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Acute Stress Enhances Emotional Face Processing in the Aging Brain

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ABSTRACT

BACKGROUND: Healthy aging has been associated with stable emotional well-being and attenuated brain responses to negative stimuli. At the same time, depressive symptoms are common in older adults. The neural mechanisms behind this paradox remain to be clarified. We hypothesized that acute stress could alter emotion processing in healthy aging brain and constitute a pathway to vulnerability.

METHODS: Using a randomized, controlled crossover design, we explored the influence of acute stress on brain responses to happy and fearful facial expressions in 25 older adults (60–75 years of age) and 25 young (18–30 years of age) control subjects. Groups were matched on trait anxiety and education. Subjects underwent two separate functional magnetic resonance imaging sessions involving acute stress or a control procedure.

RESULTS: Affective and physiological responses to the stressor were similar between the two age groups. On a whole-brain level, we revealed a significant age by stress interaction in the fusiform gyrus, indicating a selective enhancement of neural activity with stress in elderly subjects only. When specifically aiming analysis at the amygdala, we found the same stress-related increase in activity in elderly subjects only. Modulation of amygdala reactivity due to stress correlated with trait conscientiousness in elderly subjects exclusively.

CONCLUSIONS: Compared with younger adults, healthy older adults showed increased responsivity of brain regions involved in face and emotion processing while stressed. These findings suggest that increased reactivity of this neural circuitry after acute stress may constitute one mechanism by which emotional well-being during healthy aging could rapidly change into heightened vulnerability for affective disorders.

Keywords: Aging, Amygdala, Conscientiousness, fMRI, Fusiform gyrus, Stress

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Life expectancy is increasing worldwide. By 2050, the number of individuals older than 60 years is expected to have doubled (1). Unfortunately, these additional years are not always spent in good health. In fact, there is evidence of raising rates of chronic disease and disability in elderly adults (2). Mental health problems such as dementia and depression are among the most significant contributors of disability among elderly adults (3). While most research supports a general age-related decline with respect to cognition, emotional aging is thought to be more complex. In fact, in contrast to the high burden of depression in old age, there is also evidence that healthy aging can give rise to protective psychological effects, reflecting a paradox in emotional aging (4). Compared with healthy young adults, healthy older adults are better at focusing on positive stimuli, more efficient in regulating their emotions, and more biased toward positive memories [reviewed in (4,5)].

In addition, while age-related changes in learning and memory have been extensively studied using state-of-the-art neuroimaging techniques (6,7), relatively few studies have investigated the neural correlates of emotional aging. Initial neuroimaging studies have generally confirmed the behavioral findings of an increase in positive emotions in healthy aging and suggest a change in neural processing underlying emotions (8–11). For example, attenuated amygdala responses to negatively valenced pictures have been found in older adults compared with younger adults (12,13). Correspondingly, increased activity in anterior cingulate and prefrontal regions has been found when processing emotional stimuli, suggesting enhanced cognitive control (13–16).

One critical factor accounting for this paradox of resilience and vulnerability in aging could be the influence of acute stress. Age-related cellular, cerebral, and behavioral changes resemble changes found in chronically stressed individuals, and acute stress in the aging brain could be "adding fuel to the fire" (17). Possibly, acute stress could make healthy elderly adults more at risk to develop symptoms of affective disorders. Thus far, few studies have focused on possible age differences in effects of acute stress or negative mood induction in the laboratory. Some studies find better emotion regulation in older adults after acute stress, whereas other studies find a decline of positive emotions or no age differences at all

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(18–21). Effects on physiological stress parameters are also unclear (22). Unfortunately, in these studies, the role of potential confounders, such as comorbidity and use of medication, are mostly not taken into account. Moreover, we and others have shown that neural stress reactivity may be influenced by personality (23–25). Personality is generally investigated using a set of personality traits based on, for example, the five-factor model, including neuroticism, extraversion, openness to experience, agreeableness, and conscientiousness (26). Importantly, these traits change across the life span: while openness and extraversion decline with age, conscientiousness increases during adulthood, and neuroticism stays relatively stable (27). Thus, personality should be considered, as age-related changes in personality traits may influence potential age-related differences in the stress response. Lastly, to our knowledge, no prior study has investigated age differences in neural activity during experimental stress induction procedures.

In the present study, we therefore aimed to unravel the influence of acute stress on neural emotion processing in healthy aging, using a well-established experimental stress induction procedure. We hypothesized that, in line with previous research demonstrating emotional resilience with increasing age, healthy older adults would show attenuated responsivity of brain regions involved in emotion processing, in particular the amygdala, compared with healthy young adults under standard conditions. However, we anticipated that this age-related difference would become smaller or even disappear after the administration of acute psychological stress, reflecting the paradox with enhanced vulnerability in healthy aging. As we previously found that individual differences can also influence the neural stress response within this paradigm (23), we additionally explored the impact of personality traits.

METHODS AND MATERIALS

Participants

We included 25 young (18–35 years of age) and 25 old (60–75 years of age) healthy men (Table 1). Young adults were individually selected from an existing (N = 120) database (23). We carefully matched these younger subjects to the older adults based on similar trait anxiety scores and educational levels, as we estimated these factors to be potential confounders of age-related differences in neural activity (28,29).

Procedure

All participants took part in a two-session study with a randomized, counterbalanced order of the session type (stress or control) (see Supplemental Figure S1). We have described this procedure in detail elsewhere (23,30), and it was extensively standardized in order to create a highly similar experimental setting for all participants. Details of the data acquisition and processing procedures can be found in the Supplement.

Sessions were separated by on average 13 days (minimum of 5 days). All testing took place between noon and 6 PM with the aim of profiting from more stable hormone levels to limit the influence of the diurnal rhythm on our hormone assessments. In short, during 1 hour of prescanning preparation, participants received information about the study, practiced the tasks they would later have to perform in the scanner, and watched a relaxing nature documentary (31). Next, during the stress session, a state of acute stress was induced by showing highly aversive movie clips in the magnetic resonance imaging (MRI) scanner (32–34). These clips consisted of scenes from a movie (35) containing extremely aggressive behavior and violence against men and women. During a separate control session, neutral, nonarousing scenes from another movie (36) were shown. The stressful and the neutral movie clips both had a

Table 1. Demographic Characteristics of the Study Population

	Younger Adults (n = 25)	Older Adults (n = 25)	p Value[a]
Age, Years, Mean (SD) [Range]	21.5 (2.5) [18–30]	66.7 (4.3) [60–75]	< .001
Education, n (%)			NS
Primary school	0	1 (4)	
Lower secondary	3 (12)	2 (8)	
Intermediate secondary/college degree	9 (36)	6 (24)	
Higher secondary/university degree	13 (52)	16 (64)	
Trait Anxiety Score,[b] Mean (SD)	32.8 (6.3)	32.6 (8.8)	NS
NEO-FFI Scores,[b] Mean (SD) [Range]			
Altruism	42.0 (3.4) [37–48]	44.2 (3.9) [36–52]	.037
Conscientiousness	41.1 (5.5) [29–50]	44.7 (4.7) [35–53]	.018
Extraversion	44.8 (6.5) [30–53]	39.0 (5.2) [30–50]	.001
Neuroticism	26.4 (7.0) [14–40]	25.3 (6.7) [14–39]	NS
Openness	39.4 (6.8) [28–52]	37.2 (6.1) [28–51]	NS
Baseline Cortisol, nmol/L, Mean (SD)	12.3 (6.5)	13.2 (5.3)	NS
Total Brain Volume, mL (SD)	1373.9 (84.1)	1266.6 (88.0)	< .001
Amygdala Volume, mL (SD)	2.7 (0.5)	3.0 (0.4)	.064
Amygdala Volume as Percentage of Total Brain Volume, % (SD)	0.20 (0.03)	0.23 (0.04)	< .001

NEO-FFI, NEO Five-Factor Inventory; NS, not significant.
[a]All p values < .1 are reported.
[b]All scores are in the normal range for a healthy male population (62,63).

duration of 10 minutes and were similar in the amount of speech, human (face) presence, luminance, environment, and language. The participants were asked to watch the movie clips from an eyewitness perspective.

Immediately after the first movie clips, subjects performed the dynamic facial expression task, which consisted of passive viewing of photographs of emotionally neutral faces that morphed into two different emotion types: fearful or happy facial expressions (37). The morphing faces were presented in a block design (three blocks of each emotion, 25 seconds per block, 0.5 second per face, avoiding adjacent blocks of the same emotion), interleaved with blocks of fixation cross for baseline reference purposes (three blocks, 25 seconds per block). Reaction times were measured to evaluate general attention, expressed as mean time to respond to the fixation cross. After this task, the subjects participated in other studies with different questions at issue, of which the results will be reported elsewhere. A structural scan was obtained at the end of the stress session. The duration of this multistudy scanning was approximately 105 minutes per session. In between sessions, participants completed several self-report questionnaires, containing the Dutch versions of the Trait-State Anxiety Inventory (38) and the NEO Five-Factor Inventory (26).

Imaging Data Analysis

We used a factorial analysis of variance as implemented in SPM8 with stress condition and emotion type as within-subject factors and age group as between-subjects factor. This model resulted in statistical parametric maps that were superimposed on the mean anatomical image across all subjects for localization purposes. Our statistical threshold for these voxelwise analyses was set at $p < .05$ familywise error (FWE) corrected for multiple comparisons with Gaussian random field theory as implemented in SPM8.

As we were interested a priori in differences in amygdala responses between the two groups, we also performed small volume corrected analyses (threshold $p < .05$ FWE corrected) using a standard anatomical atlas for the bilateral amygdala (39). Based on previous literature reporting confounding influences of local brain atrophy on functional MRI analyses in healthy aging, we additionally made use of individual masks of the amygdala to extract β values from the individual parameter estimate maps (40). To this end, we created individual masks by means of an automatic segmentation using the FIRST module of FSL [First version 1.2 (www.fmrib.ox.ac.uk/fsl/first/index.html) (41) in FSL version 4.1.9 (www.fmrib.ox.ac.uk/fsl), developed by the Analysis Group, FMRIB, Oxford, UK]. This method is based on Bayesian statistical models of shape and appearance for bilateral amygdala from 317 manually labeled T1-weighted magnetic resonance images. Visual inspection of the segmented amygdala masks projected onto the T1-weighted MRI scans was performed using the software MRIcron Version Beta 7 (www.mricro.com/mricron). After automatic segmentation, volumes were calculated using the MarsBaR SPM toolbox version 0.42 (http://marsbar.sourceforge.net).

Other Data Analysis

All other data (baseline variables, questionnaire scores, heart rate, heart rate variability, blood pressure, cortisol levels, and Positive and Negative Affect Scale scores) were analyzed in SPSS 19 (IBM Corp., Armonk, NY). Stress × time (before and after stress induction) repeated measures analyses of variance with group as a between-subjects factor were used to evaluate stress responses in behavioral and physiological measures. The heart rate was calculated as 60/mean interbeat interval, and heart rate variability was calculated as the root mean squares of successive differences between successive interbeat intervals. Offline artifact correction and analysis of the heart rate frequency and variability were done with in-house software.

For correlation analyses, parametric tests were used as default (Pearson correlations). Nonparametric tests were used (Spearman correlations) only for correlations with reaction times, response accuracy, heart rate, and heart rate variability, as these variables were not normally distributed. All reported analyses were performed with all subjects for whom data were available. Removing possible outliers (z scores >2.5) significantly changed our results for heart rate (variability) and extracted amygdala β values only. Results for these analyses were reported with outliers excluded. Significance level was set at $p < .05$. A linear regression analysis with one model containing all NEO Five-Factor Inventory subscales as predictor of stress-related changes in amygdala responses was performed to explore whether there were any personality-related moderators that significantly predicted amygdala responses while taking the other subscales into account. Subsequently, separate correlation analyses for each predictor were performed to verify associations. Bonferroni corrections were applied for these correlational analyses (42).

RESULTS

Study Population

One subject was excluded during the screening procedure because of previously undetected hypertension and cardiac arrhythmia. A second subject decided not to continue his participation during the first scanning session. Both subjects were older adults and were replaced to maintain a sample size of 25 subjects per group.

Although we matched our two age groups on trait anxiety levels, their personality scores were still different on some subscales, consistent with current literature suggesting common personality changes with increasing age (Table 1) (43). In addition, amygdala volumes were relatively larger in older subjects compared with young subjects. Reaction times to the fixation cross presented in between the blocks did not significantly differ between the two groups (age group × stress: $F_{1,45} = 0.7$, $p = .409$; main effect of age: $F_{1,1} = 0.2$, $p = .688$; mean reaction times across sessions in young adults = 667.4 ms, mean reaction times across sessions in older adults = 702.0 ms), suggesting similar levels of attention between the two age groups.

Stress Induction

A similar state of mild, acute stress was induced in both age groups, confirmed by significant changes in heart rate and negative affect ratings (see Supplement for details). Cortisol levels (as a proportion of mean cortisol levels measured at rest

at home) showed only a main effect of time ($F_{1,48}$ = 23.9, $p < .001$; baseline = 1.0, after task = 0.8). There were no interactions, suggesting that diurnal fluctuations in cortisol levels were stronger than the influence of our mild stressor. Mean blood pressure levels, heart rate variability, and subjective positive affect ratings differed significantly between the two age groups, but our stressor did not significantly influence these preexisting differences during the task.

Functional MRI

Main Effects. The viewing of faces, independent of emotion type or session, activated the expected network of brain regions across both groups, extending from the superior occipital gyrus to the fusiform gyrus, precentral gyrus, and medial temporal lobe, including the amygdala. Deactivations compared with baseline were observed in the supramarginal gyrus and middle occipital gyrus (all $p_{FWE} < .05$) (Supplemental Table S1). Group differences in task activations were found in the occipital cortex and fusiform gyrus, where younger subjects showed higher activation levels than older adults (all $p_{FWE} < .05$) (Supplemental Table S1). There were no regions that elicited more activation in older adults than younger adults. Across both age groups, there were no brain regions that showed more activation in the stress session than in the control session. In addition, there were no brain regions with more responsivity in the control than in the stress session.

Interaction Effects of Age and Stress. A three-way interaction between stress, emotional valence, and age did not reach significance. However, we observed a significant age × stress interaction in the lingual gyrus extending into the fusiform gyrus (Table 2 and Figure 1). In this region, older adults showed a stress-related increase in activity compared with younger adults.

We explored this interaction by investigating stress effects per age group. We found a significant increase of activity in the stress condition compared with the control condition in the older adults in a more anterior region reaching into the parahippocampal gyrus (peak Montreal Neurological Institute coordinates −32 −9 −30, p = .036 cluster level corrected). When applying a small volume correction for the interaction effect in older adults, we indeed found a stress-related increase of activity in the lingual gyrus and fusiform gyrus (Table 2). In younger adults, we found a significant effect only in the opposite contrast (stress < control) in the postcentral gyrus (peak Montreal Neurological Institute coordinates 63 −2 32, p = .030 cluster level corrected). When applying a small volume correction for the interaction effect in the young group, we did not find any change of activity in this region (Table 2).

Using voxelwise analysis, we did not find an effect in the amygdala, our initial region of interest. Therefore, we used the targeted analysis of extracting β values from the individually defined anatomical amygdala of each single subject. This technique enabled us to directly compare the change of activity in the two sessions in the young and old adults separately, with an even greater certainty that normalization differences do not affect our result. Moreover, bilateral amygdala volume did not correlate with amygdala blood oxygen level–dependent responses both within and across the two groups, indicating that the group difference in amygdala volume in our study population would not drive any group effects in amygdala responsivity. Although we had a clear a priori hypothesis of stress-related increase of amygdala responsivity in the older subjects, we still tested potential interaction effects for completeness. There were no significant interactions between age group, session, or valence (age group × stress × valence: $F_{1,45}$ = 0.2, p = .643; age group × stress: $F_{1,45}$ = 1.2, p = .274; age group × valence: $F_{1,45}$ = 0.5, p = .502; stress × valence: $F_{1,45}$ = 0.3, p = .595). However, amygdala responsivity across both sessions was lower in older adults than in younger adults ($F_{1,45}$ = 4.4, p = .041; young adults = 0.2, old adults = 0.1). Similar effects were found when adding amygdala volume as covariate, again indicating these effects were not driven by volumetric changes. Given our a priori hypothesis of stress as an enhancing factor for amygdala reactivity in older adults, we further explored possible group differences in amygdala responsivity to the different stimuli. Interestingly, we found a

Table 2. Interactions of Age and Stress

Effect	BA	Region	Hemisphere	Cluster Size	Peak MNI Coordinates			Peak *F*/*t* Value
					x	y	z	
Positive Interaction Age × Stress (Old [Stress > Control] > Young [Stress > Control])	19	Lingual gyrus/ fusiform gyrus	Right	825	32	−65	0	4.53[a]
					24	−60	−4	
					22	−39	0	
Negative Interaction Age × Stress (Old [Stress > Control] < Young [Stress > Control])		None	—	—	—	—	—	—
Stress Effect in Young[b] (Stress < Control)		None	—	—	—	—	—	—
Stress Effect in Old[b] (Stress > Control)	19, 36, 37	Fusiform gyrus	Left	104	24	−44	0	—
				97	30	−65	2	
					34	−63	−1	
				10	27	−54	−9	
				16	33	−39	−6	

BA, Brodmann area; MNI, Montreal Neurological Institute.

[a]$p < .01$ cluster level corrected with an initial whole-brain voxelwise threshold of $p < .001$ uncorrected for multiple comparisons. Small volume corrections for the bilateral anatomical amygdala (39) did not yield any additional clusters.

[b]Effects are masked for the interaction effect; therefore, no statistics are performed.

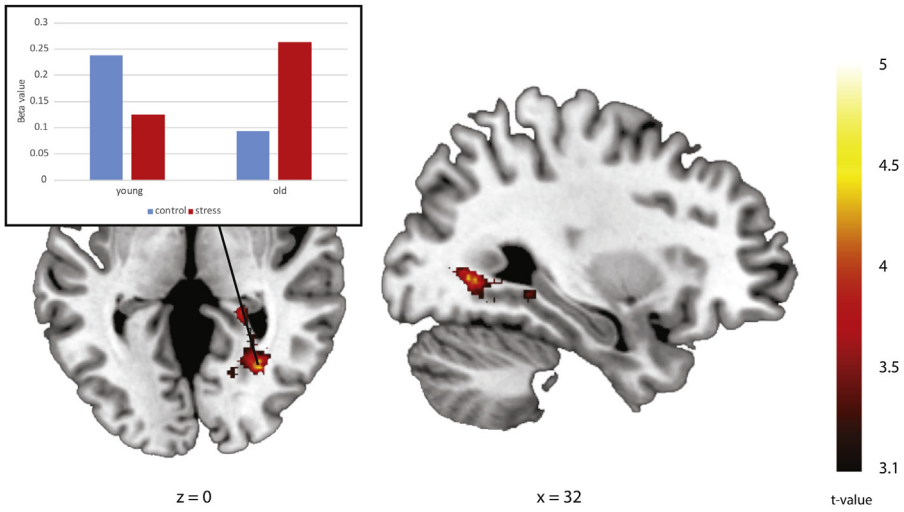

Figure 1. Positive interaction between age and stress in the lingual gyrus extending into the fusiform gyrus, representing stress-related enhancement of activity in the old subjects only. The image is thresholded at $p_{uncorrected} < .001$ and masked for the cluster level significant effect. The bar graph depicts mean beta values within the significant cluster for the age groups and sessions.

significant increase of amygdala responsivity under stress in the older adults but not in the young adults (Figure 2). We additionally explored valence-specific effects and found that this difference was mainly driven by a stress-induced increase in the response to fearful faces (Supplemental Figure S2). Because of the absence of a clear hypothesis for this valence-specific finding and the nonsignificant interactions, this finding should be interpreted with caution.

Individual Differences in Stress Responses

Finally, we were interested in age-specific differences in personality traits that could be associated with changes in amygdala responsivity under stress. Interestingly, we found that of the five subscales in the model, only conscientiousness levels predicted a stress-related difference in amygdala responsivity in older adults ($b = -.541$, $t_{16} = -2.207$, $p = .042$).

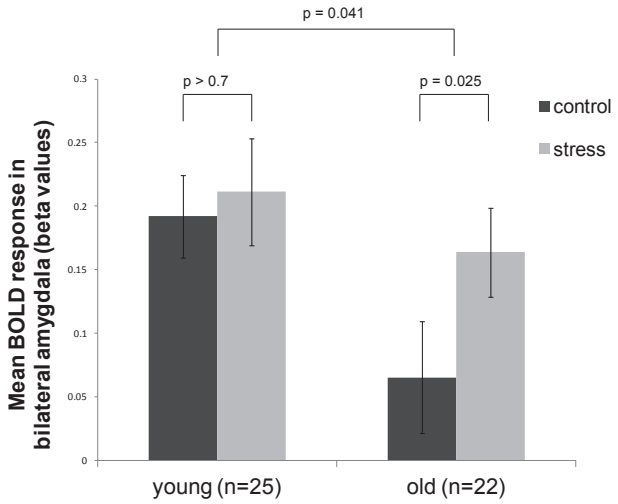

Figure 2. Mean amygdala responsivity in the two sessions per age group. Amygdala beta values were extracted from the anatomical bilateral amygdala, using individual masks for every subject. BOLD, blood oxygen level–dependent.

There were no significant predictors of amygdala responsivity in young adults. We verified this result by correlating the five different NEO Five-Factor Inventory subscale scores to the difference in amygdala responsivity between the stress session and control session. A negative correlation between conscientiousness and the impact of stress on amygdala responsivity in older adults confirmed our finding ($r_{20} = -.534$, $p = .007$) but not in young adults ($r_{23} = -.042$, $p = .840$) (Figure 3, Supplemental Results, and Supplemental Table S2). After applying Bonferroni correction for the five subscales (significance level $p = .05/5 = .01$), this result remains significant. The difference between these correlations showed a trend toward significance, indicating a more negative association in older adults ($z = 1.81$, $p = .07$).

DISCUSSION

To the best of our knowledge, this is the first study that investigated whether older age is associated with changed neural reactivity to acute stress. First, we found that overall physiological stress reactions were similar across both age groups, suggesting that our type of stressor had a similar impact across subjects of different ages. Second, we replicated earlier findings that positive affect was generally higher in healthy older adults than in younger adults. Interestingly, higher positive affect in our older subjects was accompanied by an attenuated amygdala response to emotional facial stimuli. Fundamentally, we observed that acute stress selectively enhanced neural activity in visual processing regions as well as the amygdala in older adults, bringing their neural responses to the same level as young adults. Although our study design does not permit statements about causality, this shift in neural activity in the amygdala appears to be influenced by the personality trait of conscientiousness in the older adults only.

These findings support previous research on age-related changes in brain activity underlying the positivity bias in healthy aging, in the sense that under standard conditions the healthy aging brain seems less susceptible to emotionally salient information than the healthy younger brain (9,11,14,44). However, notably under acute psychological stress, we found

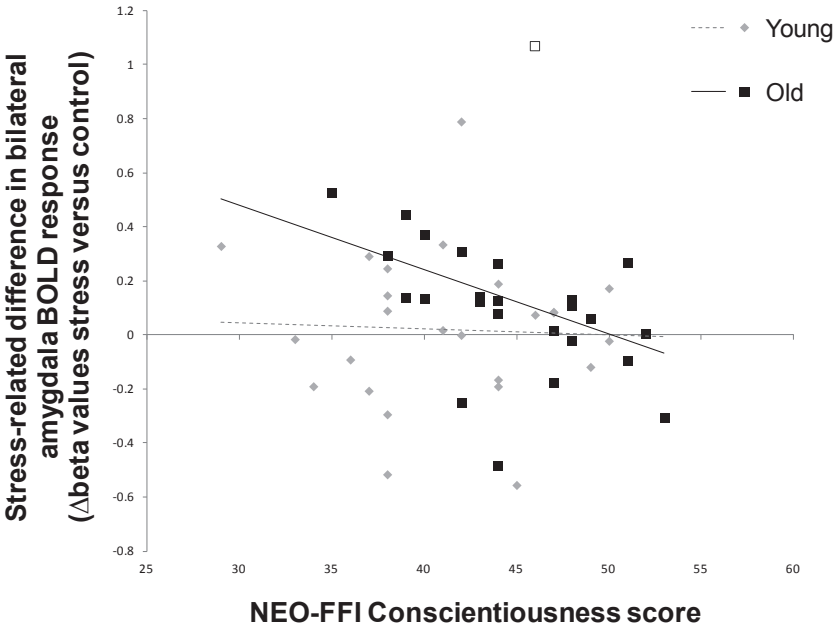

Figure 3. Conscientiousness was negatively correlated (p = .007) with the stress-related change in amygdala responsivity in older adults only. The open square represents an outlier (z value = 3.4) that was excluded from the statistical analysis. BOLD, blood oxygen level–dependent; NEO-FFI, NEO Five-Factor Inventory.

that even the healthy aging brain becomes significantly more reactive to emotional facial input. This is a novel finding, confirming the significant impact of stress in the already challenged aging brain (17). Interestingly, aging is normally accompanied by an age-related reduction in occipital activity coupled with increased frontal activity; this is known as the posterior-anterior shift in aging (PASA) (45). The PASA phenomenon is thought to reflect sensory decline in aging accompanied by prefrontal compensation and is also found for emotional stimuli (46). Although we did not find an increase of frontal activation, our results suggest that the PASA phenomenon could be influenced by acute stress. More research is needed to better understand PASA changes under stress and in particular role the amygdala has in this phenomenon.

With our paradigm, we induced only mild physiological stress, and we did not find significant stress effects in all physiological parameters. As our stress induction paradigm has been previously used in a larger sample size where it led to more significant stress effects, we believe that this is most likely due to our relatively small sample size in combination with the moderate nature of the stressor (23,30). Interestingly, bodily stress responses in our study were very similar between younger and older adults. The few studies investigating the consequences of acute psychological stress in older adults show contradicting results for cortisol responses, heart rate, and blood pressure reactivity (22,47). Of note, sex differences could potentially influence these findings. For example, the Trier Social Stress Test was found to elicit blunted heart rate responses in older versus younger adults, but the effect was largely driven by women (48). In addition, not all studies investigating autonomic reactivity after experimental stress take the potential influence of comorbidity and medication use into account (22). Lastly, different types of experimental stressors could have a dissimilar impact on young and old adults (22). By making use of emotional movie clips, we aimed to limit these potential age-related influences (49), but the present results should be replicated using other ecologically valid stress situations. For example, contextual features of testing environments have been found to differentially influence cortisol responses in young versus old adults (50). Also, age differences in violent movie or video game exposure could influence differences in stress responses after violent movie clips. Further research is needed, particularly with respect to sex differences and influence of different types of stressors, to gain better understanding of the role of physiological stress responses in age-related differences in emotion processing.

Importantly, previous studies have suggested that the positivity bias is due to a selective decrease in the neural processing of negative information with age (4). In our study, however, we did not find an interaction with stimulus type, meaning that for young as well as for older subjects, there were no differences in brain activity in response to happy versus fearful facial stimuli. Given that a similar task in young, healthy female subjects did elicit valence-specific responsivity of the amygdala, sex differences could be one reason why we did not find this effect in our male population (34). In addition, older adults have been found to have impaired recognition of basic emotions in facial expressions compared with younger adults (51). Our dynamic facial expression task could have been too challenging for older adults to successfully distinguish different types of salient facial expressions.

Furthermore, we did not observe a direct effect of acute stress on amygdala responsivity in young adults. This finding is in line with previous findings from our larger sample of 120 young adults, where we found no stress effect on amygdala responsivity in the study population as a whole but concluded that individual differences moderated amygdala responsivity to emotional faces under stress (23,30). In the present study, we found that in older adults only, trait conscientiousness modulated the amygdala response to stress. This finding is of particular relevance for elderly adults, as high levels of

conscientiousness have repeatedly been associated with attenuated cognitive decline in aging (52–54). Importantly, trait conscientiousness also seems to interact with environmental stress when conveying a risk for affective symptoms. Low levels of conscientiousness were related to higher anxiety levels in combination with stressful life events in a sample of older adults with late-life depression (55). Our finding may thus constitute a neural mechanism for the resilience associated with conscientiousness in older age.

The main strength of our study is its novelty in using a well-investigated functional MRI stress induction procedure to demonstrate stress-related changes in neural emotion processing in older adults, which to our knowledge is the first to do so. An important limitation of our study is the inclusion of only men. The aging brain shows sex-specific changes in stress responsivity, indicating that generalization to women should be done with caution (56). Furthermore, the cross-sectional nature of our study cannot exclude possible cohort effects. For example, age-related changes in frontal activity have shown opposite directions when using different study designs: while cross-sectional analyses were suggestive of age-related frontal overrecruitment, the longitudinal analyses revealed frontal underrecruitment with advancing age (57). As group differences in educational attainment are an important confounding factor in cross-sectional studies investigating aging (28) and differences in anxiety levels could bias our results as well, we aimed to limit these influences by matching our groups on educational level and trait anxiety. Finally, a larger sample with a more continuous age range would have provided additional information on the role of neural development and is recommended for future studies investigating neural stress sensitivity in aging.

Increased understanding of acute stress responses in healthy aging could help identify elderly adults at risk for mood and anxiety disorders when confronted with life stressors, as has recently been demonstrated in young adults (58). Moreover, individual differences in amygdala (re)activity may predict treatment responses in depression, highlighting the importance of gaining insight in the age-related differences in the dynamics of emotional aging (59). Interestingly, in older adults, neural responses to angry faces have previously been found to be associated with suicidal behavior (60). This is highly relevant, as suicide rates are traditionally highest in older men compared with other groups of society (61).

In conclusion, in this study we found evidence for attenuated emotional facial processing in the healthy aging brain, which seemed reversed during acute stress induction and dependent on conscientiousness levels in the older adults. Understanding how our work can be translated to more vulnerable individuals should be an important goal of future research.

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