive decline associated with getting older, it is somewhat
140 paradoxical that our twilight years are often the source of greatest satisfaction.

141 Socioemotional Selectivity Theory (SST) (Carstensen, 1992, 2006), an influential life-span
142 theory of motivation, seeks to unravel this paradox. Specifically, SST suggests that adult life
143 is governed by a core sets of goals broadly associated with either the acquisition of
144 knowledge, resources and social connections or the regulation of emotions associated with
145 well-being. The central tenet of SST is that the relative importance of these sets of goals
146 changes as a function of future time horizons. When the future is perceived as lengthy, as is
147 typical in youth, goals associated with acquisition to maximise future prospects are prioritised
148 over those associated with more immediate positive emotionality and wellbeing. Conversely,
149 when the future is perceived as constrained, typically as we get older, our goal priorities shift
150 to become less future-oriented and more focused on the emotional satisfaction that is possible
151 in the here and now. Other prominent theories in this domain similarly emphasize age-related
152 shifts in strategic priorities and consequent behavioural preferences (Labouvie-Vief, 2003;
153 Charles, 2010; Urry and Gross, 2010) .

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155 A related theoretical view is that the greater emotional stability and wellbeing in older adults
156 might also be a function of age-related increases in expertise in navigating emotive situations,
157 either through more optimal selection of appropriate emotional regulation strategies or as a

158 function of more effective and consolidated emotion-control skills (John and Gross, 2004;
 159 Birditt et al., 2005; Sims et al., 2015; Burr et al., 2020). Support for this latter view is mixed,
 160 however. Some studies are consistent, while others show no age-related changes in emotion-
 161 regulation or declines in regulation capacity with age (Phillips et al., 2008; Urry and Gross,
 162 2010; Winecoff et al., 2011; Opitz et al., 2014; Sims et al., 2015; Martins et al., 2018;
 163 Schweizer et al., 2018; Livingstone and Isaacowitz, 2019; Burr et al., 2020). One reason for
 164 these mixed data might be the reliance of several core emotion-regulation strategies – for
 165 example, reappraisal – on domain-general processes of cognitive-control (Ochsner and Gross,
 166 2005), which appear to decline with age as a function of sharp neuronal degradation of the
 167 dorsolateral prefrontal cortex (dlPFC) (Braver and Barch, 2002).
 168 However, differential structural trajectories of the sub-regions of the PFC may help to explain
 169 why older adults maintain function in other emotion processing contexts such as memory and
 170 attention. Prefrontal regions including the ventromedial prefrontal cortex (vmPFC) (Quirk
 171 and Beer, 2006; Winecoff et al., 2013) and anterior cingulate cortex (ACC) (Bush et al.,
 172 2000), both associated with emotion processing, have been shown to maintain their cortical
 173 thickness across the lifespan (Fjell et al., 2009b)
 174 A compelling and competing account suggests age-related improvements in emotion
 175 regulation are caused by an age-related change in the amygdala response to affective stimuli,
 176 the aging-brain model (ABM) (Cacioppo et al., 2011). The ABM argues that the amygdala
 177 maintains its responsiveness to positive stimuli as we age, but diminishes in response to
 178 negative stimuli, thus biasing the attention to and subsequent memory of positive stimuli.
 179 Functional imaging evidence shows older adults have reduced amygdala activity in response
 180 to negative, but not positive pictures, relative to younger adults (Mather et al., 2004), while
 181 structural imaging and post-mortem studies indicate less volumetric decline and less

182 histological effects of aging in the amygdala as a proxy of better preserved function (Allen et
183 al., 2005; Brabec et al., 2010).

184 Indeed, prominent theories of emotion regulation draw a distinction between effortful,
185 resource-demanding strategies and processes that have become automatic and thus less reliant
186 on declining cognitive control resources (Braunstein et al., 2017). There is now robust
187 support from laboratory studies that automatic cognitive processing biases in domains such as
188 attention and memory in favour of positive information – the so-called ‘positivity effect’ –
189 are augmented with older age (Charles et al., 2003; Kennedy et al., 2004; Mather and
190 Carstensen, 2005) . It therefore follows that automatized emotion regulation processes
191 (including those that drawn upon attentional and mnemonic processes) that prioritise positive
192 affect become strengthened across the lifespan, while regulation processes that rely on
193 declining effortful cognitive control may become less effective. This might help to explain
194 the mixed findings regarding emotion-regulation and aging. Another important factor that
195 may partially account for these equivocal data is the tendency to for studies to use single bi-
196 valence scales (Schweizer et al., 2018), ranging from positive to negative, to measure
197 experienced affect. Single scales could obscure age-related increases in positive affectivity if
198 there are separate age-related effects for negative affectivity that act in an opposite direction.
199 A strength of the positivity effect literature (Charles et al., 2003; Kennedy et al., 2004;
200 Mather and Carstensen, 2005), is the disaggregation of positive and negative information. A
201 similar measurement separation in the domain of valence would be consistent with research
202 in affective neuroscience confirming the utility of discrete valence dimensions (Viinikainen
203 et al., 2010; Paulus et al., 2017).

204 *Current study*

205 The present study therefore used a gold-standard laboratory-based emotional reactivity and
206 regulation task (Schweizer et al., 2013; Schweizer et al., 2016) to examine age-related

207 changes in elicited positive affectivity (assayed independently of negative affectivity) within
 208 both passive viewing and emotion regulation contexts. We used a large population-derived
 209 sample of adults aged 18-88 as part of the Cam-CAN cohort (www.cam-can.org (Shafto et
 210 al., 2014)). We evaluated the hypothesis that the robust age-related positivity effect within the
 211 cognitive domain extends to the affective domain, in the form of an age-dependent increase
 212 in core components of positive affectivity. We predicted that such enhancement in positive
 213 affectivity would be independent of any age-related changes in cognitive control measured
 214 behaviourally. Finally, we examined the relationships between age-related effects in positive
 215 affectivity and age-related differences in brain gray-matter, based on the Magnetic Resonance
 216 (MR) measurements within the Cam-CAN cohort (Taylor et al., 2017). This allowed us to
 217 evaluate whether any age-related enhancements in positive affectivity were also independent
 218 of age-related volumetric changes in fronto-parietal brain regions typically associated with
 219 more effortful emotion regulation (Fjell et al., 2009a) using structural equation modelling
 220 (SEM) to explore brain-behaviour relationships (Kievit et al., 2012).

221 **Materials and Methods**

222 **Participants**

223 Three-hundred and thirty of the Cam-CAN (Shafto et al., 2014) sample were invited to
 224 perform an emotion reactivity and regulation task. Sixteen participants chose not to undertake
 225 the task, and 26 participants were excluded as session notes indicated they did not follow the
 226 instructions of the task appropriately. Fifteen participants did not complete our measure of
 227 cognitive control – the Cattell Test (n=9) – or provide demographic data (n=6) and for an
 228 additional 24 there were problems with the analysis of their MR images (Taylor et al., 2017).
 229 Demographic data for the remaining 249 are shown in Table 1. All participants took part in a
 230 range of psychological tests (no other cognitive domains were analysed in the context of this
 231 analysis). Ethical approval for the study was obtained from the Cambridgeshire 2 (now East

232 of England—Cambridge Central) Research Ethics Committee. Participants gave full
 233 informed consent.

234 <Insert Table 1 here>

235 **Experimental Design**

236 **Emotion Reactivity and Regulation Task (ERRT)**

237 Emotion reactivity and regulation were assessed with a film-based paradigm (Schweizer et
 238 al., 2013; Schweizer et al., 2016) (Figure 1). Participants viewed a series of forty 30-second
 239 film clips that were either positive (e.g., babies laughing), neutral (e.g., weather report) or
 240 negative (e.g., documentary of the Rwandan genocide) in valence and consisted of a mixture
 241 of real-life and fictional footage. Participants received one of two different viewing
 242 instructions before each clip. Either “WATCH”, where participants were told to watch the
 243 film clips and allow themselves to feel any emotions that naturally arose without trying to
 244 deliberately distract themselves from the content of the film clip or effortfully regulate their
 245 emotions in any way. The second viewing instruction “REGULATE” was only applicable to
 246 half of the negative film clips. Here, participants were explicitly asked to try to reduce
 247 (down-regulate) any unwanted distressing affect in response to the film clip by reappraising
 248 the contents of the film clip. This gave four task conditions: Positive Watch; Negative Watch;
 249 Negative Regulate; and Neutral Watch.

250 Before each film clip, participants received a prompt to indicate the valence and viewing
 251 instruction for the clip (e.g. “WATCH NEUTRAL” or “REGULATE NEGATIVE”). This
 252 was followed by the clip itself, after which participants rated on separate ten point scales both
 253 their negative *and* positive affective reactions experienced during the clip, as well as rating
 254 how much they simply watched the clip versus regulated their affect, as a measure of
 255 compliance. This resulted in both mean negative and mean positive affective ratings for each
 256 task condition in each participant. Each of the four conditions yielded a positive affect rating

257 and a negative affect rating. Affective responses were rated on a scale ranging from ‘1’ = not
 258 at all, to ‘11’ = extremely. Instruction compliance was rated on a scale ranging from ‘1’ =
 259 Watch, to ‘11’ = Regulate. Therefore 8 ratings were utilised for modelling purposes to
 260 disaggregate the positive and negative affective ratings.

261 Instruction, film clip, and affective and compliance ratings together comprised an
 262 experimental trial. Each condition was presented twice with four trials in each block.
 263 Emotional blocks were followed by 45-s washout clips a calming film clip (e.g., waves gently
 264 rolling back and forth on a beach with a meditative soundtrack), to return affective levels to
 265 pre-stimulus baseline. Films were randomized across the WATCH and REGULATE
 266 conditions separately for each participant and the presentation order of condition was pseudo-
 267 randomized always starting with a neutral block and ending with a positive block.

268 <Insert Figure 1 here>

269 **‘Net’ Emotional Effects of Age**

270 The standard method of analysis for behavioural data of emotion regulation tasks is to
 271 subtract the raw emotion score (scaled from negative to positive) from the neutral stimulus
 272 from raw emotion score from the affective stimulus (positive or negative trials) to create
 273 positive and negative reactivity indices. In our task, we challenged affective response after
 274 each stimulus on separate positive and negative scales (ranging from ‘not at all’ to
 275 ‘extremely’). In order to examine the standard response we recreated these scales accounting
 276 for the residual emotionality for each scale and regressed them against age to determine age-
 277 related changes in ‘net’ emotionality. In each participant, we first removed the residual
 278 positivity/negativity associated with each scale. To do this, we subtracted the
 279 positivity/negativity associated with watching positive/negative films from the
 280 positivity/negativity scale associated with watching positive/negative films. We then did the

281 same for the Neutral film clips (subtracted negative from positive scales). To gain a ‘net’
 282 effect score, we then subtracted the Neutral score from either the Positive (for Positive
 283 Reactivity) or Negative (for Negative Reactivity) scores. For example;

$$284 \text{ Net Positive Reactivity} = (\text{Positive}^{\text{Watch}_{\text{positive}}} - \text{Positive}^{\text{Watch}_{\text{negative}}}) - (\text{Neutral}^{\text{Watch}_{\text{positive}}} - \text{Neutral}^{\text{Watch}_{\text{negative}}})$$

$$285 \text{ Net Negative Reactivity} = (\text{Negative}^{\text{Watch}_{\text{negative}}} - \text{Negative}^{\text{Watch}_{\text{positive}}}) - (\text{Neutral}^{\text{Watch}_{\text{negative}}} - \text{Neutral}^{\text{Watch}_{\text{positive}}})$$

$$286 \text{ Net Negative Regulation} = (\text{Negative}^{\text{Regulate}_{\text{negative}}} - \text{Negative}^{\text{Regulate}_{\text{positive}}}) - (\text{Negative}^{\text{Watch}_{\text{negative}}} - \text{Negative}^{\text{Watch}_{\text{positive}}})$$

288

289 **Demographic, cognitive and mental health measures**

290 Baseline characteristics collected from the Cam-CAN cohort include age, sex, history of
 291 depression (y/n) and a self-reported measure of highest level of education obtained, scored
 292 from 1 = ‘Basic’ (e.g., left education before 16), 2 = ‘GCSE/O-level’ (e.g., left education
 293 before 18), 3 = ‘A-level’ (e.g., left education after 18) and 4 = ‘Degree’ (e.g., left university
 294 after 21 or older). Cognitive control as indexed by fluid intelligence (Duncan, 1995) was
 295 measured by the Cattell Culture Fair Test (Scale 2, Form A), administered using pencil-and-
 296 paper according to the standard protocol.

297 **MRI data; Source-Based Morphometry (SBM)**

298 For details of the MRI sequences, see (Shafto et al., 2014); for details of the MRI pre-
 299 processing, see (Taylor et al., 2017). Voxel-based morphometry is a univariate method and
 300 does not utilize any information about the relationships among voxels. In addition, it will
 301 only detect voxels for which a specific predicted effect is present (typically, a mean
 302 difference between two groups). In contrast, a multivariate, data-driven approach can provide
 303 a way to pool information across different voxels as well as identify unpredicted patterns.
 304 The voxels that carry similar information will group to a set of regions. SBM uses

305 independent component analysis (ICA) to extract maximally spatially independent sources
 306 revealing patterns of variation that occur in structural MRI images (Xu et al., 2009).

307 Prior to ICA, we estimated the number of components for extraction using an information-
 308 theoretic approach. First, we uniformly sub-sampled the gray matter images until the
 309 estimated entropy rate equalled the entropy rate of an independent and identically distributed
 310 (i.i.d.) Gaussian random process of the same variance and data length. Next, we estimated the
 311 number of components using the Akaike's Information Criterion (AIC) resulting in 51
 312 estimated components.

313 All gray matter images were processed using spatial ICA (Calhoun et al., 2001) as
 314 implemented in the GIFT toolbox (<http://icatb.sourceforge.net>). ICA was performed using a
 315 neural network algorithm (infomax) that attempts to minimize the mutual information of the
 316 network outputs (Bell and Sejnowski, 1995). Every gray matter image is converted into a
 317 one-dimensional vector. The 249 gray matter images of each participant were arrayed into
 318 one 249-row subject-by-gray matter data matrix. This matrix was then decomposed into a
 319 mixing matrix and a source matrix. The mixing matrix expresses the relationship between
 320 249 subjects and k ($n=51$) components. The rows of the matrix are scores which indicate to
 321 what degree that the k components contribute to a given subject. The columns of the matrix
 322 indicate how one component contributes to the 249 subjects. In contrast, the source matrix
 323 expresses the relationship between the k components and the voxels within the brain. The
 324 rows of the matrix indicate how one component contributes to different brain voxels, and the
 325 columns of the matrix are scores that indicate how one voxel contributes to each of the
 326 components. We used the source matrix for visualization. We reshaped every row of the
 327 source matrix back into a 3D image (source map). These source maps were scaled to unit
 328 standard deviation (SBM Z map) and threshold at a value of $|Z| > 3:0$. The maps of the sources
 329 were then superimposed on the MNI-normalized template brain.

330 We selected 12 bilaterally represented components which anatomically (Harvard-Oxford
 331 Atlas) overlapped with emotion reactivity and regulation related regions taken from the
 332 existing functional imaging literature (Phan et al., 2002; Diekhof et al., 2011; Buhle et al.,
 333 2014; Frank et al., 2014; Kohn et al., 2014); precuneus/posterior cingulate cortex (PCC),
 334 superior frontal gyrus (SFG), middle temporal gyrus (MTG), anterior/middle cingulate cortex
 335 (A/MCC), middle frontal gyrus (MFG), inferior frontal gyrus (IFG) pars triangularis, IFG
 336 pars opercularis, ventral striatum, hippocampus/amygdala complex, medial frontal gyrus,
 337 anterior insula and angular gyrus. Data extracted from the mixing matrix for each source
 338 were entered into the SEM. Please see Figure 4 and Table 3 for visualization and covariance
 339 matrix.

340 **Statistical Analysis**

341 Analysis of the effects of age was performed by multiple regression within General
 342 Linear Models (GLMs) that treated age as a continuous variable modelled by linear and
 343 quadratic terms. We focus on effect sizes (R^2), expressed as the percentage of variance
 344 explained by a specific statistical contrast within the GLM, rather than p-values, since the
 345 latter become less appropriate for larger samples.

346 **Structural Equation Models (SEMs)**

347 SEMs were fit using the package Lavaan⁷¹ (Rosseel, 2012) in R version 3.1.2 (R
 348 Development Core Team, 2011). We used the following guidelines for judging good fit
 349 (Schermelleh-Engel et al., 2003): Root Mean Square Error of Approximation (RMSEA)
 350 below 0.05 (acceptable: 0.05-0.08) and a Comparative Fit Index (CFI) above 0.97
 351 (acceptable: 0.95-0.97). All models were fit using Maximum Likelihood Estimation using
 352 robust standard errors, for which we report the Satorra-Bentler (SB) scaled test statistic. P-
 353 values less than .05 were used to judge significance of individual paths. We initially tested a
 354 single factor model to establish whether a single general emotionality factor could explain the

individual difference in affect ratings. We then tested a four-factor measurement model based on theoretically derived notions of emotional reactivity and regulation. Once the measurement model was established, we finally tested a hierarchical model to assess whether the four factors were influence by a single general emotionality factor.

Results

Emotion Reactivity and Regulation Measurement Models

Using Confirmatory Factor Analysis, we tested a series of models which could account for the individual differences in ERRT ratings. A single factor model, with one emotion factor explaining all 8 ERRT ratings ('NEUTRAL WATCH' – *Positive* and *Negative* ratings; 'POSITIVE WATCH' – *Positive* and *Negative* ratings; 'NEGATIVE WATCH' – *Positive* and *Negative* ratings; 'NEGATIVE REGULATE' – *Positive* and *Negative* ratings) did not fit the data well (see Table 2 for fit statistics). We therefore explored a four-factor model, with each factor hypothetically representative of the theoretically plausible constructs of emotion reactivity and regulation: '*Negative Regulation*', reflecting decreases in negative affectivity in response to negative stimuli and consequently loading negatively on NEGATIVE REGULATE *negative* ratings and NEGATIVE WATCH *negative* ratings; '*Positive Regulation*', reflecting increased positive affectivity in response to negative stimuli and consequently loading positively on NEGATIVE REGULATE *positive* ratings and NEGATIVE WATCH *positive* ratings; '*Negative Reactivity*', reflecting acute increases in negative affectivity and consequently loading on *negative* ratings across all three WATCH conditions; and '*Positive Reactivity*', loading on the *positive* ratings from the POSITIVE and NEUTRAL WATCH conditions. This four-factor model fit the data significantly better than the single factor model ($\Delta\chi^2 =$

379 192, $\Delta df = 8$, $p = 2.2e-16$), but remained just below the pre-defined threshold for good fit
 380 (Table 2).

381 We next investigated the modification indices of the four-factor model to ask whether freeing
 382 any parameters would improve model fit. The value of a given modification index (m.i.) is
 383 the minimum amount that the chi-square statistic is expected to decrease if the corresponding
 384 parameter is freed. This suggested 3 minor modifications to the model : 1) To account for the
 385 residual anticorrelation between *positive* and *negative* emotion ratings elucidated from
 386 watching positive content (m.i. = 42.41); 2) to account for the anticorrelation between
 387 *positive* emotion ratings from the POSITIVE WATCH condition and the latent factor of
 388 *Negative Reactivity* (derived from all *negative* emotion ratings from the Watch conditions)
 389 (m.i. = 24.08); 3) To account for the residual covariance associated with *negative* emotion
 390 ratings from the NEGATIVE REGULATE condition and *positive* emotion ratings from the
 391 NEGATIVE WATCH condition i.e. the assertion that regulation of negative content is
 392 associated with the degree of positivity derived from simply watching negative content (m.i.
 393 = 18.26). With these 3 modifications, the model fit very well (Table 2). Finally, we tested a
 394 hierarchical factor model, whereby the four factors of the previous model underlie a single
 395 hierarchical factor of general emotionality. This model did not converge and was rejected.

396 Having established a four-factor measurement model, we then explored the significant
 397 individual path loadings for each latent factor to refine our interpretation in line with the
 398 theoretical and empirical literature. Starting with the positive affect components, our putative
 399 *Positive Reactivity* factor did load significantly and positively, as anticipated, onto the
 400 NEUTRAL WATCH *positive* ratings (0.82) and POSITIVE WATCH *positive* ratings (1.02)
 401 and so we retained the interpretation of this factor as an index of '*Positive Reactivity*' i.e. the
 402 degree of positivity derived from *both* neutral and positive content. Our putative '*Positive*
 403 *Regulation*' factor loaded positively onto *positive* scores from both the NEGATIVE

REGULATE (0.89) and NEGATIVE WATCH (0.77) conditions, representing the degree of positivity extracted from negative content and thus we also kept our interpretation of this latent factor as a form of '*Positive Regulation*'.

In terms of the negative emotion components, both the NEGATIVE REGULATE *negative* ratings (0.86) and NEGATIVE WATCH *negative* ratings (0.86) loaded comparably strongly but positively rather than negatively onto the latent factor we initially termed *Negative Regulation*. A paired t-test on the raw scores revealed no significant difference between *negative* emotion ratings when either regulating or simply watching negative content ($t=.057$, $p = .954$). This, together with the direction of associations, indicated that this latent factor is better interpreted as a measure of '*Negative Reactivity*' to negative content. Finally, the *negative* ratings from the NEUTRAL WATCH (0.90) and POSITIVE WATCH (0.58) conditions, but importantly not the *negative* ratings from the NEGATIVE WATCH condition (0.06), showed significant loadings on our original *Negative Reactivity* factor. With the strongest relationship to the negative rating from the neutral content, and moderate relationship with deriving negativity from positive films, we deemed that this initially-characterized *Negative Reactivity* factor better represents '*Basal Negative Affect*', particularly as there was no relationship with *negative* scores in the NEGATIVE WATCH condition.

<Insert Table 2 here>

Effects of Age

We next included age as a common cause of individual differences in each latent factor which produced an excellent overall model fit (Table 2) with parameters shown in Figure 2. Age had a significant relationship with all four latent factors, with factor strength (i.e. higher affect ratings) increasing with increasing age (*Positive Regulation* ($F(1, 247) = 72.5$ $p<.0001$, $r^2 = .22$; 95% c.i. = 0.14 - 0.31); *Positive Reactivity* ($F(1, 247) = 62.9$ $p<.0001$ $r^2 = .20$; 95% c.i. = 0.12 - 0.29); *Basal Negative Affect* ($F(1, 247) = 109.3$ $p<.0001$, $r^2 = .30$; 95%

c.i. = 0.22 - 0.39)), although there was a weaker (though significant) influence on *Negative Reactivity* compared to the other three factors (Negative Reactivity ($F(1, 247) = 8.1$ $p = .004$, $r^2 = .03$; 95% c.i. = 0.00 - 0.08). Indeed, this model was better than one in which the age-factor paths were constrained to be equal ($\Delta\chi^2 = 38$, $\Delta df = 3$, $p < 3.32e-08$), suggesting differential effects of age on these emotion factors. This supports our primary hypothesis that the factor constructs of both *Positive Reactivity* and *Positive Regulation* increase with age once you disaggregate emotion components by valence.

<Insert Figure 2 here>

Elucidating the contribution of cognitive control

In order to explore the contribution of cognitive or executive control, we next introduced our measure of fluid intelligence into the model, along with education level to index potential cohort effects. The model fit remained acceptable (Table 2). As expected, age was significantly negatively associated with education level (-0.21) and fluid intelligence (-0.69), reflecting shifts in UK education accessibility and policy over time. However, importantly neither educational level nor fluid intelligence showed any direct significant effects on our two positive emotion factors – *Positive Reactivity* and *Positive Regulation* – indicating that the significant increases in positive emotion processing with age are above and beyond any concomitant decline in fluid intelligence, or cohort effects. The relationship between age and *Basal Negative Affect* also remained significant. However, the positive influence of age on the *Negative Reactivity* factor was no longer significant when including fluid intelligence and education in the model suggesting that age-related increases in negative reactivity are a function of age-related declines in cognitive control.

Examining the effects of gender and depression

To further externally validate the model we next included two variables which have been reported to reliably influence emotion responding – gender, and a history of depression.

454 Based on the prior literature we anticipated that female gender would be significantly
 455 associated with both *Positive* and *Negative Reactivity* independent of the effects of age
 456 (Kring and Gordon, 1998). We also anticipated that a history of depression would be
 457 associated with higher *Basal Negative Affect* (Kanske et al., 2012) and potentially with higher
 458 *Negative Reactivity*, again independent of age.

459 The model retained its acceptable fit following the inclusion of these variables (Table 2). A
 460 history of depression was, as expected, positively associated with *Basal Negative Affect*
 461 (0.16) but no other factors. Being female was significantly associated with both *Positive*
 462 *Reactivity* (0.21) and *Negative Reactivity* (0.31) but no other factors. These relationships
 463 provide convergent validity for the latent factors in the model. Importantly, the key effects of
 464 age on *Positive Reactivity* and *Positive Regulation*, as well as on *Basal Negative Affect*,
 465 remained significant beyond the contribution of these additional variables being included in
 466 the model (Figure 3). This is important as other age-related effects on affective processing
 467 appear to be a function of age-related differences in depression (Murphy et al., 2019).

468 <Insert Figure 3 here>

469 **Evaluating the influence of volumetric indices of brain regions involved in emotion** 470 **reactivity and regulation**

471 As expected, each of the GM sources declined linearly with age, though with varying degrees
 472 (Figure 4), the Middle Temporal Gyrus (MTG) showed the greatest effect of age ($r^2 = 40\%$;
 473 95% c.i. = 0.32 - 0.48), while the Angular Gyrus showed the least effect of age ($r^2 = 4\%$; 95%
 474 c.i. = 0.01 - 0.10).

475 <Insert Figure 4 here>

476 <Insert Table 3 here>

477 We next examined whether there were meaningful relationships between variation in GM in
 478 these brain regions and our affectivity factors. Data from the GM sources were included in a

new SEM as putative causes of the factors. For these models, we retained education, depression and gender in the model but removed fluid intelligence as here we were, in principle, examining the variance associated with neural indices of cognitive control and did not want these to be obscured by including a behavioural index of cognitive control in the model. It is important to stress that significant paths represent unique covariance between the brain variable and latent variable over and above the shared covariance between the other brain variables. Thus, significant paths should not be interpreted as one-to-one region-behaviour mapping. However, model comparison can be used to test the importance of particular combinations of brain variables (Henson et al., 2016).

Model comparison allows us to test the differential partial contribution of each GMV source to the different latent factors. First, we imposed equality constraints on all brain-behaviour paths to test against the unique contributions of our brain factors. This model failed to converge, suggesting each of the brain-behaviour paths need to be estimated freely and that each has unique variability that explains some of the data. We then tested models which zeroed out each GM source to all emotion factors, to investigate the unique contribution of each region to the emotion factors. Independent models showed zeroing out the effect of the PCC ($\Delta\chi^2 = 2$, $\Delta df = 4$, $p = 0.68$), SFG ($\Delta\chi^2 = 4$, $\Delta df = 4$, $p = 0.45$), A/MCC ($\Delta\chi^2 = 3$, $\Delta df = 4$, $p = 0.62$), MFG ($\Delta\chi^2 = 1$, $\Delta df = 4$, $p = 0.88$), pars triangularis ($\Delta\chi^2 = 5$, $\Delta df = 4$, $p = 0.27$), ventral striatum ($\Delta\chi^2 = 4$, $\Delta df = 4$, $p = 0.44$), hippocampus/amygdala ($\Delta\chi^2 = 6$, $\Delta df = 4$, $p = 0.17$), medial frontal gyrus ($\Delta\chi^2 = 7$, $\Delta df = 4$, $p = 0.12$), anterior insula ($\Delta\chi^2 = 6$, $\Delta df = 4$, $p = 0.21$) and angular gyrus ($\Delta\chi^2 = 5$, $\Delta df = 4$, $p = 0.24$) did not significantly change model fit. However, separately setting IFG pars opercularis ($\Delta\chi^2 = 16$, $\Delta df = 4$, $p < 0.005$) and MTG ($\Delta\chi^2 = 20$, $\Delta df = 4$, $p < 0.001$) paths to zero did significantly reduce model fit, indicating a significant independent partial contribution of these regions to the emotion factors.

503 Five GM sources showed significant paths to our affective factors. Starting with the positive
 504 factors, *Positive Reactivity* was negatively associated with GM in the IFG pars opercularis (-
 505 .26). *Positive Regulation* was also negatively associated with GM in IFG pars opercularis (-
 506 .24) and the MTG (-.42), but positively associated with GM in the hippocampal/amygdala
 507 complex (0.19) and angular gyrus (0.16). For the negative emotion factors, *Basal Negative*
 508 *Affect* was also negatively associated with GM in IFG pars opercularis (-.24) and with GM in
 509 the medial frontal gyrus (-.29). There were no significant brain-behaviour relationships
 510 between any GM source and the *Negative Reactivity* factor.

511 **The relationship of age to emotion-related brain-behaviour associations**

512 We next entered age into the brain-behaviour model connected to each of the 4
 513 emotion factors. Model fit remained excellent (Table 2). Critically, mirroring the findings
 514 after adjusting for our behavioural index of cognitive control (fluid intelligence), the positive
 515 relationship of age with the two components of positive emotion – *Positive Regulation* and
 516 *Positive Reactivity* – remained significant after the brain variables were introduced indicating
 517 that age-related improvements in positive emotion processing are above and beyond any
 518 concomitant decline in brain volume in regions associated in the literature with emotion
 519 reactivity and regulation. The significant relationship between age and *Basal Negative Affect*
 520 was also preserved (see Figure 5).

521 Three brain-behaviour paths were no longer significant once age was included in the model.
 522 The medial FG and IFG pars opercularis were no longer significantly negatively associated
 523 with *Basal Negative Affect*, and the hippocampal/amygdala complex was no longer
 524 significantly positively associated with *Positive Regulation*, suggesting these particular
 525 relationships between brain metrics and emotion components are a function of the effects of
 526 aging. The remaining specific brain-behaviour pathways remained significant, indicating a

527 unique contribution of individual differences in the volume of these brain regions to the
 528 emotion factors over-and-above the effects of age (Figure 5 and Figure 6).

529 <Insert Figure 5 here>

530 <Insert Figure 6 here>

531 **‘Net’ Emotional Effects of Age - Results**

532 Applying this approach, the relationship between older age and increased positive affectivity
 533 disappeared. The computed ‘Net’ Positive Reactivity measure was negatively correlated with
 534 age ($r = -.188$, $p = 0.003$) highlighting the sensitivity of scaling effects in measuring
 535 emotional responding. Further, there was no significant correlation between age and the
 536 computed ‘Net’ Negative Reactivity ($r = .071$) or ‘Net’ Negative Regulation ($r = -.006$)
 537 measures. This highlights the importance of disaggregating positive and negative affectivity
 538 in the modelling in order to elucidate age-related positivity effects.

539

540 **Discussion**

541
 542 In a large population-representative sample, we show that positive affect in response to
 543 emotional and neutral stimuli increases with age, even after controlling for behavioural and
 544 neural measures of declining executive control, in line with the ‘positivity effect’ and the
 545 predictions of the SST (Carstensen, 1992, 2006) and ABM (Cacioppo et al., 2011). This was
 546 not the case for negative affect generated in response to negative stimuli, although we did
 547 find evidence of age-related increases in basal negative affect (based on negative affect
 548 ratings to neutral and positive stimuli). Furthermore, we show that emotion-related structural
 549 GM sources support and suppress affective responding and that certain brain-behaviour
 550 relationships change with age.

551

552 A key aspect of the data was the breakdown of the ‘net’ emotional effect often reported in
 553 studies of emotion regulation in older adults (Allard and Kensinger, 2014). Using this
 554 approach we were able to separate positive and negative scales in the emotion regulation
 555 condition and show that *Positive Regulation* (deriving positive affect from negative material)
 556 significantly increased with age, whereas negative regulation (downregulating negative
 557 affect) did not. The data are thus in agreement with evidence of improved emotion regulation
 558 with age (Urry and Gross, 2010). Importantly, when using the computed ‘net’ scores the
 559 relationship between older age and increased positive affectivity disappeared. The computed
 560 ‘net’ Positive Reactivity measure was negatively correlated with age, in line with our
 561 previous results (Schweizer et al., 2018). This highlights the importance of disaggregating
 562 positive and negative affectivity in the modelling in order to elucidate age-related positivity
 563 effects.

564
 565 Additionally, we included structural gray-matter sources derived from independent
 566 component analysis of structural MR data to examine brain-behaviour relationships and the
 567 influence of age on those relationships. Firstly, we observed decline in all gray matter sources
 568 associated with increasing age, in line with previous studies of age-related GMV loss (e.g.
 569 (Kalpouzos et al., 2009)). Using these sources, we then tested the diverging predictions of the
 570 amygdala-focused ABM and the frontal-focused cognitive control model at the neural level.
 571 We found no evidence in support of the ABM, with model comparison showing no unique
 572 contribution of the hippocampal/amygdala complex to any emotion factor and finding that
 573 age *devalued* the contribution of hippocampal/amygdala complex to *Positive Regulation*.
 574 Further to this, we found limited evidence for the cognitive control model, showing the pars
 575 opercularis of the IFG have a unique negative influence on *Basal Negative Affect* and *Positive*
 576 *Reactivity* as well as *Positive Regulation*. The IFG is a core emotion regulation brain region

577 typically engaged when reappraising negative information and is implicated in reducing
578 levels of negative affect (Goldin et al., 2008). However, our data suggest that the structural
579 integrity of the IFG showed a negative relationship with several of our latent factors. One
580 possibility is that the brain-behaviour relationship between the IFG and *Positive Regulation*
581 factor exerts a dampening effect on the positivity derived from negative stimuli. This is in
582 line with our previous findings using the same stimuli in an imaging version of the task on a
583 separate Cam-CAN sample, where decreased positivity was associated with decreased IFG
584 activation (Schweizer et al., 2018). These effects could feasibly extend to the *Positive*
585 *Reactivity* factor, whereby dispositional positivity is dampened as a function of
586 spontaneous/automatic emotion regulation/control. Finally, we showed that age devalued the
587 negative influence of the IFG and Medial FG on *Basal Negative Affect*, which speculatively
588 may account for the unexpected observed increases in this factor score with age. Overall, and
589 importantly in the context of the predictions of SST, age-related increases in *Positive*
590 *Regulation* and *Positive Reactivity* remained significant over and above the negative
591 influence of the neural measures.

592 In sum, this study used a population-derived sample from across the life span to investigate
593 positive and negative affective responses to ecologically valid stimuli in order to explore the
594 emotion-aging paradox. We found evidence broadly in line with the predictions of the SST,
595 finding that positive responding increased with increasing age. Furthermore, these changes
596 were related to the structural integrity of several neural regions typically associated with
597 emotion regulation. We provide neural evidence against the Aging Brain Model, instead
598 showing that age devalues the contribution of the hippocampal/amygdala complex to *Positive*
599 *Regulation*. Moreover, several of these brain-behaviour relationships remain unaffected by
600 age and may therefore constitute empirically derived neural markers to disentangle the
601 paradox of increased well-being in old age.

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604 **References**

- 605 Allard ES, Kensinger EA (2014) Age-related differences in functional connectivity during cognitive
606 emotion regulation. *J Gerontol B Psychol Sci Soc Sci* 69:852-860.
- 607 Allen JS, Bruss J, Damasio H (2005) The aging brain: the cognitive reserve hypothesis and hominid
608 evolution. *Am J Hum Biol* 17:673-689.
- 609 Bell AJ, Sejnowski TJ (1995) An information-maximization approach to blind separation and blind
610 deconvolution. *Neural Comput* 7:1129-1159.
- 611 Birditt KS, Fingerman KL, Almeida DM (2005) Age differences in exposure and reactions to
612 interpersonal tensions: a daily diary study. *Psychol Aging* 20:330-340.
- 613 Brabec J, Rulseh A, Hoyt B, Vizek M, Horinek D, Hort J, Petrovicky P (2010) Volumetry of the human
614 amygdala - an anatomical study. *Psychiatry Res* 182:67-72.
- 615 Braunstein LM, Gross JJ, Ochsner KN (2017) Explicit and implicit emotion regulation: a multi-level
616 framework. *Social cognitive and affective neuroscience* 12:1545-1557.
- 617 Braver TS, Barch DM (2002) A theory of cognitive control, aging cognition, and neuromodulation.
618 *Neurosci Biobehav Rev* 26:809-817.
- 619 Buhle JT, Silvers JA, Wager TD, Lopez R, Onyemekwu C, Kober H, Weber J, Ochsner KN (2014)
620 Cognitive reappraisal of emotion: a meta-analysis of human neuroimaging studies. *Cereb*
621 *Cortex* 24:2981-2990.
- 622 Burr DA, Castrellon JJ, Zald DH, Samanez-Larkin GR (2020) Emotion dynamics across adulthood in
623 everyday life: Older adults are more emotionally stable and better at regulating desires.
624 *Emotion*.
- 625 Bush G, Luu P, Posner MI (2000) Cognitive and emotional influences in anterior cingulate cortex.
626 *Trends in cognitive sciences* 4:215-222.
- 627 Cacioppo JT, Berntson GG, Bechara A, Tranel D, Hawkley LC (2011) Could an aging brain contribute to
628 subjective well-being? The value added by a social neuroscience perspective.
- 629 Calhoun VD, Adali T, Pearlson GD, Pekar JJ (2001) Spatial and temporal independent component
630 analysis of functional MRI data containing a pair of task-related waveforms. *Hum Brain*
631 *Mapp* 13:43-53.
- 632 Carstensen LL (1992) Motivation for social contact across the life span: a theory of socioemotional
633 selectivity. *Nebr Symp Motiv* 40:209-254.
- 634 Carstensen LL (2006) The influence of a sense of time on human development. *Science* 312:1913-
635 1915.
- 636 Carstensen LL, Pasupathi M, Mayr U, Nesselroade JR (2000) Emotional experience in everyday life
637 across the adult life span. *J Pers Soc Psychol* 79:644-655.
- 638 Carstensen LL, Turan B, Scheibe S, Ram N, Ersner-Hershfield H, Samanez-Larkin GR, Brooks KP,
639 Nesselroade JR (2011) Emotional experience improves with age: evidence based on over 10
640 years of experience sampling. *Psychol Aging* 26:21-33.
- 641 Charles ST (2010) Strength and Vulnerability Integration: A Model of Emotional Well-Being Across
642 Adulthood. *Psychological Bulletin* 136:1068-1091.
- 643 Charles ST, Mather M, Carstensen LL (2003) Aging and emotional memory: the forgettable nature of
644 negative images for older adults. *J Exp Psychol Gen* 132:310-324.
- 645 Diekhof EK, Geier K, Falkai P, Gruber O (2011) Fear is only as deep as the mind allows: a coordinate-
646 based meta-analysis of neuroimaging studies on the regulation of negative affect.
647 *Neuroimage* 58:275-285.
- 648 Duncan J (1995) Attention, intelligence, and the frontal lobes.

- 649 Fjell AM, Walhovd KB, Fennema-Notestine C, McEvoy LK, Hagler DJ, Holland D, Brewer JB, Dale AM
650 (2009a) One-year brain atrophy evident in healthy aging. *J Neurosci* 29:15223-15231.
- 651 Fjell AM, Westlye LT, Amlie I, Espeseth T, Reinvang I, Raz N, Agartz I, Salat DH, Greve DN, Fischl B,
652 Dale AM, Walhovd KB (2009b) High consistency of regional cortical thinning in aging across
653 multiple samples. *Cereb Cortex* 19:2001-2012.
- 654 Frank DW, Dewitt M, Hudgens-Haney M, Schaeffer DJ, Ball BH, Schwarz NF, Hussein AA, Smart LM,
655 Sabatinelli D (2014) Emotion regulation: quantitative meta-analysis of functional activation
656 and deactivation. *Neurosci Biobehav Rev* 45:202-211.
- 657 Goldin PR, McRae K, Ramel W, Gross JJ (2008) The neural bases of emotion regulation: reappraisal
658 and suppression of negative emotion. *Biol Psychiatry* 63:577-586.
- 659 Henson RN et al. (2016) Multiple determinants of lifespan memory differences. *Scientific Reports*
660 6:32527.
- 661 John OP, Gross JJ (2004) Healthy and unhealthy emotion regulation: Personality processes, individual
662 differences, and life span development. *Journal of Personality* 72:1301-1333.
- 663 Kalpouzos G, Chetelat G, Baron JC, Landeau B, Mevel K, Godeau C, Barre L, Constans JM, Viader F,
664 Eustache F, Desgranges B (2009) Voxel-based mapping of brain gray matter volume and
665 glucose metabolism profiles in normal aging. *Neurobiol Aging* 30:112-124.
- 666 Kanske P, Heissler J, Schonfelder S, Wessa M (2012) Neural correlates of emotion regulation deficits
667 in remitted depression: the influence of regulation strategy, habitual regulation use, and
668 emotional valence. *Neuroimage* 61:686-693.
- 669 Kennedy Q, Mather M, Carstensen LL (2004) The role of motivation in the age-related positivity
670 effect in autobiographical memory. *Psychol Sci* 15:208-214.
- 671 Kievit RA, van Rooijen H, Wicherts JM, Waldorp LJ, Kan KJ, Scholte HS, Borsboom D (2012)
672 Intelligence and the brain: A model-based approach. *Cogn Neurosci* 3:89-97.
- 673 Kohn N, Eickhoff SB, Scheller M, Laird AR, Fox PT, Habel U (2014) Neural network of cognitive
674 emotion regulation--an ALE meta-analysis and MACM analysis. *Neuroimage* 87:345-355.
- 675 Kring AM, Gordon AH (1998) Sex differences in emotion: expression, experience, and physiology. *J*
676 *Pers Soc Psychol* 74:686-703.
- 677 Labouvie-Vief G (2003) Dynamic Integration: Affect, Cognition, and the Self in Adulthood. *Current*
678 *Directions in Psychological Science* 12:201-206.
- 679 Livingstone KM, Isaacowitz DM (2019) Age Similarities and Differences in Spontaneous Use of
680 Emotion Regulation Tactics Across Five Laboratory Tasks. *Journal of Experimental*
681 *Psychology-General* 148:1972-1992.
- 682 Martins B, Sheppes G, Gross JJ, Mather M (2018) Age Differences in Emotion Regulation Choice:
683 Older Adults Use Distraction Less Than Younger Adults in High-Intensity Positive Contexts. *J*
684 *Gerontol B Psychol Sci Soc Sci* 73:603-611.
- 685 Mather M, Carstensen LL (2005) Aging and motivated cognition: the positivity effect in attention and
686 memory. *Trends Cogn Sci* 9:496-502.
- 687 Mather M, Canli T, English T, Whitfield S, Wais P, Ochsner K, Gabrieli JD, Carstensen LL (2004)
688 Amygdala responses to emotionally valenced stimuli in older and younger adults. *Psychol Sci*
689 15:259-263.
- 690 Murphy J, Millgate E, Geary H, Catmur C, Bird G (2019) No effect of age on emotion recognition after
691 accounting for cognitive factors and depression. *Quarterly Journal of Experimental*
692 *Psychology* 72:2690-2704.
- 693 Ochsner KN, Gross JJ (2005) The cognitive control of emotion. *Trends Cogn Sci* 9:242-249.
- 694 ONS (2016) Measuring National Well-being: Happiness.
- 695 Opitz PC, Lee IA, Gross JJ, Urry HL (2014) Fluid cognitive ability is a resource for successful emotion
696 regulation in older and younger adults. *Front Psychol* 5:609.
- 697 Paulus MP, Stein MB, Craske MG, Bookheimer S, Taylor CT, Simmons AN, Sidhu N, Young KS, Fan BY
698 (2017) Latent variable analysis of positive and negative valence processing focused on

- 699 symptom and behavioral units of analysis in mood and anxiety disorders. *Journal of affective*
700 *disorders* 216:17-29.
- 701 Phan KL, Wager T, Taylor SF, Liberzon I (2002) Functional neuroanatomy of emotion: a meta-analysis
702 of emotion activation studies in PET and fMRI. *Neuroimage* 16:331-348.
- 703 Phillips LH, Henry JD, Hosie JA, Milne AB (2008) Effective regulation of the experience and expression
704 of negative affect in old age. *J Gerontol B-Psychol* 63:P138-P145.
- 705 Quirk GJ, Beer JS (2006) Prefrontal involvement in the regulation of emotion: convergence of rat and
706 human studies. *Curr Opin Neurobiol* 16:723-727.
- 707 Rosseel Y (2012) lavaan: An R Package for Structural Equation Modeling. 2012 48:36.
- 708 Schermelleh-Engel K, Moosbrugger H, Müller H (2003) Evaluating the Fit of Structural Equation
709 Models: Tests of Significance and Descriptive Goodness-of-Fit Measures. *Methods of*
710 *Psychological Research* 8:23-74.
- 711 Schweizer S, Grahm J, Hampshire A, Mobbs D, Dalgleish T (2013) Training the emotional brain:
712 improving affective control through emotional working memory training. *J Neurosci*
713 33:5301-5311.
- 714 Schweizer S, Walsh ND, Stretton J, Dunn VJ, Goodyer IM, Dalgleish T (2016) Enhanced emotion
715 regulation capacity and its neural substrates in those exposed to moderate childhood
716 adversity. *Soc Cogn Affect Neurosci* 11:272-281.
- 717 Schweizer S, J. S, van Belle J, Price D, Calder AJ, T. D (2018) Age-related decline in positive emotional
718 reactivity and emotion regulation in a population-derived cohort. *PsyArXiv Preprints*.
- 719 Shafto MA, Tyler LK, Dixon M, Taylor JR, Rowe JB, Cusack R, Calder AJ, Marslen-Wilson WD, Duncan
720 J, Dalgleish T, Henson RN, Brayne C, Matthews FE, Cam CAN (2014) The Cambridge Centre
721 for Ageing and Neuroscience (Cam-CAN) study protocol: a cross-sectional, lifespan,
722 multidisciplinary examination of healthy cognitive ageing. *BMC Neurol* 14:204.
- 723 Sims T, Hogan C, Carstensen L (2015) Selectivity as an Emotion Regulation Strategy: Lessons from
724 Older Adults. *Curr Opin Psychol* 3:80-84.
- 725 Taylor JR, Williams N, Cusack R, Auer T, Shafto MA, Dixon M, Tyler LK, Cam C, Henson RN (2017) The
726 Cambridge Centre for Ageing and Neuroscience (Cam-CAN) data repository: Structural and
727 functional MRI, MEG, and cognitive data from a cross-sectional adult lifespan sample.
728 *Neuroimage* 144:262-269.
- 729 Urry HL, Gross JJ (2010) Emotion Regulation in Older Age. *Current Directions in Psychological Science*
730 19:352-357.
- 731 Viinikainen M, Jaaskelainen IR, Alexandrov Y, Balk MH, Autti T, Sams M (2010) Nonlinear
732 Relationship Between Emotional Valence and Brain Activity: Evidence of Separate Negative
733 and Positive Valence Dimensions. *Human Brain Mapping* 31:1030-1040.
- 734 Winecoff A, LaBar KS, Madden DJ, Cabeza R, Huettel SA (2011) Cognitive and neural contributors to
735 emotion regulation in aging. *Social cognitive and affective neuroscience* 6:165-176.
- 736 Winecoff A, Clithero JA, Carter RM, Bergman SR, Wang L, Huettel SA (2013) Ventromedial prefrontal
737 cortex encodes emotional value. *J Neurosci* 33:11032-11039.
- 738 Xu L, Groth KM, Pearlson G, Schretlen DJ, Calhoun VD (2009) Source-based morphometry: The use of
739 independent component analysis to identify gray matter differences with application to
740 schizophrenia. *Human brain mapping* 30:711-724.

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759 **Figure Legends**

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761 **Figure 1. A sample trial of the Emotion Reactivity and Regulation Task where**
762 **participants viewed a set of negative, neutral or positive 30-second film clips. In the**
763 **‘WATCH’ condition, participants were asked to simply watch the films and allow their**
764 **emotions to arise naturally. In the ‘REGULATE’ condition (the example shown in the**
765 **figure), participants were asked to reappraise their emotions to a negative film by**
766 **changing the way they thought about the film’s content. After each film clip,**
767 **participants rated how the positive and negative affect that they felt during the film on**
768 **two valence scale, as well as their compliance with the task instructions.**

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784 **Figure 2. Modified four-factor measurement model and the effects of age. Black paths**

785 **indicate positive associations, grey paths indicate negative associations, and dashed lines**

786 **indicate non-significant associations.**

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809 **Figure 3. SEM of Age and Emotion including cognitive control (indexed by fluid IQ),**
810 **education, gender and depression measures. Age remained significantly positively**
811 **associated with 3 emotion factors when accounting for this additional information.**
812 ***Positive Reactivity* and *Basal Negative Affect* and *Positive Regulation* all increased with**
813 **increasing age. Black paths indicate positive associations, grey paths indicate negative**
814 **associations, and dashed lines indicate non-significant associations. Dep = Depression;**
815 **Edu = Educational level; IQ = Intelligence Quotient. Coefficient weights have been**
816 **excluded for clarity (see main text).**

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834 **Figure 4. Source-Based Morphometry sources relationship to age. Each show decline**
835 **linearly with age. Effect size is shown as r^2 . PrC/PCC = Precuneus/Posterior Cingulate**
836 **Cortex; MTG = Middle Temporal Gyrus; MFG = Middle Frontal Gyrus; VS = Ventral**
837 **Striatum; MedFG = Medial Frontal Gyrus; IFG Op = Inferior Frontal Gyrus**
838 **Operculum; SFG = Superior Frontal Gyrus; A/MCC = Anterior/Middle Cingulate**
839 **Gyrus; IFG PT = Inferior Frontal Gyrus Pars Triangularis; Hc/Amyg = Hippocampal**
840 **Complex/Amygdala; AI = Anterior Insula; AG = Angular Gyrus.**

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Figure 5. Brain-Behaviour model with age included. 3 paths no longer reach significance; pars opercularis and medial frontal gyrus to *Basal Negative Affect*, and hippocampal/amygdala complex to *Positive Regulation* (dashed lines). For clarity, only significant/changed paths are shown. Black paths indicate positive associations, grey paths indicate negative associations, and dashed lines indicate non-significant associations. For full model fit, see Table 2. Dep = Depression; Edu = Educational level. IFG_Op = Inferior Frontal Gyrus Pars Opercularis; MedFG = Medial Frontal Gyrus; HC/A = Hippocampal/Amygdala Complex; MTG = Middle Temporal Gyrus; AG = Angular Gyrus. Coefficient weights have been excluded for clarity (see main text).

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884 **Figure 6. Scatterplots of the brain-behaviour pathways in the full model. Pos Reg =**

885 **Positive Regulation; Basal Neg = Basal Negative Affect; Pos Reac = Positive Reactivity;**

886 **IFG Op = Inferior Frontal Gyrus Pars Opercularis; MedFG = Medial Frontal Gyrus;**

887 **HC/A = Hippocampal/Amygdala Complex; MTG = Middle Temporal Gyrus; AG =**

888 **Angular Gyrus.**

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Table 1. Sample demographics

<i>n</i>	27	38	44	36	40	39	25
Age range; <i>M</i> (<i>sd</i>)	18-28; 24.1 (3.6)	29-38; 33.8 (2.6)	39-48; 44.6 (2.9)	49-58; 53.7 (2.9)	59-68; 64.1 (2.7)	69-88; 73.4 (3.1)	79-88; 82.3 (2.8)
Female <i>n</i> (%)	16 (59)	19 (50)	21 (48)	21 (58)	19 (47)	22 (56)	13 (52)
Education <i>n</i> (%)							
None		1 (3)	3 (7)	1 (3)	3 (7)	5 (13)	4 (16)
GCSE	5 (18.5)	3 (8)	7 (16)	6 (17)	13 (33)	9 (23)	5 (20)
A-level	5 (18.5)	5 (13)		9 (25)	4 (10)	4 (10)	4 (16)
University degree	17 (63)	29 (76)	34 (77)	20 (55)	20 (50)	21 (54)	12 (48)
Fluid intelligence <i>M</i> (<i>sd</i>)	37.3 (4.7)	37.5 (3.6)	35.8 (3.7)	33.4 (4.2)	30.9 (4.7)	27.7 (5.7)	24.9 (3.5)

M= Mean; sd = standard deviation.

Table 2. Fits of the main SEMs tested.

Model	χ^2	df	RMSEA	CFI	SB	AIC
Single Factor	305	20	0.24	0.69	1.40	5084

Hierarchical Factor	DNC	DNC	DNC	DNC	DNC	DNC
Four Factor	88	12	0.16	0.92	1.22	4781
Four Factor Modified	11	9	0.034	0.99	1.25	4694
Four Factor Modified with Age	17	13	0.038	0.99	1.30	6747
Four Factor with Age, Education & IQ	52	22	0.075	0.97	1.20	8007
Four Factor with Age, Education & IQ*	65	34	0.061	0.97	1.11	9397
Four Factor with GM & Education*	79	69	0.024	0.99	1.01	13550
Four Factor with GM & Age*	93	87	0.017	0.99	1.02	15469

938 **GM = Gray-matter; DNC = Did not converge; RMSEA = Root Mean Square Error of**
939 **Approximation; CFI = Comparative Fit Index; SB = Satorra-Bentler Scaled test**
940 **statistic; AIC = Akaike's Information Criterion; * = these models additionally contained**
941 **Gender and Depression variables (affecting all latent factors). Note that the χ^2 reported**
942 **is the Satorra-Bentler scaled χ^2 , with the scaling factor reported as SB.**

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951 **Table 3. Correlation Matrix of Source-Based Morphometry Sources.**
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Correlations		Precuneus/Posterior Cingulate Cortex	Superior Frontal Gyri	Middle Temporal Gyrus	Anterior/Middle Cingulate	Middle Frontal Gyrus	Inferior Frontal Gyrus; Pars Triangularis	Ventral Striatum	Hippocampal/Amgydala Complex	Medial Frontal Gyrus	Anterior Insula	Inferior Frontal Gyrus, Operculum
Superior Frontal Gyrus	Pearson Correlation	.371**										
	Sig. (2-tailed)	0.000										
	N	249										
Middle Temporal Gyrus	Pearson Correlation	.620**	.381*									
	Sig. (2-tailed)	0.000	0.000									
	N	249	249									
Anterior/Middle Cingulate	Pearson Correlation	.229**	.141*	.338**								
	Sig. (2-tailed)	0.000	0.027	0.000								
	N	249	249	249								
Middle Frontal Gyrus	Pearson Correlation	.518**	.590*	.502**	.244**							

	Sig. (2-tailed)	0.000	0.000	0.000	0.000							
	N	249	249	249	249							
Inferior Frontal Gyrus; Pars Triangularis	Pearson Correlation	.564**	.483*	.564**	.298**	.577**						
	Sig. (2-tailed)	0.000	0.000	0.000	0.000	0.000						
	N	249	249	249	249	249						
Ventral Striatum	Pearson Correlation	.563**	.457*	.579**	.264**	.532**	.536**					
	Sig. (2-tailed)	0.000	0.000	0.000	0.000	0.000	0.000					
	N	249	249	249	249	249	249					
Hippocampal/Amgdala Complex	Pearson Correlation	.569**	.306*	.659**	.322**	.462**	.523**	.531*				
	Sig. (2-tailed)	0.000	0.000	0.000	0.000	0.000	0.000	0.000				
	N	249	249	249	249	249	249	249				
Medial Frontal Gyrus	Pearson Correlation	.647**	.453*	.706**	.253**	.634**	.667**	.624*	.615**			
	Sig. (2-tailed)	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000			
	N	249	249	249	249	249	249	249	249			
Anterior Insula	Pearson Correlation	.584**	.422*	.623**	.358**	.495**	.618**	.601*	.569**	.688**		
	Sig. (2-tailed)	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000		

	tailed)					0				0		
	N	249	249	249	249	249	249	249	249	249	249	
Inferior Frontal Gyrus, Operculum	Pearson Correlation	.494**	.291*	.578**	.246**	.442**	.470**	.391*	.503**	.597**	.523*	
	Sig. (2-tailed)	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	
	N	249	249	249	249	249	249	249	249	249	249	
Angular Gyrus	Pearson Correlation	.284**	0.074	.327**	0.094	.189**	.188**	.161*	.280**	.284**	.230*	.186**
	Sig. (2-tailed)	0.000	0.246	0.000	0.141	0.003	0.003	0.011	0.000	0.000	0.000	0.003
	N	249	249	249	249	249	249	249	249	249	249	249
**. Correlation is significant at the 0.01 level (2-tailed).												
*. Correlation is significant at the 0.05 level (2-tailed).												

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