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Psychosocial versus physiological stress — Meta-analyses on deactivations and activations of the neural correlates of stress reactions

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ABSTRACT

Stress is present in everyday life in various forms and situations. Two stressors frequently investigated are physiological and psychosocial stress. Besides similar subjective and hormonal responses, it has been suggested that they also share common neural substrates. The current study used activation-likelihood-estimation meta-analysis to test this assumption by integrating results of previous neuroimaging studies on stress processing. Reported results are cluster-level FWE corrected.

The inferior frontal gyrus (IFG) and the anterior insula (AI) were the only regions that demonstrated overlapping activation for both stressors. Analysis of physiological stress showed consistent activation of cognitive and affective components of pain processing such as the insula, striatum, or the middle cingulate cortex. Contrarily, analysis across psychosocial stress revealed consistent activation of the right superior temporal gyrus and deactivation of the striatum. Notably, parts of the striatum appeared to be functionally specified: the dorsal striatum was activated in physiological stress, whereas the ventral striatum was deactivated in psychosocial stress. Additional functional connectivity and decoding analyses further characterized this functional heterogeneity and revealed higher associations of the dorsal striatum with motor regions and of the ventral striatum with reward processing.

Based on our meta-analytic approach, activation of the IFG and the AI seems to indicate a global neural stress reaction. While physiological stress activates a motoric fight-or-flight reaction, during psychosocial stress attention is shifted towards emotion regulation and goal-directed behavior, and reward processing is reduced. Our results show the significance of differentiating physiological and psychosocial stress in neural engagement. Furthermore, the assessment of deactivations in addition to activations in stress research is highly recommended.

Introduction

In everyday life we are confronted with social, cognitive or physiological stressors in various situations. Stress is a response to demands placed upon the body independent of the stressors' nature. Various stressor types that are associated with potential threat can induce stress (Selye, 1998; reprinted from 1936). The bodily stress reaction activates the hypothalamic–pituitary–adrenal gland (HPA) axis and subsequently the release of cortisol (Kirschbaum et al., 1993). The psychological homeostatic process is also altered by stress (Burchfield, 1979; Koob, 2009). Thus, the stress response is linked to a state of arousal and hypermobilization of the body's normal activation and emotion system (Hennessy and Levine, 1979; Koob, 2009). According to this view, two distinct types of stressors are physiological stress and psychosocial stress.

Physiological stress is indicated by an unpleasant sensoric, emotional and subjective experience that is associated with potential damage of body tissue and bodily threat (Peyron et al., 2000; Price, 2000; Tracey, 2005). Different bodily conditions may fulfill these criteria, e.g. pain, hunger, oxidative stress, etc. (see e.g., Colaianna et al., 2013). In the current study we will focus on pain processing as physiological stressor, for two main reasons. First, investigating pain as a physiological form of stress has a long lasting history (Lupien et al., 2007; Selye, 1998; Vachon-Presseau et al., 2013b). Second, pain processing is easily

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manipulated and therefore most frequently investigated in neuroimaging environments. Handling pain integrates sensory as well as affective processing (Price, 2000) and it has an arousing effect, increasing cortisol release and negative affect (Rainville, 2002; Vachon-Presseau et al., 2013a; Zubieta and Stohler, 2009). In neuroimaging environments, acute pain is induced by paradigms such as electric shocks or ice cold water which are known to increase cortisol and noradrenalin release.

Psychosocial stress is induced by situations of social threat including social evaluation, social exclusion and achievement situations claiming goal-directed performance (Dickerson and Kemeny, 2004; Pruessner et al., 2010). The need to be affiliated with others and to maintain the social-self are core psychological needs (Dickerson and Kemeny, 2004; Panksepp, 2003; Tossani, 2013). If the gratification of these needs is threatened, for example by a negative judgment of performance by others, then social threat and therefore stress is induced (Dickerson and Kemeny, 2004). Social evaluation as well as cognitive achievement with unpredictable outcome induce heightened cortisol responses, which are accompanied by increases in electrodermal activity, subjective stress reports and negative affect (Dedovic et al., 2009a; Dickerson and Kemeny, 2004; Eisenberger and Lieberman, 2004). Individuals having higher sensitivity towards social evaluation also express elevated cortisol response to acute stressors such as achievement tasks or social exclusion (Kirschbaum et al., 1995; Pruessner et al., 1999, 2008; Seidel et al., 2013; Somerville et al., 2010; Stroud et al., 2002).

Generally, neuroimaging studies refer to neural activations; however, studies investigating psychosocial stress also frequently report neural deactivations (Dagher et al., 2009; Dedovic et al., 2009a; Gradin et al., 2012; Pruessner et al., 2008). The interrelation between activated and deactivated neural areas is not well understood (Arsalidou et al., 2013b). Particularly, deactivations in limbic and cortical regions associated with emotion processing are reported (e.g., Critchley et al., 2000a; Moor et al., 2012; Onoda et al., 2009). However, some studies also report activations in these regions (e.g., Cacioppo et al., 2013; Eisenberger et al., 2003; Sebastian et al., 2011). Thus, inconsistent results regarding activation and deactivation have been reported, particularly in brain regions such as the hippocampus/amygdala, the anterior cingulate cortex (ACC) and prefrontal areas.

In contrast to psychosocial stress, the neural correlates of physiological stress are better characterized. Various meta-analyses of the neural correlates of pain processing identified a network of activated brain areas including primary and secondary motor and somatic regions, insula, dorsal ACC, thalamus, periaqueductal gray and prefrontal cortex (e.g., Apkarian et al., 2005; Friebel et al., 2011; Strigo et al., 2003). These regions process sensory-discriminative information as well as affective-cognitive pain properties (Tracey, 2005). Similar to psychosocial stress, specific deactivations during pain processing in emotion regulation areas such as the amygdala, nucleus accumbens and frontal regions, as well as in motor and sensoric-related areas have been reported (e.g., Aziz et al., 1997; Becerra et al., 2001; Derbyshire et al., 1997).

Taken together, pain as a physiological stressor and achievement situations and social exclusion as psychosocial stressors cause similar subjective, emotional and peripheral stress responses (e.g., Eisenberger et al., 2003; MacDonald and Leary, 2005; Mee et al., 2006; Meerwijk et al., 2013). Both psychosocial and physiological stress are associated with situations that threaten survival (Karremans et al., 2011), and both stressors alter the mesolimbic dopamine transmission in the striatum and the prefrontal cortex (Adler et al., 2000; Pruessner et al., 2008; Saal et al., 2003; Scott et al., 2006). Additionally, it has been argued that similar neural regions, such as the limbic-prefrontal circuit, are activated in processing psychosocial as well as physiological stress (Zubieta and Stohler, 2009). However, until now, this assumption has not been tested quantitatively. The primary interest of the current study lies in assessing the neural correlates of human stress responses to different stressors. In addition to neural activations, we wanted to further determine deactivations from both psychosocial and physiological stress. Therefore, the current meta-analysis set out to test whether psychosocial and physiological stress share overlapping and also distinct neural deactivations and/or activations. To do so, we used an activation-likelihood-estimation (ALE) meta-analysis approach (Eickhoff et al., 2012).

Based on previous results, we expected to find overlaps in deactivations between psychosocial and physiological stress in the amygdala, prefrontal regions and distinct somatosensory areas. Contrarily, brain regions associated with peripheral arousal, emotion processing and avoidance (e.g. prefrontal regions, insula, ACC) were suspected to be activated during both psychosocial and physiological stress.

Material and methods

Selection criteria for used data

Literature research was conducted using PubMed (www.pubmed.com) searching for combinations of the keywords: “fMRI”, “PET”, “neuroimaging”, “stress”, “achievement/cognitive stress”, “psychosocial stress”, “social exclusion”, “social stress”, “social rejection”, “ostracism”, “social pain”, “physiological stress”, “pain”, or “pain regulation”. Additional studies were identified by review articles, other meta-analyses and by tracing references from retrieved studies. Furthermore, in the case that a study did not sufficiently report the results, the corresponding authors were contacted and asked to provide more information on their data. In the following the term “experiment” refers to any single contrast analysis, and the term “study” refers to a scientific publication, usually reporting more “experiments” (Laird et al., 2011).

Only data of healthy adults (aged 18 and older) with no prior report of neurological, psychiatric or pain-related disorders were considered for the current meta-analysis, while results of patient or group effects (e.g., gender differences) were excluded. Furthermore, only neuroimaging studies which utilized either functional magnetic resonance imaging (fMRI) or positron emission tomography (PET) on a whole-brain level and reported the coordinates of brain region activation or deactivation in standard anatomical reference space (Talairach/Tournoux; Montreal Neurological Institute [MNI]) were included. We excluded articles that conducted solely region-of-interest (ROI) analyses or did not report all significant peak-voxels at a specific threshold as well as receptor-PET studies. At last, we excluded studies in which any stress type served as an independent factor affecting further cognitive domains (e.g., fear conditioning, decision making), any pharmacological/placebo studies and correlation or resting-state analyses.

For psychosocial stress we included social exclusion and rejection studies as well as studies investigating cognitive achievement under time pressure or concurrent social evaluation. For physiological stress we included paradigms manipulating pain experience (e.g., extreme heat or cold, electrical stimulation, etc.). As we focused on both activation and deactivation of brain regions during a stressful event compared to a control or baseline condition, activation peaks were defined as brain regions more strongly activated during stress than during control or baseline (stress > control/baseline) and deactivation peaks as less activated during stress compared to control or baseline (control/baseline > stress). As of January 29th, 2014, this resulted in inclusion of 43 experiments for psychosocial (26 activation/17 deactivation; $n = 1130$) and 82 experiments for physiological (69 activation/13 deactivation; $n = 967$) stress (Table 1).

Activation-likelihood (ALE) estimation

All meta-analyses were performed according to the standard analysis method used in previous studies (cf. Bzdok et al., 2012; Langner and Eickhoff, 2013; Rottschy et al., 2012). In particular, analyses were based on the revised ALE algorithm for coordinate-based meta-analysis of neuroimaging results (Eickhoff et al., 2012). This algorithm aims at identifying topographic clusters of activation/deactivation that show significantly higher convergence across experiments than expected under random spatial distributions. Importantly, the reported foci are

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Table 1
Overview of included studies.
A) Physiological stress. B) Psychosocial stress. (Included PET-studies are marked with an asterisk.).

Study	n	Deactivation/activation	Task
A) Physiological stress			
Seminowicz and Davis (2007)	23	Deactivation/activation	Electrical stimulation
Aziz et al. (1997)*	8	Deactivation/activation	Esophageal distension
Torta et al. (2013)	17	Deactivation/activation	Mechanical stimulation
Carlsson et al. (2006)	9	Deactivation/activation	Electrical stimulation
Lui et al. (2008)	14	Deactivation/activation	Mechanical stimulation
Becerra et al. (2001)	8	Deactivation/activation	Thermal stimulation
Oshiro et al. (2009)	12	Deactivation/activation	Thermal stimulation
Coghill et al. (1994)*	9	Deactivation/activation	Thermal stimulation
Derbyshire et al. (1994)*	6	Deactivation/activation	Thermal stimulation
Derbyshire et al. (1997)*	12	Deactivation/activation	Thermal stimulation
Derbyshire and Jones (1998)*	12	Deactivation/activation	Thermal stimulation
Perini et al. (2013)	18	Deactivation/activation	thermal stimulation
Strigo et al. (2003)	7	Activation	Thermal stimulation/esophageal distension
Ladabaum et al. (2001)*	15	Activation	Gastric distension
Benson et al. (2012)	30	Activation	Rectal distension
Dunckley et al. (2005)	10	Activation	Rectal distension/thermal stimulation
Niddam et al. (2002)	10	Activation	Electrical stimulation
Wiech et al. (2006)	12	Activation	Electrical stimulation
Singer et al. (2004)	32	Activation	Electrical stimulation
Ibinson et al. (2004)	6	Activation	Electrical stimulation
Xu et al. (1997)*	6	Activation	Mechanical stimulation
Pujol et al. (2009)	9	Activation	Mechanical stimulation
Farrell et al. (2006)*	10	Activation	Mechanical stimulation
Rolls et al. (2003)	8	Activation	Mechanical stimulation
Iadarola et al. (1998)*	13	Activation	Capsaicin pain
Mochizuki et al. (2007)	14	Activation	Thermal stimulation
Seifert and Maihöfner (2007)	12	Activation	Thermal stimulation
Botvinick et al. (2005)	12	Activation	Thermal stimulation
Lorenz et al. (2002)*	14	Activation	Thermal stimulation
de Leeuw et al. (2006)	9	Activation	Thermal stimulation
Valet et al. (2004)	7	Activation	Thermal stimulation
Bornhövd et al. (2002)	10	Activation	Thermal stimulation
Talbot et al. (1991)*	8	Activation	Thermal stimulation
Vachon-Presseau et al. (2013a)	18	Activation	Thermal stimulation
Hofbauer et al. (2001)*	10	Activation	Thermal stimulation
Kurata et al. (2002)	5	Activation	Thermal stimulation
Peyron et al. (1999)*	12	Activation	Thermal stimulation
Tracey et al. (2000)	6	Activation	Thermal stimulation
Svensson et al. (1998)*	10	Activation	Thermal stimulation
Coghill et al. (2001)*	9	Activation	Thermal stimulation
Brooks et al. (2002)	18	Activation	Thermal stimulation
Dubé et al. (2009)	12	Activation	Thermal stimulation
Kong et al. (2006)	16	Activation	Thermal stimulation
Oshiro et al. (2007)	12	Activation	Thermal stimulation
Coghill et al. (1999)*	16	Activation	Thermal stimulation
Derbyshire et al. (2002)*	16	Activation	Thermal stimulation
Geuze et al. (2007)	12	Activation	Thermal stimulation
Paulson et al. (1998)*	10	Activation	Thermal stimulation
Schmahl et al. (2006)	12	Activation	Thermal stimulation
Smith et al. (2002)	8	Activation	Thermal stimulation
Keltner et al. (2006)	13	Activation	Thermal stimulation
Becerra et al. (2004)	9	Activation	Thermal stimulation
B) Psychosocial stress			
Dedovic et al. (2009b)	28	Deactivation/activation	MIST
Dagher et al. (2009)	15	Deactivation	MIST
Derntl et al. (submitted for publication)	80	Deactivation/activation	MIST
Kogler et al. (2015)	43	Deactivation/activation	MIST
Critchley et al. (2000a)*	6	Deactivation	Mental arithmetic + isometric exercise
Kern et al. (2008)*	14	Deactivation/activation	TSST
Moor et al. (2010)	16	Deactivation	Social evaluation
Gradin et al. (2012)	16	Deactivation/activation	Cyberball
Bolling et al. (2011)	23	Deactivation/activation	Cyberball
Bolling et al. (2012)	20	Deactivation/activation	Cyberball
Sebastian et al. (2011)	16	Deactivation/activation	Cyberball
Seidel et al. (submitted for publication)	80	Deactivation/activation	Cyberball
Maurage et al. (2012)	22	Deactivation/activation	Cyberball
Moor et al. (2012)	15	Deactivation/activation	Cyberball
Lederbogen et al. (2011)	32	Activation	MIST
Soliman et al. (2011)	40	Deactivation/activation	MIST
Fechir et al. (2010)	16	Activation	STROOP
Koric et al. (2012)	15	Deactivation/activation	PASAT
Eisenberger et al. (2003)	13	Activation	Cyberball

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Table 1 (*continued*)

Study	n	Deactivation/activation	Task
B) Psychosocial stress			
Kawamoto et al. (2012)	22	Activation	Cyberball
Masten et al. (2011)	18	Activation	Cyberball
DeWall et al. (2012)	25	Activation	Cyberball
Karremans et al. (2011)	15	Activation	Cyberball
Lelieveld et al. (2012)	72	Activation	Cyberball
Onoda et al. (2010)	26	Activation	Cyberball
Masten et al. (2012)	21	Activation	Cyberball

not treated as single points, but rather as centers of 3D Gaussian probability distributions. This acknowledges spatial uncertainty and reliability by weighting studies according to their sample sizes through the width of the 3D Gaussian probability distribution. Thus, larger sample sizes provide more reliable approximations of the true activation/deactivation effect and are therefore modeled by smaller Gaussian distributions (Eickhoff et al., 2009). The resulting probabilities of all reported foci in a given experiment are combined for each voxel yielding a modeled activation (MA) map (Turkeltaub et al., 2012). The union of all MA maps from all experiments included in the analysis then results in voxel-wise ALE scores, which describe the convergence of results at each particular location in the brain. These ALE scores are then compared to an empirical null-distribution reflecting a random spatial association between experiments' MA maps (Eickhoff et al., 2012). Hereby, a random-effects inference was invoked, focusing on inference on the above-chance convergence between studies, rather than clustering of foci within a particular study.

The null-hypothesis was derived by sampling a random voxel from each of the MA maps and taking the union of these values. The *p*-value of a "true" ALE score is given by the proportion of equal or higher values obtained under the null-distribution. The resulting non-parametric *p*-values were then thresholded at a cluster-level family-wise error (FWE) corrected threshold of $p < .05$ (cluster-forming threshold at voxel-level $p < 0.001$) (Bzdok et al., 2012; Eickhoff et al., 2011; Rottschy et al., 2012).

Additionally, we conducted contrast and conjunction analysis between the meta-analyses of psychosocial and physiological stress. Minimum conjunction analyses (Nichols et al., 2005) were computed in order to isolate the intersection of the thresholded z-maps of two separate meta-analyses. Thus, any voxel determined to be significant by the conjunction analysis constitutes a region in the brain which survived inference corrected on cluster-level FWE in each of the individual meta-analyses. Differences between psychosocial and physiological stress were tested by comparing the two ALEs to a random distribution. First, the true difference between two individual analyses was determined by computing the voxel-wise difference between the unthresholded ALE maps of each analysis (cf. Eickhoff et al., 2012). Second, we determined a null-distribution of differences. This was done by pooling all experiments contributing to either analysis and randomly dividing them into two groups of the same size as the two original sets of experiments. ALE-scores for these two randomly assembled groups were calculated and the difference between these ALE-scores was recorded for each voxel in the brain. Repeating this process 25,000 times then yielded an expected distribution of ALE-score differences under the assumption of exchangeability. The "true" difference in ALE scores was then tested against this null-distribution yielding a probability that the true difference was not due to random noise in an exchangeable set of labels, based on the proportion of lower differences in the random exchange. The resulting probability values were thresholded at $p > .95$ (95% chance for true difference) and inclusively masked by the respective main effects, i.e., the significant effects of the ALE analysis for the particular condition. For both the conjunction and the contrast analyses only clusters larger than 10 voxels were considered. Anatomical labeling was conducted with SPM Anatomy Toolbox version 1.8 (Eickhoff et al., 2005, 2007).

Follow-up analyses

In order to specifically determine functional networks for regions of interest derived from the current meta-analyses, we additionally conducted resting-state functional connectivity analyses as well as functional characterization (e.g., Müller et al., 2014).

Resting-state functional connectivity analysis

Resting-state images were obtained from the Nathan Kline Institute "Rockland" sample (available online as part of the International Neuroimaging Datasharing Initiative; http://fcon_1000.projects.nitrc.org/indi/pro/nki.html), consisting of 132 healthy subjects (representing the U.S. population in key demographic measures; 18–85 years; mean age: 42.3 ± 18.08 years; 78 male, 54 female). 260 images were acquired on a Siemens 3 T TrioTim scanner using BOLD contrast (gradient echo EPI pulse sequence, repetition time (TR) = 2.5 s, echo time (TE) = 30 ms, flip angle = 80°, in-plane resolution = 3.0 × 3.0 mm, 38 axial slices (3.0 mm thickness) covering the entire brain). Data was processed using SPM8 (Wellcome Trust Centre for Neuroimaging, London, http://www.fil.ion.ucl.ac.uk/spm/software/spm8/). The first four scans were discarded from each subject prior to further analyses. EPI images were corrected for head movement by affine registration using a two-pass procedure. In a first step, images were aligned to the initial volumes and subsequently to the mean of all volumes. Next, the mean EPI image was spatially normalized to the MNI single-subject template for each subject (Holmes et al., 1998) using the "unified segmentation" approach (Ashburner and Friston, 2005). Ensuing deformation was applied to the individual EPI volumes. Images were smoothed by a 5 mm full-width-at-half-maximum Gaussian kernel to improve signal-to-noise ratio and to compensate for residual anatomical variations. Time-series of each voxel were processed as follows (Müller et al., 2013): Spurious correlations were reduced by excluding variance which could be explained by the following variables: (1) the six motion parameters derived from image realignment; (2) their first derivatives; (3) mean gray-matter (GM), white-matter (WM), and cerebral blood flow (CBF) intensity (each tissue-signal-class related signal separately). All nuisance variables entered the model as first and second order terms. Finally, data was band-pass filtered (cut-off frequencies of 0.01 and 0.08Hz). The time-courses of all voxels within each seed of interest were extracted for each subject as the first eigenvariate of all GM voxels within the respective seed. Linear (Pearson) correlation coefficients were computed between the resulting characteristic time series of the seed and the time series of all other GM voxels of the brain to quantify resting-state functional connectivity. The voxel-wise correlation coefficients of each subject were transformed into Fisher's z-scores and fed into a second-level ANOVA including an appropriate non-sphericity correction implemented in SPM8. Results were again thresholded at a cluster-level FWE corrected threshold of $p < .05$ (cluster-forming threshold at voxel-level $p < .001$; $k > 10$).

Functional characterization

Functional characterization of regions of interest derived from the meta-analyses was performed by using meta-data categories that

classify each single experimental contrast from the BrainMap database according to the assessed "behavioral domain" (such as emotion, cognition or perception) and "paradigm class" (such as flanker task, mental rotation or reward task) (Turner and Laird, 2012; see http://brainmap.org/scribe/ for the complete list of behavioral domains and paradigm classes). For the analyses the forward and reverse inference approaches were calculated (Müller et al., 2013). The forward inference approach determines the probability of observing activity in a brain region when a mental process is present. We tested whether the conditional probability of activation given a particular task [P(Activation|Task)] was higher than the baseline probability of activation [P(Activation)]. The baseline denotes the probability of finding a (random) activation from BrainMap in the region of interest. Significance was tested using a binominal test ($p < .05$, corrected for multiple comparisons). Additionally, the reverse inference approach tests the probability of the presence of a mental process given knowledge of activation in a particular region of interest. This likelihood [P(Task|Activation)] can be derived from P(Activation|Task) as well as P(Task) and P(Activation) using Bayes' rule. Significance was assessed by means of a chi-square test ($p < .05$, corrected for multiple comparisons) (Amft et al., 2015).

Results

Activation

Physiological stress

The analysis across experiments reporting activations during physiological stress revealed convergent activity in bilateral insula extending to the putamen (PUT), caudate nucleus (CN), pallidum (PA) and temporal pole. Additionally, activation of bilateral supramarginal cortex and rolandic operculum, bilateral thalamus, as well as the right supplementary motor cortex (SMA) extending to the left middle cingulate cortex (MCC), left cerebellum, and right middle frontal gyrus (MFG) emerged (for details see Table 2).

Psychosocial stress

Investigation of consistent activation across experiments assessing psychosocial stress revealed activation of the right superior temporal gyrus (STG) and the right inferior frontal gyrus (IFG) (pars triangularis) extending to the insula (Table 2).

Physiological vs. psychosocial stress

This direct comparison revealed stronger convergence of activation for physiological stress in the bilateral insula extending to PUT, PA and IFG, bilateral supramarginal gyrus extending to the right rolandic operculum, bilateral MCC, bilateral thalamus, right MFG, and left cerebellum (Fig. 1 and Table 2).

Psychosocial vs. physiological stress

For psychosocial compared to physiological stress stronger convergence of activation for psychosocial stress emerged in the right STG (Fig. 1 and Table 2).

Physiological and psychosocial stress

The conjunction analysis revealed common activation for both stressor types in the right IFG (pars triangularis) extending into the insula lobe (Fig. 2 and Table 2).

Deactivation

Physiological stress

The meta-analysis across experiments reporting deactivation upon *physiological* stress revealed significant convergence in the right paracentral lobule (Table 3).

Psychosocial stress

Convergent deactivation across experiments of psychosocial stress was found in one cluster extending from the left CN to the PUT (Table 3).

Physiological vs. psychosocial stress

Physiological stress directly compared to psychosocial stress showed stronger convergence of deactivation in the right paracentral lobule (see Fig. 3 and Table 3).

Psychosocial vs. physiological stress

The direct comparison of psychosocial and physiological stress revealed significantly stronger convergence of deactivations for psychosocial stress in the left CN (see Fig. 3 and Table 3).

Physiological and psychosocial stress

Conjunction analysis did not reveal any common deactivations for both stressor types.

The striatum in physiological and psychosocial stress

Interestingly, engagement of the striatum was found for both stress conditions: for physiological stress activation was reported while for psychosocial stress deactivation emerged. Notably, this activation–deactivation pattern engaged distinct parts of the striatum. Therefore, we additionally compared the results of *activation during physiological* stress with the results of *deactivation during psychosocial* stress. Contrast analysis revealed stronger convergence of activation during physiological stress compared to deactivation during psychosocial stress in the dorsal striatum [maximum peak: −20 2 8], while deactivation during psychosocial stress compared to activation during physiological stress showed significantly more convergence in the ventral part of the striatum [maximum peak: −12 20 −8] (Fig. 4*A*).

From clusters that derived from the comparison of convergent *activation of physiological* and *deactivation of psychosocial* stress, we extracted ROIs according to in-house cytoarchitectonic maps of the striatum (Ludwig-Zahl et al., 2014) implemented in SPM Anatomy Toolbox (Eickhoff et al., 2005, 2007). Both ROIs were then further investigated with regard to their functional connectivity profile as well as their functional properties.

Comparison of resting-state functional connectivity between dorsal and ventral striatum

The left dorsal striatum showed higher functional connectivity than the ventral striatum with numerous regions such as precentral areas and middle cingulate gyrus, supramarginal gyrus extending to the STG, left middle and inferior temporal gyrus or right middle frontal gyrus (see Fig. 4*B* and Table 4). The left ventral striatum demonstrated higher functional connectivity than the dorsal striatum with regions such as mid orbital gyrus and ACC or angular and supramarginal gyri (see Fig. 4*B* and Table 4).

Functional characterization of dorsal and ventral striatum

In addition, the derived clusters of dorsal and ventral striatum were functionally characterized using the "behavioral domain" and "paradigms class" meta-data of the BrainMap database.

Dorsal striatum. Based on the forward inference approach, activation of this cluster was particularly related to behavioral domains of action execution, perception of pain, and action imagination. Paradigm classes significantly associated with dorsal striatum were finger tapping, imagined movement, pain monitor/discrimination, and flexion/tension (Fig. 4*C*). Similar results (additionally including speech execution and overt recitation/repetition) were observed when calculating reverse inference.

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Table 2
Brain regions and activation peaks showing convergence in activation for the main effects for psychosocial and physiological stress, for the contrasts activation in psychosocial vs. physiological and physiological vs. psychosocial stress and for the conjunction of psychosocial and physiological stress.

Contrasts	Cluster	Macroanatomical location	Cytoarchitectonic location	X	Y	Z	t value
Activation physiological							
	Cluster 1 (k = 2181)	Right insula lobe		38	18	0	=8.25
		Superior temporal gyrus		52	12	−4	=6.90
		Right temporal pole		60	6	2	=6.40
		Right pallidum		22	0	−4	=5.59
		Right putamen		22	8	−2	=4.75
	Cluster 2 (k = 2011)	Left insula lobe		−38	14	4	=7.92
		Left rolandic operculum	OP 4	−58	0	6	=5.69
		Left putamen		−20	6	2	=5.07
		Left caudate		−14	8	6	=3.61
	Cluster 3 (k = 1683)	Right SMA	Area 6	4	6	46	=7.97
		Left middle cingulate cortex		0	14	36	=7.81
	Cluster 4 (k = 934)	Left rolandic operculum	OP 3	−42	−18	18	=6.62
		Left supramarginal gyrus	IPC (PFop)	−54	−24	24	=6.50
		Left insula lobe	Insula (Ig2)	−36	−20	2	=4.07
	Cluster 5 (k = 916)	Left thalamus	Th-Prefrontal	−14	−12	10	=8.25
		Right thalamus	Th-Prefrontal	10	−18	4	=7.20
				10	−16	−8	=3.43
			Th-Temporal	2	−6	6	=3.25
		Left thalamus		−20	−16	−2	=3.88
	Cluster 6 (k = 893)	Right supramarginal Gyrus	IPC (PFop)	56	−24	24	=7.86
		Right rolandic operculum	OP 3	44	−14	16	=4.91
	Cluster 7 (k = 277)	Right middle frontal gyrus		38	50	12	=5.83
	Cluster 8 (k = 131)	Left Cerebellum	Lobule VI (Hem)	−24	−66	−26	=4.18
			Lobule VIIa Crus I (Hem)	−26	−70	−28	=4.04
Activation psychosocial							
	Cluster 1 (k = 126)	Right inferior frontal gyrus (p. triangularis)		38	22	8	=4.91
		Right Inferior frontal gyrus (p. triangularis)		42	18	4	=4.05
		Right insula lobe		38	26	−4	=3.61
	Cluster 2 (k = 120)	Right superior temporal gyrus	Ipc (pf)	62	−40	22	=5.22
Contrast							
Activation							
Physiological > psychosocial							
	Cluster 1 (k = 1558)	Left insula lobe		−36	6	6	=5.77
		Left rolandic operculum	OP 4	−58	0	6	=5.69
		Left insula lobe		−38	4	−4	=5.26
		Left Inferior frontal gyrus (p. opercularis)		−50	8	8	=4.92
		Left putamen		−24	0	4	=4.58
		Left rolandic operculum		−62	2	2	=3.94
		Left putamen		−24	8	6	=3.81
				−30	10	2	=3.80
				−20	6	8	=3.77
				−26	2	−10	=3.25
		Left pallidum		−18	−2	0	=3.23
	Cluster 2 (k = 1401)	Right insula lobe		36	10	6	=7.84
		Superior temporal gyrus		54	14	−4	=6.65
		Right pallidum		26	−2	−2	=4.74
		Right putamen		24	8	−2	=4.57
		Right pallidum	Amyg (CM)	24	−8	−6	=3.02
				18	−4	−4	=2.54
		Right insula lobe		40	18	−4	=2.11
	Cluster 3 (k = 1169)	Left SMA		2	12	46	=6.75
		Left middle cingulate cortex		0	12	32	=6.65
		Right middle cingulate cortex		4	12	34	=6.44
		Left middle cingulate cortex		−8	8	38	=5.60
		Right middle cingulate cortex		4	16	30	=3.94
		Left middle cingulate cortex		−12	6	40	=3.94
		Right middle cingulate cortex		6	12	40	=3.67
	Cluster 4 (k = 767)	Left supramarginal gyrus	IPC (PFop)	−54	−24	24	=6.50
		Left postcentral gyrus	IPC (PFop)	−50	−22	30	=3.94
		Left Heschls gyrus	Insula (Ig2)	−38	−18	8	=3.80
		Left Insula lobe	Insula (Ig2)	−36	−18	2	=3.78
		Left Heschls gyrus	TE 1.0	−40	−22	10	=3.49
		Left supramarginal gyrus	IPC (PF)	−58	−32	28	=3.35
			IPC (PFt)	−56	−28	34	=3.02
	Cluster 5 (k = 695)	Right supramarginal gyrus	IPC (PFop)	58	−18	24	=5.57
			IPC (PFop)	58	−22	30	=4.87
			IPC (PFop)	54	−20	28	=4.30
			IPC (PF)	58	−26	32	=3.94
		Right rolandic operculum	OP 1	52	−20	18	=3.86
			OP 4	52	−16	16	=3.81
		Right supramarginal Gyrus	IPC (PF)	60	−30	32	=3.78
			IPC (PF)	56	−32	34	=3.62

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Table 2 (*continued*)

Contrasts	Cluster	Macroanatomical location	Cytoarchitectonic location	X	Y	Z	t value
Contrast							
Activation							
Physiological > psychosocial							
			IPC (PFop)	66	−18	24	=3.60
		Right Insula lobe	OP 3	36	−12	8	=3.13
		Insula (Ig2)	34	−14	6	=3.07	
	Cluster 6 (k = 583)	Left thalamus	Th-Prefrontal	−12	−12	8	=7.05
				−20	−16	−2	=3.49
		Right thalamus	Th-Prefrontal	16	−12	6	=3.45
			Th-Premotor	16	−14	2	=3.38
			Th-Prefrontal	10	−10	0	=3.33
			Th-Temporal	8	−8	8	=3.28
			Th-Temporal	2	−6	6	=3.24
			Th-Parietal	16	−20	10	=2.67
			Th-Somatosensory	16	−20	−2	=2.66
				12	−18	−8	=2.63
			Th-Prefrontal	4	−12	0	=2.31
	Cluster 7 (k = 224)	Right middle frontal gyrus		44	44	14	=3.26
				46	52	14	=3.19
	Cluster 8 (k = 61)	Left cerebellum	Lobule VI (Hem)	−26	−64	−28	=3.16
		Lobule VIIa Crus I (Hem)	−28	−68	−30	=2.07	
Contrast							
Activation							
Psychosocial > physiological							
	Cluster 1 (k = 96)	Right superior temporal gyrus	IPC (PFm)	62	−44	18	=3.13
			IPC (PF)	60	−34	16	=2.20
Conjunction							
Activation							
Physiological & psychosocial							
	Cluster 1 (k = 120)	Right inferior frontal gyrus (p. triangularis)		38	22	8	=4.91
				42	18	4	=4.05
		Right insula lobe		38	26	−4	=3.61

Note. Coordinates x, y, z of local maxima refer to Montreal Neurological Institute space (MNI) (k > 10). k = number of voxels in cluster.
References for histological assignments: Amyg (CM): Amunts et al. (2005); Insula lg2: Kurth et al. (2010a,b); IPC (PF, PFop): Caspers et al. (2006); Lobules VI (Hem), Lobule VIIa Crus I (Hem): Diederichsen et al. (2009); Thalamus (connectivity zones): Behrens et al. (2003); TE 1.0: Morosan et al. (2001); OP1, OP 3, OP 4: Eickhoff et al. (2006a,b).

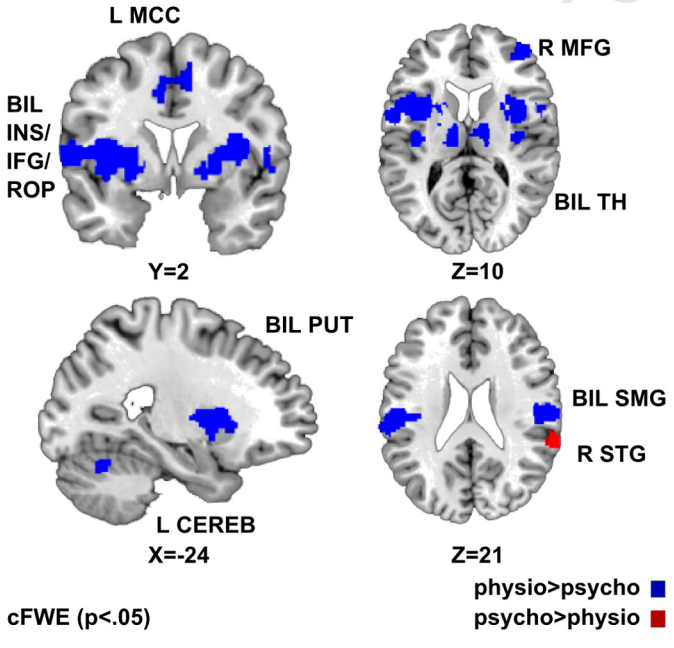

Fig. 1. Activations for physiological and psychosocial stress. Contrasts showing stronger convergence in activation in psychosocial stress than in physiological stress (red) and stronger convergence in activation in physiological stress than in psychosocial stress (blue). (Abbreviations: L = left; R = right; BIL = bilateral; INS = insula; IFG = inferior frontal gyrus; ROP = rolandic operculum; MCC = middle cingulate gyrus; MFG = middle frontal gyrus; TH = thalamus; CEREB = cerebellum; PUT = putamen; SMG = supramarginal gyrus; STG = superior temporal gyrus.) Results are cluster-level FWE corrected (p < .05).

Ventral striatum. Activation of the ventral striatum was significantly associated with the behavioral domains of cognition and emotion as well as the paradigm class reward using the forward inference approach (Fig. 4C). The same results emerged when applying the reverse inference.

Dorsal vs. ventral striatum. These findings were further supported by direct contrast analysis revealing significantly stronger association of the behavioral domains of emotion and cognition and the paradigm class reward with the ventral than the dorsal striatum. Additionally, the

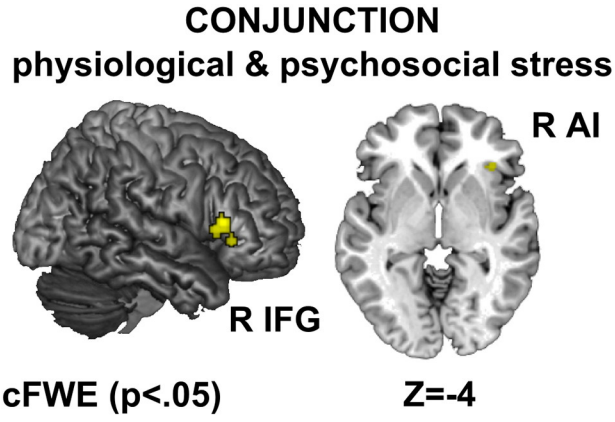

Fig. 2. Conjunction of activations. Conjunction of the results of the meta-analysis on activation in psychosocial stress and the one on activation in physiological stress revealing a cluster in the right inferior frontal gyrus (IFG) extending into the anterior insula (AI). Results are cluster-level FWE corrected (p < .05).

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Table 3
Brain regions and activation peaks showing convergence in deactivation for the main effects for physiological and psychosocial stress as well as for the contrasts deactivation in physiological vs. psychosocial and psychosocial vs. physiological stress.

Contrasts	Cluster	Macroanatomical location	Cytoarchitectonic location	X	Y	Z	t value
Deactivation physiological							
	Cluster 1 (k = 107)	Right paracentral lobule	Area 4a	2	−28	64	=5.29
Deactivation psychosocial							
	Cluster 1 (k = 148)	Left caudate nucleus		−10	18	−6	=4.73
		Left putamen		−18	14	−4	=3.99
Contrast							
Deactivation							
Physiological > psychosocial							
	Cluster 1 (k = 45)	Medial frontal gyrus		0	−30	58	=2.96
		Right paracentral lobule	Area 4a	2	−30	62	=2.30
		Left paracentral lobule		0	−26	62	=1.90
		Right SMA		4	−24	62	=1.81
		Right paracentral lobule	Area 4a	4	−30	66	=1.73
Contrast							
Deactivation							
Psychosocial > physiological							
	Cluster 1 (k = 67)	Left caudate nucleus		−14	20	−4	=2.30
				−10	20	−8	=1.99
				−6	20	−4	=1.94

Note. Coordinates x, y, z of local maxima refer to Montreal Neurological Institute space (MNI) (k > 10). k = number of voxels in cluster.
References for histological assignments: Area 4a: Geyer et al. (1996).

behavioral domains of vision and motion perception, action execution and imagination, speech execution, cognition of language and speech as well as the paradigm classes finger tapping, overt reading, saccades, tone monitor/discrimination, and overt recitation/repetition showed significantly stronger associations with the dorsal than with the ventral striatum.

Discussion

The current study used a meta-analytic approach for quantitatively summarizing activations and deactivations of fMRI and PET studies on psychosocial and physiological stress processing. For this purpose we included studies on achievement stress and social exclusion as indicators for psychosocial stress. Pain processing was used as an indicator for physiological stress. Our results show that physiological and psychosocial stressors deactivate as well as activate distinct neural regions. Furthermore, the striatum appeared to be especially involved in physiological and psychosocial stress. The results are discussed in detail in the following.

Activation in physiological and psychosocial stress

Physiological stress

The meta-analysis of activation during physiological stress revealed convergence in regions typically activated during pain processing (e.g., Peyron et al., 2000), so our findings are in concordance with existing literature on pain experience. The ACC, prefrontal cortex and thalamic nuclei belong to the affective–cognitive–evaluative pain system whereas motor and somatosensory cortices are part of the discriminative-sensoric pain system (Friebel et al., 2011; Iannetti and Mouraux, 2010). The insula seems to mediate systems that are coding intensity and lateralization as well as emotional processing of pain, making it a suspect for coordinating emotional and sensoric properties during physiological stress processing (Friebel et al., 2011). Therefore, the detection of sensoric qualities, the handling of affective information and the integration of those sensoric and affective-emotional sensations are particularly significant in physiological stress processing.

Stronger convergence of activation in physiological than in psychosocial stress. Stronger convergence of activation in physiological compared to psychosocial stress in regions such as the posterior insula, dorsal striatum, IFG (pars opercularis) or MCC indicate sensory-motoric processing (Arsalidou et al., 2013a; Friebel et al., 2011; Kurth et al., 2010b). This specific part of the IFG is reported to be involved in action control (Binkofski and Buccino, 2006), indicating motoric processing and preparation of behavioral tendencies. Additionally, rostral MFG activation is associated with control of negative as well as self-referential processing and episodic working memory (e.g., Gilbert et al., 2006; Yang et al., 2013). The observed activation of regions involved in self-referential working memory and action control indicates a preparation of motoric and behavioral patterns that were acquired in previous situations of bodily threat. This specific induction of a "fight-or-flight" response in situations of physiological stress (Cannon, 1932; Taylor et al., 2000) should be taken into account when using this approach to induce stress for research purposes. Furthermore, people suffering from physiological

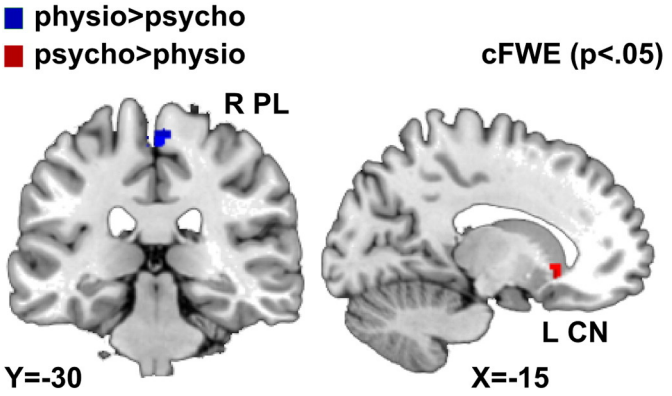

Fig. 3. Deactivations for physiological and psychosocial stress. Contrasts showing stronger convergence in deactivation in psychosocial stress than in physiological stress (red) and stronger convergence in deactivation in physiological stress than in psychosocial stress (blue). (Abbreviations: L = left; R = right; CN = caudate nucleus; PL = paracentral lobule.) Results are cluster-level FWE corrected (p < .05).

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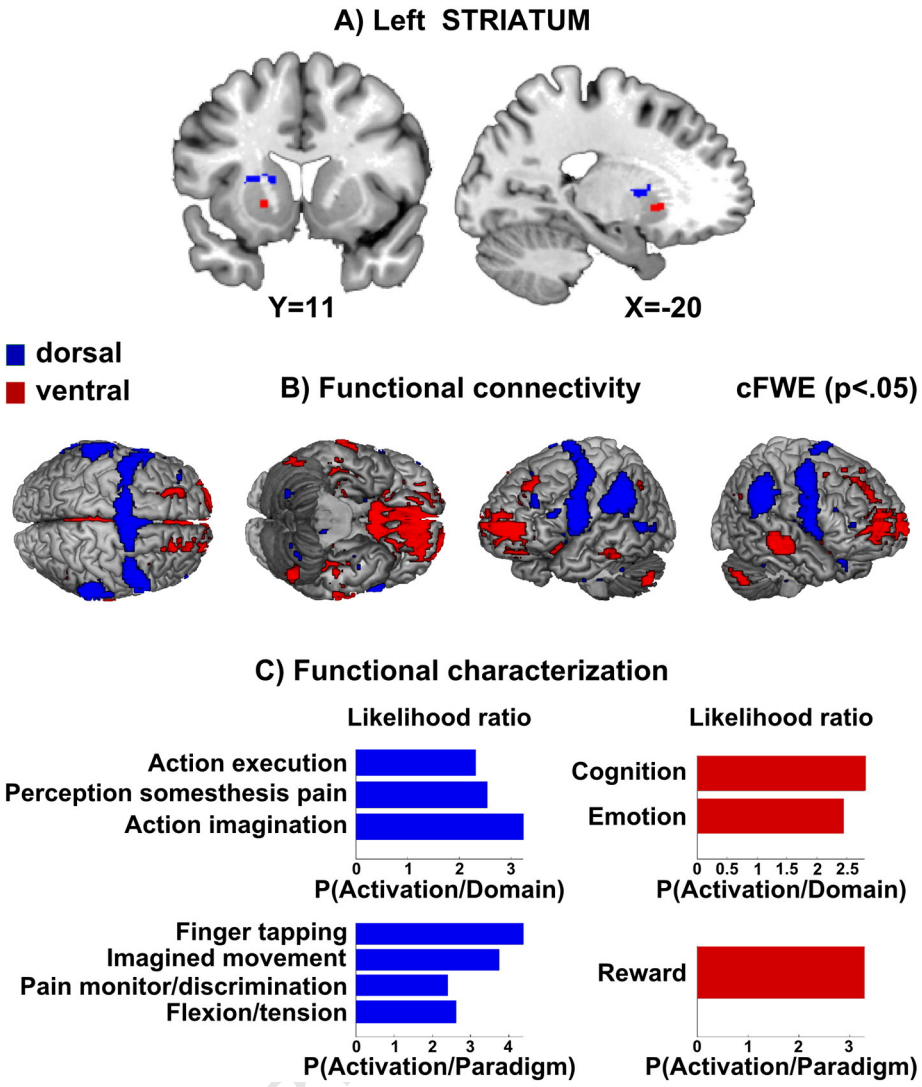

Fig. 4. Dorsal and ventral striatum in stress processing. A) Contrasts showing convergence of deactivation in psychosocial in the ventral striatum (red) and activation in physiological stress in the dorsal striatum (blue). B) Regions showing functional resting-state connectivity with the ventral (red) and the dorsal (blue) striatum. C) Likelihood ratio for significant behavioral domains (graphs in the upper panel) and paradigm classes (graphs in the lower panel) for the ventral (red) and the dorsal (blue) striatum for the forward inference approach.

stress complaints may benefit specifically from targeting these sensoric processing and "fight-or-flight" reactions in stress coping interventions.

Psychosocial stress

Analysis of consistent activation across experiments of psychosocial stress revealed convergence in the right IFG and the right posterior STG. The posterior STG cluster of the current meta-analyses widely overlaps with the anterior temporo-parietal junction, which is strongly involved in attentional processes and shows negative connectivity with a network involved in social cognition (Bzdok et al., 2013). In association with early life and social stress, this region shows decreased resting-state activity and greater gray matter volume (De Bellis et al., 2002; Philip et al., 2013). A recent meta-analysis on emotion regulation (Kohn et al., 2014) indicates the involvement of the right STG in cognitive regulation of emotion. In terms of psychosocial stress, the activation of the right posterior STG indicates enhanced attention processing and regulation of emotional arousal, probably resulting in focused and (ego-centric) goal-directed behavior in situations of psychosocial stress.

Stronger convergence of activation in psychosocial than in physiological stress. Here, the right STG showed stronger convergence in activation for psychosocial stress than for physiological stress. Given its role in attention processing during emotion regulation and goal-directed behavior (Bzdok et al., 2013; Kohn et al., 2014) as well as negative connectivity to networks involved in social cognition (Bzdok et al., 2013), our results indicate that during psychosocial stress attention is focused towards ego-centric emotional arousal while social processing is concurrently reduced. In contrast to pain experience, the competitive nature of challenging tasks in psychosocial stress may reduce social processing but increase attention as well as emotion control, which is subserving the goal-directed orientation when performing the task.

Common activation in physiological and psychosocial stress

A cluster in the right IFG (pars triangularis) extending to the insula showed convergence of activation in the meta-analyses of both stressor types. This part of the IFG, often referred to as VLPFC, is essential for action and cognitive control such as the inhibition of behavior (Aron et al., 2014) as well as for suppressing emotions and emotional memory (Depue et al., 2007; Quirk and Beer, 2006). In addition, IFG activity is associated with processing and regulating particularly negative affective states (e.g., negative affect/emotion processing during negative experiences: Eisenberger et al. (2003); Wang et al. (2005); cognitive emotion

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Table 4
Comparison of resting-state functional connectivity between dorsal and ventral striatum. Note. Coordinates x, y, z of local maxima refer to Montreal Neurological Institute space (MNI) (k > 10). k = number of voxels in cluster.

Contrasts	Cluster	Macroanatomical location	Cytoarchitectonic location	X	Y	Z	t value
Dorsal > ventral							
	Cluster 1 (k = 21,190)	Left putamen		−24	2	6	= 35.46
		Right putamen		26	0	6	= 18.97
				28	−4	8	= 18.57
				28	−10	8	= 17.03
				28	2	0	= 15.99
				30	−8	0	= 15.99
		Left putamen		−28	−16	6	= 15.83
				−18	0	14	= 14.79
				−28	2	−14	= 12.51
				18	0	14	= 12.05
		Left Inferior frontal gyrus (p. Opercularis)	Area 44	−54	8	6	= 12.02
	Cluster 2 (k = 1481)	Right supramarginal gyrus	IPC (PF)	62	−40	24	= 10.33
			IPC (PF)	62	−32	30	= 9.77
			IPC (PF)	60	−40	34	= 9.53
			IPC (PF)	60	−32	36	= 9.52
			OP 1	60	−24	24	= 7.53
		Right superior temporal gyrus	IPC (PFcm)	48	−32	22	= 7.28
		Right supramarginal gyrus	IPC (PFt)	66	−20	34	= 5.97
		Right superior temporal gyrus		68	−42	12	= 5.71
				60	−30	12	= 4.91
	Cluster 3 (k = 398)	Right cerebellum	Lobule VI (Hem)	34	−58	−24	= 5.64
			Lobule VI (Hem)	26	−64	−22	= 5.49
			Lobule VI (Hem)	38	−46	−32	= 5.06
			Lobule VI (Hem)	12	−76	−20	= 4.89
			Lobule VI (Hem)	12	−70	−18	= 4.52
			Lobule VI (Hem)	16	−68	−26	= 4.12
		Right lingual gyrus	Lobule VI (Hem)	10	−64	−10	= 3.76
	Cluster 4 (k = 361)	Left cerebellum	Lobule VIIIa (Hem)	−30	−60	−56	= 5.79
			Lobule VIIIa (Hem)	−24	−66	−52	= 5.41
				14	−56	−34	= 5.10
		Left cerebellum	Lobule VIIb (Hem)	−38	−54	−54	= 4.99
				−14	−50	−36	= 4.91
		Cerebellar vermis	Lobule IX (Vermis)	2	−54	−32	= 4.73
		Left cerebellum		−26	−52	−44	= 4.70
				−18	−48	−40	= 4.60
			Lobule VIIb (Hem)	−38	−50	−52	= 4.23
				−12	−56	−34	= 3.91
				−8	−56	−32	= 3.87
	Cluster 5 (k = 282)	Left cerebellum	Lobule VI (Hem)	−32	−60	−26	= 6.24
			Lobule VI (Hem)	−10	−76	−20	= 5.44
			Lobule VI (Hem)	−26	−66	−22	= 5.37
			Lobule VI (Hem)	−36	−46	−36	= 5.14
			Lobule VI (Hem)	−18	−70	−20	= 4.75
			Lobule VI (Hem)	−36	−48	−30	= 4.35
	Cluster 6 (k = 248)			−8	−38	−42	= 6.84
				8	−38	−44	= 5.51
				4	−28	−40	= 5.15
				2	−18	−38	= 4.26
				−10	−28	−34	= 3.69
				−6	−28	−38	= 3.61
	Cluster 7 (k = 239)	Left middle temporal gyrus		−62	−52	2	= 7.94
		Left inferior temporal gyrus		−58	−54	−6	= 7.00
		Left middle temporal gyrus		−60	−62	0	= 6.80
				−50	−46	10	= 5.07
		Left superior temporal gyrus		−62	−60	8	= 4.85
	Cluster 8 (k = 204)	Right cerebellum		24	−70	−50	= 6.01
				24	−64	−52	= 5.44
				26	−62	−50	= 5.17
				16	−62	−50	= 4.28
	Cluster 9 (k = 89)	Left cerebellum	Lobules I–IV (Hem)	−4	−46	−12	= 4.98
		Cerebellar vermis	Lobule V	6	−48	−12	= 4.89
	Cluster 10 (k = 85)	Right middle frontal gyrus		40	50	24	= 4.70
				44	50	16	= 3.28
Ventral > dorsal							
	Cluster 1 (k = 26,469)	Left caudate nucleus		−12	18	−6	= 56.36
		Right caudate nucleus		10	18	−6	= 23.18
		Left anterior cingulate cortex		−12	38	−4	= 13.79
		Left mid orbital gyrus		0	50	−10	= 13.54
		Right mid orbital gyrus		6	42	−12	= 13.39
		Left mid orbital gyrus		−2	58	−4	= 13.38
				−10	50	−8	= 13.31
		Left anterior cingulate cortex		−18	44	−4	= 13.24
				−6	42	−4	= 13.22

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Table 4 (*continued*)

Contrasts	Cluster	Macroanatomical location	Cytoarchitectonic location	X	Y	Z	t value
Ventral > dorsal							
		Left mid orbital gyrus		−10	42	−6	=13.13
	Right mid orbital gyrus		10	40	−4	=12.98	
	Cluster 2 (k = 629)	Right cerebellum	Lobule VIIa Crus I (Hem)	52	−64	−40	=6.98
			Lobule VIIa Crus I (Hem)	48	−60	−46	=6.03
			Lobule VIIa Crus I (Hem)	42	−56	−44	=5.76
			Lobule VIIa Crus I (Hem)	30	−84	−34	=4.05
			Lobule VIIa Crus I (Hem)	38	−62	−36	=3.74
			Lobule VIIa Crus I (Hem)	30	−76	−34	=3.56
	Cluster 3 (k = 597)	Left angular gyrus	IPC (PFm)	−42	−52	34	=5.92
		Left supramarginal gyrus		−36	−50	30	=5.48
		Left angular gyrus	IPC (PFm)	−44	−56	42	=5.30
		Left inferior parietal lobule		−38	−48	26	=5.21
			IPC (PGa)	−50	−56	36	=5.13
		Left angular gyrus	IPC (PGa)	−50	−64	36	=4.83
			IPC (PFm)	−50	−64	46	=4.44
				−42	−74	48	=4.38
				−36	−58	32	=4.34
			IPC (PGa)	−42	−64	34	=4.22
		Left inferior parietal lobule		−40	−44	26	=3.99
	Cluster 4 (k = 579)	Left cerebellum		−48	−64	−46	=7.78
				−48	−70	−36	=5.43
				−28	−84	−32	=4.79
				−34	−82	−34	=4.54
				−40	−76	−40	=4.21
				−16	−88	−40	=4.19
				−58	−58	−42	=4.02
				−46	−46	−50	=3.89
				−54	−46	−50	=3.85
				−26	−86	−38	=3.71
				−46	−52	−48	=3.56
	Cluster 5 (k = 537)	Right angular gyrus	IPC (PGa)	50	−58	28	=6.47
				40	−62	30	=5.81
		Right middle temporal gyrus		36	−62	28	=5.78
		Right angular gyrus	IPC (PGp)	44	−70	30	=5.61
		Right supramarginal gyrus	hIP1	36	−52	28	=5.25
		Right angular gyrus	IPC (PGp)	48	−68	44	=4.60
		Right angular gyrus	IPC (PGp)	46	−72	44	=4.23
		Right angular gyrus	IPC (PGa)	46	−66	50	=3.95
		Right angular gyrus	IPC (PGp)	44	−74	36	=3.65
Right inferior parietal lobule		IPC (PGa)	56	−60	42	=3.59	

References for histological assignments: Areas 44: Amunts et al. (1999); IPC (PF, PFcm, PFm, PFop, PFt, PGa): Caspers et al. (2006); Thalamus (connectivity zones): Behrens et al. (2003); OP 1, 4: Eickhoff et al. (2006a,b); Lobules I–IV (Hem), V, VI (Hem), VIIa Crus I (Hem): Diederichsen et al. (2009); Intraparietal sulcus (hIP1): Choi et al. (2006).

regulation: Lieberman et al. (2007); Ochsner and Gross (2005); social rejection: Cacioppo et al. (2013)). IFG activation during stress processing may indicate processing negative, subjective experiences, which result from and/or are accompanied by the inhibition of behavioral impulses such as a potential flight reaction.

Besides IFG, activation of the ventral, anterior part of the insula (AI) emerged in the analyses of both psychosocial and physiological stress. The insula merges nociceptive, thermoregulatory, and cardiovascular-related activation, and regulates peripheral activation and autonomic arousal (Critchley et al., 2000b; Rainville, 2002). It is therefore suspected to mediate sensoric and affective processing (Critchley, 2004; Critchley et al., 2000b; Rainville, 2002). The ventral anterior part in particular is engaged during reliving and processing strong emotions (Kober et al., 2008; Kurth et al., 2010b; Touroutoglou et al., 2012), and it is assumed to encode the affective and autonomic features of the current state, i.e. how unpleasant or aversive one feels in a certain situation (Singer and Lamm, 2009; Singer et al., 2004). Therefore, the engagement of the AI during physiological and psychosocial stress may reflect mapping and evaluation of emotions.

Hence, activation of the cluster spanning from IFG to AI found for both stressor types may reflect a global, neural stress reaction. This cluster may be a potential target for stress regulation trainings. Modulating its activation via neurofeedback or non-invasive stimulation and investigating the effect on stress reaction may be the focus of future research (Bauer et al., 2011; Linden et al., 2012; Votinov et al., 2013).

Deactivations in physiological and psychosocial stress

While most neuroimaging studies focus on neural activation, some studies additionally report neural deactivations during stress processing compared to a control condition or baseline activity.

Physiological stress

Convergent deactivation during physiological stress was found in a region covering the right paracentral lobule. The paracentral lobule is especially engaged during activation of inner body organs (Blok et al., 1997; Seseke et al., 2006; Zhang et al., 2005). Additionally, subjective pain sensitivity modulates activation of this region, with stronger activation in subjects who experience pain as more intense (Coghill et al., 2003). Deactivation of the paracentral lobule during physiological stress may cease the acute functioning of essential body organs, suggesting an expedient reaction to situations of bodily threat, which requires the prevention of potentially threatened resources and the preparation of fast motoric reactions.

Stronger convergence of deactivation in physiological than in psychosocial stress. It follows that physiological stress showed stronger convergence in deactivation than psychosocial stress in the paracentral lobule. Again we speculate that the ceasing of acute functioning of essential body organs seems to be more significant for processing sensoric information in

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the threatening situations of physiological stress compared to situations of psychosocial stress.

Psychosocial stress

Psychosocial stress resulted in consistent deactivations in one cluster within the striatum extending from the left CN to the PUT. In line with our results, Nikolova et al. (2012) reported a negative association between recent life stress and a concomitant decrease in striatal activation which in turn was associated with lower positive affect. In general, the CN is associated with various behavioral and cognitive domains, including reward processing and motivation (Arsalidou et al., 2013a). With regard to the latter function, Kumar et al. (2014) showed that psychosocial stress influences reward processing, with decreased CN activation during reward consumption. This taken together with our results indicates that psychosocial stress induces an anhedonic behavior and decreases processing of positive reinforcement as well as motivation (Wang et al., 2007). This is also consistent with reports that emotional complaints such as lack of motivation are associated with stress experience (e.g., American Stress Report, American Psychological Association, 2010; German Stress Report, Lohmann-Haislah, 2012). The PUT, in contrast to the CN, is classically assigned to motor processes and control (Arsalidou et al., 2013a; Leisman and Melillo, 2013). It was also shown to be directly related to pain sensation (Davis et al., 2002; Favilla et al., 2014) and intensity discrimination (Oshiro et al., 2009). At first glance, motor as well as pain related properties seem to be diminished, and reward processing and the capacity of cognitive resources seems to be reduced during psychosocial stress processing.

Stronger convergence of deactivation in psychosocial than in physiological stress. The CN cluster also showed stronger convergence in deactivation during psychosocial stress than during physiological stress. The deactivation of reward related areas may be associated with task engagement particularly during psychosocial stress. Paradigms used to elicit psychosocial stress instructed participants to engage in either a demanding achievement or a social goal-directed task. Contrarily, physiological stress tasks were mainly passive without immediate overt response or cognitive engagement. The effortful nature of the demanding task and the self-relevant evaluation of a situation seem to induce anhedonic mood and decrease processing of positive reinforcements (Pizzagalli et al., 2009; Wang et al., 2007). The engagement of cognitive resources should be taken into account when using different induction methods to assess stress reaction.

The role of the striatum in psychosocial and physiological stress

The striatum appeared to be involved in both psychosocial and physiological stress processing, with activation during physiological stress and deactivation during psychosocial stress. This result may be explained by the assumption that physiological stress engages motor preparation and sensory processing, whereas psychosocial stress down-regulates these processing states. The distinct involvement of the striatum displayed by the current results led us to examine this region in more detail. We conducted additional exploratory analysis, which revealed functionally divided sub-regions of the left striatum: While physiological stress activated dorsal parts, psychosocial stress deactivated ventral parts of the striatum.

Dorsal striatum

The current analyses revealed consistent engagement of the left dorsal striatum during physiological stress. Further functional connectivity analysis revealed that this cluster was functionally connected to other nuclei of the basal ganglia as well as parietal and frontal areas. The functional characterization of this region showed significant association with action execution, pain and sensory processing. Our finding of consistent activation of the dorsal striatum across experiments of physiological stress indicates a dominant role of this region for motor and sensory processes. This, together with results of other studies (Arsalidou et al., 2013a), suggests increased sensory processing and supports our assumption of the preparation of motor programs during physiological stress.

Ventral striatum

In contrast, during psychosocial stress the ventral striatum showed consistent deactivation. Functional connectivity analysis of this region revealed a network involved in emotion processing including other striatal and frontal regions. Additionally, functional characterization of the ventral striatum showed significant associations with cognition, emotion and reward. Therefore, psychosocial stress seems to correlate with deactivation of the ventral striatum, which is involved in processing reinforcement, motivation and executive functioning (Arsalidou et al., 2013a).

Dorsal vs. ventral striatum

Functional division of the striatum has already been suggested by some authors, in particular into three different subzones: a dorsal sensorimotor, a medial cognitive–associative, and a ventral limbic and emotional–motivational striatum (e.g., Lehéricy et al., 2004; Middleton and Strick, 2000; Postuma and Dagher, 2006). The ventral striatum is classically assigned to reward processing (Arsalidou et al., 2013a), and the current results as well as previous literature indicate that it has strong connections with regions associated with emotion processing and executive functioning (Lehéricy et al., 2004; Postuma and Dagher, 2006). Deactivation of the ventral striatum during psychosocial stress points to suppression of functions important for cognitive and emotion processing, in particular reward processing. In contrast, previous and current result show that the dorsal striatum is connected to motor and premotor areas as well as sensory processing brain regions (Postuma and Dagher, 2006). Therefore, activation of the dorsal striatum and its connectivity to motor regions in situations of physiological stress may indicate the preparation of musculoskeletal systems to induce either a fight or a flight response in life threatening situations (Cannon, 1932).

Limitations and suggestions for future studies

The current study has some limitations that might influence data interpretation.

First, we focused on acute stress reactivity without considering chronic psychosocial or physiological stress. Chronic stress is known to have long-lasting effects that are manifested on neural levels such as anatomical volume changes and even neural reorganization (e.g., Admon et al., 2013; Birbaumer et al., 1997). So far, the amount of neuroimaging studies on processing chronic stress is modest, but this factor should be taken into account in future research.

Second, it has to be noted that emotion-induction may induce negative affect and mood states as well, blurring the borders between emotional arousal and stress processing. However, it has been shown that mere emotion-induction studies do not trigger stress responses (Dickerson and Kemeny, 2004). Thus, in the current meta-analyses studies using stress-induction by emotional triggers (e.g., viewing of emotional pictures) were excluded to avoid confusion of mere emotional arousal and stress processing.

Third, the current study focused on pain manipulation as a physiological stressor. Investigating similarities with further possible operationalization of physiological stress, such as hunger or oxidative stress, is of high interest to broaden the knowledge on the neural correlates of stress processing.

Fourth, it may be argued that physiological stress possesses limited variance, whereas psychosocial stress may have more variability. One of the reasons to conduct the current analyses was to define regions that are involved in these diversified stress constructs of physiological and psychosocial stress. The term “stress” is often used without

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specifically differentiating the induction methods. We hope that the current analyses contribute to a better methodological separation of the different possibilities of stress induction in stress research.

Fifth, it is intriguing that regions often reported to be involved in stress reaction, such as the ACC or the amygdala, did not appear to be relevant for stress processing in the current meta-analyses. Inclusion of studies reporting whole-brain analyses was one precondition of the current study. We therefore excluded a fair amount of studies due to region-of-interest analyses or small-volume corrections. This may be a factor explaining the missing effects within these regions.

Sixth, the amount of studies in the field of psychosocial stress is modest and in both psychosocial and physiological stress only a few studies report deactivations. This is the first meta-analysis on deactivation and we hope that reports on psychosocial stress and deactivations will accumulate within coming years to enable more robust results on these data.

At last, future studies may deal with the neural correlates of different stressors when a cortisol reaction was observed vs. when there was no accompanying cortisol reaction. The specific contribution of the HPA axis on the neural deactivations and activations of stress processing is of great interest and may be addressed via this comparison.

Summary and conclusion

The current meta-analyses provide new insights into the neural correlates of stress processing which is strongly dependent on stressor type. Both physiological and psychosocial stress share activation in a cluster extending from the IFG into the AI; therefore, processing and regulation of negative subjective feelings is crucial for both stressor types. Besides that, rather distinct regions underlie the processing of both stress types. During the life threatening nature of physiological stress, the brain adapts by ceasing the functioning of essential body organs. It also engages motoric-sensoric processing and self-referential working memory to prepare a fight-or-flight reaction. Contrarily, the demanding character of psychosocial stress shifts attention to cognitive control of emotion and serves a goal-directed behavior. The effortful nature of psychosocial stress additionally deactivates reward processing and induces anhedonia. Overlaps in deactivations for physiological and psychosocial stress are missing.

Our results have several implications as daily stress varies from health concerns to social and emotional complaints. Increases in prevalence rates in a variety of stress-related disorders have been reported (Keller et al., 2012; World Health Organization, 2001), which demonstrates the importance of investigating reactions to different stressors in more detail. The current analyses show the importance of differentiating physiological and psychosocial stress for specific conclusions on neural stress processing. Furthermore, the assessment of deactivations in addition to activations in stress research is highly recommended.

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Conflict of Interest

All authors declare no conflict of interest in relation to the manuscript.

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