Archival Report

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 (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 ($n = 25$)	<i>p</i> 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.

^aAll *p* values < .1 are reported.

^bAll scores are in the normal range for a healthy male population (62,63).

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