



Review

Neural correlates of alexithymia: A meta-analysis of emotion processing studies



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ABSTRACT

Alexithymia is a personality trait characterized by difficulties in the experience and cognitive processing of emotions. It is considered a risk factor for a range of psychiatric and neurological disorders. Functional neuroimaging studies investigating the neural correlates of alexithymia have reported inconsistent results. To integrate previous findings, we conducted a parametric coordinate-based meta-analysis including fifteen neuroimaging studies on emotion processing in alexithymia. During the processing of negative emotional stimuli, alexithymia was associated with a diminished response of the amygdala, suggesting decreased attention to such stimuli. Negative stimuli additionally elicited decreased activation in supplementary motor and premotor brain areas and in the dorsomedial prefrontal cortex, possibly underlying poor empathic abilities and difficulties in emotion regulation associated with alexithymia. Positive stimuli elicited decreased activation in the right insula and precuneus, suggesting reduced emotional awareness in alexithymia regarding positive affect. Independent of valence, higher (presumably compensatory) activation was found in the dorsal anterior cingulate possibly indicating increased cognitive demand. These results suggest valence-specific as well as valence-independent effects of alexithymia on the neural processing of emotions.

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1. Introduction

Alexithymia (“no words for feelings”) is a personality trait characterized by difficulties in identifying, analyzing and verbalizing feelings, restricted imaginal capacities and limited emotional experience (Sifneos, 1973; Vorst and Bermond, 2001). Its prevalence rate lies around 10% in the general population (Salminen et al., 1999). In the past, there has been some debate on whether alexithymia should be conceptualized as a distinct clinical type or as a dimensional personality construct. However, recent studies provided strong support in favor of alexithymia as a dimensional personality construct (Mattila et al., 2010; Parker et al., 2008). Alexithymia is considered to be a risk factor for various psychiatric and psychosomatic disorders, including substance abuse, depression and schizophrenia (Taylor et al., 1997; van’t Wout et al., 2007). Furthermore, individuals with alexithymia report lower life satisfaction (Mattila et al., 2007) and are more likely to commit suicide (Hintikka et al., 2004). Therefore, it is of great clinical importance to gain more insight in the neural basis underlying this personality trait.

Difficulties in emotion processing are at the core of alexithymia. Both the ability to experience and cognitively process emotions is reduced. For example, individuals with alexithymia show impaired performance in remembering emotional words (Luminet et al., 2006), problems during the identification of facial expressions (Grynbeg et al., 2012; Parker et al., 1993) and impaired higher order mentalizing (Swart et al., 2009). Hence, theories on neural correlates of alexithymia mainly focus on brain areas involved in emotion processing. One theory states that alexithymia might be associated with a right hemisphere deficit or a left hemisphere preference (Bermond et al., 2005; Buchanan et al., 1980) because the right hemisphere plays an important role in the perception and regulation of emotional behavior (Adolphs et al., 2000).

Lane et al. (1997) hypothesized a central role for the anterior cingulate cortex (ACC) in alexithymia. According to their ‘blindfeel’ hypothesis, the conscious experience of emotion is compromised in individuals with alexithymia, assumed to result from a dysfunction in the ACC (Lane et al., 1997). In addition to the ACC, the insula is another relevant brain region in generating emotional experience. This structure receives information from internal bodily states and integrates these into a subjective feeling state (Craig, 2009). Furthermore, subcortical areas, such as the amygdala and striatum, are proposed to underlie emotion processing difficulties in alexithymia because of their role in the detection of emotional significance and the generation of emotional feelings (Bermond et al., 2006; Goerlich et al., 2013; Kano and Fukudo, 2013; Larsen et al., 2003; Moriguchi and Komaki, 2013; Taylor and Bagby, 2004; Wingbermühle et al., 2012). Thus, alexithymia-related difficulties in perceiving and experiencing emotions may be associated with dysfunction of the ACC, insula, amygdala and striatum. According to two recent reviews, this decrease in limbic and paralimbic activation is associated with a decrease in prefrontal activation when individuals with high alexithymia scores are presented with external emotional stimuli (Kano and Fukudo, 2013; Moriguchi and Komaki, 2013). Especially, altered activation in the orbitofrontal cortex and medial prefrontal cortex is proposed to underlie alexithymia (Bermond et al., 2006; Larsen et al., 2003; Wingbermühle et al., 2012) because of their involvement

in the cognitive control of emotions, including emotion regulation (Ochsner and Gross, 2005) and emotional decision making (Rogers et al., 2004). In fact, lesions in these regions have been shown to result in restricted capacities for the cognitive processing of emotions (Glascher et al., 2012). Difficulties in the restricted imaginal capacities in alexithymia, on the other hand, are thought to be related to reduced activation in the posterior cingulate cortex (Aleman, 2005; Bermond et al., 2006; Kano and Fukudo, 2013; Larsen et al., 2003; Moriguchi and Komaki, 2013; Wingbermühle et al., 2012), because of its role in emotional memory (Maddock, 1999) and the imagination of future events (D’Argembeau et al., 2008).

To date, neuroimaging studies have tried to identify these proposed brain regions as neural correlates of alexithymia. In 2010, Pougă and colleagues compared alexithymia-related brain activation in the medial frontal gyrus, cingulate gyrus and amygdala across studies. They identified lower activation in the medial frontal gyrus and the amygdala in alexithymia, while results on the cingulate gyrus were mixed (i.e. both higher and lower activation was associated with alexithymia). Furthermore, results regarding other brain areas in alexithymia are also inconsistent across studies. Most existing reviews on the neural correlates of alexithymia highlight the variability in findings across studies but do not integrate these findings (Aleman, 2005; Bermond et al., 2006; Larsen et al., 2003; Taylor and Bagby, 2004; Wingbermühle et al., 2012). Furthermore, only a selected group of brain regions are mentioned in these reviews, leaving other possible neural correlates unremarked.

The aim of the present study was to integrate findings from the literature and identify brain regions underlying emotion processing difficulties in alexithymia across studies. Therefore, studies examining the neural correlates of alexithymia during the processing of either positive or negative emotional stimuli were included in this meta-analysis. Positive and negative emotional processing presumably differ in their neural correlates (Wager et al., 2003). Furthermore, previous studies have indicated that there appears to be a valence-specific effect on the neural correlates of emotion processing in alexithymia (Berthoz et al., 2002; Kano et al., 2003; Pollatos and Gramann, 2011; Reker et al., 2010). For example, studies investigating alexithymia reported decreased activity in the amygdala for negative, but not for positive stimuli (Kugel et al., 2008; Reker et al., 2010). Moreover, Berthoz et al. (2002) reported decreased activation in the ACC for negative stimuli while activation in this area was increased for positive stimuli in alexithymia. Besides these indications of possible differences between the neural correlates of positive and negative emotion processing in alexithymia, a recent meta-analysis indicated that emotional valence modulates neural abnormalities in depression (Groenewold et al., 2012). Given the fact that alexithymia is significantly related to depression (Honkalampi et al., 2000), one could hypothesize that valence might also modulate alexithymia-related brain activation. Therefore, brain activation associated with alexithymia was examined for the processing of negative and positive stimuli separately. A novel parametric coordinate-based meta-analysis (PCM) approach (Costafreda, 2012) was employed as this method allowed for the inclusion of both whole brain and region of interest (ROI) studies as well as for the inclusion of different thresholds across studies.

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