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Research report

An investigation of facial emotion recognition impairments in alexithymia and its neural correlates

Q1 Sebastian Jongen[a], Nikolai Axmacher[b,c], Nico A.W. Kremers[b], Holger Hoffmann[d], Kerstin Limbrecht-Ecklundt[d], Harald C. Traue[d], Henrik Kessler[a,e,*]

[a] Department of Psychiatry and Psychotherapy, University of Bonn, Germany
[b] Department of Epileptology, University of Bonn, Germany
[c] German Center for Neurodegenerative Diseases (DZNE), Bonn, Germany
[d] Department of Medical Psychology, University of Ulm, Germany
[e] Department of Psychosomatic Medicine and Psychotherapy, LWL University Hospital, Ruhr-University Bochum, Germany

HIGHLIGHTS

- We examine neural correlates of emotion recognition impairments in alexithymia.
- High degree of alexithymia is associated with impaired facial emotion recognition.
- High degree of alexithymia is associated with less activity in ACC and other regions.
- High alexithymia is associated with more activity in the superior parietal lobule.

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ABSTRACT

Alexithymia is a personality trait that involves difficulties identifying emotions and describing feelings. It is hypothesized that this includes facial emotion recognition but limited knowledge exists about possible neural correlates of this assumed deficit. We hence tested thirty-seven healthy subjects with either a relatively high or low degree of alexithymia (HDA versus LDA), who performed in a reliable and standardized test of facial emotion recognition (FEEL, Facially Expressed Emotion Labeling) in the functional MRI. LDA subjects had significantly better emotion recognition scores and showed relatively more activity in several brain areas associated with alexithymia and emotional awareness (anterior cingulate cortex), and the extended system of facial perception concerned with aspects of social communication and emotion (amygdala, insula, striatum). Additionally, LDA subjects had more activity in the visual area of social perception (posterior part of the superior temporal sulcus) and the inferior frontal cortex. HDA subjects, on the other hand, exhibited greater activity in the superior parietal lobule. With differences in behaviour and brain responses between two groups of otherwise healthy subjects, our results indirectly support recent conceptualizations and epidemiological data, that alexithymia is a dimensional personality trait apparent in clinically healthy subjects rather than a categorical diagnosis only applicable to clinical populations.

1. Introduction

Alexithymia describes difficulties to identify and describe feelings, a functional and externally oriented way of thinking and reduced emotional awareness in interpersonal interactions. Originally coined in the 1970s, the term alexithymia was introduced as a typical trait in psychosomatic patients, complaining about multiple somatic symptoms due to the lack of a symbolic language to explain their feelings [1]. In the original view, alexithymia represented a categorical diagnostic entity equalling a clinically relevant condition. On the other hand, recent conceptualizations and epidemiological data support the notion that alexithymia is a dimensional personality trait showing a normal distribution in the general population with significantly higher levels in male subjects [2]. Although alexithymic traits can be found in healthy subjects, high levels of alexithymia still represent an independent risk factor

* Corresponding author at: Department of Psychosomatic Medicine and Psychotherapy, LWL-University Clinic Bochum, University Hospital of the Ruhr University of Bochum, Alexandrinenstrasse 1–3, D-44791 Bochum, Germany.
Tel.: +49 234 5077 3176; fax: +49 234 5077 3759.
E-mail addresses: henrik.kessler@ruhr-uni-bochum.de, henrik.kessler@rub.de (H. Kessler).

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for different medical and psychiatric conditions and can best be viewed within the framework of dysfunctional emotion regulation and recognition [3,4]. Empirically though, the latter aspect – impaired recognition of others' emotions in alexithymia – is still discussed controversially. The major instrument to measure alexithymia is the Toronto Alexithymia Scale (TAS-20), which relies on self-rating of a subject's ability to identify and describe feelings [5]. It is not clear, to what extent this self-rating correlates with objectively measured capacities to actually recognize emotions in others [6] and empirical studies showed mixed results [7]. Since the recognition of emotions from *facial expressions* plays an important role in interpersonal communication and is well-studied on the behavioural and neuronal level, we will focus on this aspect. A recent review argues that alexithymia is actually linked with deficits to recognize facially expressed emotions in healthy as well as clinical groups [8]. It is important to notice that although there are studies showing impaired emotion recognition from faces in healthy subjects with alexithymic features [9–12], others have shown no such correlations [13–16]. Interestingly, the only study controlling for verbal abilities found no significant differences in facial emotion recognition between healthy subjects relatively high or low in alexithymia [17]. Even in research with various patient groups with clinically assigned alexithymic features the picture is heterogeneous [18–22].

Regarding this controversy, it is of interest if subjects with alexithymic traits show differential recruitment of brain areas associated with emotion processing when confronted with facially displayed emotions. The literature influencing our hypotheses can roughly be divided into three types of neuroimaging studies: emotion processing (across various tasks) in alexithymia, studies investigating the concept of emotional awareness and facial recognition in general. As for the neural correlates of emotion processing in alexithymia, various single studies and a recent meta-analysis will be reported. Presenting masked emotional faces to healthy subjects with varying degrees of alexithymia in the fMRI, the study of Reker et al. [23] showed activity in insula, superior temporal gyrus, middle occipital gyrus and parahippocampal gyrus to correlate with alexithymia. The study of Duan et al. [24] presented surprised faces subliminally and found activity in parahippocampal gyrus and fusiform gyrus. Finally, the studies of Eichmann et al. [25] and Kugel et al. [26] found a negative correlation between the degree of alexithymia and activity in the fusiform gyrus [25] and the right amygdala [26] when confronted with masked sad facial expressions. The recent meta-analysis by van der Velde et al. [27] examined 15 studies across various task types and valence of emotions and provides converging evidence of a relative hypofunction in alexithymia in amygdala, fusiform gyrus, premotor areas, dorsomedial prefrontal cortex (dMPFC), Insula and precuneus. An interesting case is the activity of the anterior cingulate cortex (ACC) in alexithymia. ACC hypofunction has been associated with alexithymia in many neuroimaging studies (e.g. [28–30]) and is also evident in reduced emotional awareness (see below). In contrast though, the meta-analysis by van der Velde et al. [27] reported a relative ACC *hyper*function in alexithymia. This discrepancy will be considered in the discussion. On the methodological side, two recent meta-analyses show that the majority of alexithymia neuroimaging studies do not explicitly assess the ability to actually recognize emotions [8,27] when using emotional faces as stimuli but rather present faces subliminally. Additionally, only a limited set of emotions was typically used in each study (mostly only up to three). Since we were interested in explicit recognition of a representative array of emotions (i.e. the six basic emotions), previous studies are difficult to compare. On the other hand, the review by van der Velde et al. reported activity independent of task type, which encourages us to derive hypotheses regarding brain areas with hypofunction in alexithymia from the above mentioned literature.

A second group of studies guiding our hypotheses is centred around the concept of emotional awareness, which is a type of cognitive processing of emotions undergoing five levels in rising order [32]. Deficits in emotional awareness are part of the broader concept of alexithymia but not identical to it. This theoretical distinction is supported by empirical findings showing limited correlations between the two concepts (e.g. [33,34]). Many neuroimaging studies of emotional awareness use measures of subjective attention to feelings by studying affective films and pictures. Within this experimental framework, recent neurobiological models posit a deficit of the anterior cingulate cortex (ACC) in the processing of emotions [35,36]. Accordingly, in the general population, the central role of the anterior cingulate and medial prefrontal cortices in emotion processing has been well established [37,38]. Therefore, despite the reported ACC hyperfunction in alexithymia in the meta-analysis by van der Velde et al. [27], we still hypothesize less ACC activation in alexithymia derived from its generally important role in emotion processing [38], the concept of its functional deficits in reduced emotional awareness [39], and neuroimaging studies of alexithymia showing ACC hypofunction (e.g. [28–30]).

The last body of literature concerns the neural correlates of facial recognition in general. The most influential model by Haxby et al. proposes both, a core and an extended system [40,41]. The core system (occipital face area, fusiform face area and posterior superior temporal sulcus) is involved in basic face processing (independently of emotional content) and hence not probable to show strong abnormalities in alexithymia. Of more interest for our study is the extended system which is primarily concerned with extracting meaning from faces, i.e. all the aspects of social communication and emotion. Areas of the extended system processing facial emotions include amygdala, insula and striatum [40,41]. Additionally, the inferior frontal gyrus (IFG) [42] and thalamus as a "sensory gateway" [43] have been implied in emotion recognition from faces. Finally, in an extensive meta-analysis of over 100 studies comparing processing of emotional versus neutral faces, Sabatinelli et al. [44] showed emotion-specific activity in the amygdala, fusiform gyrus, medial prefrontal cortex (mPFC), inferior, superior and middle frontal gyrus, parahippocampal gyrus and middle temporal gyrus.

From this background, we searched for neural correlates of hypothesized deficits in facial emotion recognition in alexithymia. To this end, we assessed the ability to recognize facially expressed emotions with a standardized and reliable test system using functional magnetic resonance imaging (fMRI) in two groups: healthy subjects with relatively high (HDA) or low degree of alexithymia (LDA). The discrepancy between the modern concept of alexithymia as a dimensional trait and our methodological approach categorizing subjects into HDA and LDA merits some explanation: On the one hand, we do believe that alexithymia is a dimensional trait that is present in the general population. In order to avoid confounding factors such as psychopathological symptoms (e.g. anhedonia in depression), we deliberately chose to only recruit healthy participants. On the other hand, we opted for a between-group design comparing two relative extremes within the healthy subjects to improve testing of differences in brain activity. Although this categorizes alexithymia again, it is done within a non-clinical group and for the sake of hypothesis testing. Our methodological decision is backed by the approaches apparent in previous research: Six out of 15 studies of emotion processing in alexithymia mentioned in the meta-analysis by van der Velde et al. [27] use this approach contrasting high versus low alexithymia subjects, and in all of those studies "healthy" participants without any clinically relevant psychopathological symptoms were investigated.

We hypothesized that HDA subjects would perform worse in the facial emotion recognition task compared to the LDA group and that HDA subjects would show differential neuronal activity in key brain

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Table 1
Demographics.

Measure			HDA	LDA	t-Test
Demographics					
N	Total		17	20	
Gender	Women:men		8:9	17:3	
Age	Mean (SD)		25.8 years (6.7)	26.5 years (7.7)	$T = -0.306$; n.s.
Education	Secondary school level I		2	1	
	Secondary school diploma		13	17	
	University		2	2	
Diagnostics					
TAS	Total	Mean (SD)	54.82 (3.9)	30.7 (2.8)	$T = 21.7$; $p < 0.001$
		Range	50–64	24–36	
	Difficulty describing feelings	Mean (SD)	27 (2.6)	13.95 (2.6)	$T = 15.2$; $p < 0.001$
		Range	23–31	10–19	
	Externally oriented thinking	Mean (SD)	13.8 (3.0)	8.5 (1.6)	$T = 6.9$; $p < .001$
		Range	9–19	6–13	
	Difficulty identifying feelings	Mean (SD)	14 (2.4)	8.3 (1.9)	$T = 8.0$; $p < 0.001$
		Range	11–19	5–11	

HDA: High degree of alexithymia group; LDA: low degree of alexithymia group.

areas associated with emotion processing, emotional awareness and facial recognition during the task. In detail, we hypothesized the following areas to show a hypofunction in HDA subjects. The anterior cingulate cortex (ACC) should be hypoactive in accordance with its role in emotion processing, emotional awareness and alexithymia. Additionally, the areas of the extended system of facial emotion recognition (amygdala, insula and striatum), inferior frontal gyrus and thalamus should be relatively hypoactive. Finally, regions reported in other studies comparing emotional versus neutral face processing and partially covered in the meta-analyses by Sabatinelli et al. [44] and van der Velde et al. [27], i.e. fusiform gyrus, medial prefrontal cortex (mPFC), inferior, superior and middle frontal gyrus, parahippocampal gyrus and middle temporal gyrus should also be hypoactive. Those hypothesized areas were tested in a region-of-interest (ROI) analysis if they show less activity in HDA than in LDA subjects.

Furthermore, we performed an explorative analysis checking for any other differences in brain activity between HDA and LDA subjects that might have been missed with the hypothesis-based ROI approach.

2. Materials and methods

2.1. Subjects and alexithymia assessment

The German 20-item version of the Toronto Alexithymia Scale (TAS-20) was used to assess alexithymia [45]. This scale measures (a) difficulty identifying feelings, (b) difficulty describing feelings and (c) externally oriented thinking by assessing the degree of self-reported agreement with 20 statements on a 5-point Likert scale (total scores from 20 to 100). The authors of the original TAS-20 [5] suggested a cut-off criterion of scores ≥ 61 to represent "alexithymia" as a categorical diagnosis. However, the authors themselves describe that cut-off criterion as "preliminary" [46] since there is a lack of empirical data supporting it. Based on a large representative German sample, the 66th percentile (TAS sum score ≥ 52) is suggested as a threshold for the inclusion of alexithymic subjects in experimental settings [2]. We screened 110 study participants (39 males and 71 females) for alexithymia using the TAS-20 in order to obtain two extreme groups with high versus low scores of alexithymia. Based on the TAS sum score, we selected 20 participants with the lowest score (low degree of alexithymia, LDA) and 20 with the highest score (high degree of alexithymia, HDA). Three participants from the HDA group were later excluded from the study due to high-frequency artifacts in fMRI images.

The subjects in the HDA and LDA groups were aged 26.5 (SD = 7.7) and 25.8 (SD = 6.7) years, respectively, and their TAS-20 scores were 54.82 (SD = 3.9; range 50–64) and 30.70 (SD = 2.8; range 24–36), respectively (for demographics, see Table 1). Only one subject in the HDA group had a TAS score of 50, all the others were above the cut-off criterion, i.e. the 66th percentile (≥ 52) suggested by Franz et al. [2]. Subjects were right-handed, native German speakers with normal or corrected-to-normal vision, no MRI exclusion criterion and without current or past neurological or psychiatric disorders. To ensure the latter, every subject was interviewed carefully checking for psychiatric disorders in the history or present and was additionally screened for psychiatric symptoms using Beck Depression Inventory (BDI) and the Symptom Check List (SCL-90) (none above clinical cut-offs). Importantly, there were no significant differences between groups in terms of BDI scores ($t = 0.85$; $p = 0.40$) and TAS-20 scores and measures of negative affect (BDI, SCL-90) were uncorrelated across the whole sample implying no need for the adjustment of TAS-20 scores.

The participants were paid 10€ per hour (total time for the experiment 1–1.5 h). Subjects gave written informed consent and the investigation was carried out in accordance with the latest version of the Declaration of Helsinki. The study protocol was approved by the local ethical committee.

2.2. Facially Expressed Emotion Labelling (FEEL) Test

Participants' emotion recognition ability was assessed with the FEEL Test (Facially Expressed Emotion Labeling) [14,47]. The FEEL Test was originally developed as a computer-based psychometric test aiming to quantify one's ability to recognize facially displayed basic emotions via a forced-choice paradigm. The presented pictures were based upon the JACFEE series (Japanese and Caucasian Facial Expressions of Emotion) developed by Matsumoto and Ekman [48], that showed high validity as they were produced using the Facial Action Coding System (FACS) [49]. The JACFEE photo is comprised of 48 photos, including eight photos of each basic emotion (anger, disgust, fear, happiness, sadness, and surprise). Four photos of each emotion depicted subjects of either Japanese or Caucasian descent (two men, two women). No poser appears more than once for each emotion. Another 48 color photographs of the subjects found in the JACFEE were presented portraying neutral facial

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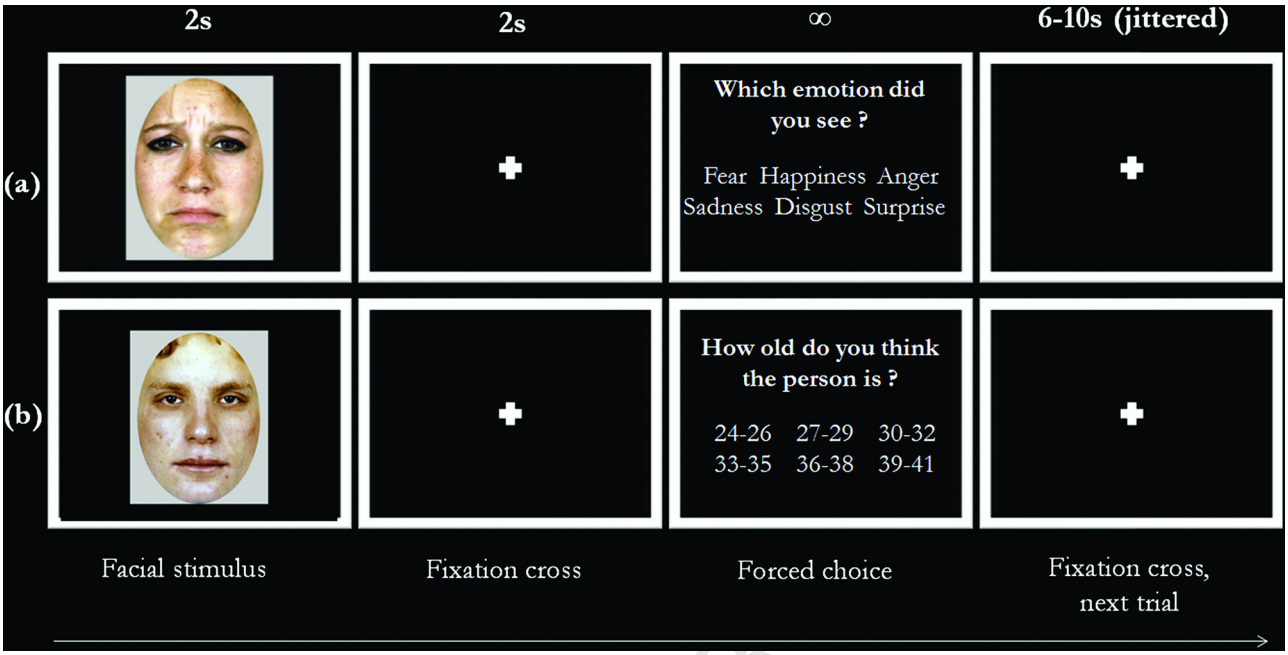

Fig. 1. Outline of the experimental design with exemplary stimuli and presentation times. The two main conditions include (a) presentation of an emotional face and the task to judge the expression, and (b) presentation of a neutral face and the task to estimate the age.

expressions. This equaled a total number of 96 pictures that were shown during our experiment. All images were matched regarding brightness and contrast and were edited to remove hair, ears, shirt collars, in order to force participants to focus on features of the face, i.e. the eyes, mouth, nose and facial geometry.

In our experimental design, we aimed at identifying brain regions related to the detection of emotion in faces. This process depends on two variables: the presence of an emotional face, and the task to identify the emotion in this face. Therefore, we introduced control conditions for these two variables. Participants were shown either emotional or neutral faces (factor "face") and either had to identify the emotional expression or to estimate the age of the face (factor "task"). As a result, we obtained a two-by-two experimental design with four experimental conditions:

(1) emotional face & task to identify the emotion;
(2) emotional face & task to estimate the age;
(3) neutral face & task to identify the emotion;
(4) neutral face & task to estimate the age.

The presentation of stimuli and the subsequent task followed the same temporal pattern for all 96 trials (see Fig. 1 for an overview of the paradigm). A picture of a neutral or emotional face was displayed on the screen in front of the subject for 2 s. The order of pictures was randomized across participants. After a delay of two seconds with a fixation cross, six options appeared on the screen corresponding to the six basic emotions (labelled accordingly) or indicating six age ranges. Subjects had to choose - without a time limit - the emotion or age range they judged to be correct (forced-choice) using a three-button response box (left, right, selection). After selection, a fixation cross appeared with a jittered interval of six to ten seconds before presentation of the next stimulus. It is important to notice, that only the 24 runs included in condition (1) contributed to the FEEL score. With 24 emotional faces that were followed by the task to identify the emotion, the FEEL Score ranges between 0 (no emotion recognized) and 24 (all emotions recognized).

2.3. Image acquisition

Blood oxygenation level-dependent (BOLD) contrast fMRI data were acquired on a 1.5-T Siemens Avanto scanner (Siemens, Erlangen, Germany) with a head matrix coil at the Life & Brain Center in Bonn, Germany. Subjects were placed in the MRI scanner in a supine position with their heads immobilized with cushions to reduce motion artefact and ear plugs to attenuate scanner noise (pre-processing analyses revealed that none of the subjects displayed movement of more than 2 mm in any direction).

A gradient-echo T2*-weighted echo-planar MR sequence was used (TR = 2700 ms, TE = 40 ms, 34 slices, in-plane resolution: 3 mm × 3 mm × 3 mm) and resulted in approximately 600 volumes per participant. After acquisition of functional images, an anatomical image data set was acquired with a standard T_1-weighted high-resolution anatomic scan (TR = 2700 ms, TE = 40 ms, 34 slices, resolution of 1 mm × 1 mm × 1 mm).

2.4. Data analysis

Prior to the statistical analyses the following pre-processing steps were applied to the images: slice timing, motion correction and spatial smoothing using a Gaussian kernel of 8 mm FWHM. Furthermore each subject's T2*-weighted functional images were registered to the T1-weighted structural image, which was then normalized to a standard image (a T1-weighted image in standard space, based on 152 brains from Montréal Neurological Institute). Finally, the normalization matrix was applied to the fMRI images. Pre-processing was conducted using SPM8 (SPM 8, Wellcome Department of Cognitive Neurology, Institute of Neurology, University College, London).

In the second-level analyses, we specifically pursued two paths: (a) analysing contrasts in a priori regions of interest (ROI) that have been associated with alexithymia and emotion recognition tasks and (b) an explorative analysis of BOLD responses (whole-brain) to check for other areas. We used the four conditions as regressors with an onset time at the beginning of facial picture presentation and a variable duration until subjects made their choice, as well

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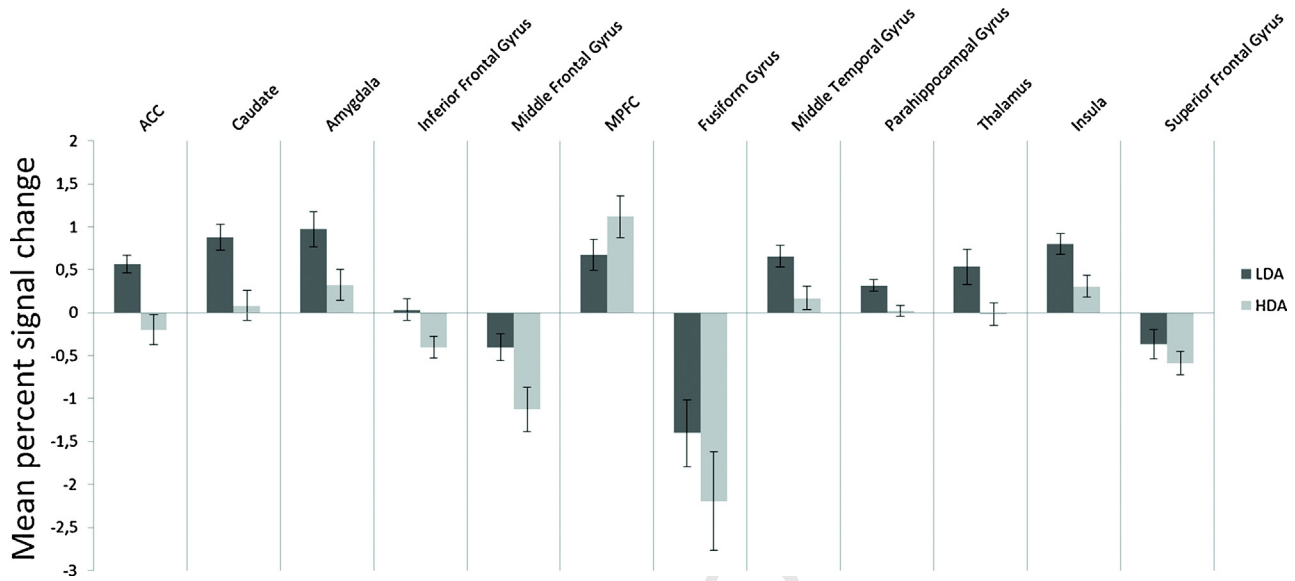

Fig. 2. Brain activity in all 12 regions-of-interest for the contrast Emo_Emo > Neu_Age comparing the low degree of alexithymia (LDA) and high degree of alexithymia (HDA) subjects.

as one regressor modelling scanner drift. We defined the following contrasts:

1) Emotional face and task to identify the emotion (Emo_Emo) versus neutral face and task to estimate the age (Neu_Age).
2) Emotional face and task to identify the emotion (Emo_Emo) versus neutral face and task to identify the emotion (Neu_Emo).
3) Emotional face and task to identify the emotion (Emo_Emo) versus emotional face and task to guess the age (Emo_Age).

The first contrast is of primary interest to test our hypothesis. We compared LDA and HDA subjects with the contrast (Emo_Emo > Neu_Age × LDA > HDA) and calculated this within predefined ROIs (see below) and as a whole-brain analysis. This main contrast, though, cannot isolate a specific process, as the result could be driven by stimulus as well as task characteristics. To control for the influence of stimulus and task on neural activity, the ROI analyses were repeated with contrasts (2) and (3) in regard to differences between both groups.

Both whole-brain and tailored ROI analyses were conducted using SPM8 and Marsbar [50]. For second-level random effects analyses, the single-subject beta-estimates were entered into a full factorial design ANOVA. The ANOVA calculating group differences over all 12 ROIs was implemented in a ROI (12) × GROUP (2) design. In the whole-brain analysis, we adjusted for false positive errors by using a cluster threshold of at least 30 voxels (determined by the Alphasim routine in AFNI; alpha level of $p < 0.001$) [51]. For the ROI analyses, we created anatomically defined regions of interest derived from the literature on alexithymia, emotional awareness and emotion recognition in general. In the case of the striatum as part of the extended System of face recognition, we chose the caudate as a major region of the striatum implicated in emotion processing as our ROI. In addition, we applied spherical ROIs, consisting of 8 mm spheres around the peak activation coordinates of regions that had been reported as being involved in emotion recognition from faces in a recent meta-analysis [44]. In the latter case, we intentionally chose restricted limits, as an anatomically correct inclusion of the whole gyri that encompass peak activation coordinates would have been to unspecific and assess areas with broad functions (e.g. superior frontal gyrus).

The following ROIs were selected (all bilateral if applicable):

Anatomical borders: anterior cingulate cortex (ACC), thalamus, amygdala, caudate, insula, parahippocampal gyrus;

8 mm sphere: superior frontal gyrus, middle frontal gyrus, inferior frontal gyrus, middle temporal gyrus, fusiform gyrus and medial prefrontal cortex.

3. Results

3.1. Behavioral data

In the emotion recognition test (FEEL), a maximum score of 24 was obtainable. Participants in the HDA group performed significantly worse than subjects low in alexithymia (20.65 [SD = 1.41] versus 21.84 [SD = 1.57]; unpaired *t*-test: $T = -2.403$; $p = 0.022$; Mann-Whitney $U = 91.5$; $p = 0.025$).

3.2. Brain activations

We investigated group differences between HDA and LDA subjects over all 12 ROIs for the contrast "emotional face & task to identify the emotion (Emo_Emo) versus neutral face and task to estimate the age (Neu_Age)". The ROI (12) × GROUP (2) ANOVA revealed a significant main effect for GROUP ($F = 13.35$; $p < 0.001$). Fig. 2 shows activity differences between both groups separately for all 12 ROIs for the contrast Emo_Emo > Neu_Age. Therefore, our second hypothesis was confirmed with subjects high in alexithymia having relatively less activity in a cluster of regions comprising anterior cingulate cortex, amygdala, insula, inferior frontal gyrus, thalamus, caudate and others when judging the emotional content of a face.

In principle, activity in our main contrast may either be due to the stimuli (emotional versus neutral) or the task (emotion assessment versus age assessment). We hence calculated two additional contrasts and repeated the ROI analyses to differentiate between these two possibilities. Both contrasts yielded a significantly higher activity over all 12 ROIs in the LDA group (Emo_Emo > Emo_Age × LDA > HDA: $F = 27.35$; $p < 0.001$; Emo_Emo > Neu_Emo × LDA > HDA; $F = 20.66$; $p < 0.001$). Those results indicate that both, stimulus and task, have an impact on the primary results and lead to activity differences in the regions of interest that differentiates between LDA and HDA subjects.

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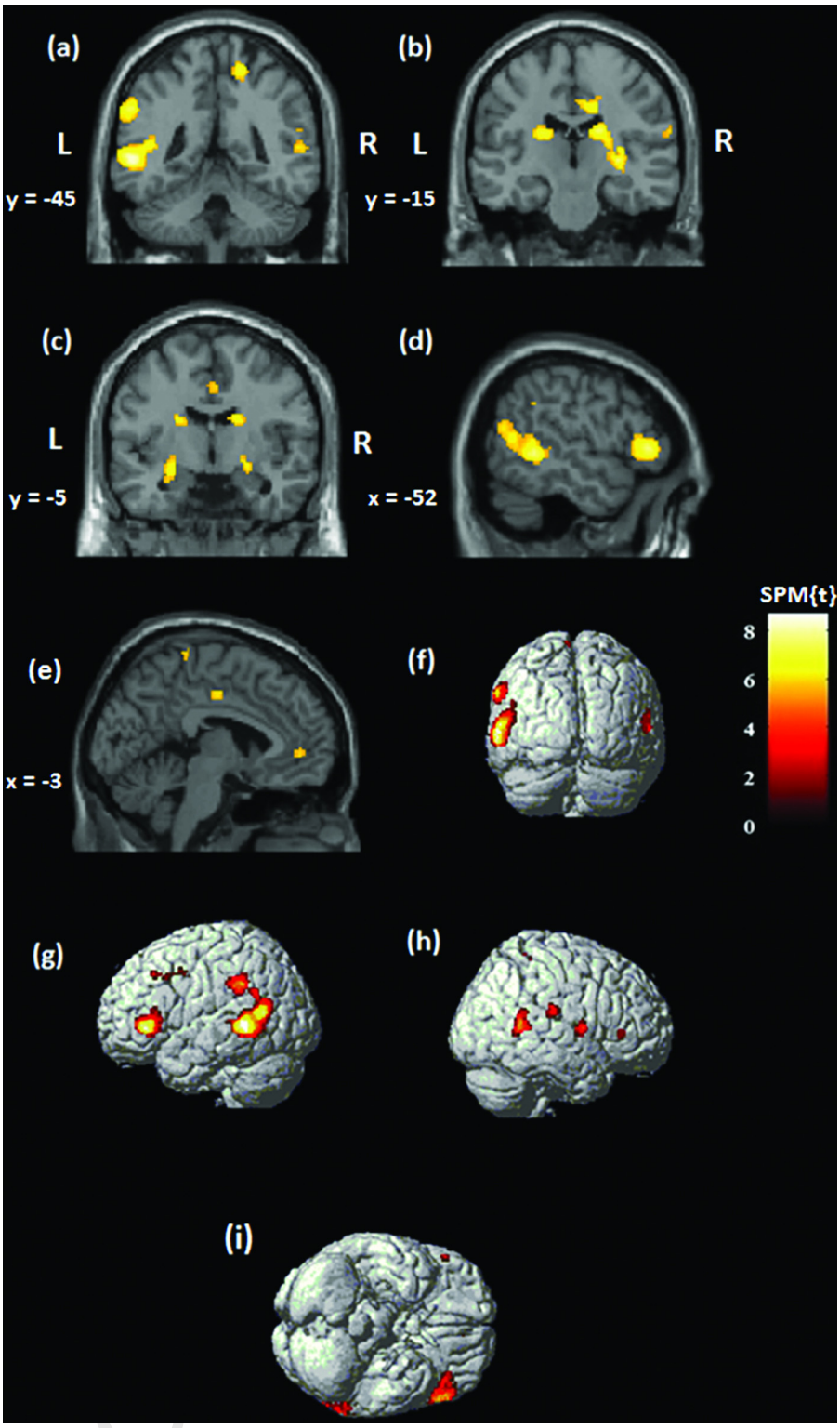

Fig. 3. Brain images of the differential BOLD responses (LDA versus HDA) elicited by the emotional pictures and the task to judge the emotion compared with neutral pictures and the task to estimate the age in our primary contrast Emo_Emo > Neu_Age × LDA > HDA. The bar on the right shows the range of *t*-scores for SPM. The height threshold for illustrating the clusters was $p = 0.001$ (FDR corrected) with a cluster-threshold of 30 voxels (as determined by the Alphasim routine in AFNI).

Next, we aimed at identifying differences between HDA and LDA subjects in neural activation during emotion recognition on an explorative whole-brain level. The contrast (Emo_Emo > Neu_Age × LDA > HDA) yielded significant activations in the inferior frontal gyrus, middle temporal gyrus, supramarginal gyrus, cuneus/precuneus, thalamus, insula, anterior cingulate cortex, caudate, parahippocampal gyrus and others ($p < 0.001$ (FDR) with a cluster threshold of 30 voxels; see Fig. 3; a list of all significant clusters can be found in Table 2). The whole-brain analysis confirms the results of our ROI analyses with regions associated with alexithymia and emotional awareness (ACC) and areas related to facial emotion recognition (inferior frontal gyrus,

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Table 2
Activated regions and *T* scores for the contrast Emo_Emo > Neu_Age × LDA > HDA in a whole-brain analysis.

Regions	Brodmann area	MNI coordinates *x*, *y*, *z* (mm)			Side	*t* or *F*	Cluster size
Emo_Emo > Neu_Age × LDA > HDA							
Middle temporal gyrus	21	−58	−48	−2	L	6.2	1435
Middle temporal gyrus	22	−46	−44	−10	L	4.5	
Superior temporal gyrus	22	−44	−58	14	L	4.6	
Caudate	*	20	−12	22	R	5.9	724
Parahippocampal gyrus	*	22	0	−10	R	4.7	
Putamen	*	30	−20	2	R	4.2	
Thalamus	*	−18	−25	16	L	5.8	313
Caudate	*	−16	−16	20	L	5.0	
Caudate	*	−20	−8	20	L	4.3	
Inferior frontal gyrus	45	−50	34	2	L	5.7	533
Inferior frontal gyrus	47	−38	32	0	L	5	
Supramarginal gyrus	40	−58	−42	36	L	5.2	258
Amygdala	*	−28	−4	−14	L	4.8	142
Cingulate gyrus	24	12	−18	38	R	4.5	255
Precentral gyrus	6	54	0	8	R	4.5	84
Middle frontal gyrus	6	−36	4	42	L	4.3	142
Middle frontal gyrus	8	−28	26	38	L	4.0	
Precentral gyrus	9	−36	14	40	L	3.7	
Superior temporal gyrus	22	56	−48	10	R	4.2	320
Middle temporal gyrus	39	46	−52	4	R	4.0	
Middle temporal gyrus	39	58	−48	18	R	3.8	
Insula	13	35	−4	14	L	4.0	60
Hippocampus	*	−34	−34	−4	L	4.0	52
Anterior cingulate	32	−6	44	0	L	3.9	58
Inferior frontal gyrus	45	54	32	4	R	3.8	30
Postcentral gyrus	40	62	−24	20	R	3.7	75
Emo_Emo > Neu_Age × HDA > LDA							
Superior parietal lobule	7	−28	−60	50	L	4.7	472
Superior parietal lobule	7	−30	−52	46	L	4.2	
Superior parietal lobule	*	−28	−44	32	L	3.6	

Areas which are significant for the contrast Emo_Emo > Neu_Age × LDA > HDA are reported at the level of $p = 0.001$ (FDR corrected) and with a cluster-threshold of 30 voxels. *X*, *Y*, *Z* values indicate center of gravity of the cluster. Number of voxels gives the number of active voxels in this specific region and/or in this Brodmann area. Column "*t* or *F*" represents maximal *t*-value or *F*-value for the given cluster.

thalamus, insula, caudate, etc.) being more active in subjects low in alexithymia. The contrast (Emo_Emo > Neu_Age × HDA > LDA) showed one large cluster in the superior parietal lobule where subjects high in alexithymia have more activity when recognizing emotions (see Table 2). Similar as for the ROI analysis, the two additional contrasts differentiating between stimulus and task influences yielded comparable results as the main contrast (Emo_Emo versus Neu_Age) for the whole-brain analysis. This further confirms that both, stimulus and task, have an impact on the primary results.

4. Discussion

This study showed that healthy individuals with alexithymic traits have deficits in their ability to recognize facially expressed emotions. During the task they also exhibited less recruitment of specific brain regions hypothesized to be hypoactive in alexithymia: the anterior cingulate cortex, areas that are part of the extended system of face recognition (amygdala, insula, striatum) and other regions previously implicated in facial emotion recognition (e.g. inferior frontal gyrus, middle temporal gyrus, thalamus, parahippocampal gyrus, and middle frontal gyrus). Interestingly, subjects high in alexithymia showed enhanced activity in the left superior parietal lobule.

Two distinguishing features from other neuroimaging studies of alexithymia concern the presentation of a broad spectrum of emotions (as opposed to one to three emotions in the majority of previous studies) and the need for explicit emotion recognition in order to score in the task (as opposed to masked presentation of emotional faces in many other studies). This was done in order to closer mirror real-life situations and enhance ecological validity.

4.1. Behavioral data

In the controversy whether healthy subjects with alexithymic features do show impaired emotion recognition from faces or not, our results confirm the first position with HDA subjects scoring significantly lower in a standardized and reliable test to assess facial emotion recognition (FEEL). We are hence in line with a recent review arguing for deficits in facial emotion labelling in alexithymia [8]. The small absolute differences between groups (20.7 versus 21.8), may be a reason for the heterogeneity between studies, with confounding variables and other "noise" easily changing results.

4.2. Brain activations

The discussion of brain areas differentially active between groups follows their hypothesized importance in alexithymia and is separated into functional groups. In line with the concept of *reduced emotional awareness* (being part of but not identical to alexithymia), the ACC as the major structure involved in emotional awareness showed relative hypofunction in our HDA subjects. Early studies reported that ACC activity is associated with attention to subjective emotional responses [52]. Later studies found that in otherwise healthy subjects activity in ACC was negatively correlated with emotional awareness when confronted with emotional stimuli [35,36,53]. These empirical findings and theoretical considerations led to the assumption that reduced emotional awareness in alexithymia may be associated with decreased ACC function [39,54]. Apart from the alexithymia literature, the important role of the ACC in emotion processing is also well-established [37,38]. It is important to notice, though, that the ACC is also an area involved in a wide range of other functions including conflict monitoring [55], self-regulation [56], and decision-making [57]. Nevertheless,

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our result of decreased ACC function in HDA subjects confirms its important role in reduced emotional awareness associated with alexithymia. Additionally, studies on emotion processing in alexithymia are in line with our results, showing ACC hypofunction [28–30]. In contrast to our result, though, a recent meta-analysis [27] reported *increased* ACC activity in alexithymia. From a methodological perspective, it has to be noted, that the majority of studies included in the meta-analysis only implemented a reduced set of emotional facial stimuli (up to three) and had no instruction to explicitly recognize the emotion shown (e.g. masked presentation of faces). Those studies are hence difficult to compare with our approach. An alternative explanation is mentioned in the meta-analysis itself. Some authors suggest that differences in task paradigms may account, among other aspects, for the difference between ACC hyper- and hypofunction [58,59]. Interestingly, most of the studies that found ACC hypofunction applied tasks that called for cognitive processing of emotional stimuli (as opposed to passive viewing) [28–30]. Our task of explicit emotion recognition is conceptually similar to those paradigms.

Extracting emotional content from faces is the second function supposedly impaired in alexithymia. This function is associated with the extended system of face recognition [40,41] that includes amygdala, insula and striatum. In our study, ROI analyses in those areas showed relatively less activity in HDA subjects and provide a potential neural correlate of their impaired ability to extract emotion from faces as exhibited in the lower FEEL scores. Apart from their involvement in the extended system, each of those regions has previously been implicated in important aspects of emotion processing [60]. The amygdala, for instance, has been described as a threat-indicator [61] or on a broader level as an indicator of salience and biological relevance [62–64]. Interestingly, considering 100 studies investigating facial emotion recognition, the amygdala is the area of greatest overlap [44]. The insula is associated with the representation of internal bodily responses to enable subjective feelings [65–71]. Bridging the insula's general function with alexithymia, Silani et al. [72] presented evidence for a correlation between the activation of the anterior insula during an interoceptive task and the level to which study participants were aware and understood their own emotions (which was lower in alexithymia). Lesion studies confirm this by showing reduced recognition of emotions (specifically disgust) after injuries to the insular cortex [73,74]. The caudate (as being part of the striatum) was associated with the recognition of emotions already in early studies [75,76]. More specifically, functional impairments in the caudate of the fronto-striatal circuitry have been implied in the pathophysiology of alexithymia [77].

The large cluster in the middle temporal gyrus (whole-brain) closely matches the posterior part of the superior temporal sulcus (pSTS, see, e.g. Fig. 3g). Hence, enhanced activity in this area in LDA subjects could be interpreted within the framework of *social perception*. Amongst other functions, the STS operates as a higher order visual area relevant for biological motion and social perception [78,79], a function that is in theory likely to show some variability in individuals with alexithymic features. The ROI in the middle temporal gyrus was defined following coordinates for face-specific emotion perception provided by a meta-analysis [44] and is hence in line with those results. Interestingly, the STS was reported to be hypoactive in many studies investigating patients with autism spectrum disorders (overview in [80]), sharing some characteristics with alexithymic individuals.

The next three areas, thalamus, parahippocampal gyrus, and middle frontal gyrus have also shown the hypothesized hypofunction in HDA individuals. The thalamus has been implied in emotion recognition before (e.g. [43]) and was reported in the meta-analysis of emotion activation studies of Phan et al. [81]. Given its description as a "sensory gateway", its role in emotion processing is plausible. Parahippocampal gyrus, and middle frontal gyrus have been reported in Sabatinelli's meta-analysis [44] indicating the involvement of those structures in the perception of facially expressed emotions. To our knowledge, though, no specific theory or explanation exists as to why those structures are a specific part of emotion processing.

Finally, hypoactivation of the inferior frontal cortex (IFC) in HDA subjects merits some further discussion. Apart from the ROI analysis, there was also a large cluster in the IFC apparent in the whole-brain analysis. In Sabatinelli's meta-analysis [44], the IFC is among the areas with most overlap for studies of emotion perception in general. More specifically, many studies found parts of the IFC to be active in facial emotion recognition (e.g. [42]). Interestingly, more recent studies interpret this activity within a *mirror neuron system* (MNS) framework [82–84]. Since the existence of a human mirror neuron system is still not established firmly and we have no further data to back our interpretation, the following assumptions are speculative in nature. According to the theory of human mirror neurons, when we watch and imitate motor behaviour the frontal MNS broadly located in the ICF is involved (higher-order goals or concepts associated with the motor action) [85]. According to the "simulation theory" of empathic behaviour we recognize others' emotions better when we "simulate" the observed emotion internally making use of the MNS (e.g. [86]). More specifically, in the case of emotional facial expressions, a similar mechanism has been proposed long ago in the "facial-feedback" hypothesis (e.g. [87]): Perception of our own facial expression actually makes us "feel" the emotion expressed. In various studies, it has been shown that we automatically imitate observed facial expressions in subtle ways, which sometimes leads to emotional contagion [88,89]. In this vein, we speculate that higher activity in the frontal MNS (IFC) in LDA subjects could be a possible correlate of their enhanced use of the MNS in order to "simulate" the facial emotions seen in our experiment for the sake of better performance. In future studies, subjective ratings of recognition techniques and facial EMG to measure mimicry should be included to test this speculative interpretation.

The only region with relatively more activity in HDA subjects was the left superior parietal lobule detected in the whole-brain analysis. Our interpretation of this surprising result follows the above mentioned speculation considering the involvement of the MNS. The superior parietal lobule that is part of the parietal MNS, being primarily concerned with detailed aspects of the motor action itself. One has to consider that, although scoring significantly lower than LDA subjects, subjects with alexithymic features still recognized about 85% of the expressed emotions. We hence interpret enhanced activity in the parietal MNS as a compensatory mechanism: If HDA subjects lack the ability to "simulate" the emotion seen as a whole (reduced frontal MNS activity in the IFC, see above), they may rely more on the detection of local facial features (enhanced parietal MNS activity) to identify the emotion. In accordance with its role in the parietal MNS, the superior parietal lobule is also involved in the direction of visual attention and visuospatial processing [90–93]. For patients with autism, who share to some degree deficits in emotion recognition with alexithymics, the superior parietal lobule has already been discussed as part of a compensatory network [94]. Compared to healthy controls, autistic individuals use different strategies for visual processing when they have to analyze faces relying on local modes of information processing. In line with this interpretation, autistic subjects showed relatively higher activity in the superior parietal lobule after a successful facial affect recognition training supporting the compensatory function of this area [95].

Interestingly, we found significant differences in behaviour and BOLD responses comparing two healthy groups with the main difference being their relative degree of alexithymia. Hence, our

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results indirectly support recent conceptualizations and epidemiological data, that alexithymia is a dimensional personality trait apparent in clinically healthy subjects rather than a categorical diagnosis only applicable to clinical populations [2]. With no psychiatric diagnoses acting as confound in our sample and the HDA group closely matching the suggested 66th percentile of "alexithymia", the ecological validity of our study can be considered as relatively high.

4.3. Limitations

The major limitation of our study is the distribution of alexithymia scores among subjects. The subjects we term "high degree of alexithymia" are mostly within a range that is considered as "possible alexithymia" (52–60) within original TAS standards. Hence, our results cannot be easily generalized for alexithymic subjects in a strict sense (TAS > 60). Our approach, though, was guided by the concept of alexithymia as a continuous variable within the general population [2,96,97]. Following this, the differences between HDA (54.8) and LDA subjects' (30.7) mean TAS scores are sufficiently high to assume two distinct groups along the continuum of alexithymia.

The second limitation considers the sex distribution of our sample. With relatively more female subjects in the LDA group, differences could theoretically be attributed to sex rather than alexithymic features per se. An extensive meta-analysis [44] did not report sex differences considering brain activation in emotion perception, though, rendering this attribution less likely. To better rule this out, future studies should weight the sex distribution equally within subject groups.

Presenting six emotions leads to another methodological issue. With only few faces shown per emotion (due to the limited amount of different faces and repetition being critical in a recognition test) we cannot make inferences about participants' recognition of a single emotion but rather have to remain within the broad concept of "emotion recognition" as a whole.

Finally, there exist methodological issues with the TAS itself. Although it is the most widely used instrument to assess alexithymia and its validity has been established [5], it is seriously questionable to what extent a self-report measure can reflect difficulties in the identification of emotions [6,88–99,34]. Another main point of criticism, the correlation between TAS and scores of negative affect (e.g. depression) in general often apparent in studies, was ruled out in our study, though.

5. Conclusions

Our study provides evidence that healthy subjects with a relatively high degree of alexithymia perform significantly worse in a standardized test of facially expressed emotion recognition, and, while doing so, have relatively less activity in a multitude of brain areas that cover important aspects of emotion processing: the ACC as a structure involved in emotional awareness, the amygdala, insula and striatum as part of the extended system of face perception concerned with extracting meaning from faces (e.g. emotions). Additionally, enhanced activation of the IFC in healthy individuals with a relatively low degree of alexithymia could reflect an elevated involvement of the frontal MNS, possibly to "simulate" the emotion seen on a higher order level to increase recognition. On the other side, enhanced activation of the superior parietal lobule in subjects relatively high in alexithymia could reflect an involvement of the parietal MNS as a compensatory mechanism (focus on local facial features). The later speculations should be tested in future studies applying facial EMG and self-ratings of recognition techniques.

Conflict of interest

All authors declare that there are no conflicts of interest regarding this study.

Uncited reference

[31].

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.bbr.2014.05.069.

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