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Dissociation of age and ability on a visual analogue of the University of Pennsylvania Smell Identification Test in children with autism

W.J. Brewer [a,*], A. Brereton [b], B.J. Tonge [b]

[a] *ORYGEN Research Centre, Department of Psychiatry, University of Melbourne, Locked Bag 10, Parkville, 3052 Victoria, Australia*
[b] *Department of Psychological Medicine, Monash University, Australia*

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Abstract

Early olfactory identification deficits have been associated with neurodevelopmental arrest of limbic-prefrontal networks. These same networks are implicated in development of autistic-spectrum disorders. We aimed to investigate olfactory identification ability in children with high functioning autism (HFA).

Fifteen children with HFA (aged 5–9 years) and 15 age-, gender- and IQ-matched controls (CTL) were compared on their performance on a visual analogue of the University of Pennsylvania Smell Identification Test (UPSIT).

The hypothesis that children with HFA would exhibit impaired olfactory identification ability was not supported. However, contrary to the relationship found in the CTLs, smell identification ability was negatively associated with age in HFA.

The results suggest some disruption of normal developmental association between olfactory ability and age in HFA. The visual analogue of the UPSIT warrants further investigation to determine its validity and reliability in normal and other clinical populations.

Keywords: Autism; Olfactory identification; UPSIT

Autism is an early-onset, neurodevelopmental disorder that implicates neural dysfunction of prefrontal brain regions (Dawson, Meltzoff, Osterling, & Rinaldi, 1998). It is composed of three major features: (i) restricted, repetitive and stereotyped patterns of behaviour; (ii) impairments

* Corresponding author. Tel.: +61 3 9342 2800; fax: +61 3 9342 2858.
E-mail address: w.brewer@unimelb.edu.au (W.J. Brewer).

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in social interaction; (iii) and impaired communication (American Psychiatric Association, 1994). Unusual reactions to sensory stimuli are also associated features of autism, and occur in all sensory modalities (American Psychiatric Association, 1994). They may appear as either under- or over-reactivity, and most children with autism display one or both of these reactions (Ornitz, 1989) although more recently, there is little support for hyper-arousal and failure of habituation assumptions (see Rogers and Ozonoff, 2005). Current literature reports sensory processing problems in children with autism in the auditory and visuo-spatial domains; findings which are thought to reflect impaired sensory integration and/or arousal modulation in the central nervous system (as reviewed in Baranek, 2002). However, a sensory modality in autistic spectrum disorders that has not received significant attention to date is that of olfaction.

Consensus regarding the brain abnormalities that mediate autistic behaviour has not yet been reached, although fronto-striatal dysfunction, including the dorsolateral prefrontal cortex (DLPFC), and the orbitofrontal cortex (OFC; Bradshaw & Sheppard, 2000), has been implicated (Dawson et al., 1998). For example, it has been suggested that the impairment in social communication that commonly affects autistic-spectrum individuals could be accounted for by damage to the anterior temporal cortex, the amygdala and the orbitofrontal cortex (as reviewed in Emery, 2000). Further, neuropsychological studies have shown that patients with autism perform poorly on tests of executive function that depend upon the integrity of these regions (Ozonoff, Strayer, McMahon, & Filloux, 1994; Rinehart, Bradshaw, Tonge, Brereton, & Bellgrove, 2002).

Despite research implicating the orbitofrontal cortex in the pathophysiology of autism, there have been no studies that specifically probe the integrity of this region. In this regard, it has been found that normal olfactory *identification* ability is contingent upon the normal functioning of the OFC once the integrity of lower-order *sensitivity/detection* processes that are mediated more by neural pathways from the olfactory bulbs to the primary olfactory cortex has been confirmed (Potter & Butters, 1980; see also Brewer, Wood, & Pantelis, 2006). Tests of smell identification are a well-recognised means of indirectly assessing the integrity of the OFC. Intact OFC is essential for olfactory identification processing which involves the recognition and naming of a perceived odour (see Martzke, Kopala, & Good, 1997 for review). A reliable body of research confirms that identification of odours represents a secondary stage in olfactory processing mediated by the OFC (Martzke et al., 1997; Qureshy et al., 2000; Suzuki, Critchley, Rowe, Howlin, & Murphy, 2003). For example, Potter and Butters (1980) examined patients with lesions of the OFC, and demonstrated an association between this area and the identification of both pleasant and unpleasant odours. Conversely, Jones-Gotmann and Zatorre (1988) linked olfactory *acuity* (detection as measured by sensitivity to threshold) to the medial temporal lobe structures following examination of patients with excisions to the temporal lobe. Similarly, it has been found that damage to the temporal olfactory cortex leads to deficits in both odour recognition and identification; while conversely, damage to the orbitofrontal cortex (OFC) gives rise mainly to odour identification deficits (Savage et al., 2002).

Clinical populations in which compromise of the limbic-prefrontal axis (particularly the orbitofrontal cortex) has been implicated have consistently been linked to behavioural deficits in olfactory identification. These include schizophrenia (Kopala, Clark, & Hurwitz, 1992), first episode psychosis (Brewer et al., 2001) and obsessive–compulsive disorder (Barnett et al., 1999). Similarly, Suzuki and colleagues (Suzuki et al., 2003) concluded that Asperger's Disorder was associated with dysfunction of the orbitofrontal cortex rather than the medial temporal lobe,