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Unimpaired discrimination of fearful prosody after amygdala lesion ☆

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ABSTRACT

Prosody (i.e. speech melody) is an important cue to infer an interlocutor's emotional state, complementing information from face expression and body posture. Inferring fear from face expression is reported as impaired after amygdala lesions. It remains unclear whether this deficit is specific to face expression, or is a more global fear recognition deficit. Here, we report data from two twins with bilateral amygdala lesions due to Urbach-Wiethe syndrome and show they are unimpaired in a multinomial emotional prosody classification task. In a two-alternative forced choice task, they demonstrate increased ability to discriminate fearful and neutral prosody, the opposite of what would be expected under an hypothesis of a global role for the amygdala in fear recognition. Hence, we provide evidence that the amygdala is not required for recognition of fearful prosody.

1. Introduction

The perception of a conspecific's emotional state is an important aspect of social communication. In humans this ability relies heavily on non-verbal signals such as facial expression (Ekman & Oster, 1979), emotional speech melody (i.e., prosody) (Banse & Scherer, 1996), and bodily posture (Dael, Mortillaro, & Scherer, 2012).

Extraction of emotional state from a conspecific's facial expression is widely reported to involve the amygdala (Adolphs et al. 1999). Numerous neuroimaging studies have demonstrated amygdala responses to emotional and in particular to fearful expression (Breiter et al. 1996; Fischer et al. 2003; Morris et al. 1996; Whalen et al. 2004; Whalen et al. 1998). Successful identification of fearful facial expression is reported to be impaired following amygdala lesions (Adolphs et al., 1999). This observation could reflect a specific deficit for extraction of emotional meaning from faces, in line with an hypothesised function of the amygdala in face processing, encompassing, but extending beyond, emotional meaning (Atkinson & Adolphs, 2011). On the other hand it is possible that a function of the amygdala includes extraction of information about a conspecific's emotional state, independent of its source.

Here, we capitalised on another source of emotional information, emotional prosody (i. e. speech melody), and investigated whether its identification was impaired in two patients with amygdala lesions. As yet, the role of the amygdala for extraction of emotional meaning from prosody is unclear. Some functional magnetic resonance imaging studies have reported amygdala responses to emotional prosody (Bach et al., 2008; Dolan, Morris, & de Gelder, 2001; Ethofer et al. 2009; Fruhholz, Ceravolo, & Grandjean, 2012; Fruhholz & Grandjean, 2013; Grandjean et al. 2005; Mothes-Lasch, Mentzel, Miltner, & Straube, 2011; Wiethoff, Wildgruber, Grodd, & Ethofer, 2009), but not to fearful voices in particular. Most lesion studies report cases with either unselective, or incomplete, amygdala damage. Impaired fear prosody recognition has been observed in patients with unselective bilateral (Adolphs & Tranel, 1999; Brierley, Medford, Shaw, & David, 2004; Sprengelmeyer et al. 1999) and unilateral (Brierley et al., 2004; Dellacherie, Hasboun, Baulac, Belin, & Samson, 2011) temporal lobe damage, and in one patient with selective, but incomplete, bilateral amygdala resection (Scott et al., 1997). On the other hand, unimpaired fear prosody recognition has been reported in cases with unselective unilateral temporal lobe lesions (Adolphs & Tranel, 1999; Adolphs, Tranel, & Damasio, 2001) or unilateral selective amygdala combined with contralateral extended temporal lobe lesion (Anderson & Phelps, 1998). Furthermore, a large 3D lesion mapping study has shown no clear contribution of medial temporal cortex to prosody recognition (Adolphs, Damasio, & Tranel, 2002), although this might be biased by sampling of lesions. In summary, both the impairments and the heterogeneity of results could reflect lesions to temporal lobe structures outside the amygdala which were differentially affected

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in the different samples, due to their underlying aetiology (surgical lesions, hippocampal sclerosis, paraneoplastic encephalitis, stroke, and others). Hence, a case of bilateral selective amygdala lesion (SM) showing no impairment in emotional prosody identification might be taken as the most specific finding to date (reported together with other cases in (Adolphs & Tranel, 1999)).

However the small sample sizes studied necessarily entails low power, Further, all studies to date have relied on accuracy measures, i.e. hit rates in a multinomial classification task. This is a common approach in emotion recognition studies which has long been criticised due to a lack of control for false alarms (Wagner, 1993). In an extreme example, a person indiscriminately labelling all stimuli as "angry" will appear impaired in all other emotions, but not in the "angry" category. Or a person with reduced sensitivity to distinguish fearful expression, but with increased bias to label any expression as fearful, might not show any impairment because the preponderance of false alarms, evenly distributed across all other emotion categories, might not exceed the noise level in the control population.

Hence, we sought to extend previous findings reported on patient SM (Adolphs & Tranel, 1999) in three ways: first by examining two further patients with focal amygdala lesions due to congenital Urbach–Wiethe disease; second by using a more powerful and precise metric for prosody identification, namely by means of a two-alternative forced choice task which allows for independent analysis of sensitivity and bias (or criterion) as prescribed by signal detection theory. Finally, because impairments might not be detected due to floor or ceiling effects when normal performance is very low (as for fear in Adolphs and Tranel (1999)) or very high (as for anger in Adolphs and Tranel (1999)), we used a validated stimulus set comprising low and high intensity of emotional expression.

2. Methods

2.1. Design

Task 1 was a multinomial emotion identification task, for comparison with the previous literature, previously validated on a large clinical sample (Bach, Buxtorf, Grandjean, & Strik, 2009). A subset of the stimuli (angry, fearful, and neutral) was used for the 2-alternative forced choice (2AFC) task 2. Task 1 followed a nested 6 (emotional category: anger, fear, disgust, surprise, happiness, neutral) × 2 (emotion intensity: low, high) × 2 (group) factorial design. Due to the construction of the initial stimulus set, stimuli for disgust and neutral were not intensity-graded. Task 2 followed a completely crossed 2 (emotions pair: neutral-fearful, neutral-angry) × 2 (emotion intensity: low, high) × 2 (group) factorial design.

2.2. Participants

AM (previously also labelled patient 1) and BG (patient 2) (Becker et al., 2012) are monozygous female twins diagnosed at the age of 12 with congenital Urbach–Wiethe disease (lipoid proteinosis) due to a *de novo* mutation (Becker et al., 2012). This disorder in some cases leads to specific calcification of the amygdala that is thought to encroach on this structure gradually over the course of childhood and adolescence (Newton, Rosenberg, Lampert, & O'Brien, 1971). Despite these lesions, both twins exhibit only minor deficits in a standard neuropsychological test battery (Talmi, Hurlemann, Patin, & Dolan, 2010). At the time this research was conducted, they were 35 years old. The calcified volumes on high-resolution computer assisted tomography images include the whole basolateral amygdala and most other amygdala nuclei, only sparing anterior amygdaloid and ventral cortical amygdaloid parts at an anterior level, as well as lateral and medial parts of the central amygdaloid nucleus and the amygdalo-hippocampal area at posterior levels.

For experiment 1, we compared the patients against a control group acquired in the context of a different study (Bach et al., 2009); comprising 25 healthy participants (13 male, 12 female) with an age (mean ± standard deviation) of 35.4 ± 13.1 years. For experiment 2, we collected a sample more closely matched to the patients; these were 16 healthy females with an age of 33.6 ± 3.4 years.

2.3. Stimuli

Task 1: Stimuli were taken from a validated set of Banse & Scherer (1996). The original work was concerned with acoustic profiles in vocal emotion expression that addressed the emotions fear, sadness, anger, disgust, neutral affect, and happiness. In the original set, 12 professional actors vocalised the emotions. There were two sentences for each emotion and intensity level, and each sentence was vocalised twice in two different eliciting scenarios. From the whole set, items were selected on the basis of expert ratings by an independent group of 12 actors. Those items were then included in a recognition study with naive participants. In the recognition study, stimuli were also included from actors who did not perform well on all emotions. To minimise variance caused by low-level acoustic features, we used only stimuli from the two actors (one male, one female) who performed the whole set of emotions. Therefore, the stimulus set used in the present study comprised only a part of the original set. Nine additional stimuli vocalised by a different actor were used as practice items for experiment 1. Hence, there were eight items for each intensity level of intensity-graded emotions, for two actors, two sentences, and two scenarios. For neutral and disgust, there were two different items from each actor/sentence/scenario combination, adding up to 16 items, to keep the total number of items per emotion category constant. The sentences were 'Hat sundig pron you venzy' and 'Fee gott laish jonkill gosterr '. These meaningless sentences comprise phonemes from several Indo-European languages and resemble normal speech. According to the validation study, 'listeners generally have the impression of listening to an unknown foreign language' (Banse & Scherer, 1996). Thus, experiment 1 used 96 stimuli expressing fear, sadness, anger, disgust, neutral affect, and happiness. Only stimuli for fear, sadness, anger, and happiness were graded in two intensity categories. Hence, a first analysis was performed on all six emotion categories while not accounting for intensity, and a second analysis on the four intensity-graded emotion categories.

Task 2: Stimuli for the second task were the subset of 16 angry, 16 fearful, and 16 neutral items from task 1.

2.4. Apparatus and procedure

Task 1: All stimuli were played on a standard PC, using eprime software (Psychology Software Tools, Pittsburgh PA, USA). Listeners could adjust the loudness ad libitum. Each stimulus was about 2 s in length. Stimuli were presented in randomized order. Participants responded by selecting the appropriate emotion category with a computer mouse. They had as much time to respond as they needed, but the presentation could not be repeated.

Task 2: Stimuli were played on a standard PC, using Matlab software (MathWorks, Natick MA, USA), with the Cogent toolbox (www.vislab.ucl.ac.uk). Each stimulus was presented once in each of two response contexts for 2 s in randomized order. Afterwards, participants were required to choose from a pair of emotions (fearful-neutral, angry-neutral, fearful-angry). Angry/fearful pairs were included in order to not bias the selection of the neutral response as a default response, without specific hypotheses. These were not included in the main analysis. Exploratory inclusion into the analysis of sensitivity did not result in any additional effects involving group, and there were no significant effects involving group in an intensity × group ANOVA of sensitivity only involving these pairs.

2.5. General procedure

Because patients performed both tasks one after the other, whereas control participants received only one of the tasks, we balanced task order in the patients to control for training effects. BG received first task 1, then task 2; AM received first task 2, then task 1.

2.6. Statistical analysis

Data extraction was implemented using R and Matlab. In task 1, we computed a measure of accuracy as hit rate for each emotion category. For task 2, we computed a measure of sensitivity as $d' = Z(\text{hit rate}) - Z(\text{false alarm rate})$, and a measure of the response criterion, as $c = .5 \times (Z(\text{hit rate}) + Z(\text{false alarm rate}))$ where Z is the quantile function of the standard normal distribution. Preliminary statistical analysis to localise effects was implemented in SPSS 20, using repeated-measures ANOVA in the General Linear Model routine, assuming equal variance. For interaction effects involving group, this approach might inflate type I error if variance in the control population is unequal between cells (Crawford, Garthwaite, & Howell, 2009); hence significant results were confirmed on a single case level in a Bayesian approach using Crawford's single case tests (Crawford & Garthwaite, 2007) as implemented in the authors' program `dissocsbayes.exe`. Non-significant results in the ANOVA approach do not require confirmation.