



Face working memory deficits in developmental prosopagnosia: Tests of encoding limits and updating processes



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ABSTRACT

Developmental prosopagnosia (DP) is a condition in which individuals experience life-long problems recognising faces. In recent years, unpacking the nature of the impairments of this population has been the focus of numerous studies. One focus has been on the nature of face-based memory impairments for such individuals, with the onus being mainly on long-term memory deficits. Far fewer have considered the nature of face-based working memory (WM) impairments for DP cases, and the current study seeks to address this. One recent WM study (Shah et al., 2015) reported that the maintenance of faces over time in WM was spared among DPs, and argued instead that face encoding was limited in some way. Here we further explore the nature of face-based WM impairments in DP across two experiments designed to probe encoding limits (Experiment 1) and WM updating processes (Experiment 2). In Experiment 1 we manipulated the number of faces (1–4) to encode into WM and presented these simultaneously. We reasoned that if face encoding among DPs was inefficient or imprecise, then increasing encoding demands (WM load) would disproportionately impair WM accuracy compared to controls. However, we found that DP cases were consistently poorer than controls across all face load conditions, suggesting that front-end encoding problems are only part of the deficit. In Experiment 2, to measure updating four faces were shown sequentially for encoding into WM and accuracy was analysed as a function of whether the test face had been presented first, second, third or last in the encoding sequence. DPs had significantly poorer WM than controls for later faces but not the first face encoded in the sequence, and showed an attenuated recency effect. To account for these findings, we discuss the potential role of comparison processes at retrieval, impairments in configural face processing, and the impact of noise in the face identification system of individuals with DP.

1. Introduction

Developmental prosopagnosia (DP) is a lifelong hereditary condition in which face recognition is severely impaired, while low level visual processing, intelligence, and general social cognition remain intact (for recent overviews see Bate and Tree (2016), and Dalrymple and Palermo, (2016). Also known as ‘face blindness’, it is a disorder affecting approximately 2% of the population (Kennerknecht et al., 2006). It can impact on daily life and impede social interactions. The condition is still not fully understood, and the face processing challenges experienced by DP individuals warrants further exploration.

DP is heterogeneous, but is typically characterised by impairments in perceptual face matching tasks and/or face memory tasks. Regarding perceptual face processing, reported deficits are variable across different tasks. Using faces shown in different viewpoints, DP individuals can show impaired identity or gender matching of two simultaneously

presented unfamiliar faces, producing more errors and slower response times than controls (same/different response; Behrmann et al., 2005). Similar evidence was provided by Duchaine et al. (2007a) who reported impaired DP face perception using the Cambridge Face Perception Test (CFPT; Duchaine et al., 2007b), which requires individuals to sort 6 morphed face images (frontal view) in order of similarity to a three quarter profile view target face within one minute (see also White et al., 2016). However, although DP cases can be impaired at fundamental testing of face perception, this is not true of all such cases (Duchaine and Nakayama, 2004, 2006a). A more consistent pattern is reported on tests that involve memory, indicating that DP cases can show a dissociation between face perception and face memory. Using the CFPT and the Old/New Faces task (Duchaine and Nakayama, 2005), Dalrymple et al. (2014) found that all DP participants showed memory deficits while only half showed perceptual deficits, thus the DP population may be sub-divisible into two subtypes. As such, it has recently

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been suggested that DP be defined as a specific face memory deficit that may or may not be accompanied by abnormal face perception (Dalrymple and Palermo, 2016).

However, characterising the form of face based memory impairments DP cases have is by no means clear. For example, it is well established that there are different forms of memory (long-term, short-term) and a large variety of ways in which memory can be tested. Regarding long-term memory (LTM) there is evidence of impairments for familiar or famous faces among DP individuals (Behrmann et al., 2005; Duchaine et al., 2003; Le Grand et al., 2006). LTM impairments for unfamiliar faces have also been shown using the ‘Face one in ten’ test, in which participants were required to recognise 15 images of a target face (studied during a brief learning phase) among a set of 150 face alternatives presented sequentially during a test phase (identity old/new speeded response; Duchaine et al., 2003). Short-term or working memory (WM) deficits are also reported. In one version of the Cambridge Face Memory Test (CFMT; Duchaine and Nakayama, 2006b), three target faces in different views are sequentially presented for three seconds each, immediately after which participants must choose which face matches in identity to one of the previously studied faces. This task requires the temporary maintenance of each study face in WM for a few seconds until the match response is required. DP participants have been shown to perform 36% worse than controls on this task (Duchaine et al., 2007a). On balance then, there is clear evidence of LTM face based impairments for DP cases. However, we argue that more in-depth exploration of WM for faces is still needed, and that it is particularly important to understand how WM for faces is affected for DP cases as this shapes how these individuals interact with others from moment to moment. As a consequence, the focus of the work presented here is to further explore the nature of face based WM impairments in the context of DP.

1.1. Working memory and face processing in developmental prosopagnosia

Working memory is a fundamental aspect of human cognition. It is conceived as the glue which temporarily maintains and binds perceptual information during brief input disruptions in order to provide a cohesive and integrated representation of what is happening and unfolding from second to second. Without a functioning WM system we could not read, follow a conversation, or keep track of social interactions. While WM has been extensively studied over the decades using non-face stimuli (for overviews see Baddeley (2012), and Logie and Cowan (2015)), WM for faces has only more recently been examined (Curby and Gauthier, 2007; Eng et al., 2005; Jackson and Raymond, 2008; Meconi et al., 2014; Scolari et al., 2008) and predominantly in the context of emotional expression effects on WM accuracy (Becker et al., 2014; Jackson et al., 2012, 2008, 2009, 2014; Sessa et al., 2011; Stiernströmer et al., 2015; Thomas et al., 2014).

The successful recognition of faces using visual WM requires a combination of perceptual and mnemonic processes. Front-end processing requires perceptual encoding of face information, followed by temporary maintenance of encoded representations in WM until such time at which memory is tested (normally 1–10 s after encoding). Retrieval requires the ability to accurately compare the visible test item (s) with the stored WM representation(s) held in the mind's eye. For comparison processes at retrieval to be accurate therefore requires that faces are encoded sufficiently and also effectively maintained.

Due to the short time-course of WM, the stages of encoding, maintenance, and retrieval can be manipulated, controlled, and examined independently in various different ways to determine more specific elements of the process (e.g., with faces Jackson et al., 2012, 2014). This makes the WM paradigm an ideal task to probe both perceptual and memory face processing deficits among prosopagnosics, yet only one study has explicitly examined this to date. Shah et al. (2015) explored whether face recognition deficits among developmental prosopagnosics were driven by deficits in maintaining face information in

WM. In their study, participants were required to encode one face into WM and maintain its representation for either two or eight seconds. Immediately after the maintenance period, six test faces were presented simultaneously from which participants chose the one that matched in identity to the encoding face just seen. Shah and colleagues reasoned that if DP individuals were specifically impaired in maintaining face representations in WM, the longer interval would disproportionately impair their memory performance compared to controls. However, this was not found. Both groups showed a similar detrimental effect of the extended maintenance period and DP participants performed significantly worse than controls at both short and long maintenance intervals. They concluded that maintenance of face information was spared, but perceptual face encoding was impaired. They did not find a WM deficit among DPs for hands, butterflies, or chairs, indicating a face-specific impairment.

Shah et al.'s (2015) DP sample also showed impaired face perception using the CFPT, so these DP cases fit the sub-type of this condition in which both face memory and face perceptual impairments go hand in hand. It is perhaps not surprising therefore that WM was impaired, argued to be driven by poor perceptual encoding of faces into WM. However, if perceptual encoding of faces into WM was impaired, it may be considered unusual that this did not lead to a disproportionately larger maintenance deficit (but see Bogartz (1990) who argued that there is no link between the depth of encoding and the rate of forgetting). More generally, prior research with healthy individuals has shown a link between the effectiveness of encoding and the accuracy of recall, in that WM accuracy for faces improved given longer and more sufficient encoding time (Curby and Gauthier, 2007; Eng et al., 2005). Curby and Gauthier concluded that insufficient encoding time impaired perceptual encoding processes which thus impaired recall. If we make the assumption that sufficient representation in WM relies on effective encoding, then inadequate encoding (whatever the cause) could render the comparison between the test faces at retrieval with the face stored in WM particularly difficult.

More relevant in the current context of prosopagnosia is the consideration of *clinical* impairments in perceptual processing and how these impact WM. Individuals with schizophrenia have impaired WM that is not stimulus-specific or WM-domain specific (it presents in visual-spatial and verbal tasks). It has been proposed that inefficient encoding is partly responsible for this WM deficit, as it leads to poor or imprecise internal representations of the memoranda being stored in WM (Lee and Park, 2005). Furthermore, encoding among schizophrenia patients is considered to be imprecise because they fail to efficiently select or attend to the most relevant information for optimal processing (e.g., Braver et al., 1999; Adler et al., 1998). Using abstract shapes, Haenschel and colleagues found that increasing WM load from 1 to 3 shapes (using a serial presentation) disproportionately impaired WM among schizophrenia patients compared to controls (Haenschel et al., 2007, 2009). This suggests that poor perceptual encoding in a clinically impaired sample leads to more severe encoding limits than controls. Similarly, using an *n*-back task in which coloured stimuli were serially presented, Jansma et al. (2004) found an interaction between load (how many items back a repeat occurred) and participant group, wherein schizophrenia patients became more impaired than controls as load increased. Interestingly, increasing the maintenance interval did not result in disproportionately larger WM deficits in schizophrenia than controls (Tek et al., 2002), which mirrors the findings from Shah et al.'s (2015) study of DP patients. Lee and Park point out that inefficient encoding may not be the sole contributor of WM deficits in schizophrenia, and that other mechanisms need to be considered.

1.2. The present study – exploring face based WM performance in a DP population

In the current study we sought to directly examine WM encoding deficits among DPs in two ways, by assessing encoding limits

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