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Is the emotion recognition deficit associated with frontotemporal dementia caused by selective inattention to diagnostic facial features?☆

Lindsay D. Oliver[d,e], Karim Virani[d], Elizabeth C. Finger[c,d], Derek G.V. Mitchell[a,b,c,d,*]

[a] Departments of Psychiatry, University of Western Ontario, London, ON, Canada
[b] Anatomy & Cell Biology, University of Western Ontario, London, ON, Canada
[c] Clinical Neurological Sciences, University of Western Ontario, London, ON, Canada
[d] Graduate Program in Neuroscience, University of Western Ontario, London, ON, Canada
[e] Schulich School of Medicine & Dentistry, Department of Psychology, Brain and Mind Institute, University of Western Ontario, London, ON, Canada

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ABSTRACT

Frontotemporal dementia (FTD) is a debilitating neurodegenerative disorder characterized by severely impaired social and emotional behaviour, including emotion recognition deficits. Though fear recognition impairments seen in particular neurological and developmental disorders can be ameliorated by reallocating attention to critical facial features, the possibility that similar benefits can be conferred to patients with FTD has yet to be explored. In the current study, we examined the impact of presenting distinct regions of the face (whole face, eyes-only, and eyes-removed) on the ability to recognize expressions of anger, fear, disgust, and happiness in 24 patients with FTD and 24 healthy controls. A recognition deficit was demonstrated across emotions by patients with FTD relative to controls. Crucially, removal of diagnostic facial features resulted in an appropriate decline in performance for both groups; furthermore, patients with FTD demonstrated a lack of disproportionate improvement in emotion recognition accuracy as a result of isolating critical facial features relative to controls. Thus, unlike some neurological and developmental disorders featuring amygdala dysfunction, the emotion recognition deficit observed in FTD is not likely driven by selective inattention to critical facial features. Patients with FTD also mislabelled negative facial expressions as happy more often than controls, providing further evidence for abnormalities in the representation of positive affect in FTD. This work suggests that the emotional expression recognition deficit associated with FTD is unlikely to be rectified by adjusting selective attention to diagnostic features, as has proven useful in other select disorders.

1. Introduction

Frontotemporal dementia (FTD) is a devastating progressive neurodegenerative disorder characterized by atrophy of the frontal and temporal lobes, for which there is currently no cure. Perhaps the most debilitating early symptoms of FTD and most troubling to caregivers are the profound decline in social and emotional behaviour (Diehl-Schmid et al., 2013; Levenson & Miller, 2007; Neary, Snowden, & Mann, 2005). One of the key features of the behavioural symptoms of FTD is impaired recognition of emotional expressions (Kumfor & Piguet, 2012). Emotion recognition capabilities are related to both empathy and social functioning. For example, emotion recognition accuracy is associated with prosocial behaviour (Marsh, Kozak, & Ambady, 2007), and measures of trait empathy are positively correlated with activation in emotion-related neural regions while viewing emotional faces (Jabbi, Swart, & Keysers, 2007). Thus, neurocognitive abnormalities related to impaired emotion recognition are a potential key treatment target for social dysfunction and empathy deficits in FTD. Though there are different anatomical variants of FTD, emotion recognition impairments have been demonstrated in both patients with frontal and temporal variants of the disorder (Keane, Calder, Hodges, & Young, 2002; Rosen et al., 2004, 2002b). FTD is also broken up into behavioural variant and semantic dementia, which largely map onto frontal and temporal variants respectively. However, in the present paper, we have elected to use the anatomical designations given that temporal and frontal lesions are often

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* Corresponding author at: Departments of Psychiatry, University of Western Ontario, London, ON, Canada. Tel.: +1 519 661 2111x84862.
E-mail address: dmitch8@uwo.ca (D.G.V. Mitchell).

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associated with distinct facial expression recognition profiles (e.g., see Blair, 2003). Numerous studies report a generalized emotion recognition deficit in patients with FTD, including recognition difficulties with both positive and negative emotional expressions, while recognition of non-emotional features, such as gender, is preserved (Couto et al., 2013; Keane et al., 2002; Snowden et al., 2008). However, the degree to which the impairment extends to happiness is unclear, with several studies reporting normal happy expression recognition (Fernandez-Duque & Black, 2005; Kipps, Mioshi, & Hodges, 2009a; Kumfor et al., 2011; Lavenu, Pasquier, Lebert, Petit, & Van der Linden, 1999; Lough et al., 2006; Rosen et al., 2002b).

The facial features that are most diagnostic for emotion recognition vary by emotion. Specifically, whereas the eyes convey the most important cues for recognizing fearful and angry faces (Adolphs et al., 2005), the lower half of the face provides the most significant diagnostic information for disgusted and happy expressions (Boucher & Ekman, 1975; Calder, Young, Keane, & Dean, 2000; Smith, Cottrell, Gosselin, & Schyns, 2005). It has recently been shown that fear recognition deficits in some patient populations can be remedied by manipulating attention to diagnostic facial cues. For example, patients with focal amygdala damage exhibit impaired recognition of emotional expressions, particularly for fear (Adolphs, Tranel, Damasio, & Damasio, 1994; Adolphs et al., 1999). This deficit has been shown to be associated with a failure to utilize critical information from the eye region of fearful faces in S.M., a patient with bilateral amygdala lesions, and can be ameliorated by instructing her to focus on the eyes (Adolphs et al., 2005). Similarly, youth with high callous-unemotional psychopathic traits suffer from a selective fear recognition deficit (Blair et al., 2004; Stevens, Charman, & Blair, 2001), and also show reduced attention to the eye region of fearful expressions (Dadds, El Masry, Wimalaweera, & Guastella, 2008). Crucially, instructing these youth to attend to the eye region also reverses their fear recognition deficit (Dadds et al., 2006). Such findings in populations with neurological and developmental disorders implicating the amygdala raise the question of whether enhancing the processing of critical facial features will abate the emotion recognition deficit associated with FTD. It is important to note, however, that patients with FTD exhibit a more generalized emotion recognition deficit, featuring impairments for fearful, sad, disgusted, angry, and sometimes happy facial expressions. Furthermore, this impairment has been associated with the degree of atrophy in not only the amygdala, but also the orbitofrontal cortex (OFC) and insula (Couto et al., 2013; Kipps et al., 2009a; Omar, Rohrer, Hailstone, & Warren, 2011; Rosen et al., 2002b). Thus, the FTD-related expression recognition deficit, and its remedy, may differ from that of neurological disorders featuring focal amygdala damage or developmental disorders affecting empathy.

In the current study, we examined the impact of isolating distinct regions of the face (i.e., the eyes versus the remaining facial features) on the ability to recognize expressions of anger, fear, disgust, and happiness in a sample of individuals with FTD. Notably, this paradigm resembles one of the experiments used to delineate the emotion recognition deficit in S.M. (Adolphs et al., 2005). Based on the existing literature, we predicted that, relative to a matched control group, individuals with FTD would show a deficit in recognizing both positive and negative emotional expressions. We also anticipated that healthy adults would demonstrate a relative decrease in emotion recognition accuracy for angry and fearful expressions with the eye region occluded, and for eyes-only presentations of disgusted and happy expressions, due to the lack of respective critical identification features being present. The task also allowed us to test two alternate predictions concerning the impact of manipulating exposure to diagnostic facial features on the recognition of fear. One possibility is that, similar to patients with amygdala damage and high psychopathic traits (Adolphs et al., 2005; Dadds et al., 2008), patients with FTD fail to utilize information in the eye region of faces during attempts to recognize emotional expressions. If this is the case, then like S.M., patients with FTD should show limited recognition performance costs when the eyes of fearful faces have been erased. In addition, they may also show a disproportionate advantage in terms of accuracy when provided with fearful eyes-only stimuli. Of course, patients with FTD show deficits for other emotions. The current design allowed us to determine whether manipulating the most diagnostic features of these other emotions (the eyes for anger and the mouth for disgust and happiness) would have similar effects. Alternatively, if the facial emotion recognition impairment is more general (i.e., not driven by selective inattention to critical facial features), we reasoned that patients with FTD would show impaired emotional expression recognition irrespective of which facial features were available. Crucially, we were not examining general attention; instead, we were interested in whether reduced processing of specific critical facial features of emotional expressions underlies the emotion recognition deficit characteristic of patients with bvFTD. This study was implemented to test these predictions in order to gain insight into potential treatment and compensatory options for targeting some of the socioemotional impairments associated with FTD.

2. Materials and methods

2.1. Participants

Forty-eight participants took part in this study, including 24 patients with FTD (11 male, 13 female) and 24 healthy volunteers (10 male, 14 female). Participants in the FTD group included 18 patients who met the revised consensus diagnostic criteria for probable behavioural variant FTD (Rascovsky et al., 2011) and 6 patients who met the Neary et al. (1998) diagnostic criteria for semantic dementia. All patients had magnetic resonance imaging (MRI; $N=17$) and/or computed tomography (CT; $N=14$) scans consistent with the diagnoses, as well as single-photon emission computed tomography (SPECT) in some cases ($N=9$; i.e., no diagnoses were made solely on the basis of SPECT). Based on the pattern of atrophy and perfusion evident in the clinical anatomical scans, a trained behavioural neurologist (ECF) classified 16 patients as frontal variant FTD and 8 patients as temporal variant FTD. Patients were classified as frontal variant FTD if they presented with predominantly frontal atrophy, whereas patients were classified as temporal variant FTD if they showed predominantly temporal atrophy. Patients were divided into groups based on anatomic criteria, because emotion recognition deficits have been demonstrated in both behavioural variant FTD and semantic dementia, but appear to largely follow the pattern of atrophy in patients with FTD (Kumfor, Irish, Hodges, & Piguet, 2013; Kumfor & Piguet, 2012; Rosen et al., 2004). As would be expected, none of the patients with frontal variant dementia showed evidence of semantic dementia. All but two of the patients with temporal variant FTD were diagnosed with semantic dementia. These two patients had behavioural variant FTD with predominantly right-sided temporal atrophy. To ensure that the inclusion of these participants did not alter our results, accuracy analyses were performed with these two patients excluded. Significant effects did not differ from the whole group analysis.

Participant demographic and neuropsychological characteristics are presented in Table 1. Chi-square analyses unveiled no significant differences in sex or handedness between FTD and control groups. Independent *t*-tests also revealed that FTD and control groups did not differ significantly in age at testing or years of education. However, patients with FTD performed significantly worse than controls on the Mini-Mental State Examination (MMSE). Within the FTD group, patients with frontal versus temporal variant FTD did not differ significantly in sex, handedness, age at testing, years of education, or disease duration. Additionally, the FTD subgroups did not differ significantly in performance on administered neuropsychological tests, aside from the temporal variant patients performing significantly worse than the frontal variant patients on the Naming subscale of the Western Aphasia Battery (WAB). See Table 1 for statistical details.

Patients with FTD were recruited through the Cognitive Neurology and Alzheimer Research Centre at Parkwood Hospital in London, Ontario, Canada. Age-matched control participants were recruited through volunteer databases of the centre, and through advertisements to caregivers at local FTD family support groups. All participants provided written informed consent. This study was approved by the Health Sciences Research Ethics Board at the University of Western Ontario, London, Ontario, Canada.

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