Neuropsychologia 50 (2012) 2926–2932

Contents lists available at SciVerse ScienceDirect

Neuropsychologia

journal homepage: www.elsevier.com/locate/neuropsychologia

Reduced face identity aftereffects in relatives of children with autism

Chiara Fiorentini [a,b,c,*], Laura Gray [d], Gillian Rhodes [c], Linda Jeffery [c], Elizabeth Pellicano [c,d]

[a] Institute of Child Health, University College London, London
[b] Department of Psychology, The Australian National University, Australia
[c] ARC Centre of Excellence in Cognition and its Disorders, School of Psychology, University of Western Australia, Australia
[d] Centre for Research in Autism and Education (CRAE), Institute of Education, University of London, London

ARTICLE INFO

Article history:
Received 23 February 2012
Received in revised form
23 August 2012
Accepted 24 August 2012
Available online 5 September 2012

Keywords:
Autism
Endophenotype
Faces
Aftereffects
Adaptation

ABSTRACT

Autism is a pervasive developmental condition with complex aetiology. To aid the discovery of genetic mechanisms, researchers have turned towards identifying potential endophenotypes – subtle neurobiological or neurocognitive traits present in individuals with autism and their "unaffected" relatives. Previous research has shown that relatives of individuals with autism exhibit face processing atypicalities, which are similar in nature albeit of lesser degree, to those found in children and adults with autism. Yet very few studies have examined the underlying mechanisms responsible for such atypicalities. Here, we investigated whether atypicalities in adaptive norm-based coding of faces are present in relatives of children with autism, similar to those previously reported in children with autism. To test this possibility, we administered a face identity aftereffect task in which adaptation to a particular face biases perception towards the opposite identity, so that a previously neutral face (i.e., the average face) takes on the computationally opposite identity. Parents and siblings of individuals with autism showed smaller aftereffects compared to parents and siblings of typically developing children, especially so when the adapting stimuli were located further away from the average face. In addition, both groups showed stronger aftereffects for adaptors far from the average than for adaptors closer to the average. These results suggest that, in relatives of children with autism, face-coding mechanism are similar (i.e., norm-based) but less efficient than in relatives of typical children. This finding points towards the possibility that diminished adaptive mechanisms might represent a neurocognitive endophenotype for autism.

1. Introduction

Autism spectrum disorders (ASD) are pervasive developmental conditions characterised by often striking difficulties in social communication and social interaction in addition to repetitive and unusually focused behaviours and interests (American Psychiatric Association, 2000). Early twin and family history studies firmly established that genetic factors play a crucial role in the aetiology of autism (Bailey et al., 1995; Bolton et al., 1994; Folstein & Rutter, 1977). Yet, despite numerous studies using linkage or candidate gene approaches, the discovery of a single genetic locus of major effect has not been forthcoming. Instead, there is now clear consensus among geneticists that autism is both oligogenic – resulting from the action of multiple interacting genes – and multifactorial – resulting from interactions between genes and environmental factors, which have yet to be fully identified (see Geschwind, 2011, for review).

A significant minority of parents and siblings of individuals with ASD show behavioural traits that are qualitatively similar to the defining features of ASD, albeit in more subtle form (see Bailey, Palferman, Heavey, & Le Couteur, 1998, for review). Many studies have attempted to identify the various components of this so-called "broad autism phenotype", which can include rigid or aloof personality traits, difficulties initiating and maintaining friendships, limited communicative use of language, and overly focused and unusual interests and activities (Bishop, Maybery, Wong, Maley, & Hallmayer 2006; Losh, Childress, Lam, & Piven, 2008; Losh & Piven, 2007; Piven, Palmer, Jacobi, Childress, & Arndt, 1997).

Focusing on the behavioural level alone, however, is not an ideal basis for identifying genetic mechanisms: the same genotype can give rise to different behavioural phenotypes, and the same phenotype can arise from a range of genotypes (Gottesman & Gould, 2003). Researchers have therefore turned their attention towards discovering neurobiological or cognitive markers that are initially unobservable but which are more proximal to the underlying

* Corresponding author at: Department of Psychology, The Australian National University, Canberra ACT 0200, Australia. Tel.: +61 0 2 612 5416; fax: +61 0 2 612 50499.
E-mail address: fiorentinichiara@gmail.com (C. Fiorentini).

0028-3932/$ - see front matter © 2012 Elsevier Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.neuropsychologia.2012.08.019

aetiology of autism than overt behaviours themselves. Such "endophenotypes" are thought to index genetic liability to autism in otherwise apparently "unaffected" individuals (Flint & Munafò, 2007).

Atypical face-processing mechanisms have been proposed to be one such candidate endophenotype for autism (Dawson et al. 2002). Difficulties in perceiving and discriminating faces have been well documented in individuals with ASD (e.g., see Dawson, Webb, & McPartland, 2005, for review). Children and adults with ASD show poorer performance on a variety of face processing tasks compared with non-autistic individuals, including face recognition (Boucher, Lewis, & Collis, 1998; Ewing, Pellicano, & Rhodes, 2011a), face discrimination (Ewing et al., 2011a; Wallace, Coleman, & Bailey, 2008a), expression recognition (Rump, Giovannelli, Minshew, & Strauss, 2009; Wallace, Coleman, & Bailey, 2008b), and eye-gaze perception (Wallace, Coleman, Pascalis, & Bailey, 2006). Even when their performance is similar to that of non-autistic individuals, individuals with ASD appear to use atypical strategies, such as paying more attention to the mouth than the eyes (Klin, Jones, Schultz, Volkmar, & Cohen, 2002; Neumann, Spezio, Piven, & Adolphs, 2006; although see Falck-Ytter & von Hofsten, 2011, for a critique), and applying a local rather than holistic processing style (Joseph & Tanaka, 2002).

A similar range of face processing atypicalities has also been reported in parents and siblings of individuals with ASD. Such atypicalities include less time spent looking at the eyes during a face processing task (Dalton, Nacewicz, Alexander & Davidson, 2005), difficulties discriminating subtle differences between faces, identifying facial expressions of fear and disgust, and judging direct eye contact (Wallace, Sebastian, Pellicano, Parr, & Bailey, 2010) and problems on a standardized test of facial identity recognition (Wilson, Freeman, Brock, Burton, & Palermo, 2010), compared to parents and siblings of typically developing individuals. These studies clearly show that atypicalities in various behavioural aspects of face processing are shared by individuals with autism and their relatives.

Studies that go one step further to pinpoint the underlying mechanisms responsible for such atypicalities in relatives of individuals with autism should therefore bring us closer for isolating a potential endophenotype for autism at the neurocognitive level. One study has demonstrated distinct face-processing strategies during emotion recognition in relatives of individuals with ASD (Adolphs, Spezio, Parlier, & Piven, 2008). These authors showed that parents of typical children showed substantial use of the eyes when judging emotions like fear or happiness. Yet parents of autistic children, especially those with an aloof personality, made much less use of the eyes when making these judgments, using more cues from the mouth (Adolphs et al., 2008), a strategy that closely mirrors the behaviour of individuals diagnosed with ASD (Spezio, Adolphs, Hurley, & Piven, 2007).

The present study extends the search for candidate endophenotypes for autism by focusing on mechanisms underlying another important aspect of face processing the recognition of facial identity.

Typical children and adults code faces relative to an implicitly-stored internal *average* or norm, which is continuously updated by experience to represent the central tendency of the population of faces experienced (for review see Rhodes & Leopold, 2011). The strongest evidence for this adaptive norm-based coding mechanism comes from *aftereffect* paradigms. Aftereffects occur throughout perceptual systems and are illustrative of how perceptual attributes, such as colour and motion, are coded by these systems. For example, the motion aftereffect occurs when adaptation (prolonged exposure) to a stimulus moving in a particular direction causes a subsequently viewed stationary stimulus to be perceived as moving in the opposite direction. Similarly, in face identity aftereffects, adapting to a face biases us to see a subsequent face as having opposite properties (e.g., Leopold, O'Toole, Vetter, & Blanz, 2001).

In a typical face identity aftereffect task, participants learn some target identities (e.g., Ted and Rob; see Fig. 1), and are then tested on their recognition of faces with weaker identity strengths of Ted (e.g., 30%, 60%, etc., including 0% average face) both before and after adaptation to anti-Ted. In typical adults (e.g., Leopold et al., 2001; Rhodes & Jeffery, 2006) and children (e.g., Nishimura, Maurer, Jeffery, Pellicano, & Rhodes, 2008), identification of the target identity (e.g., Ted) is facilitated following adaptation to its antiface (antiTed), that is, a face with opposite properties. For example, if Ted has smaller lips than average, antiTed will have larger lips than average, and so on for many other facial attributes. In terms of face space, anti-faces lie along the same vector as the target identity, but are situated on the other side of the average face. After adaptation to antiTed, the previously neutral 0% (average) face will be perceived as Ted. Adaptation to antiTed "shifts" the observer's *internal* average toward anti-Ted, causing the *actual* average (identity neutral) to look more like Ted. Furthermore, the identity aftereffect is selective for opposite face pairs (e.g., Ted/anti-Ted but not Ted/anti-Rob), suggesting that facial identity is coded opponently, with pairs of neural populations coding for above- and below-average values along particular dimensions in face space (Rhodes & Jeffery, 2006; Rhodes et al., 2005; Robbins, McKone, & Edwards, 2007).

Pellicano, Jeffery, Burr and Rhodes (2007) investigated adaptation to facial identity in children with and without ASD aged between 8 and 13 years. They found that the extent to which the children shifted their perception following adaptation was significantly attenuated in those with autism. Furthermore, the degree of adaptation correlated significantly and negatively with children's current levels of autistic symptoms, such that the children with the smallest aftereffects exhibited greater levels of symptoms. These findings suggest that the mechanisms responsible for coding facial identity might be less flexible or adaptable in children with autism, so that, relative to typical children, they

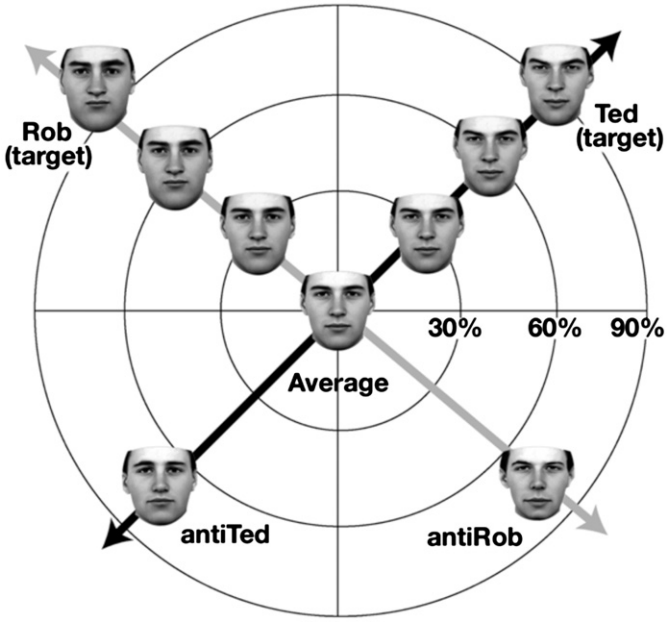

Fig. 1. A simplified face-space showing an average face, two target faces, Ted and Rob, and their opposite "antifaces". "Weaker" versions of each target were made by morphing each target with the average face by different amounts e.g., 60% Ted and 30% Ted as shown here. Adapting to antiTed facilitates recognition of Ted, so that "weaker" versions of Ted are more accurately identified and the average face takes on the appearance of Ted, but recognition of Rob is not facilitated.