HHS Public Access

Author manuscript

Brain Res. Author manuscript; available in PMC 2017 August 01.

Published in final edited form as:

Brain Res. 2016 August 1; 1644: 22–31. doi:10.1016/j.brainres.2016.05.007.

Dedifferentiated face processing in older adults is linked to lower resting state metabolic activity in fusiform face area

Leslie Zebrowitz[a], **Noreen Ward**[b], **Jasmine Boshyan**[a], **Angela Gutchess**[a], and **Nouchine Hadjikhani**[b,c,*]

[a]Department of Psychology, Brandeis University, Waltham, MA 02453, USA

[b]MGH/HST Athinoula A. Martinos Center for Biomedical Imaging, Harvard University, Boston, MA 02129 USA

[c]Gillberg Neuropsychiatry Center, Gothenburg University, Sweden

Abstract

We used multimodal brain imaging to examine possible mediators of age-related neural dedifferentiation (less specific neural activation) to different categories of stimuli that had been shown in previous research. Specifically, we examined resting blood flow and brain activation in areas involved in object, place and face perception. We observed lower activation, specificity, and resting blood flow for older adults (OA) than younger adults (YA) in the fusiform face area (FFA) but not in the other regions of interest. Mediation analyses further revealed that FFA resting state blood flow mediated age differences in FFA specificity, whereas age differences in visual and cognitive function and cortical thickness did not. Whole brain analyses also revealed more activated voxels for all categories in OA, as well as more frontal activation for faces but not for the other categories in OA than YA. Less FFA specificity coupled with more frontal activation when passively viewing faces suggest that OA have more difficulty recruiting specialized face processing mechanisms, and the lower FFA metabolic activity even when faces are not being processed suggests an OA deficiency in the neural substrate underlying face processing. Our data point to a detuning of face-selective mechanisms in older adults.

Keywords

Aging; Face processing; Dedifferentiation; FMRI; Fusiform face area; Cerebral blood flow

1. Introduction

Theories of life span cognition propose that development is marked by the differentiation of abilities from childhood through adulthood with dedifferentiation in older adulthood when cognitive abilities become less distinctive and more homogeneous (Balinsky, 1941; Baltes, 1987; Ghisletta and de Ribaupierre, 2005). Age-related changes in differentiation have been evidenced at both the behavioral and neural levels. The present study extended previous

*Correspondence to: Athinoula A. Martinos Center for Biomedical Imaging, 149 13th Street, Charlestown, MA 02129, USA., nouchine@nmr.mgh.harvard.edu (N. Hadjikhani).

research examining older adults (OA) neural dedifferentiation in visual processing by investigating whether it can be explained by age-related declines in visual acuity, cognitive function, cortical thickness, and/or cerebral blood flow.

Neural dedifferentiation can be conceptualized as an increasingly shared neural substrate for particular stimuli or tasks that yield less specificity in the activation pattern. Age related increases have been observed in various paradigms, and two non-mutually exclusive explanations for these age-related changes in specificity have been suggested: (1) functional compensation for neural decline that may yield age-related changes in recruitment of additional regions of activation and (2) difficulty recruiting specialized neural mechanisms that may yield age-related decreases in the 'tuning' of activation.

Evidence for neural dedifferentiation in OA has been empirically demonstrated in more brain activation in OA than younger adults (YA) during various cognitive tasks (Burianova et al., 2013; Grady, 2002; Zarahn et al., 2007). Greater activation in OA has been noted most frequently in frontal regions, consistent with the idea that this is a compensatory mechanism reflecting increased effort or increased demands on executive functions (Cabeza et al., 1997; Grady et al., 1994). Other research has provided evidence for dedifferentiation within the visual system. OA show within category dedifferentiation of faces, as evidenced by greater OA than YA adaptation in the FFA to faces that are moderately similar (Goh et al., 2010). This effect parallels behavioral research demonstrating that OA make fewer distinctions among faces when forming trait impressions (Ng et al., 2014), and show reduced accuracy in both face recognition (Bartlett and Leslie, 1986; Bartlett et al., 1989; Goh et al., 2010) and emotion recognition (Orgeta and Phillips, 2008; Ruffman et al., 2008, 2009a; Slessor et al., 2010), although the latter age effect may depend on the methods used (Hahn et al., 2006; Mather and Knight, 2006; Murphy et al., 2010; Ruffman et al., 2009b). OA also show between category neural dedifferentiation when attempting to remember a series of visual stimuli (Park et al., 2004), or when passively viewing them (Park et al., 2012), with a smaller difference in activation to faces than to other stimulus categories in voxels specialized for faces, and a parallel age difference for activation to buildings or chairs vs. other stimulus categories in voxels specialized for places or objects, respectively. These effects are consistent with the idea that OA dedifferentiation reflects difficulty recruiting specialized neural mechanisms.

In the present study, we examined age-related neural dedifferentiation during passive viewing of different categories of stimuli that included faces, buildings, objects and cars. We examined activation in whole brain as well as in ROIs located in the areas that should respond maximally to faces, buildings, or objects: the FFA (Kanwisher et al., 1997), the parahippocampal place area (PPA; (Epstein et al., 1999)), and the medial fusiform object area (FOA; (Hadjikhani et al., 2004; Ishai et al., 1999)), respectively. We also included a category of front views of car stimuli, because some research indicates that the face-like appearance of car grills can elicit activation in the FFA (Kuhn et al., 2014). Neural dedifferentiation in OA was conceptualized as: 1) a less specific, broader activation pattern to stimuli from each of the categories in the whole brain 2) a less specific, more uniform pattern of activation in each ROI, as evidenced by weaker differences between the preferred stimulus category and the other categories. Finally, in addition to the aim of replicating

previous research evidence for OA neural dedifferentiation, we investigated possible mediators of these effects, including visual acuity, cognitive function, cortical thickness, and blood flow at rest.

Since OA show reduced visual acuity and contrast sensitivity, these peripheral mechanisms may mediate OA neural dedifferentiation to visual stimuli. OA deficits in executive function or speed also could mediate, as Grady (2002) found greater dedifferentiation with increased cognitive load, and Park et al. (2010) found that greater fluid processing ability predicted greater neural specificity of the BOLD response to faces and houses. Gray matter cortical thinning also has been shown in healthy OA (Fjell et al., 2009; Liu et al., 2010; Salat et al., 2004). Here we examined whether age-related cortical thinning mediated age-related BOLD dedifferentiation. Recent research has shown that weaker BOLD activation in OA than YA at rest is due not to age-related differences in cerebral vasculature alone but also to cognitive and structural factors (Marstaller et al., 2015). In order to examine whether lower cerebral blood flow (CBF) was at the origin of differences in BOLD signal, we also measured CBF at rest using asymmetric spin labeling, and determined whether any age differences in CBF mediated age differences in BOLD dedifferentiation within the ROIs we examined.

2. Results

2.1. Vision and cognition measures

As shown in Table 1, OA performed significantly worse than YA on visual acuity and contrast sensitivity, but not on the Benton face recognition test. OA also showed significantly slower performance on a speeded pattern comparison task, consistent with decreases in processing speed in older adulthood (Salthouse, 1993). On the card sorting task assessing executive control, OA showed marginally more perseverative errors, and significantly more non-perseverative errors. In contrast to poorer performance by OA on preceding measures, they performed better on a vocabulary task, consistent with the maintenance of crystallized intelligence in older adulthood and previous research with community dwelling OA who volunteer as research participants in our lab (Horn and Cattell, 1967; Zebrowitz et al., 2013). There were no differences between groups in task performance (pressing a key every time a red crossed appeared) during the scanning.

2.2. Cortical thickness

Using QDEC and a whole brain analysis approach, we observed regional thinning of the cortex in OA compared with YA, replicating results reported by many other studies (e.g. (Fjell et al., 2009; Liu et al., 2010; Salat et al., 2004)). Thinning was bilateral and most prominent in motor and premotor cortex, superior and inferior frontal gyri, angular gyri, superior and middle temporal lobes and pole, and posterior cingulate, as well as in the calcarine sulcus, the lingual gyri and the ventro-lateral prefrontal cortex (see Fig. 1).

In addition, a 2 (age group) × 3 (ROIs: FFA, FOA, PPA) ANOVA on measures of cortical thickness revealed a significant main effect for age group, $F(1,36)=24.14$, $p<.001$, $\eta_P^2=.401$, reflecting lower thickness for OA than YA that was not qualified by an interaction with ROI, $F(2,72)=1.26$, $p=.291$, $\eta_P^2=.034$.

2.3. Blood flow patterns

Whole brain analysis revealed regional cortical decrease of CBF bilaterally in the posterior and lateral occipital cortex, the fusiform gyrus, the inferior temporal gyrus, the dorsolateral prefrontal cortex, and the medial prefrontal cortex replicating results reported by others (e.g. (Chen et al., 2011)). Reduced CBF was also observed in subcortical structures including the thalamus, the caudate and the brainstem.

ROI analysis: a 2 (participant age) × 3 (ROIs: FFA, FOA, PPA) revealed no significant main effect for age, (YA M=51.45, SE=3.49 and OA M=43.83, SE=3.58), $F(1,35)=2.32$, $p=.136$, $\eta_p^2=.062$. However there was a significant main effect for ROI, $F(2,70)=3.82$, $p=.027$, $\eta_p^2=.098$ that was qualified by a significant interaction with age, $F(2,70)=3.99$, $p=.023$, $\eta_p^2=.102$. Planned comparisons revealed greater blood flow in FFA for YA (M=55.76, SE=3.56) than OA (M=40.87, SE=3.66), $p=.006$, with no significant age differences in blood flow in either PPA or FOA, both $ps > .423$.

2.4. BOLD activation

2.4.1. Whole brain analysis—Consistent with the broader, less specific activation associated with dedifferentiation, whole brain analysis showed that OA had more extensive activation than YA for all stimulus categories (Fig. 2 and Table 2). Comparing the proportion of all voxels activated for YA and OA revealed significant differences for: **faces:** Z=87.96, $p<.01$; **buildings**: Z=87.85, $p<.01$; **objects**: Z=165.55, $p<.01$; and **cars**: Z=25.47, $p<.01$. OA showed not only broader activation than YA, but also significantly stronger activation for three of the four categories. For faces, this was shown in an anterior cluster (frontal pole, medial and dorsolateral prefrontal cortex; anterior cingulate) and a posterior cluster (parietal cortex; cuneus). For buildings and objects, OA stronger activation was in posterior clusters (pericalcarine cortex for both plus lingual gyrus for buildings). YA showed stronger activation than OA in the temporal fusiform cortex for all categories, although the age difference was significant only for cars.

2.4.2. ROI analyses

2.4.2.1. Overview of analyses: We assessed dedifferentiation in the three a priori ROIs (FFA, PPA, and FOA) in two ways. First, we performed ANOVAs to examine percent signal change with participant age (2) as a between groups variable and stimulus category (4) as a within groups variable, and we examined the rater age × stimulus category effects to determine whether there was less specificity of activation for OA than YA. Second, we examined age differences for three differentiation indices that were computed for each participant following the procedure used by Voss et al. (2008). FFA differentiation was the difference between activation to faces and the mean activation to buildings, objects, and cars divided by the average standard deviation of FFA activation to all four categories. PPA differentiation was the difference between activation to buildings and the mean activation to faces, objects, and cars divided by the average standard deviation of PPA activation to all four categories. FOA differentiation was the difference between the predicted activation to objects and the mean activation to faces, buildings, and cars divided by the average standard deviation of FOA activation to all four categories.

2.4.2.2. FFA: The 2-way ANOVA on signal change revealed a significant main effect for age, $F(1,36)=4.81$, $p=.035$, $\eta_p^2=.118$, with greater FFA activation for YA (M=40.12, SE=5.29) than OA (M=23.28, SE=5.57). There was also a stimulus category effect in FFA activation, $F(3,108)=9.03$, $p<.001$, $\eta_p^2=.201$ that was qualified by a significant participant age × category interaction, F (3,108)=4.55, $p=.005$, $\eta_p^2=.112$ (Fig. 3). Planned comparisons within participant age revealed strong differentiation for YA, with the FFA showing greater activation to faces than to all other categories, *ps*<.01. Also as expected, FFA activation for OA was less differentiated, with activation to faces greater than cars, $p=.007$, but not significantly different from buildings, $p=.468$ and objects, $p=.354$. Comparing the OA and YA FFA differentiation indices corroborated the conclusion that YA showed more differentiation ($M=2.32$, $SE=.51$) than OA ($M=.74$, $SE=.53$), $F(1,36)=4.63$, $p=.038$, $\eta_p^2=.114$.

2.4.2.3. PPA: The 2-way ANOVA on signal change revealed no significant age difference in PPA activation, $F(1,36)=.74$, $p=.395$, $\eta_p^2=.020$. However, there was a significant effect for stimulus category, $F(3,108)=10.23$, $p<.001$, $\eta_p^2=.221$, that was qualified by a significant participant age × category effect, $F(3,108)=2.88$, $p=.039$, $\eta_p^2=.074$ (Fig. 3). Planned comparisons within participant age revealed strong differentiation for YA, with the PPA showing greater activation to buildings than to all other categories, *ps*<.01. PPA activation for OA was less differentiated, with greater activation to buildings than cars, $p=.004$, but only marginally greater activation to buildings than to faces, $p=.096$, and no significant difference in activation to buildings and objects, $p=.309$. Comparing OA and YA PPA differentiation indices revealed that despite a somewhat less specific activation pattern for OA than YA, the age difference was not significant (YA $M=1.98$, $SE=.60$; OA $M=1.57$, $SE=.63$), $F(1,36)=.22$, $p=.644$, $\eta_p^2=.006$.

2.4.2.4. FOA: The 2-way ANOVA on signal change revealed a significant main effect for age, $F(1,36)=6.26$, $p=.017$, $\eta_p^2=.148$, with greater FOA activation for YA (M=30.51, SE=5.18) than OA (M=11.69, SE=5.46). There was also a significant main effect for stimulus category in FOA activation, F (3,108)=19.02, $p<.001$, $\eta_p^2=.346$, but only a marginally significant participant age × category effect, F (3,108)=2.46, $p=.067$, $\eta_p^2=.064$. As shown in Fig. 3, neither YA nor OA showed the expected pattern of greater activation to objects than other categories. Comparing the OA and YA FOA differentiation indices revealed equally low differentiation for YA ($M=.67$, $SE=.83$) and OA ($M=.60$, $SE=.87$), $F(1,36)=.00$, $p=.957$, $\eta_p^2=.000$.

2.5. Mediation of age differences in BOLD differentiation in ROIs

2.5.1. Overview of analyses—We used the differentiation indices for FFA, PPA, and FOA to test mediation of age–related dedifferentiation by vision and cognitive measures, cortical thickness, and blood flow. The mediation analyses followed guidelines provided by Kenny and Judd (2014) together with an online calculator to implement the bootstrapping approach (Preacher and Hayes, 2004) that yielded confidence intervals for significance of the change in the direct effect of the causal variable when the mediator variable was controlled (Selig and Preacher, 2008). Necessary conditions for mediation are that the putative causal variable (i.e. age) be significantly related to the mediator (Step 2) and that

the mediator be significantly related to the outcome variable (i.e. differentiation) with the causal variable controlled (step 3) (Kenny and Judd, 2014). We examined mediation of age-related dedifferentiation between age groups as well as within each age group.

2.5.2. Mediation by vision and cognition—As reported above, across both age groups, OA showed less FFA differentiation than YA, with no age group differences in PPA or FOA differentiation (Step 1). OA also showed poorer performance than YA on many measures of vision and cognition (Step 2). However, Step 3 was satisfied in only one case, which yielded a paradoxical suppressor effect: age differences in non-perseverative errors on the BCST suppressed, rather than mediated, age group differences in FFA differentiation, 95% CI [. 161, 2.021].

Analyses within age groups revealed that increasing age in OA was associated with a marginally significant decrease in differentiation in FFA, β=−.423, p=.08, but not PPA or FOA, both ps>.10 (Step 1). None of the control variables satisfied the conditions for mediation of age effects on OA differentiation. Although increasing age in YA was not significantly associated with decreasing differentiation in FFA or FOA, both ps>.16, it was associated with decreasing differentiation in PPA, β=−.554, p=.011 (Step 1). Steps 2 and 3 were satisfied in only one case which yielded another paradoxical suppressor effect: age differences in non-perseverative errors on the BCST tended to suppress, rather than mediate effects of age on YA PPA differentiation, 90% CI [.016, .338]. In sum, age differences in measures of vision and cognition within each age group failed to mediate relationships between age and neural differentiation in any of the three ROIs.

2.5.3. Mediation by cortical thickness—Although OA showed less FFA differentiation than YA (Step 1) and less cortical thickness in FFA, PPA, and FOA (Step 2), cortical thickness was unrelated to neural differentiation with age group controlled (Step 3), all ps>. 44. Analyses within age groups further revealed that age was not related to cortical thickness in any of the ROIs (Step 2), all ps>.591, and that cortical thickness was unrelated to neural differentiation with actual age controlled (Step 3), all ps>.41. Thus, age differences in cortical thickness failed to mediate the differences in neural differentiation.

2.5.4. Mediation by blood flow—Not only did OA show less FFA differentiation than YA (Step 1) as well as lower FFA blood flow (Step 2), but also blood flow was marginally related to FFA but not PPA or FOA differentiation, with age group controlled (step 3), β=. 296, p=.099, for FFA; ps>.44 for PPA and FOA. Although the effect of age group on FFA differentiation lost significance with FFA blood flow controlled, β=−.188, p=.288, the reduction in the effect of age group was only marginally significant, 90% CI [−1.398, −. 015].

Analyses within age groups revealed that none of the mediation steps was significant for YA, all ps>.568. Among OA, age was marginally related to less FFA differentiation, β=−.423, p=.08 (Step 1). Age also predicted lower FFA blood flow, β=−.486, p=.041 (step 2), and resting state blood flow was significantly related to FFA but not PPA or FOA differentiation with age controlled (step 3), β=.538, p=.033 for FFA; ps>.22 for PPA and FOA. Moreover, the reduction in the effect of age on OA FFA differentiation when controlling blood flow

was significant, 95% CI [−.212, −.002]. In summary, age differences in resting state blood flow mediated age group differences in FFA differentiation as well as age differences within OA.

3. Discussion

Replicating previous research investigating neural dedifferentiation in OA, we found neural dedifferentiation during passive viewing of stimuli both at the level of the whole brain and within the FFA. We further found that neither age differences in visual and cognitive function nor age differences in cortical thickness could explain the age-related dedifferentiation of FFA activation to various stimulus categories. The mediation that our data did support was resting state blood flow, with age differences in resting state FFA blood flow predicting the differentiation of neural activation to stimuli within the FFA. This is the first evidence to indicate that OA may have a basic deficiency in the FFA.

Consistent with the hypothesis that neural dedifferentiation in OA is marked by a broadening of activation, whole brain activation data revealed that OA had more voxels significantly activated than YA when passively viewing faces, buildings, objects, and cars, and also showed significantly stronger activation to faces, buildings, and objects than YA in many brain regions. More, specifically, as in previous research documenting more OA than YA activity in frontal regions (Li et al., 2015) OA showed more frontal activation than YA when viewing faces. In addition, like previous research showing a decrease in fusiform activation with increasing age (Li et al., 2015), OA showed less activity in the fusiform gyrus than YA for all categories of stimuli in the whole brain analysis, although the age difference reached significance only for cars. The ROI analyses also showed significantly less activation for OA than YA in the fusiform gyrus (FFA and FOA). These analyses further revealed a significantly less differentiated pattern of FFA activation in OA than YA, consistent with previous research (Goh et al., 2010; Park et al., 2004). Whereas YA showed greater FFA activation to faces than buildings, objects, or cars, OA showed a ‘detuning’ of face selective cells to the preferred stimuli with greater FFA activation to faces than to cars, but no significant difference between faces and buildings or objects. Moreover, the summary differentiation index showed that this age difference in the specificity of FFA activation was significant, whereas there was no significant age difference in the PPA or FOA. Finally, although FFA activation was significantly lower overall in OA than YA, both OA and YA showed FFA activation significantly greater than zero for all categories, a finding that is consistent with other evidence that FFA responds strongly to categories other than faces (Mur et al., 2012; Rossion et al., 2012).

Despite strong age-related differences in visual and cognitive function, cortical thickness, and resting state blood flow, blood flow was the only significant mediator of the foregoing age-related neural dedifferentiation in the FFA. Resting state blood flow was lower for OA than YA in the FFA, but not in the other ROIs where there also were no significant age differences in differentiation. Resting state FFA blood flow also was negatively related to age in years within the OA group. Finally, resting state FFA blood flow was a marginally significant mediator of age group differences in FFA differentiation and a significant mediator of age differences within the OA group. This finding that OA loss of specificity in

FFA reactivity to faces is linked to lower metabolic activity in FFA even when not processing faces suggests that OA have a basic deficiency in the FFA.

Although one might suggest that diminished attention to stimuli in our passive viewing task rather than some deficiency in the FFA mechanism contributed to the dedifferentiation of FFA activation in OA, this alternative explanation is problematic. First, it would require arguing that YA looked at the faces more than OA did, whereas both YA and OA looked equally at the buildings and objects, an argument that seems implausible. Second, we maintained attention by requiring key press responses to red fixation crosses inserted randomly throughout the stimulus presentation. Third, dedifferentiation of neural responses to faces in OA has been shown in paradigms involving both passive viewing (Park et al., 2012) and active tasks (Burianova et al., 2013; Carp et al., 2011; Goh et al., 2010; Park et al., 2004, 2010; Voss et al., 2008). Fourth, the greater frontal activation to faces in OA, which is indicative of cognitive effort, also argues against the notion that OA were attending less to faces than YA. Finally, lower attention to faces by OA cannot explain the finding that resting state FFA blood flow mediated age differences in FFA differentiation.

The finding that the difference between the FFA response to faces and other categories is smaller for OA than YA as well as the finding that the FFA responds significantly to stimuli other than faces may be interpreted within the context of research on non-human primates. That work has revealed that several areas of the brain contain cells with higher firing rates to images of faces than other stimuli/objects. The middle face patch is the suggested homolog of FFA, and cells in this area also respond to non-face objects (Freiwald and Tsao, 2010; Issa et al., 2013; Meyers et al., 2015). At the population level, however, the responses elicited by face images are stronger than those by non-face images (Kiani et al., 2007; Tsao, 2006). A recent study in humans demonstrated that the FFA can respond strongly to images of the non-preferred category, and that some non-face images could activate the FFA more strongly than some face images (Mur et al., 2012). These results seem to indicate that the category membership of natural objects is encoded at the population level (Kiani et al., 2007; Kriegeskorte et al., 2008; Mur et al., 2012; Vogels, 1999). The FFA has activation profiles showing category steps, but graded within and outside the preferred category (Mur et al., 2012). In OA, the specificity for the preferred category (faces) diminishes, yielding comparatively more activation to the non-preferred categories, suggesting that OA have difficulty recruiting the specialized FFA neural mechanism. Previous studies have indeed demonstrated that aging affects the brain's ability to integrate elements that are crucial for facial identification (Habak et al., 2008), and recently Wilson et at. (2011) and colleagues have reported the presence of significant broadening of cortical bandwidths for face perception, providing additional evidence for a detuning phenomenon of face cells with aging.

While our design had sufficient power to detect age differences in the whole brain analyses and in FFA differentiation, it is possible that the non-significant age differences in PPA and FOA differentiation would become significant with greater statistical power. However, this seems unlikely given the absence of any trend, with the differentiation index Fs<1 for both of these ROIs. Also, although multiple statistical comparisons were made in the ROI analyses, the single summary differentiation index supports the conclusions drawn from the

multiple comparisons, which argues against the criticism that our conclusions are capitalizing on chance.

Although our study did not include behavioral measures, our finding that OA show a deficit in resting state activation in the FFA has behavioral significance. Specifically, resting state functional connectivity between spontaneous neural activity in occipital face area (OFA) and FFA predicts performance on a variety of face recognition tasks (Zhu et al., 2011). Thus, lower resting state FFA blood flow in OA may be associated not only with dedifferentiated FFA activation when viewing stimuli, but also with the inferior performance of OA on face recognition tasks that has been demonstrated in previous research (Bartlett and Leslie, 1986; Bartlett et al., 1989; Goh et al., 2010).

4. Experimental procedure

4.1. Participants

Forty-five participants were scanned. Of these, two OA and two YA participants were removed due to truncated data, 1 OA did not complete the study due to claustrophobia, 1 OA was removed due to excessive movement and 1 OA was removed due to BOLD responses ranging from 3.96 to 4.07 standard deviations above the mean. Of the remaining 38 participants, 20 (10 males) were YA aged 19–33 (M=24.55, SD=3.61), and 18 (8 males) were OA aged 65–88 (75.56, SD=7.14). OA were screened using the Mini-Mental State Examination (Folstein et al., 1975) all scoring above 28 out of 30 (M=30, SD=.60). All participants provided written informed consent before they took part in the study. The study was approved by the Massachusetts General Hospital Human Studies Committee under Protocol #2013P000707 and by the Brandeis University IRB #11,010, and all procedures followed the Declaration of Helsinki. All participants had normal or corrected-to-normal vision using magnet-compatible glasses.

4.2. Stimuli

Participants viewed 112 grayscale images equally divided between human faces, buildings (churches and houses), objects from different categories (e.g., screwdriver, shovel, vase), as well as objects from one category (cars), plus 32 Fourier phase-scrambled control images (8 scrambled faces, 8 scrambled objects, 8 scrambled cars and 8 scrambled houses) (Fig. 4). These stimuli were designed to minimize between-category sensory differences in size, luminosity, or spatial frequency (Hadjikhani and de Gelder, 2002). Control images were created by scrambling the phase information present in all the experimental stimuli so that the spatial frequency information was preserved but the visual information was meaningless (Hadjikhani and de Gelder, 2002).

4.3. Vision and cognition measures

Participants completed a set of measures to assess possible mediators of the predicted age differences in differentiation of neural responses to visual stimuli: visual acuity (Snellen Eye Chart); contrast sensitivity (Mars Letter Contrast Sensitivity Test, Mars Perceptrix, Chappaqua, NY); and facial recognition ability on the Benton Facial Recognition Test (Benton et al., 1983). Measures of general cognitive and executive abilities included the

Shipley Vocabulary Test assessing crystallized intelligence (Shipley, 1946); a timed Pattern Comparison Task assessing processing speed (Salthouse, 1993); a short-form 48 item computerized version of the Wisconsin Card Sort Task assessing executive functioning (the Berg Card Sort Task (BCST)) downloaded from http://pebl.sourceforge.net/battery.html and validated by Piper et al. (2012).

4.4. Cortical thickness

Cortical thickness was measured using FreeSurfer (Fischl et al., 1999). Group comparison was conducted using Qdec, part of the FreeSurfer suite (https://surfer.nmr.mgh.harvard.edu/fswiki/FsTutorial/QdecGroupAnalysis_freeview). Qdec fits a general linear model at each surface vertex to explain the data from all subjects in the study. We also examined cortical thickness in the ROIs selected for the BOLD activation study.

4.5. Blood flow

Pulsed arterial spin labeling (pASL) was used to measure cerebral blood flow. pASL allows the estimation of cerebral blood flow (CBF) using blood water as an endogenous tracer. The pASL imaging sequence uses short adiabatic pulses to tag the spins in blood. During the sequence, alternating tag and control images are obtained. The quantitative CBF value per voxel is calculated using the Standard Kinetic Model (Buxton, 2005). The script created to perform this calculation was based on Jean Chen's ASL processing steps (www.nmr.mgh.harvard.edu/~jjchen/ASL.html).

4.6. Design

Stimuli presented during MR scanning were generated with a Macintosh G4 computer using E-prime 2.0, and back-projected via an LCD projector and a mirror attached to the head coil onto an acrylic rear-projection screen (DaTex, Da-Lite Corp.) providing a visually-activated stimulus screen of 48° × 36°. Button press responses were collected using an MR-compatible button box connected to the Macintosh via a custom USB interface, providing both accuracy and reaction time information. A trigger mechanism was used to lock stimulus presentation to the start of each TR during the fMRI scan.

Subjects were asked to maintain fixation on a central fixation spot, where a green cross was superimposed in the center of every image. To maintain their attention, subjects were also asked to press a button in response to red fixation crosses (which appeared 12.5% of the time). Images were presented during one run (6 min 40) in 20 s blocks, at a rate of 2.5 s per image. Four blocks of each category (faces, buildings, objects, cars, scrambled stimuli) were presented in one run for a total of 20 blocks. The order of the blocks was randomized for each participant.

4.7. Imaging data acquisition and analysis

Anatomical and functional MR images were acquired with a 12-channel RF coil in a Siemens 3 T scanner (Siemens TrioTim, Erlangen) at the Martinos Center for Biomedical Imaging. The first scanning sequence consisted of Siemens's auto-align scout for the head allowing an automatic positioning and alignment of slices. Anatomical images were acquired using a multi-echo magnetization prepared rapid gradient echo sequence (ME-

MPRAGE: matrix=256 × 256; echo time (TE): TE1=1.64 ms, TE2=3.5 ms, TE3=5.36 ms, TE4=7.22 ms; repetition time (TR)= 2530 ms; flip angle=7°; slice thickness=1.33 mm; in-plane resolution=1 mm × 1 mm). After the structural scan, a series of 7 functional scans were acquired, including the one reported here, which was acquired at the end of the functional series. The total time for functional scans was approximately 60 min. The parameters for the current functional data were the following: echo planar imaging (EPI) sequence (41 axial slices with 3 mm thickness; in plane resolution=3.125 mm by 3.125 mm, matrix=64 × 64; FOV=200; TE=30 ms; TR=2500 ms; flip angle=90°, 160 timepoints) with a duration of 6.03 min. Finally, a pulsed ASL (pASL) sequence, (Siemens ep2d_pASL, FOV=220, matrix=320 × 320; slice thickness=4 mm, number slices=24, in-plane resolution=3.44 × 3.44, TR=4000, flip angle=90°, TE=13 ms, TI1=600 ms, TI2=1800 ms, number of frames=104, scanning time 7:02), was acquired at the end, while subjects were resting with eyes closed, to enable computation of cerebral blood flow (CBF).

Functional imaging data were preprocessed and analyzed with FSL (FEAT Version 5.98). Non-brain tissue was removed from high-resolution anatomical images using FSL BET and fed into FEAT. Data were motion corrected using MCFLIRT and motion parameters added as confound variables to the model. Participants with motion exceeding 3 mm were excluded from further processing (n=0 YA, 1 OA). Preprocessing further included spatial smoothing using a Gaussian kernel of 8 mm. The final analysis comprised 18 subjects in the OA group, and 20 in the YA group.

Subject-level statistical analysis was carried out in a block-design analysis for each category vs. baseline (faces, buildings, objects and cars vs. a scrambled version of all these stimuli) using FILM with local autocorrelation correction. Registration to high-resolution structural images was carried out using FLIRT. Registration to MNI standard space was then further refined using FNIRT nonlinear registration. Group-level analyses were carried out using mixed effects GLM analysis using FLAME 1 with automatic outlier detection. In modeling subject variability, this kind of analysis allows inference about the population from which the subjects are drawn. For within-group whole brain analyses, significance threshold was set at $p<0.05$, cluster corrected. Between-group differences were assessed using an independent t-test available in FSL. Statistical maps were registered to the FreeSurfer template brain and displayed on the surface.

4.8. ROI analyses

All ROIs were defined in independent tests conducted in independent subjects in previous studies on the MNI brain and back-projected onto each subject's individual space.

The FFA ROI was defined using an independent test of faces vs. scrambled faces. A 4*4*4 mm cube was drawn around a peak located at x=42, y=−46, z=−26 in MNI space. The PPA ROI was defined using an independent test of buildings vs. scrambled buildings. A 4*4*4 mm cube was drawn around a peak located at x=22, y=−38, z=−16 in MNI space. The FOA ROI was defined using an independent test of objects vs. scrambled objects. A 4*4*4 mm cube was drawn around a peak located at x=32, y=−48, z=−16 in MNI space. Mean percentage BOLD signal change was extracted from the contrast of parameter estimate at the

subject-level using FSL featquery. Effect of category (faces, cars, objects, buildings), and group (YA, OA) were assessed with ANOVA.

Acknowledgments

This work was supported by NIH Grant 5 RO1 AG038375 to LZ. The funder played no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript. The authors want to thank Eri Ichijo for her help with the cortical thickness analysis, and Luke Hanlin for his help with the data acquisition, as well as the participants who took part in the experiments.

References

Balinsky B. An analysis of the mental factors of various age groups from nine to sixty. Genet Psychol Monogr. 1941; 23:191–234.

Baltes MM. Theoretical propositions of life span developmental psychology: on the dynamics between growth and decline. Dev Psychol. 1987; 23:611–626.

Bartlett JC, Leslie JE. Aging and memory for faces versus single views of faces. Mem Cognit. 1986; 14:371–381.

Bartlett JC, Leslie JE, Tubbs A, Fulton A. Aging and memory for pictures of faces. Psychol Aging. 1989; 4:276–283. [PubMed: 2803620]

Benton, AL.; Hamsher, K.; Varney, NR.; Spreen, O. Test of Facial Recognition: Form SL. Oxford University Press; New York: 1983.

Burianova H, Lee Y, Grady CL, Moscovitch M. Age-related dedifferentiation and compensatory changes in the functional network underlying face processing. Neurobiol Aging. 2013; 34:2759–2767. [PubMed: 23870836]

Buxton RB. Quantifying CBF with arterial spin labeling. J Magn Reson Imaging. 2005; 22:723–726. [PubMed: 16261574]

Cabeza R, McIntosh AR, Tulving E, Nyberg L, Grady CL. Age-related differences in effective neural connectivity during encoding and recall. Neuroreport. 1997; 8:3479–3483. [PubMed: 9427311]

Carp J, Park J, Polk TA, Park DC. Age differences in neural distinctiveness revealed by multi-voxel pattern analysis. Neuroimage. 2011; 56:736–743. [PubMed: 20451629]

Chen JJ, Rosas HD, Salat DH. Age-associated reductions in cerebral blood flow are independent from regional atrophy. Neuroimage. 2011; 55:468–478. [PubMed: 21167947]

Epstein R, Harris A, Stanley D, Kanwisher N. The parahippocampal place area: recognition, navigation, or encoding? Neuron. 1999; 23:115–125. [PubMed: 10402198]

Fischl B, Sereno MI, Dale AM. Cortical surface-based analysis II: inflation, flattening, and a surface-based coordinate system. NeuroImage. 1999; 9:195–207. [PubMed: 9931269]

Fjell AM, Westlye LT, Amlien I, Espeseth T, Reinvang I, Raz N, Agartz I, Salat DH, Greve DN, Fischl B, Dale AM, Walhovd KB. High consistency of regional cortical thinning in aging across multiple samples. Cereb Cortex. 2009; 19:2001–2012. [PubMed: 19150922]

Folstein MF, Folstein SE, McHugh PR. “Mini-mental state”. A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res. 1975; 12:189–198. [PubMed: 1202204]

Freiwald WA, Tsao DY. Functional compartmentalization and viewpoint generalization within the macaque face-processing system. Science. 2010; 330:845–851. [PubMed: 21051642]

Ghisletta P, de Ribaupierre A. A dynamic investigation of cognitive dedifferentiation with control for retest: evidence from the Swiss Interdisciplinary Longitudinal Study on the Oldest Old. Psychol Aging. 2005; 20:671–682. [PubMed: 16420141]

Goh JO, Suzuki A, Park DC. Reduced neural selectivity increases FMRI adaptation with age during face discrimination. Neuroimage. 2010; 51:336–344. [PubMed: 20139012]

Grady CL, Maisog JM, Horwitz B, Ungerleider LG, Mentis MJ, Salerno JA, Pietrini P, Wagner E, Haxby JV. Age-related changes in cortical blood flow activation during visual processing of faces and location. J Neurosci. 1994; 14:1450–1462. [PubMed: 8126548]

Grady CL. Age-related differences in face processing: a meta-analysis of three functional neuroimaging experiments. Can J Exp Psychol. 2002; 56:208–220. [PubMed: 12271751]

Habak C, Wilkinson F, Wilson HR. Aging disrupts the neural transformations that link facial identity across views. Vision Res. 2008; 48:9–15. [PubMed: 18054981]

Hadjikhani N, de Gelder B. Neural basis of prosopagnosia: an FMRI study. Hum Brain Mapp. 2002; 16:176–182. [PubMed: 12112771]

Hadjikhani N, Joseph RM, Snyder J, Chabris CF, Clark J, Steele S, McGrath L, Vangel M, Aharon I, Feczko E, Harris G, Tager-Flusberg H. Activation of the fusiform gyrus when individuals with autism spectrum disorder view faces. NeuroImage. 2004; 22:1141–1150. [PubMed: 15219586]

Hahn S, Carlson C, Singer S, Gronlund SD. Aging and visual search: automatic and controlled attentional bias to threat faces. Acta Psychol. 2006; 123:312–336.

Horn JL, Cattell RB. Age differences in fluid and crystallized intelligence. Acta Psychol. 1967; 26:107–129.

Ishai A, Ungerleider LG, Martin A, Schouten JL, Haxby JV. Distributed representation of objects in the human ventral visual pathway. Proc Natl Acad Sci USA. 1999; 96:9379–9384. [PubMed: 10430951]

Issa EB, Papanastassiou AM, DiCarlo JJ. Large-scale, high-resolution neurophysiological maps underlying FMRI of macaque temporal lobe. J Neurosci. 2013; 33:15207–15219. [PubMed: 24048850]

Kanwisher N, McDermott J, Chun MM. The fusiform face area: a module in human extrastriate cortex specialized for face perception. J Neurosci. 1997; 17:4302–4311. [PubMed: 9151747]

Kenny DA, Judd CM. Power anomalies in testing mediation. Psychol Sci. 2014; 25:334–339. [PubMed: 24311476]

Kiani R, Esteky H, Mirpour K, Tanaka K. Object category structure in response patterns of neuronal population in monkey inferior temporal cortex. J Neurophysiol. 2007; 97:4296–4309. [PubMed: 17428910]

Kriegeskorte N, Mur M, Bandettini P. Representational similarity analysis - connecting the branches of systems neuroscience. Front Syst Neurosci. 2008; 2:4. [PubMed: 19104670]

Kuhn S, Brick TR, Muller BC, Gallinat J. Is this car looking at you? How anthropomorphism predicts fusiform face area activation when seeing cars. Plos One. 2014; 9:e113885. [PubMed: 25517511]

Li HJ, Hou XH, Liu HH, Yue CL, Lu GM, Zuo XN. Putting age-related task activation into large-scale brain networks: a meta-analysis of 114 fMRI studies on healthy aging. Neurosci Biobehav Rev. 2015; 57:156–174. [PubMed: 26318367]

Liu T, Wen W, Zhu W, Trollor J, Reppermund S, Crawford J, Jin JS, Luo S, Brodaty H, Sachdev P. The effects of age and sex on cortical sulci in the elderly. Neuroimage. 2010; 51:19–27. [PubMed: 20156569]

Marstaller L, Williams M, Rich A, Savage G, Burianova H. Aging and large-scale functional networks: white matter integrity, gray matter volume, and functional connectivity in the resting state. Neuroscience. 2015; 290:369–378. [PubMed: 25644420]

Mather M, Knight MR. Angry faces get noticed quickly: threat detection is not impaired among older adults. J Gerontol B Psychol Sci Soc Sci. 2006; 61:P54–P57. [PubMed: 16399942]

Meyers EM, Borzello M, Freiwald WA, Tsao D. Intelligent information loss: the coding of facial identity, head pose, and non-face information in the macaque face patch system. J Neurosci. 2015; 35:7069–7081. [PubMed: 25948258]

Mur M, Ruff DA, Bodurka J, De Weerd P, Bandettini PA, Kriegeskorte N. Categorical, yet graded–single-image activation profiles of human category-selective cortical regions. J Neurosci. 2012; 32:8649–8662. [PubMed: 22723705]

Murphy NA, Lehrfeld JM, Isaacowitz DM. Recognition of posed and spontaneous dynamic smiles in young and older adults. Psychol Aging. 2010; 25:811–821. [PubMed: 20718538]

Ng SY, Zebrowitz LA, Franklin RG Jr. Age differences in the differentiation of trait impressions from faces. J Gerontol B Psychol Sci Soc Sci. 2014

Orgeta V, Phillips LH. Effects of age and emotional intensity on the recognition of facial emotion. Exp Aging Res. 2008; 34:63–79. [PubMed: 18189168]

Park DC, Polk TA, Park R, Minear M, Savage A, Smith MR. Aging reduces neural specialization in ventral visual cortex. Proc Natl Acad Sci USA. 2004; 101:13091–13095. [PubMed: 15322270]

Park J, Carp J, Hebrank A, Park DC, Polk TA. Neural specificity predicts fluid processing ability in older adults. J Neurosci. 2010; 30:9253–9259. [PubMed: 20610760]

Park J, Carp J, Kennedy KM, Rodrigue KM, Bischof GN, Huang CM, Rieck JR, Polk TA, Park DC. Neural broadening or neural attenuation? Investigating age-related dedifferentiation in the face network in a large lifespan sample. J Neurosci. 2012; 32:2154–2158. [PubMed: 22323727]

Piper BJ, Li V, Eiwaz MA, Kobel YV, Benice TS, Chu AM, Olsen RHJ, et al. Executive function on the psychology experiment building language tests. Behav Res Methods. 2012; 44:110–123. [PubMed: 21534005]

Preacher KJ, Hayes AF. SPSS and SAS procedures for estimating indirect effects in simple mediation models. Behav Res Methods Instrum Comput. 2004; 36:717–731. [PubMed: 15641418]

Rossion B, Hanseeuw B, Dricot L. Defining face perception areas in the human brain: a large-scale factorial fMRI face localizer analysis. Brain Cognit. 2012; 79:138–157. [PubMed: 22330606]

Ruffman T, Henry JD, Livingstone V, Phillips LH. A meta-analytic review of emotion recognition and aging: implications for neuropsychological models of aging. Neurosci Biobehav Rev. 2008; 32:863–881. [PubMed: 18276008]

Ruffman T, Halberstadt J, Murray J. Recognition of facial, auditory, and bodily emotions in older adults. J Gerontol B Psychol Sci Soc Sci. 2009a; 64:696–703. [PubMed: 19805486]

Ruffman T, Ng M, Jenkin T. Older adults respond quickly to angry faces despite labeling difficulty. J Gerontol B Psychol Sci Soc Sci. 2009b; 64:171–179. [PubMed: 19202144]

Salat DH, Buckner RL, Snyder AZ, Greve DN, Desikan RS, Busa E, Morris JC, Dale AM, Fischl B. Thinning of the cerebral cortex in aging. Cereb Cortex. 2004; 14:721–730. [PubMed: 15054051]

Salthouse TA. Speed and knowledge as determinants of adult age differences in verbal tasks. J Gerontol. 1993; 48:29–36.

Selig, JP.; Preacher, KJ. Monte Carlo method for assessing mediation: An interactive tool for creating confidence intervals for indirect effects. 2008. ⟨http://quantpsy.org/⟩

Shipley, WC. Institute of living scale. Western Psychological Services; Los Angeles: 1946.

Slessor G, Miles LK, Bull R, Phillips LH. Age-related changes in detecting happiness: discriminating between enjoyment and nonenjoyment smiles. Psychol Aging. 2010; 25:246–250. [PubMed: 20230145]

Tsao D. Eppendorf 2006 winner. A dedicated system for processing faces. Science. 2006; 314:72–73. [PubMed: 17023644]

Vogels R. Categorization of complex visual images by rhesus monkeys. Part 2: single-cell study. Eur J Neurosci. 1999; 11:1239–1255. [PubMed: 10103119]

Voss MW, Erickson KI, Chaddock L, Prakash RS, Colcombe SJ, Morris KS, Doerksen S, Hu L, McAuley E, Kramer AF. Dedifferentiation in the visual cortex: an fMRI investigation of individual differences in older adults. Brain Res. 2008; 1244:121–131. [PubMed: 18848823]

Wilson HR, Mei M, Habak C, Wilkinson F. Visual bandwidths for face orientation increase during healthy aging. Vision Res. 2011; 51:160–164. [PubMed: 21074549]

Zarahn E, Rakitin B, Abela D, Flynn J, Stern Y. Age-related changes in brain activation during a delayed item recognition task. Neurobiol Aging. 2007; 28:784–798. [PubMed: 16621168]

Zebrowitz LA, Franklin RG, Hillman S, Boc H. Older and younger adults' first impressions from faces: similar in agreement but different in positivity. Psychol Aging. 2013; 28:202–212. [PubMed: 23276216]

Zhu Q, Zhang J, Luo YL, Dilks DD, Liu J. Resting-state neural activity across face-selective cortical regions is behaviorally relevant. J Neurosci. 2011; 31:10323–10330. [PubMed: 21753009]

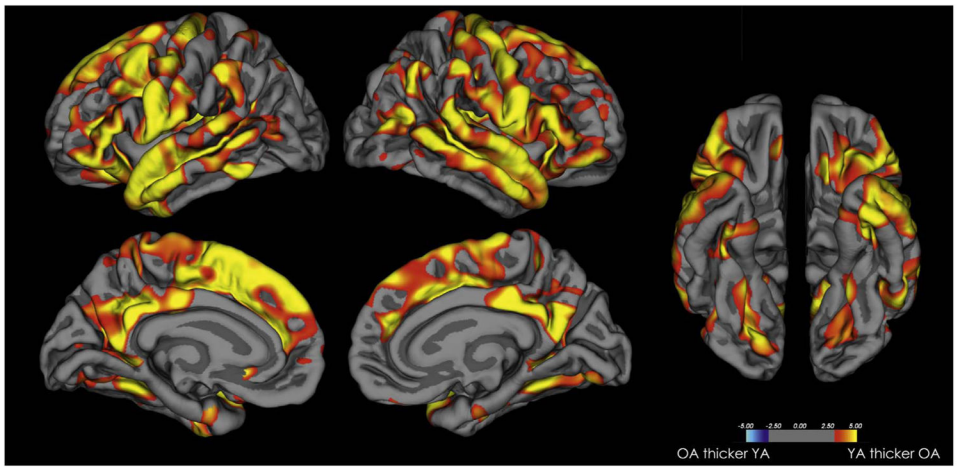

Fig. 1.

Mean thickness difference significance maps. Lateral, median and inferior views of the brain showing areas presenting cortical thinning in OA compared with YA. Gyri are represented in light gray, and sulci in dark gray. The medial wall is masked (white) reflecting the fact that it is not included in the cortical measures. Statistical maps are projected onto the folded FreeSurfer template brain. Areas that are significantly thinner in OA vs. YA are represented in red-to-yellow, and areas that are significantly thinner in YA in blue-to-cyan. Differences are shown at $p<0.01$. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

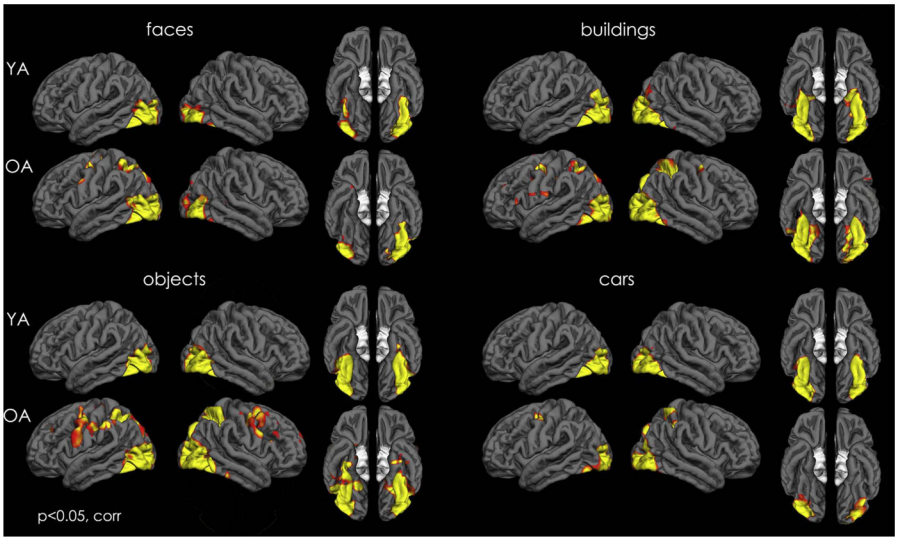

Fig. 2.

Mean statistical maps significance maps. Lateral, median and inferior views of the brain showing activation for faces vs. scrambled (top left), buildings vs. scrambled (top right), objects vs. scrambled (bottom left) and cars vs. scrambled (bottom right) in YA and in OA, projected on the pial surface of the FreeSurfer template brain. The medial wall is masked (white) reflecting the fact that surface data are not included in that region. Data are shown at $p<0.05$, corrected.

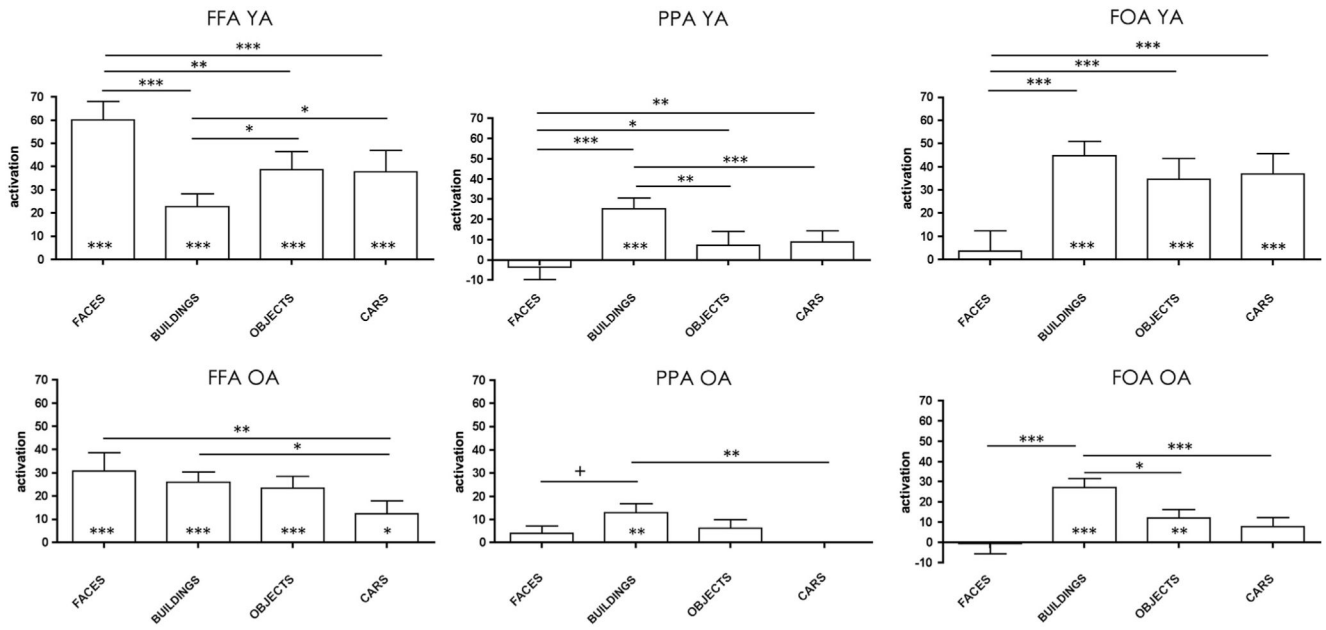

Fig. 3.

YA and OA BOLD activation to each stimulus category across the ROIs. The top three panels show activation for YA in the FFA (left panel), the PPA (middle panel) and the FOA (right panel). The bottom three panels show activation for OA in the FFA (left panel), the PPA (middle panel) and the FOA (right panel). The asterisks in the columns represent the significance of the activation for each of the categories, and the asterisks between the columns represent the significance of the difference of activation between categories. *p<0.05; **p<0.01; ***p<0.005.

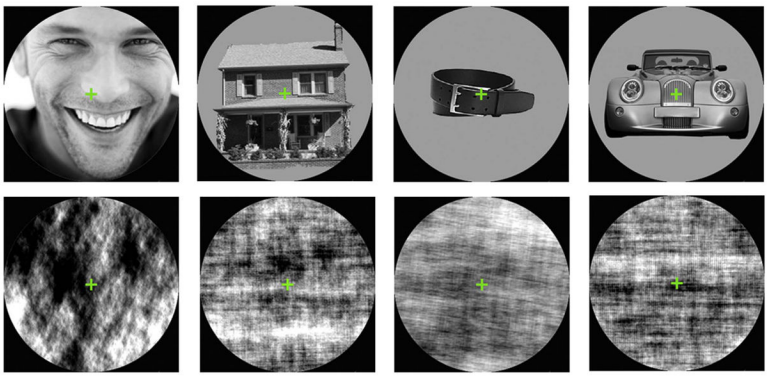

Fig. 4.

Example of the stimuli used for each category (face, building, object, car) on the top row, and their corresponding scrambled version (bottom row). Each stimulus was contained in a circle within a square, to limit retinotopic differences between categories, and a fixation cross was located in the center of each stimulus.

Table 1

Older and younger adults' scores on control measures.

Measure	Older adults		Younger adults		F-value	p-value
	M	SD	M	SD		
Snellen Visual Acuity (denominator)	27.50	9.89	14.68	5.32	24.46	<.001
Mars Letter Contrast Sensitivity (Mars Perceptrix, Chappaqua, NY)	1.60	.12	1.74	.03	20.90	<.001
Benton Facial Recognition Test (Benton et al., 1983)	45.67	5.75	47.68	2.91	1.84	.18
Timed Pattern Comparison Test (Salthouse, 1993)	29.56	7.59	43.26	8.97	25.04	<.001
Wisconsin Card Sorting Test (the Berg Card Sort Task (BCST validated by Piper et al., 2012)) http://pebl.sourceforge.net/battery.html	7.33	4.26	5.42	1.30	3.49	.07
Perseverative errors						
Non-Perseverative errors	10.44	9.20	4.21	1.75	8.42	.006
Shipley Vocabulary Test (Shipley, 1946)	36.11	2.87	34.16	2.79	4.99	<.001
Education	4.50	1.46	4.50	1.24	0	

Level of Education was coded for highest level attained: 1 – no high school diploma, 2 – high school diploma, 3 – some college, 4 – Bachelor's degree, 5 – some graduate work, 6 – Masters degree, 7 – Doctorate degree. Medians and range are reported.

Table 2

whole brain activation

YA faces:				
	Z	**x**	**y**	**z**
Cluster 1: 6158 voxels				
L Inferior occipital gyrus	7.06	−44	−76	−16
L Lateral occipital cortex	6.41	−40	−80	−8
L Temporal occipital fusiform	6.31	−42	−54	−24
L Fusiform gyrus	5.66	−38	−68	−18
Cluster 2: 6181 voxels				
R Lateral occipital cortex	6.34	42	−86	−8
R Inferior occipital gyrus	5.27	36	−90	−16
R Temporal occipital fusiform	5.63	42	−44	−26
R Fusiform gyrus	5.62	40	−44	−22
OA faces:				
	Z	x	y	z
Cluster 3: 13552 voxels				
R Lateral occipital cortex	5.68	50	−66	−2
R Inferior occipital gyrus	5.57	48	−78	−14
R Lateral occipital gyrus	5.11	44	−80	−2
R Fusiform gyrus	4.20	46	−50	−26
R Amygdala	4.29	26	−4	−20
Cluster 2: 13040 voxels				
L Fusiform gyrus	6.25	−34	−54	−24
L Lateral occipital cortex	6.15	−44	−72	10
L Inferior occipital gyrus	5.46	−40	−86	−20
L Inferior temporal gyrus	5.35	−46	−54	−26
L Temporal occipital fusiform	5.40	−36	−46	−24
L Fusiform gyrus	5.06	−38	−52	−24
Cluster 1: 2959 voxels				
L Precentral gyrus	4.54	−26	−12	62
YA>OA faces				
Nothing				
OA>YA faces				
	Z	x	y	z
Cluster 2: 3140 voxels				
L frontal pole	3.56	−30	58	18
L Dorsolateral prefrontal cortex	3.08	−32	28	32
L midcingulate	2.81	−8	22	28
L anterior cingulate	2.69	−6	34	8
L medial prefrontal cortex	2.66	−6	46	−6
Cluster 1: 1967 voxels				

YA faces:				
	Z	**x**	**y**	**z**
R superior parietal cortex	5.25	14	−84	40
R cuneus	2.74	4	−74	36
L superior parietal cortex	3.09	−10	−68	56
L cuneus	2.88	−2	−84	32
YA buildings				
	Z	x	y	z
Cluster 2: 10293 voxels				
R Inferior occipital cortex	5.81	46	−78	−18
R Lateral occipital cortex	5.45	32	−94	6
R Parahippocampal gyrus	4.07	26	−34	−18
Cluster 1: 9704 voxels				
L Lateral occipital cortex	6.42	−36	−84	6
L Occipital fusiform gyrus	6.38	−34	−80	−16
L Inferior occipital cortex	5.78	−48	−80	−10
L Parahippocampal gyrus	4.66	−32	−42	−12
OA buildings				
	Z	x	y	z
Cluster 1: 40171 voxels				
R Temporal occipital fusiform	5.78	24	−50	−18
L Temporal occipital fusiform	5.72	−26	−58	−16
R Inferior occipital cortex	5.48	26	−90	−24
L Occipital fusiform gyrus	5.39	−22	−86	−20
L Superior parietal cortex	4.51	−26	−62	56
R Superior parietal cortex	4.38	30	−62	58
R Midcingulate gyrus	4.19	6	6	48
L Precentral gyrus	4.01	−60	−2	32
R Parahippocampal gyrus	3.30	22	−34	−22
L R Parahippocampal gyrus	3.19	−22	−44	−18
YA>OA buildings				
Nothing				
OA>YA buildings				
	Z	x	y	z
Cluster 1: 1835 voxels				
L lingual gyrus	3.38	−10	−76	−14
R pericalcarine cortex	2.99	2	−92	12
L pericalcarine cortex	2.94	−14	−90	−4
YA Objects				
	Z	x	y	z
Cluster 2: 7945 voxels				
R Lateral occipital cortex	6.07	46	−66	−8
R Occipital fusiform gyrus	5.06	38	−68	−14

YA faces:				
	Z	**x**	**y**	**z**
R Temporal occipital fusiform	4.68	36	−42	−26
Cluster 1: 7277 voxels				
L Lateral occipital cortex	6.02	−42	−78	−8
L Temporal occipital fusiform	5.63	−36	−62	−16
OA Objects				
	Z	x	y	z
Cluster 1: 56061 voxels				
R Temporal occipital fusiform	6.29	−26	−52	−22
R Occipital fusiform gyrus	5.36	−34	−64	−20
L Lateral occipital cortex	5.06	50	−84	−18
R Superior parietal gyrus	4.97	24	−68	60
L Superior parietal gyrus	4.49	−40	−42	42
R inferior temporal gyrus	4.46	60	−36	−22
R Precentral gyrus	4.35	48	−2	58
R middle temporal gyrus	4.09	60	−60	−4
R inferior frontal gyrus	4.06	54	18	22
R Midcingulate gyrus	3.70	0	8	48
YA>OA objects				
Nothing				
OA>YA objects				
	Z	x	y	z
Cluster 1: 3451 voxels				
L pericalcarine cortex	3.51	−14	−98	12
R pericalcarine cortex	3.39	8	−96	0
YA Cars				
	Z	x	y	z
Cluster 2: 7059 voxels				
R inferior occipital cortex	6.36	36	−84	−22
R Occipital fusiform gyrus	6.25	38	−70	−20
R Temporal occipital fusiform	6.11	34	−66	−16
Cluster 1: 6591 voxels				
L Occipital fusiform gyrus	6.37	−34	−78	−16
L Lateral occipital cortex	5.99	−42	−80	−10
L Inferior occipital cortex	5.82	−40	−80	−14
L Temporal occipital fusiform	5.41	−38	−48	−24
OA Cars				
	Z	x	y	z
Cluster 3: 9828 voxels				
R Superior lateral occipital	5.17	40	−74	18
R Superior parietal lobule	5.03	26	−52	58
R Inferior lateral occipital	4.80	50	−82	−12

YA faces:				
	Z	**x**	**y**	**z**
R Inferior occipital gyrus	4.64	48	−88	−12
Cluster 2: 6169 voxels				
L Superior lateral occipital	4.71	−28	−94	28
L Inferior lateral occipital	3.65	−32	−94	−10
L Occipital pole	3.61	−32	−96	−12
Cluster 1: 2036 voxels				
L Precentral gyrus	4.24	−48	−10	52
R/L Supplementary motor area	3.71	0	2	54
L Midcingulate gyrus	3.56	−10	−2	40
YA>OA cars				
	Z	x	y	z
Cluster 1: 1928 voxels				
R inferior occipital cortex	4.46	44	−72	−14
R Temporal occipital fusiform	3.58	44	−48	−20
OA>YA cars				
nothing				