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Recognition of disgust is selectively preserved in Alzheimer's disease

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Abstract

The neural substrates that subserve decoding of different emotional expressions are subject to different rates of degeneration and atrophy in Alzheimer's disease (AD), and there is therefore reason to anticipate that a differentiated profile of affect recognition impairment may emerge. However, it remains unclear whether AD differentially affects the recognition of specific emotions. Further, there is only limited research focused on whether affect recognition deficits in AD generalize to more ecologically valid stimuli. In the present study, relatively mild AD participants ($n = 24$), older controls ($n = 30$) and younger controls ($n = 30$) were administered measures of affect recognition. Significant AD deficits were observed relative to both the younger and older control groups on a measure that involved labeling of static images of facial affect. AD deficits on this measure were observed in relation to all emotions assessed (anger, sadness, happiness, surprise and fear), with the exception of disgust, which was preserved even relative to the younger adult group. The relative preservation of disgust could not be attributed to biases in the choice of labels made, and it is suggested instead that this finding might reflect the relative sparing of the basal ganglia in AD. No significant AD effect was observed for the more ecologically valid measure that involved dynamic displays of facial expressions, in conjunction with paralinguistic and body movement cues, although a trend for greater AD difficulty was observed.

Keywords: Emotion recognition; Facial affect recognition; Basal ganglia

1. Introduction

In the neuropsychological literature, considerable emphasis has been placed on the potential role of dissociable neural substrates in recognizing specific emotions (Adolphs, Tranel, Damasio, & Damasio, 1994; Calder, Keane, Lawrence, & Manes, 2004). For instance, when it comes to facial expressions, the orbitofrontal cortex and the ventral striatum have been particularly linked to decoding expressions of anger (Blair & Cipolotti, 2000; Blair, Morris, Frith, Perrett, & Dolan, 1999; Fine & Blair, 2000; Iidaka et al., 2001; Sprengelmeyer, Rausch, Eysel, & Przuntek, 1998), the amygdala (Adolphs & Tranel, 2004; Blair et al., 1999; Breiter et al., 1996; Lennox, Jacob, Calder, Lupson, & Bullmore, 2004; Yang et al., 2002), fusiform gyrus (Surguladze et al., 2003, 2005), and the anterior cingulate cortex (Blair et al., 1999; Killgore & Yurgelun-Todd, 2004; Lennox et al., 2004; Phan, Wager, Taylor, & Liberzon, 2002) to sadness, and the basal ganglia and insula to disgust (Calder, Keane, Manes, Antoun, & Young, 2000).

In Alzheimer's disease (AD), prominent atrophy and tau deposition is observed in limbic regions (including the amygdala), as well as temporal and frontal neocortices with subcortical structures such as the basal ganglia typically less affected until later in the disease process (Boller & Duykaerts, 2003; Braak & Braak, 1991; Delacourte et al., 1999; Hyman & Gomez-Isla, 1998). Thus, because the neural substrates that subserve decoding of different emotions are subject to different rates of degeneration and atrophy in AD, a differentiated profile

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of affect recognition impairment may be anticipated. In support of this prediction, Rosen et al. (2006) found that poor recognition of anger, sadness and fear in a mixed dementia sample was associated with specific regional grey matter shrinkage in areas of the temporal lobes. Although this study does not provide direct evidence for such a link in AD in that Rosen et al.'s sample included heterogeneous dementia diagnoses, it does provide grounds for thinking that a differentiated profile of affect recognition might exist in AD due to different rates of brain change.

Importantly, differential difficulty recognizing specific emotions has been observed in normal aging and has been linked to brain changes (e.g., Calder et al., 2003; Sullivan & Ruffman, 2004a; Sullivan & Ruffman, 2004b). In a meta-analytic review of this literature, Ruffman et al. (in press) concluded that the predominant pattern across all emotions and modalities was of age-related decline, that recognition of anger and sadness was particularly impaired, but that older adults may be *better* at recognizing facial expressions of disgust compared to young adults. Age-related neural volume loss occurs earliest and most rapidly in frontal and temporal lobe structures (Allen, Bruss, Brown, & Damasio, 2005; Grieve et al., 2005; Raz, 2000), with the orbitofrontal cortex experiencing particularly rapid decline (Convit et al., 2001; Lamar & Resnick, 2004; Raz et al., 1997; Resnick, Pham, Kraut, Zonderman, & Davatzikos, 2003), and the anterior cingulate cortex also experiencing consistent decline (Convit et al., 2001; Garraux et al., 1999; Ohnishi, Matsuda, Tabira, Asada, & Uno, 2001; Pardo et al., 2007; Petit-Taboué, Landeau, Desson, Desgranges, & Baron, 1998; Resnick et al., 2003; Tisserand et al., 2002). There are also consistent volume reductions in temporal areas such as the amygdala (Allen et al., 2005; Grieve et al., 2005; Mu, Xie, Wen, Weng, & Shuyun, 1999; Tisserand, Visser, van Boxtel, & Jolles, 2000; Wright, Wedig, Williams, Rauch, & Albert, 2006; Zimmerman et al., 2006). Ruffman et al. (in press) therefore related age-related difficulties identifying anger to changes in the orbitofrontal region, sadness to changes in the anterior cingulate cortex and temporal areas such as the amygdala, and fear to changes in the amygdala. In contrast, the relative sparing of some structures within the basal ganglia are argued to underlie the absence of deficits recognizing disgust (Calder et al., 2003; Williams et al., 2006).

Only three studies have assessed how AD affects recognition of each of the six basic emotions relative to age-matched controls (Burnham & Hogervorst, 2004; Hargrave, Maddock, & Stone, 2002; Lavenu, Pasquier, Lebert, Petit, & Van der Linden, 1999). Whilst the results for individual emotions were generally inconsistent, only deficits recognizing fear and sadness were identified in more than one study, providing some support for the notion that different emotions may be subject to differential rates of decline in AD. In terms of potential reasons for the inconsistencies, these AD studies used a variety of facial affect recognition stimuli. Since Ekman and Friesen's (1976) *Pictures of Facial Affect* are the most widely used stimuli, Edwards, Jackson, and Pattison (2002) advise that facial affect recognition studies should use these stimuli to increase the comparability of their results. However, Hargrave et al. (2002) used photographs from a different stimulus set. This study also included a relatively small predominantly female control sample ($n = 14$), and a larger, but predominantly male AD sample ($n = 22$). Whilst Lavenu et al. (1999) used Ekman and Friesen's stimuli, only four exemplars of each emotion were shown, and again, a relatively small control sample ($n = 12$) was used. Burnham and Hogervorst (2004) also used Ekman and Friesen stimuli, but again, a relatively small number of participants were sampled (13 AD and 13 controls). Thus, it seems likely that prior inconsistencies may reflect artefactual variance, but also substantive differences in terms of the nature and number of stimuli used.

The present study was the first to use the well validated *Ekman 60 Faces Test* (Young, Perret, Calder, Sprengelmeyer, & Ekman, 2002) to index facial affect identification in an AD population. The images in this measure are taken from the *Pictures of Facial Affect* and consist of 10 models expressing each of the six basic emotions. Further, in addition to age-matched controls, the present study was the first to also include a *younger* control group. This provides a unique point of comparison by assessing how difficulties decoding specific emotions vary across AD, older and younger groups. Of particular interest is whether the AD and younger groups differ with regard to the recognition of disgust, given the noted relative preservation of the basal ganglia in AD, which has been argued to underpin intact (and possibly even enhanced) disgust recognition in older adulthood (Ruffman et al., in press).

Finally, virtually all studies to date that have investigated affect recognition in relation to AD have used static stimuli. Where AD deficits have been reported in relation to other emotion cues such as auditory and paralinguistic cues (Allender & Kaszniak, 1989; Bucks & Radford, 2004; Koff, Zaitchik, Montepare, & Albert, 1999; Testa, Beatty, Gleason, Orbelo, & Ross, 2001), different emotion cues were presented in isolation of one another. Thus, the final aim was to test how AD impacts on affect recognition using a more ecologically valid measure that integrates different affective cues. In addition to being of theoretical interest, assessment of this issue has potentially important practical implications since such stimuli represent a better approximation of real-life emotion recognition processes.

2. Methods

2.1. Participants

Eighty-four community dwelling adults in Sydney participated, 24 of whom met DSM-IV and NINCDS-ADRDA criteria for AD, 30 of whom were older adults matched demographically to the AD participants, and 30 of whom were younger adults. Demographic characteristics for all three groups are presented in Table 1. The AD participants were recruited via geriatricians based at hospitals in Sydney. The older control participants were either partners of the AD participants, or volunteers recruited from the general community, and did not differ significantly in age, $t(52) = 0.51$, $p = 0.61$, or years of education, $t(52) = 0.51$, $p = 0.62$, from the AD participants. Some of the younger control participants were recruited from the general community, and others were undergraduate students who took part in return for course credits. The three groups did not differ significantly in gender (50, 53 and 43% male, respectively). Exclusionary criteria for all participants were the presence of uncorrected hearing or visual loss, psychotic symptoms, and a history of substance abuse. An additional exclusionary criterion for the older control participants was a Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975) score of less than 27. For the AD