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Regular Article

Adolescents' neural reactivity to acute psychosocial stress: dysfunctional regulation habits are linked to temporal gyrus response

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Abstract

Mid-adolescence is a critical time for the development of stress-related disorders and it is associated with significant social vulnerability. However, little is known about normative neural processes accompanying psychosocial stress at this time. Previous research found that emotion regulation strategies critically influence the relationship between stress and the development of psychiatric symptoms during adolescence. Using functional magnetic resonance imaging (fMRI), we examined neural responses to acute stress and analyzed whether the tendency to use adaptive or maladaptive emotion regulation strategies is related to neural and autonomic stress responses. Results show large linear activation increases from low to medium to high stress levels mainly in medial prefrontal, insulae and temporal areas. Caudate and subgenual anterior cingulate cortex, neural areas related to reward and affective valuations, showed linearly decreasing activation. In line with our hypothesis, the current adolescent neural stress profile resembled social rejection and was characterized by pronounced activation in insula, angular and temporal cortices. Moreover, results point to an intriguing role of the anterior temporal gyrus. Stress-related activity in the anterior temporal gyrus was positively related to maladaptive regulation strategies and stress-induced autonomic activity. Maladaptive coping might increase the social threat and reappraisal load of a stressor, relating to higher stress sensitivity of anterior temporal cortices.

Keywords: emotion regulation strategies, fMRI, mid-adolescence, Montreal imaging stress task (MIST), psychosocial stress

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Introduction

In adolescence, onsets of stress-associated disorders such as depression or substance use disorders peak (Kessler & Wang, 2008; Paus, Keshavan, & Giedd, 2008). In this context, restructuring of important functional brain networks such as the prefrontal–amygdala circuitry (Casey et al., 2010; Tottenham & Galván, 2016), rapid gray matter changes (Giedd, 2004; Gogtay et al., 2004) and imbalances in neuroendocrine axes and resulting hormonal output (Romeo, 2010; Romeo & McEwen, 2007) are believed to create a window of neural stress vulnerability.

In addition, the transition into adulthood brings about significant environmental challenges. Adolescents begin exploring new social groups and networks outside their familiar home (Nelson, Leibenluft, McClure, & Pine, 2005). At school, they are confronted with increasing academic pressure (Kouzma & Kennedy, 2004). Simultaneously, high emotional reactivity (Casey et al., 2010; Dahl & Gunnar, 2009) and hypersensitivity to social acceptance and rejection (Platt, Kadosh, & Lau, 2013; Sebastian, Viding, Williams, & Blakemore, 2010) further raise the potency of (psychosocial) stressors on the adolescent nervous system.

Consequently, the adolescent brain is becoming strikingly sensitive to stress-induced alterations (Gee & Casey, 2015). Acute stress negatively affects working memory and performance (Gärtner, Rohde-Liebenau, Grimm, & Bajbouj, 2014). In the long run, severe or chronic stress-related alterations during adolescence show close links to the emergence of depressive and anxiety disorders in adulthood (Dahl & Gunnar, 2009). As such, stress-related processes in the adolescent brain are of pivotal relevance for an individual's mental health. In general, the literature on stress-related functional brain changes as investigated in adults suggests neural resources shift towards areas involved in salience detection (e.g., anterior insula) and affective processing (e.g., medial prefrontal cortex [mPFC]), while modulating neural top-down regulation (e.g., ventrolateral PFC) in a specific and time-dependent manner (for a review see van Oort et al., 2017).

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Stress, moreover, changes activation and processing of stimuli in reward-related areas such as the striatum and orbitofrontal cortex (OFC; Porcelli, Lewis, & Delgado, 2012). For example, Porcelli et al. (2012) exposed participants to acute stress prior to examining neural responses to monetary rewards and punishments. They could show that participants exposed to acute stress demonstrated decreased dorsal striatum and OFC reward sensitivity. Behaviorally, acute stress appears to increase attention towards threat-related cues and heighten the salience of negative emotional material (Hermans, Henckens, Joëls, & Fernández, 2014; Lupien, Maheu, Tu, Fiocco, & Schramek, 2007; Oei et al., 2012). However, neural correlates of acute stress vary with the type of stress induced, as well as with the paradigm employed (for a review, see Noack, Nolte, Nieratschker, Habel, & Derntl, 2019). Reviews summarizing functional brain changes associated with acute psychosocial stress induced by the Montreal imaging stress task (MIST; Dedovic, D'Aguiar, & Pruessner, 2009; Pruessner et al., 2010) reported increased activation in the left medial PFC, cingulum, occipital cortex and left premotor area and deactivation of the limbic system, particularly in the medio-orbitofrontal cortex, hippocampus, and hypothalamus. Meta-analytic data moreover show that psychosocial stress in adults is associated with heightened activation in the right superior temporal gyrus as well as the inferior frontal gyrus extending into insula across tasks. Convergent decreased activation was observed in the caudate (Kogler et al., 2015).

In adolescence, these neural systems are undergoing dynamic changes. Therefore, the stress response might be unique (Romeo, 2010). Highly plastic and developing brain systems are more sensitive to adverse and challenging environmental stimuli (Eiland & Romeo, 2013; Lupien, McEwen, Gunnar, & Heim, 2009). For instance, cortical volume increases in frontal and temporal areas are observed from childhood to puberty, followed by cortical thinning during adolescence (Gogtay et al., 2004). Importantly, current models of adolescent neural development propose that descending projections from the prefrontal (particularly ventromedial prefrontal cortex [vmPFC]) to limbic subcortical areas (e.g., amygdala) are maturing at this time. A shift from positive to negative cortico–subcortical coupling is still being completed during adolescence (Casey, Heller, Gee, & Cohen, 2019), which is likely to impact reactivity to and regulation of psychosocial stress during adolescence.

As studies on stress in adolescence are rare, previous investigations of social rejection as a stressor in adolescence might be informative regarding relevant neural mechanisms. Here, models assume that, on the one hand, dorsal anterior cingulate cortex (ACC), insula, fusiform gyrus, superior temporal sulcus, and anterior temporal gyrus detect social threat, while, on the other, amygdala, mPFC, and subgenual ACC, as well as areas of the neural reward system (orbitofrontal cortex, striatum), are involved in affective responses (Blakemore, 2008; Nelson et al., 2005; Sebastian et al., 2010; Somerville, 2013; Somerville, Whalen, & Kelley, 2010). More specifically, during socially evaluative and stressful situations, dorsal ACC and insula are directly involved in the detection of negative social stimuli and generation of negative affect in adolescents and adults (Masten et al., 2009; Slavich, Way, Eisenberger, & Taylor, 2010). For lower level processing, interpretation and reappraisal of socio-emotional stimuli, superior temporal cortex and fusiform gyrus play important roles (Blakemore, 2008; Pfeifer & Blakemore, 2012). Affective reactivity and processing in these challenging social situations have been linked with subgenual ACC and ventromedial PFC activation. Reactivity in these areas is, moreover, believed to be higher during adolescence compared to childhood and adulthood (Blakemore, 2008; Gunther Moor, van Leijenhorst, Rombouts, Crone, & Van der Molen, 2010; Somerville, 2013). While research in adults suggests significant involvement of the lateral PFC including the inferior frontal cortex in the response to acute stress (Kogler et al., 2015), there are indications that these regulatory brain areas might not be recruited in the adolescent stress response (Sebastian et al., 2011).

Furthermore, acute psychosocial stress is negatively linked to activation in orbitofrontal and striatal regions in adults (Dedovic et al., 2009; Kogler et al., 2015; Porcelli et al., 2012), and similar interactions might be relevant for adolescents (Lincoln et al., 2019). The striatum is a core area of the mesocorticolimbic reward circuit (Walker et al., 2017) and has been found to moderate reward sensitivity (Van Leijenhorst et al., 2010) and reward-related learning (Delgado, 2007). The orbitofrontal cortex is believed to be involved in reward and hedonic experience (Kringelbach, 2005).

Taken together, despite the critical role of psychosocial stress during adolescence for mental health, the amount of studies on the effects of acute stress on the adolescent brain is limited. Previous neuroimaging research on social rejection has pointed to effects of rejection stress on widespread neural areas associated with socio-emotional processing during adolescence. In some areas, such as subgenual ACC and vmPFC, neural reactivity has been suggested to be particularly high, whereas in others, such as inferior frontal gyrus, reactivity might tend to be lower. In the current study, we therefore aimed to advance this existing knowledge by employing an effective parametric design that systematically varied stress level to examine neural correlates linearly associated with acute psychosocial stress in the adolescent brain.

Furthermore, despite immanent instabilities and vulnerabilities during adolescence, it is also a time of intense learning. For example, a second wave of synaptic pruning (after infancy) is believed to underlie rapid gray matter changes during adolescence (Selemon, 2013). Moreover, whereas in children effective prefrontal regulation of amygdala reactivity seems to depend on maternal presence, adolescents' effective neural emotion regulation in the prefrontal–amygdala circuit has been found to be more independent (Gee et al., 2014). With increasing developmental age, PFC-related amygdala modulation and inter-connections within the PFC increasingly facilitate cognitive reappraisal of emotion (Silvers et al., 2017). Similarly, from a cognitive perspective, new abilities allow the use of complex strategies aimed at regulating emotions (LeBlanc, Essay, & Ollendick, 2017), which modulate physiological stress responses (Krkovic, Clamor, & Lincoln, 2018; Nasso, Vanderhasselt, Demeyer, & De Raedt, 2018). These emotion regulation strategies are often defined as "processes responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensive and temporal features, to accomplish one's goals" (Thompson, 1994). Different theoretical models have conceptualized functional strategies as adaptive (e.g., re-evaluation) and dysfunctional as maladaptive (e.g., rumination), dating back to literature on stress coping (e.g., Carver, Scheier, & Weintraub, 1989) and early cognitive behavioral therapists (e.g., Beck, 1979). For developmental and clinical researchers alike, a central criterion for categorizing strategies as adaptive or maladaptive has been their association with health outcome, that is, psychological well-being versus the development of psychopathology such as depression or anxiety disorders (Saarni, Campos, Camras, & Witherington, 2006; Schäfer,

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Naumann, Holmes, Tuschen-Caffier, & Samson, 2017; Zeman, Klimes-Dougan, Cassano, & Adrian, 2007). Usage of adaptive versus maladaptive emotion regulation strategies has been found to be the critical factor for the relationship between stress experience and the development of psychiatric symptoms during adolescence (Kullik & Petermann, 2013; Nolen-Hoeksema & Aldao, 2011; Shapero, Stange, McArthur, Abramson, & Alloy, 2019; Silk, Steinberg, & Morris, 2003; Stevens et al., 2013). In addition to systematically investigating linear neural effects of acute stress by employing an effective parametric design, it was, therefore, another important aim to analyze associations between neural stress reactivity and trait level emotion regulation tendencies.

To address these two research aims as described, we administered an adapted version of the MIST (Dedovic et al., 2005; Zschucke, Renneberg, Dimeo, Wüstenberg, & Ströhle, 2015) to a sample of mid-adolescent individuals during functional magnetic resonance imaging (fMRI). Owing to the heightened salience of social stimuli at this developmental stage (Nelson et al., 2005), we expected that the neural profile of psychosocial stress would be similar to the one found for social rejection during adolescence. Therefore, we hypothesized significantly increased neural reactivity in the medial prefrontal and dorsal ACC, insula, fusiform gyrus, superior temporal sulcus and anterior temporal gyrus under stress. Moreover, based on previous studies using the MIST in adults (Dedovic et al., 2009; van Oort et al., 2017) and studies investigating the effects of acute stress on reward processing (Porcelli et al., 2012; Van Leijenhorst et al., 2010), we expected neural activity in limbic and reward-related areas, particularly in the hippocampus, hypothalamus, orbitofrontal cortex, and striatum, to decrease. Although previous research is rare, we further speculated that use of maladaptive strategies might be linked to more stress-associated activation in limbic areas and adaptive strategies to attenuated neural stress response.

Method

Participants and procedure

The study was approved by the ethics committee of the German Psychological Society; all participants as well as legal guardians provided written informed consent. Fifty-three adolescents from 14 to 17 years took part in the experiment ($M = 15.70 \pm 0.61$, 24 girls).

Participants were screened for the following exclusion criteria via standardized (in-house) questionnaires: (a) adverse health conditions, (b) neurological or psychological disorders, (c) use of medication that influences central nervous system, (d) nonremovable ferromagnetic material. Before the fMRI experiment, habitual use of emotion regulation strategies was measured by self-report (see section on emotion regulation strategies below). Afterwards, participants performed a psychosocial stress task (adapted MIST) in the MRI scanner. Pulse oximetry was used to record heart rate (HR) over the course of the experiment (see section on subjective and autonomic stress response measures for a detailed description). Ten participants were excluded from the fMRI sample due to excessive head movement (>3° mm translation or 3° rotation) and two due to technical problems during fMRI recordings. Thus, fMRI-based analyses relate to a sample of $N = 41$ adolescents ($M = 15.63 \pm 0.59$, 22 girls); 1 left-handed and 40 right-handed (according to the Edinburgh Handedness Inventory) (Oldfield, 1971).

Emotion regulation strategies

We selected the German FEEL-KJ questionnaire (Fragebogen zur Erhebung der Emotionsregulation bei Kindern und Jugendlichen; Grob & Smolenski, 2005) to measure emotion regulation strategies. It assesses a wide range of emotion regulation strategies and was specifically developed for children and adolescents from 10 to 19 years of age. 90 items rated on a 5-point scale (ranging from 1 = *never* to 5 = *always*) index adaptive and maladaptive strategies in response to feelings of anxiety, sadness, and anger. Adaptive strategies consist of problem solving, distraction, forgetting, acceptance, humor enhancement, cognitive problem solving, and re-evaluation, whereas giving up, withdrawal, rumination, self-devaluation, and aggressive actions represent the maladaptive strategies. The mean values of the relative use of adaptive and maladaptive strategies are the two main outcome values. The questionnaire has been shown to be reliable and to reflect a valid self-report measure of emotion regulation strategies in adolescents (Cracco, Van Durme, & Braet, 2015). The scales also showed excellent internal consistency in the current study (adaptive strategies $\alpha = .92$, maladaptive strategies $\alpha = .88$).

Psychosocial stress task

The MIST (Dedovic et al., 2005; Pruessner et al., 2008) as modified by Zschucke et al. (2015) was employed. In summary, the aim of the MIST is to induce psychosocial stress during a mental arithmetic challenge (with difficulty and time limit being manipulated to increase individual failure rates) in combination with an artificial social evaluative threat. Each trial began with the depiction of the mathematical equation. The equation was presented either until the participant submitted a response by choosing an integer via button press or the time allotted was elapsed. Afterwards, the feedback screen was presented for 2.5 s until the next trial started. Each trial lasted from the depiction of the equation until the end of the feedback period.

In detail, the general interface of the modified MIST consisted of the following elements: a mental arithmetic question, a horizontal line of integers from 0 to 9 (used for response submission), a text field that provides feedback on the submitted response ("correct," "incorrect" or "timeout") and a time bar indicating how much time is left for the current trial. The task had three conditions: (a) low stress, (b) moderate stress, and (c) high stress, presented in a fixed sequence. In the low-stress condition (40 trials), participants were given sufficient time (20 s) to solve each arithmetic problem while being told that this part served for training only. These 40 trials were used to calculate participants' individual baseline response time. During the moderate stress condition (32 trials), participants were told that responses were now being recorded and monitored by the experimenter. Participants were given 25% less time than their baseline reaction time to solve each question. Two performance indicators were also added to the interface. One indicator tracked the participants' performance and the other one displayed a faked average of all previous study participants. This faked group average was programmed to be consistently above the current participant's performance. After the moderate stress condition, the experimenter delivered scripted negative feedback over the microphone. Participants were told to improve their performance for a last run, otherwise their data would not be usable. During the high-stress condition (32 trials), the algorithm further restricted the allotted time and increased task difficulty to raise failure rates

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to approximately 50%. See Zschucke et al. (2015) for a detailed description of the version of the MIST that was used.

Participants used the outer button (relative to participant's body) of a four-button fiber-optic response box (Current Design Inc., Philadelphia, USA) in their left hand to move the highlight to the left, and the right-hand button box to move the highlight to the right (outer button) and to submit the answer (inner button). After the task, participants were fully debriefed, told that the task was designed to be impossible to accomplish, and that it did not access their math abilities.

Subjective and autonomic stress response measures

Participants were asked (via microphone) immediately before and after the MIST to evaluate how (a) stressed and (b) strained they felt on a scale from 1 to 10. In addition, we used pulse oximetry to record HR to validate autonomic arousal over the course of the MIST. For this purpose, the integrated photoplethysmograph of the Siemens Physiological Monitoring Unit with an infrared emitter was placed under the pad of the left index finger. Processing of the pulse oximetry data was performed with the TAPAS PhysIO toolbox, version r671, implemented in SPM12 (Kasper et al., 2017). The adaptive "auto-matched" peak detection algorithm of the PhysIO toolbox was used for peak detection. Diagnostic plots of the heartbeat interval time course (as provided by the toolbox) were used to check for missed and wrongly detected heart beats (due to movement or scanner pulses). All data sets with more than three wrongly detected heart beats (as indicated by the toolbox) were excluded from the analyses.

Statistical analysis of behavioral and autonomic stress measures

Two paired sample *t* tests were calculated to assess changes in subjective stress and strain. With regard to HR measurements, we used data derived from peak detection to calculate mean beats per minute (bpm) for all three conditions (low, moderate, and high stress). A repeated analysis of variance (ANOVA) with the factor condition was computed. Furthermore, we calculated mean values for the usage of adaptive and maladaptive emotion regulation. Given previous reports of gender- and age-related differences in emotion regulation (e.g., Zimmermann & Iwanski, 2014), we compared usage of emotion regulation strategies between genders in a mixed-effects ANOVA adding gender (girls, boys) as a group factor and emotion regulation (adaptive, maladaptive) as a between-subject factor. In addition (b), we calculated bivariate correlations between emotion regulation and age of the adolescents.

fMRI acquisition

The fMRI data were acquired in a 3 Tesla Magnetom Trio scanner system (Siemens Medical Systems, Erlangen, Germany) with a 12-channel radiofrequency head coil. The experiment was programmed with Presentation software (Version 14.9, Neurobehavioral Systems Inc., Albany, CA, USA) and projected onto a screen at the end of the magnet bore by a video projector (NEC GT950, NEC Corporation, Itasca, IL, USA, resolution 1,024 × 768 pixels), which participants viewed via a mirror on the head coil. A T1-weighted three-dimensional magnetization-prepared rapid gradient echo sequence with 192 continuous sagittal slices (image matrix = 256 × 256, repetition time [*TR*] = 1,900 ms, echo time [*TE*] = 2.52 ms, flip angle = 9°, field of view [FoV] = 256 × 256, voxel size = 1 × 1 × 1 mm^3) was conducted. Functional images were collected using axial aligned T2*-weighted gradient echo planar imaging (EPI) sequence sensitive to blood-oxygen-level-dependent (BOLD) contrast (33 transversal slices, image matrix = 64 × 64, *TR* = 2,000 ms, *TE* = 30 ms, flip angle = 78°, FoV = 192 × 192, voxel size = 3 × 3×3 mm^3).

fMRI data processing and analysis

Preprocessing

The fMRI data were analyzed using SPM12 (Statistical Parametric Mapping software package; Wellcome Department of Imaging Neuroscience, London, UK). EPIs were corrected for acquisition time delay and motion artifacts and transformed into the stereotactic normalized standard space of the Montreal Neuroimaging Institute (MNI) using the unified segmentation algorithm as implemented in SPM12. Finally, EPIs were resampled (voxel size = 3 × 3 × 3 mm^3) and smoothed with a three-dimensional Gaussian kernel of 7 mm full-width-at-half-maximum.

Single subject analysis

The time series were high-pass filtered (1/256 Hz cut-off frequency). Aliasing effects and serial correlations were modelled using an autoregressive model of first order and the parameters of the model were estimated with a restricted maximum likelihood algorithm as implemented in SPM. A general linear model was implemented at the single-subject level, in accordance with the approach described in Zschucke et al. (2015). We modelled the different conditions convolved with a hemodynamic response function as explanatory variables on a voxel-by-voxel basis. Further, we used the constant of the general linear model (i.e., the mean signal within each run) as our main neural parameter representing stress-associated neural activity of each condition. Motor response, task and feedback screen, as well as the six movement parameters were additionally included as regressors of no interest. For flexible factorial modelling at the group level, we generated three different contrast images at the individual level, which represented the sustained mean signal of the (a) low-, (b) medium-, or (c) high-stress run, respectively. In addition, we generated two contrast images of parameter estimates that compared the high- and low-stress run at the individual level (i.e., [high > low], [low > high]). These were then used in a one-sample *t* test and voxel-wise regression analysis at the group level (see next section).

Group analysis

Individual contrast images were taken to fMRI group random effects analyses using the flexible factorial modelling procedure that included the within-subject factor stress level (low, moderate, high stress). For correction of multiple comparison, voxel-wise familywise error (FWE) correction with $p < .05$ at the peak level was applied for whole brain analyses. It is important to note that this conservative, voxel-wise approach was chosen due to reported issues around cluster-based thresholding (Eklund, Nichols, & Knutsson, 2016) and because it appears to provide an appropriate balance between false positives and false negatives for social neuroscience studies (Han & Glenn, 2017). In addition, we only report resulting clusters with a minimum size of $k \geq 5$ voxels. We then conducted a whole brain voxel-wise regression to analyze the relationship between neural stress reactivity and maladaptive emotion regulation (FEEL-KJ). In a follow-up analysis, we explored potential effects of gender or age differences. A

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two-sample t test with differential contrast images (low vs. high stress) from individual subjects contrasting both genders was computed. By means of a voxel-wise regression analysis with the same contrast images, we tested for associations between age- and stress-induced neural activation. Results were FWE-corrected with $p < .05$ at the peak level within the regions that showed a main effect of stress and only report clusters with a minimum cluster size of $k \geq 5$ voxels.

For completeness, we examined the relationship between significant neural activation clusters in response to stress and subjective as well as autonomic stress response (HR) by extracting mean parameter estimates from these regions for exploratory, post-hoc analysis.

For the purpose of extracting individual beta values contrasting two conditions (low vs. high stress), a one-sample t test with differential contrast images (low vs. high) from individual subjects was computed. This more conservative approach offers several advantages in terms of error partitioning (Henson, 2015; McFarquhar, 2019). From all resulting suprathreshold activation clusters (after whole brain voxel-wise FWE-correction with $p <$ .05 at the peak level) that were 5 voxels or larger, mean parameter estimates for the whole cluster were extracted for each participant for correlation with subjective stress and HR values in SPSS (statistical package for the social sciences) software.

Results

Subjective and autonomic stress response validation, emotion regulation statistics

Self-reported stress and strain significantly increased after performance of the MIST task (stress: $M_{pre} = 2.96 \pm 2.33$, $M_{post} = 6.10 \pm 2.59$, $t\ (49)_{stress} = 9.17$, $p < .001$; strain: $M_{pre} = 3.44 \pm 2.19$, M_{post} 6.23 ± 2.28, $t\ (46)_{strain} = 8.46$, $p < .001$).

Excessive hand movement corrupted the pulse oximetry data of 17 participants (see Supplementary Table 1 in the Supplementary Material for information of sex ratio and mean age in this subsample). The repeated measures ANOVA on the remaining 36 participants showed a main effect of condition ($M_{low} = 64.95 \pm 11.68$ bpm, $M_{moderate} = 70.35 \pm 14.38$ bpm, $M_{high} = 76.73 \pm 18.50$ bpm, $F\ (2) = 17.45$, $p < .001$). Bonferroni corrected post-hoc paired t tests confirmed significant increases in HR from baseline to moderate ($t\ (35) = 2.97$, $p = .010$ adjusted) and from moderate to high stress condition ($t\ (37) = 3.70$, $p = .002$ adjusted).

The mean value for usage of adaptive emotion regulation strategies, which was rated on a 5-point scale ranging from 1 = never to 5 = always, was $M_{adaptive} = 3.26 \pm 0.49$ (variance = 0.19, range = 1.87), the mean value for maladaptive strategies $M_{maladaptive} = 2.68 \pm 0.43$ (variance = 0.20, range = 2.02). The mixed-effects ANOVA regarding emotion regulation showed no significant effect of gender on type of emotion regulation (non-significant main and interaction effects). Age of participants did not significantly correlate (Pearson) with emotion regulation (for both maladaptive and adaptive emotion regulation).

Whole brain stress-induced neural activation

We compared neural activation across the brain between the three MIST conditions (i.e., low, moderate, and high stress) in a flexible factorial design. Linearly increasing activation from low to moderate to high stress was observed in the mPFC/ACC, insula, inferior frontal gyrus, middle orbital gyrus, angular gyrus, anterior temporal gyrus/temporal pole, middle and superior occipital gyrus, and thalamus. Decreasing activation was found in the caudate gyrus and subgenual ACC (Figure 1). Details (laterality, MNI coordinates, and t values) for the comparison between the low- and high-stress condition can be found in Table 1. Please refer to Supplementary Tables 2 and 3 in the Supplementary Material for details on contrasts between moderate- and high- as well as low-stress conditions. One-sample t tests with differential contrast images from individual subjects (low vs. high) resulted in activation clusters within highly similar brain areas (see Supplementary Table 4 in the Supplementary Material).

Voxel-wise regression analysis with differential contrast images from individual subjects (low vs. high) and mean scores of the tendency to use maladaptive emotion regulation showed a significant positive correlation between the tendency of using maladaptive emotion regulation strategies and right temporal pole activation (MNI peak 51/8/−29, $t = 4.29$, $p < .035$, $k = 11$). There was no significant association with adaptive emotion regulation strategies. Adding age as a covariate did not significantly change the results (MNI peak 51/8/−29, $t = 3.80$, $p < .041$, $k = 11$).

The two-sample t test comparing genders did not yield any significant results. Similarly, we did not observe any significant associations between age- and stress-induced neural activation (whole brain voxel-wise regression). When gender and age were added as covariates to the one-sample t test with differential contrast images (low vs. high stress), results were highly similar (see Supplementary Table 5 in the Supplementary Material).

Post-hoc correlations: Neural activation areas, HR and subjective response

Beta values were extracted from the one-sample t test (from all eight significant activation clusters) for exploratory post-hoc correlations with stress-related HR increases. We observed negative correlations of stress-induced right temporal gyrus activation [high > low stress] with stress-induced HR increase (MNI peak 60/−13/−23: Pearson $r = -.502$, $p = .006$; MNI peak 54/5/−29<: Pearson $r = -.377$, $p = .048$, Figure 2). There were no significant associations between subjective stress, stress-induced neural activity or between subjective stress and stress-induced HR activity.

Discussion

Mid-adolescence is a critical period for the development of stress-associated disorders and characterized by vulnerability to social stressors. We therefore investigated neural response to acute psychosocial stress in a sample of mid-adolescent individuals. Largely in line with our hypothesized regions, results showed linear activation increases from low to medium to high psychosocial stress levels in the mPFC/ACC, insula, angular gyrus, inferior/anterior temporal gyrus, and middle occipital gyrus, among other regions. Caudate and subgenual ACC showed linearly decreasing activation. Furthermore, stress-related anterior temporal gyrus activation increase was positively linked to the use of maladaptive emotion regulation and negatively linked to autonomic arousal. In contrast to our expectation, adaptive strategies of emotion regulation were not significantly associated with brain responses to acute stress. Results support the possibility that gender might not significantly affect the response to psychosocial stress at this developmental stage.

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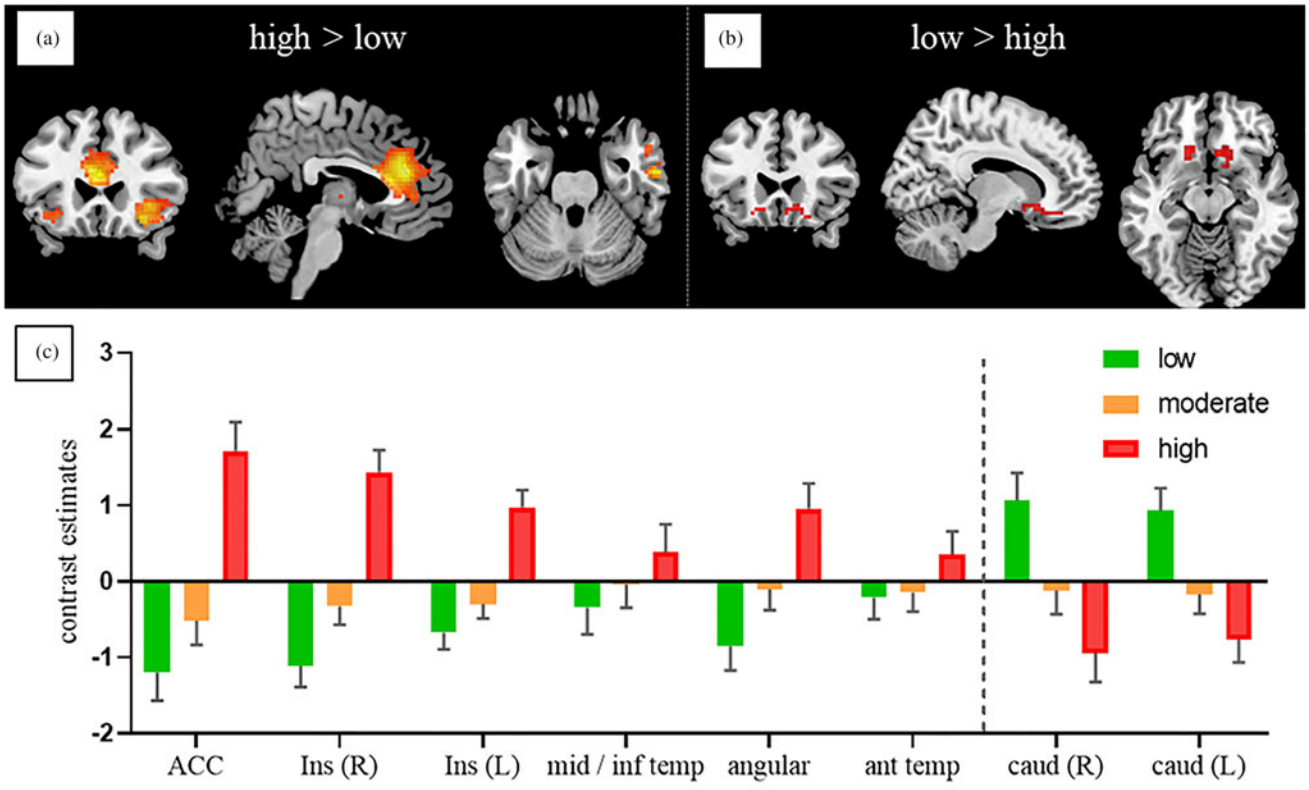

Figure 1. (a) Depiction of significant activation clusters from the contrast high > low stress with flexible factorial analysis of variance (whole brain familywise error [FWE] corrected with $p < .05$ at the peak level, $k \geq 5$ voxels). (b) Depiction of significant activation clusters from the contrast low > high stress with flexible factorial analysis of variance (same statistical threshold as in (a)). (c) Contrast estimates for peak voxels of the main activation clusters from the flexible factorial analysis of variance showing stress-associated activation increases (left) or decreases (right). Low = low stress condition; moderate = moderate stress condition; high = high stress condition; ACC = anterior cingulate cortex; Ins (R) = right insular cortex; Ins (L) = left insular cortex; mid/inf temp = middle/inferior temporal gyrus; angular = angular gyrus; ant temp = anterior temporal gyrus; caud (R) = right caudate nucleus; caud (L) = left caudate nucleus.

Stress-associated pattern of linearly increasing activation

We found increasing activation in mPFC and occipital gyrus and decreasing activation in the reward system. This general activation pattern is in line with the findings that were reported by earlier studies that employed the MIST paradigm in adult participants (for reviews see Dedovic et al., 2009; Pruessner et al., 2010). In line with our expectation, the observed neural circuitry of psychosocial stress in adolescents is similar to adolescents experiencing the distress of being socially rejected (Masten et al., 2009; Sebastian et al., 2011; Slavich et al., 2010). We assume that – at this developmental stage – situations of increased performance pressure that include negative social feedback may be processed similarly to situations of social rejection, leading to the same neural responses. Concordantly, a study by Slavich et al. (2010) highlights the overlap between biological systems for social threat and rejection. They found that in young adults, inflammatory response to a social stressor was linked to neural responses to social rejection in the ACC and insula. Moreover, adolescents show high vigilance to social evaluation and several studies report that social evaluative situations lead to greater self-consciousness (for a review see Somerville, 2013). In line with this idea, the insula is a region known to be involved in visceral and social pain perception, as well as in negative affect attributions (Orenius et al., 2017; Phillips, Drevets, Rauch, & Lane, 2003; Wiech et al., 2010) and represents a key region affected by the experience of traumatic stress (Stark et al., 2015). Furthermore, mPFC is believed to serve the processing of affectively important, self-relevant information in adults and adolescents (Etkin, Egner, & Kalisch, 2011; Pfeifer & Blakemore, 2012; Romund et al., 2017).

Stress-associated deactivation

Contrary to our hypothesis and earlier MIST studies in adults (Dedovic et al., 2005; Khalili-Mahani, Dedovic, Engert, Pruessner, & Pruessner, 2010; Pruessner et al., 2008; Soliman et al., 2011), we did not find large limbic deactivation. In contrast, the adolescent stress pattern was largely characterized by pronounced neural activation rather than deactivation. However, there are also studies in adults using the MIST or similar designs that did not find the frequently reported limbic deactivation in adults (Lederbogen et al., 2011; Wang et al., 2005; Zschucke et al., 2015), and results of several imaging studies show a mixed pattern of increasing and decreasing neural activation in limbic areas during psychosocial stress (e.g., Fan et al., 2015; Grimm et al., 2014). However, the predominance of neural activation rather than deactivation and, in particular, the activation of anterior temporal cortices is intriguing and should be explored in future studies.

Of note, however, increasing intensity of stress was associated with a cluster of linearly decreasing activation in the caudate nucleus encompassing the subgenual ACC and reaching into the medial orbital gyrus. These areas are involved in different

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Table 1. Comparison between high and low stress conditions with flexible factorial analysis of variance

Region	H	Cluster size (voxels)	*t* (peak)	*p* (peak FWE)	MNI coordinates (peak)		
					x	*y*	*z*
t contrast: high > low							
ACC/mPFC	**L**	**817**	**10.00**	**<.0001**	**−3**	**32**	**19**
	L		9.07	<.0001	−6	23	22
	L		7.85	<.0001	−6	32	4
Insula	**R**	**317**	**11.51**	**<.0001**	**33**	**17**	**−11**
IFG (pars orbitalis)	R		6.47	<.0001	39	32	−11
Middle orbital gyrus	R		6.28	<.0001	39	44	−8
Insula	**L**	**155**	**9.51**	**<.0001**	**−27**	**14**	**−8**
IFG (pars orbitalis)	L		6.48	<.0001	−30	29	−11
IFG (pars orbitalis)	L		6.39	<.0001	−39	26	−8
Angular gyrus	**L**	**104**	**7.25**	**<.0001**	**−54**	**−55**	**25**
Inferior temporal gyrus	**R**	**62**	**8.35**	**<.0001**	**57**	**−10**	**−26**
Middle temporal gyrus	R		6.56	<.0001	63	−16	−17
Superior temporal gyrus	R		5.42	.011	51	−7	−11
Middle occipital gyrus	**R**	**16**	**5.49**	**.009**	**30**	**−73**	**28**
Anterior temporal gyrus/temporal pole	**R**	**13**	**6.33**	**.000**	**54**	**5**	**−29**
Thalamus	**R**	**11**	**6.22**	**.001**	**6**	**−10**	**1**
Superior occipital gyrus	**L**	**10**	**5.49**	**.009**	**−21**	**−73**	**40**
Middle occipital gyrus	L		5.09	.036	−27	−73	31
Middle temporal gyrus	**R**	**10**	**5.60**	**.006**	**48**	**−61**	**7**
t-contrast: low > high							
Caudate	**R**	**62**	**6.99**	**<.0001**	**9**	**14**	**−8**
Subgenual ACC	R		6.97	<.0001	6	23	−14
Medial orbital gyrus	R		6.54	<.0001	21	23	−17
Subgenual ACC/Caudate	**L**	**16**	**7.28**	**<.0001**	**−15**	**23**	**−11**

Note. In bold type, peak voxel coordinates and *t* values for significant activation clusters at $p < .05$ familywise error (FWE) corrected across the whole brain, cluster size > 5 voxels; in non-bold type, peak voxel coordinates and *t* values of local maxima within significant activation clusters. ACC = anterior cingulate cortex; mPFC = medial prefrontal cortex; H = hemisphere; L = left; R = right; MNI = Montreal Neurological Institute.

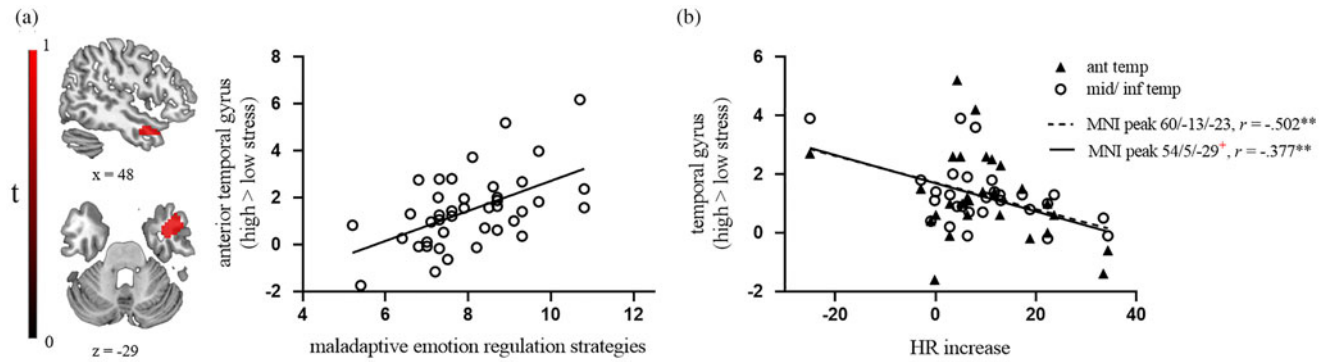

Figure 2. (a) Voxel-wise regression analysis: significant positive correlation between stress-associated anterior temporal gyrus activation and the tendency to use maladaptive strategies to regulate negative emotions. Depicted are activation clusters after whole brain familywise error (FWE) correction with $p < .05$ at the peak level (brain) and extracted parameter estimated from 5 mm sphere around peak (scatter). (b) Significant positive Pearson correlation between stress-associated temporal gyrus activation and stress-induced increase in HR. *Please note that the correlation between HR and activation cluster in the temporal gyrus peaking at Montreal Neuroimaging Institute (MNI) 60/−13/−23 did not survive correction for multiple comparisons. Mid/inf temp = middle/inferior temporal gyrus; ant temp = anterior temporal gyrus; HR = heart rate.

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components of the reward-related neural circuitry in adolescence (Silverman, Jedd, & Luciana, 2015). This converges with earlier studies in adults, where reduced reward processing was observed under stress (Born et al., 2010; Porcelli et al., 2012). Subgenual ACC moreover plays an important role in affective valuations, mood states, and depression (Hamani et al., 2011; Holtzheimer et al., 2017; Laxton et al., 2013). Previous studies also point to its dysregulation in adolescent depression (Ho et al., 2014, 2015). The largest proportion and peak of this decreasing activation cluster was, however, located in the right caudate nucleus. This part of the dorsal striatum is mainly involved in reward-related learning (Delgado, 2007). Associations between a response, on the one hand, and reward versus punishment, on the other, have been found to be represented here (Burton, Nakamura, & Roesch, 2015; Spreckelmeyer et al., 2009). Deactivation under psychosocial stress might thus lead to a decreased association between a response and (social) reward or punishment when an adolescent is under stress (Porcelli et al., 2012). In other words, since the caudate is involved in coding reward prediction during goal-directed behavior (Delgado, Miller, Inati, & Phelps, 2005; Haruno & Kawato, 2006), the negative effect of acute stress on caudate activation might indicate a deterioration of reward expectation and reinforcement of goal-directed behavior under stress. This interpretation would be in line with prior studies reporting lower caudate response for reward anticipation in adolescents with stress-related disorders (i.e., major depression) (Forbes et al., 2006, 2009). More generally, acute stress has been shown to reduce reward responsiveness, which is a promising candidate mechanism linking stressful experience to depression (Bogdan & Pizzagalli, 2006). Overall, these results concur with a body of previous adult and animal research suggesting that exposure to acute stress affects reward sensitivity (e.g., Cao et al., 2010; Ironside, Kumar, Kang, & Pizzagalli, 2018).

Furthermore, in light of heightened social vulnerability in adolescence (Dahl, 2004; Dahl & Gunnar, 2009), it seems likely that this effect might be particularly pronounced for psychosocial stress compared to other kinds of stress. A meta-analysis by Kogler et al. (2015), which directly compared the effects of psychosocial and physiological stress across tasks, lends support to this idea. The authors reported that this deactivation of the caudate is more strongly associated with acute psychosocial than physiological stress. In addition, a recent study on the effects of psychosocial stress on reward processing during adolescence similarly observed decreased activation in the dorsal striatum under acute stress (Lincoln et al., 2019).

The role of anterior temporal gyrus in the adolescent stress response and its link to emotion regulation

Our results indicate that the anterior temporal gyrus may play a significant role in the adolescent psychosocial stress response. Voxel-wise regression showed a positive association with maladaptive emotion regulation. In addition, its activation was negatively correlated with stress-related autonomic arousal (HR increase). Adolescents that report using maladaptive strategies such as self-devaluation, aggression or simply giving up (not responding) more frequently show higher activation of the anterior temporal gyrus during stress induction. This partly confirms our hypothesis by showing that maladaptive emotion regulation strategies are associated with more intense neural stress response. Contrary to our expectation, regions such as amygdala or mPFC that are often primarily linked with emotional responses were not linked to maladaptive strategies. There is, however, substantive evidence from research in adults suggesting a role of the anterior temporal gyrus, including the temporal pole, in socio-emotional processing and regulation (for a review see Olson, Plotzker, & Ezzyat, 2007). Moreover, a study by McRae et al. (2012) who compared neural bases of emotion regulation across the life span found that, compared to children and adults, activation in the anterior temporal gyrus in adolescents more strongly serves the purpose of reappraising negative stimuli and emotions. It is possible that in those adolescents who tentatively revert to maladaptive strategies to regulate negative affect that arises during psychosocial stress induction, socio-emotional threat is more salient and possibly reappraisal facilitated by anterior temporal gyrus is more effortful, resulting in more intense activation increase. However, this interpretation is highly speculative and needs to be further explored in future studies. Moreover, we should note that in the current type of research, which records neural activity to an acute stressor, it remains fundamentally challenging to clearly disambiguate neural reactivity from regulation. There is no scientific consensus on how and whether these two can be usefully distinguished (here) at all. Prominent psychological perspectives see generation and regulation as closely intertwined in these kinds of responses, and not mechanistically distinguishable from one another. Some might argue that they can be differentiated by subtle aspects such as whether the individual has a sense of agency during the experience or at what point in time the activity occurs (e.g., at the very beginning of an emotional episode vs. later on) (Gross & Feldman Barrett, 2011), which can hardly be determined in this type of study design. It is thus difficult to ultimately conclude whether heightened anterior temporal gyrus activation (that was found to be correlated with maladaptive emotion regulation habits) reflects heightened regulatory activity (i.e., reappraisal) or heightened stress reactivity.

We moreover found a negative correlation between stress-associated anterior temporal gyrus activation and autonomic activation. In general, the anterior temporal gyrus has been linked to autonomic reactivity based on its anatomical connections to the hypothalamus. For example, temporal gyrus seizures, which consist of excessive and uncontrolled activity in the temporal gyrus, commonly impair autonomic cardiovascular regulation, particularly in response to stress (e.g., Allendorfer & Szaflarski, 2014; Choudhary et al., 2017; Weil, Arnold, Eisensehr, & Noachtar, 2005). Consistent with the current results, the anterior temporal gyrus is believed to integrate inputs from complex, emotionally evocative stimuli and to assist with regulating the resulting visceral emotional responses (Kondo, Saleem, & Price, 2003; Olson et al., 2007).

In contrast to our expectation, we did not observe any correlation between the habit of using adaptive emotion regulation strategies and neural stress responses. Adaptive strategies assessed here might not have a significant effect on negative emotionality that was created by the current MIST task, and therefore did not correlate with neural stress. This would be in line with a previous study by Silk et al. (2003) observing little effect of primary control strategies, such as problem solving, on feelings of anxiety and anger during adolescence. Moreover, coping literature suggests that these strategies are more helpful in stressful situations that are controllable rather than uncontrollable (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001). It is also possible that the adaptive strategies measured could not be sufficiently used. This would be in line with studies finding low levels of adaptive emotion regulation, and increased use of

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maladaptive emotion regulation during adolescence (Cracco, Goossens, & Braet, 2017; Zimmermann & Iwanski, 2014). In addition, adolescents might not have been able to apply strategies such as cognitive problem solving or humor enhancement (categorized as adaptive) within the context of the MIST. Future studies might examine effects of habitual maladaptive and adaptive strategy use on different kinds of stressors and associated emotional reactions.

Associations between subjective and physiological (autonomic/neural) stress responses

We did not find a significant association between subjectively experienced stress and physiological measures (HR/neural activation). In contrast, an early study on the neural correlates of psychological stress reported a positive correlation between subjective stress ratings and neural responses in several areas, including the right ventral PFC, the left insula/putamen, and the superior temporal gyrus (Wang et al., 2005). However, while several studies examining neural stress responses to the MIST report task-related increases in subjective stress (Boehringer et al., 2015; Grimm et al., 2014; Lederbogen et al., 2011; Zschucke et al., 2015), very few explicitly examine the correspondence between neural and subjective stress responses. A study by Ali, Nitschke, Cooperman, and Pruessner (2017) found that suppression of autonomic and endocrine stress responses through pharmacological interventions did not lower participants' subjective stress responses. This points to a considerable dissociation between subjective experience of stress and physiological stress systems. It is also in line with a previous review by Campbell and Ehlert (2012) that reported an association between biological and subjective measures in only about one quarter of all studies. As has previously been suggested (Ali et al., 2017; Andrews & Pruessner, 2013), this might be due to several methodological issues. Explicit measurements of subjective stress responses, like the one applied in the current study, are often not found to be linked with physiological stress responses. A promising approach towards gaining a better understanding between subjective stress responses and physiological measures might be to use implicit measurements, which have been found to correspond more consistently with biological stress systems (e.g., Mossink, Verkuil, Burger, Tollenaar, & Brosschot, 2015; Verkuil, Brosschot, & Thayer, 2014; Wegner, Schüler, Schulz Scheuermann, Machado, & Budde, 2015).

Strengths and limitations

Finally, we would like to offer some additional considerations concerning our study. In our approach to study the neural substrates of psychological stress, we assumed that stress induces tonic changes of neural activity. First, we did not collect endocrinological data on activation of the hypothalamic–pituitary–adrenal axis or sympathetic nervous system during the psychosocial stress induction, which is a clear limitation of the current study. Nonetheless, the MIST task is a well-validated task for psychosocial stress induction and subjective stress ratings, as well as the increase in HR clearly confirms a pronounced stress response to the MIST. Furthermore, previous studies have reported baseline drifts and carry-over effects of stress-induced psychoneurendocrine activation (cf. Wang et al., 2005). We therefore followed Zschucke et al. (2015) and refrained from using a classical block design BOLD contrast, but rather chose a design of successively increasing stress levels. However, this might somewhat reduce comparability of the current study with effects of other studies in adults using more conventional approaches. Furthermore, when interpreting the present results, we compared with other studies in adults, since information about acute stress processing in adolescents is sparse or even absent. Although our findings indicate a difference in the neural circuitry of acute psychosocial stress and associations with emotion regulation in adolescents versus adults, this assumption must be explored in future studies considering direct comparisons of adolescents and adults. Moreover, there was a relatively high amount of movement in our data, leading to the exclusion of 10 participants from the fMRI sample and 17 from HR analysis. This is rather typical and might be related to intrinsic behavioral properties of the sample in terms of age. Movement artefacts are a common problem in stress induction paradigms and likely to be more pronounced in developmental samples. We used a relatively conservative threshold for fMRI correction (whole brain, voxel-wise FWE correction with $p < .05$ at the peak level) and conservative exclusion criterion for HR artefacts to minimize inadvertent effects. Lastly, variance of the FEEL-KJ data was relatively low, which is likely to have factored into non-significant results, particularly when analyzing associations between stress-related neural activations and adaptive emotion regulation strategies.

Conclusion and Outlook

The neural profile of psychosocial stress highlights the close affinity of psychosocial stress and social rejection experiences for the individual during mid-adolescence. The extent of pronounced linear neural activation rather than deactivation, particularly in the insula, angular, and anterior temporal cortices, emerges when contrasting with adult MIST studies. Based on observed associations with maladaptive regulation and autonomic activity, the anterior temporal gyrus is likely to play a more central role in psychosocial stress processing during mid-adolescence. Its activation during acute psychosocial stress might facilitate adaptive stress processing by means of reappraisal and by binding complex stressors to regulate visceral emotional responses. We speculate that the usage of maladaptive emotion regulation might increase the social threat and reappraisal load of an acute psychosocial stressor. We suggest that future studies should explore the role of the anterior temporal gyrus in stress buffering during adolescence.

Overall, neural stress response and processing of associated emotions during adolescence is likely to impact mental health over the lifetime (Young, Sandman, & Craske, 2019). For example, lower temporal gyrus activation to negative emotional stimuli has been associated with depression during adolescence (Quevedo et al., 2018). In addition, the reward-mediation model poses that stress potentiates the risk of depression via impairing reward processing (Auerbach, Admon, & Pizzagalli, 2014). Future research should probe whether neural response to acute psychosocial stressors at this vulnerable developmental stage predicts development of depression and anxiety disorders and whether dysfunctional strategies to regulate emotions influence this potential link.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0954579421000572

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Conflicts of Interest. None.

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