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Intact facial adaptation in autistic adults

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Lay Abstract

Looking at particular facial identities or expressions, for prolonged periods, can give rise to compelling visual illusions. For example, if we look continuously at a sad facial expression (an 'adapting stimulus'), and then look at a neutral face (a 'test stimulus') the latter seems to take on a happy expression. This illusion is known as a 'facial aftereffect'. Recent experiments have found that children with Autism Spectrum Disorders (ASD) show atypical facial aftereffects, prompting some researchers to speculate that face processing systems work in a different way in all individuals with ASD – both children and adults. However, caution is required when attempting to generalize findings from children with ASD to the wider ASD population. An alternative possibility is that people with autism are simply slower to develop the mechanisms responsible, and therefore only show smaller aftereffects during childhood. The present study thus sought to measure the strength of facial aftereffects in high-functioning adults with autism. We report two experiments conducted with a sample of adults with ASD, which reveal substantial identity and expression aftereffects, comparable to those of matched controls. That adults with autism can show robust facial aftereffects, raises the possibility that group differences are seen only at particular points during development, and may not be a life-long feature of the condition.

Scientific Abstract

Adaptation paradigms seek to bias subsequently viewed stimuli through prolonged exposure to an adapting stimulus, thereby giving rise to an aftereffect. Recent experiments have found that children with Autism Spectrum Disorders (ASD) show reduced facial aftereffects, prompting some researchers to speculate that all individuals with ASD exhibit deficient facial adaptation. However, caution is required when generalizing findings from samples of children with ASD to the wider ASD population. The reduced facial aftereffects seen in child samples may instead reflect delayed or atypical developmental trajectories, whereby individuals with ASD are slower to develop adaptive mechanisms. In the present study, two experiments were conducted to determine whether high-functioning adults with ASD also show diminished aftereffects for facial identity and expression. In Experiment 1, using a procedure that minimized the contribution of low-level retinotopic adaptation, we observed substantial aftereffects comparable to those seen in matched controls, for both facial identity and expression. A similar pattern of results was seen in Experiment 2 using a revised procedure that increased the contribution of retinotopic adaptation to the facial aftereffects observed. That adults with autism can show robust facial aftereffects, raises the possibility that group differences are seen only at particular points during development, and may not be a life-long feature of the condition.

Key words: Autism, Adaptation, Aftereffects, Facial identity, Facial expressions

INTRODUCTION

Autism Spectrum Disorders (ASD) are characterized by abnormalities of social interaction, impaired verbal and nonverbal communication, and a restricted repertoire of interests and activities (American Psychiatric Association, 1994). Characteristic problems with social interaction in individuals with ASD have prompted considerable interest in potential deficits of facial identity (Weigelt, Koldewyn, & Kanwisher, 2012) and expression (Harms, Martin, & Wallace, 2010) recognition in ASD populations. More recently, reports of atypical adaptation-induced facial aftereffects in individuals with ASD (Pellicano, Jeffery, Burr, & Rhodes, 2007; Rutherford, Troubridge, & Walsh, 2011; Ewing, Pellicano, & Rhodes, 2013) have also received a great deal of attention (Simmons et al., 2009; Webster & Macleod, 2011; Pellicano & Burr, 2012; Weigelt et al., 2012).

Adaptation paradigms seek to bias perception of subsequently viewed stimuli through prolonged exposure to an adapting stimulus. If neurotypical participants fixate on a particular identity or expression, subsequently viewed test faces are perceived as containing less of that identity (Leopold, O'Toole, Vetter, & Blanz, 2001; Rhodes & Jeffery, 2006) or expression (Skinner & Benton, 2010; Cook, Matei, & Johnston, 2011). Facial aftereffects have been interpreted as evidence of adaptive recalibration, whereby prolonged exposure to a given input causes the visual system to readjust, optimizing detection and description within ever-changing environments (Clifford & Rhodes, 2005; Webster & Macleod, 2011; Pellicano & Burr, 2012). The study of facial aftereffects has been instrumental in developing the 'face space' framework. According to this view, faces are coded as mean-relative vectors in a multi-dimensional space through the relative excitation of opponent neural populations (Leopold et al., 2001; Susilo, McKone, & Edwards, 2010; Webster & Macleod, 2011). By modulating the responses of some populations, relative to their opponents, facial adaptation is thought to systematically bias perception towards the opposite side of face space. Crucially, facial aftereffects are therefore thought to be causally related to the recognition and interpretation of faces (Dennett, McKone, Edwards & Susilo, 2012; Rhodes, Watson, Jeffery & Clifford, 2010).

Pellicano et al. (2007) reported that children (8- to 13-years) with ASD show diminished facial identity aftereffects relative to age-matched neurotypical controls. Having adapted to anti-

identities (identities on the diametrically opposite side of face-space), children with ASD showed reduced perceptual bias towards the veridical identities. Moreover, ASD severity was negatively correlated with the magnitude of the aftereffects – those individuals with more severe autism showed less adaptation. This finding was replicated and extended by Ewing et al. (2013). Children (aged 7- to 15-years) with autism again exhibited smaller face shape aftereffects than matched typically developing controls. Reduced aftereffects were seen only with upright faces; children with autism and controls demonstrated equivalent adaptation for inverted faces and cars. Reports of atypical facial aftereffects have been taken as evidence of disturbed face-specific representation; that ASD individuals have a face space organized differently to that of neurotypical individuals (Weigelt et al., 2012), or that their face-representation mechanisms exhibit deficient adaptive recalibration (Pellicano et al., 2007).

The aim of the present study was to determine whether adults with autism also show diminished facial aftereffects. Caution is required when attempting to generalize atypical facial adaptation observed in samples of ASD children to the wider ASD population. In the typically developing population, recognition of facial identity (e.g., Carey, Diamond, & Woods, 1980; Germine, Duchaine, & Nakayama, 2011) and facial expressions (e.g., Johnston et al., 2011), continues to improve throughout adolescence into early adulthood. Moreover, many aspects of neurotypical face perception are thought to undergo qualitative changes throughout childhood and adolescence. For example, whereas younger infants are disproportionately reliant on the external facial contours (e.g., Carey & Diamond, 1977; Campbell, Walker, & Baron-Cohen, 1995) and individual features (e.g., Mondloch, Leis, & Maurer, 2006), older children and young adults make more use of internal feature configurations (e.g., Mondloch, Le Grand, & Maurer, 2002). Similarly, the volume of the fusiform face area (FFA; a region of inferotemporal cortex implicated in face recognition) is disproportionately larger in adults than children (Golarai et al., 2007; Scherf, Behrmann, Humphreys, & Luna, 2007). Differential facial adaptation seen in ASD children might therefore reflect developmental delay, whereby individuals take longer to achieve typical levels of adaptation, rather than a stable difference that persists into adulthood.

To date, little is known about facial adaptation in adults with autism: no studies have addressed facial identity adaptation, and the only reported investigation of facial expression adaptation, is hard to interpret. Rutherford et al. (2011) required adults with ASD (aged 22-39) to fixate on an emotional facial expression – the adapting stimulus – before the presentation of a test face exhibiting a neutral expression. Participants were then asked to attach an emotion label (anger, disgust, fear, happy, sad, surprise) to their percept. While the authors found evidence of adaptation in both groups, the ASD adults selected different labels to describe the aftereffects they experienced. However, the procedure employed places considerable demands on the emotion recognition abilities of individuals with autism, many of whom have difficulties attaching emotional labels to facial expressions (e.g., Harms et al., 2010; Bird & Cook, 2013; Cook, Brewer, Shah, & Bird, 2013). Indeed, it is not clear whether the atypical attributions reflect adaptive processes or pre-existing differences in the use of emotional labels. Moreover, the protocol used by Rutherford et al. (2011) did not allow estimation of the strength of the aftereffects observed. Because the strength of the illusory bias induced in the ASD sample is unknown, comparison with the diminished aftereffects seen in samples of ASD children (Pellicano et al., 2007; Ewing et al., 2013), is not possible.

EXPERIMENT 1

To better understand the nature of adaptive visual processes in adults with ASD, Experiment 1 sought to compare the size of aftereffects, in autistic adults and matched neurotypical controls, following adaptation to emotional facial expressions and identities. A psychophysical procedure was used, whereby participants made binary choice judgments about stimuli drawn from facial identity or expression morph continua, having adapted to particular facial identities or expressions. This approach allows estimation of the degree of perceptual bias in different adaptation conditions; i.e., the magnitude of the facial aftereffects induced.

Methods

Participants

Participants were 32 adults, 16 with (15 males; mean age = 39.2 years, *SD* = 12.2 years) and 16 without (12 males; mean age = 33.9 years, *SD* = 11.1 years) a clinical diagnosis of ASD. ASD and control participants did not differ significantly in age [$t(30) = 1.63$, $p = .11$] or proportion

of females [χ^2 (1) = 2.13, p = .14]. IQ scores did not differ significantly [t(30) = 1.54, p = .13] between the ASD (M = 115.8, SD = 10.3) and control groups (M = 121.7, SD = 11.2). All participants in the ASD group had received diagnoses from an independent clinician and met the criteria for ASD on the Autism-Spectrum Quotient (AQ; Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001). All but one also met the criteria for ASD on the Autism Diagnostic Observation Schedule (ADOS-G; Lord et al., 2000). The participant who did not was not an outlier in any analysis, and correlations with ASD severity were not altered by exclusion of this participant. Autistic features were assessed in all participants using the AQ. The presence of autism, as indexed by AQ scores, was significantly greater in the ASD (M = 33.1, SD = 8.2) than control (M = 16.0, SD = 7.8) group [t(30) = 6.05, p < .001]. Further details of the ASD group are provided in Table 1.

Table 1

Stimuli and Procedure

Morph continua were produced by blending images with Morpheus Photo Morpher version 3.11 (Morpheus Software LLC, Indianapolis, USA). An emotion continuum was derived by morphing the facial emotions of disgust and anger, expressed by an average identity. A complimentary identity continuum was derived by morphing the facial identities 'Harold' and 'Felix' (identities M4 and M6; Ekman & Friesen, 1976), expressing an average facial emotion (Figure 1a). Each continuum comprised seven images ranging from 20-80% of each attribute in equidistant 10% steps (Figure 1b).

Figure 1

The same adapting stimuli were used to elicit identity and expression aftereffects; Felix expressing anger and Harold expressing disgust. The identities and emotions depicted in the adapting stimuli were presented at 100% of their veridical intensity. Because the same adapting stimuli were used to produce identity and expression aftereffects, an attribute-specific group difference (e.g., atypical facial expression adaptation in the ASD group), could not have been due to the particular adapting stimulus used. If a stimulus was 'a bad adaptor' – if for example

individuals with ASD found it less salient – diminished aftereffects would be expected for both expression and identity. The present morph design therefore allows direct comparison of the magnitude of expression and identity aftereffects across the groups.

The experimental program was written in MATLAB using Psychtoolbox (Brainard, 1997; Pelli, 1997). Participants completed four blocks of 70 adaptation trials (judging identity having adapted to Harold expressing disgust; judging identity having adapted to Felix expressing anger; judging emotion having adapted to Harold expressing disgust; judging emotion having adapted to Felix expressing anger). Block order was fully counterbalanced. Each of the seven stimuli comprising each continuum was presented ten times per block. When judging emotion, participants made attributions of test stimuli drawn from the identity-invariant emotion continuum, and when judging identity, test stimuli were drawn from the emotion-invariant identity continuum. Because this procedure measures the strength of perceptual shifts in orthogonal identity and expression planes, any aftereffects seen are 'pure' measures of identity and expression adaptation.

The onset of each test stimulus was preceded by a period of adaptation and a 400 ms inter-stimulus-interval. On the first trial, the adapting stimulus was presented for an interval of 20 seconds. On all subsequent trials, for the remainder of the block, the adapting stimulus was presented for a 'top-up' interval of 8 seconds. Test stimuli were presented for 800 ms followed immediately by a prompt to attribute either expression ('Disgust or Anger?') or identity ('Harold or Felix?'), depending on the block. During the period of adaptation a fixation cross was presented equidistantly between the eyes and the mouth of the adapting stimulus. Adapting and test stimuli were presented at different scales and different locations, to minimize retinotopic adaptation. Adapting stimuli subtended 8° × 6° and were presented centrally. Test stimuli subtended 6° × 4.5° and were presented at random points on a notional circle of radius 4°, located around the display center.

Results and discussion

Separate psychometric functions were modeled for each adaptation condition by fitting cumulative Gaussian distributions to individuals' discrimination data, using the Palamedes

toolbox (Prins & Kingdom, 2009). Participants' susceptibility to adaptation-induced recalibration was inferred from the difference between the point of subjective equivalence (PSE) parameters following adaptation to the different identities and expressions (Figure 2). The PSE is a measure of bias and describes the point on the identity or expression dimension where participants are equally likely to make either attribution.

The magnitude of adaptation-induced shifts in identity and expression attributions exhibited by the two groups is shown in the left-hand panel of Figure 2c. Both the ASD [$t(15) = 5.36$, $p < .001$] and neurotypical [$t(15) = 11.38$, $p < .001$] groups demonstrated robust identity aftereffects ($M = 10.3\%$, $SD = 7.7\%$ and $M = 11.9\%$, $SD = 4.2\%$, respectively). The magnitude of the identity aftereffects did not differ between the groups [$t(30) = .76$, $p = .45$], or correlate with autism severity, as indexed by either AQ [$r(30) = -.30$, $p = .099$] or ADOS score [$r(14) = .46$[1], $p = .075$]. Similarly, both the ASD [$t(15) = 3.64$, $p = .002$] and neurotypical [$t(15) = 3.63$, $p = .002$] groups demonstrated substantial expression aftereffects ($M = 6.8\%$, $SD = 7.5\%$ and $M = 6.0\%$, $SD = 6.6\%$, respectively), but no group difference emerged [$t(30) = .35$, $p = .73$]. The magnitude of the expression aftereffects also failed to correlate with either AQ [$r(30) = -.03$, $p = .89$] or ADOS [$r(14) = -.29$, $p = .27$] scores.

Figure 2

Contrary to previous reports of diminished aftereffects in samples of children with ASD (Pellicano et al., 2007; Ewing et al., 2013), the results of Experiment 1 suggest that adults with autism and matched neurotypical controls exhibit similar facial adaptation. If differential facial adaptation is seen only in samples of ASD children, this raises the possibility that the effect is due to delayed or atypical developmental trajectories, and might not be a stable, universal, feature of the condition.

[1] A weak trend was observed whereby increasing symptom severity was associated with *increased* adaptation. This non-significant trend was not predicted and we note the direction for clarification only.

EXPERIMENT 2

The interpretation of facial aftereffects is not straightforward (e.g., Afraz & Cavanagh, 2008). As well as eliciting calibration of high-level, face-specific areas, fixating on a facial image also causes low-level retinotopic adaptation; for example, adaptation to particular patterns of contrast (e.g., Movshon & Lennie, 1979), spatial frequencies and orientations (e.g., Blakemore & Campbell, 1969). This low-level adaptation originates early in the visual stream, in retinotopically-organized areas, and not in the occipitotemporal regions thought to support domain-specific face representation. Although reports of diminished facial aftereffects in samples of ASD children (Pellicano et al., 2007; Ewing et al., 2013), have been taken as evidence of disturbed face-specific representation, it is also possible that differences reflect reduced low-level retinotopic adaptation. It has been suggested previously that individuals with ASD may show reduced adaptation for a range of stimulus attributes, extending beyond facial identity (Pellicano & Burr, 2012). Moreover, retinotopic adaptation – which requires stable gaze fixations so that each portion of the retina receives a consistent visual input – may be particularly sensitive to atypical patterns of facial fixation, thought by some to be a feature of ASD (e.g., Senju & Johnson, 2009).

If reduced aftereffects in child samples (Pellicano et al., 2007; Ewing et al., 2013) are due to diminished retinotopic adaptation, the protocol used in Experiment 1 would be unlikely to detect these differences. In the first experiment we varied the scale and position of adapting and test stimuli; a technique used widely to minimize the contribution of retinotopic adaptation (e.g., Leopold et al., 2001; Cook et al., 2011), in order to draw inferences about recalibration of face-specific mechanisms. If the differential aftereffects seen in samples of ASD children reflect reduced retinotopic adaptation, the change of scale and position may have prevented the detection of a similar group difference. Instead, experiments may be more likely to detect group differences between ASD and control samples when adapting and test stimuli are presented at identical scales (Pellicano et al., 2007; Rutherford et al., 2011) and at the same location (Pellicano et al., 2007; Rutherford et al., 2011; Ewing et al., 2013). To test this possibility, twelve members of the original ASD sample used in Experiment 1 were recalled, 13-14 months after completion of the first experiment, to complete a second adaptation procedure.

Methods

Participants

Participants were 24 adults, 12 with (11 males; mean age = 38.3 years, *SD* = 10.7 years) and 12 without (11 males; mean age = 42.7 years, *SD* = 11.1 years) a clinical diagnosis of ASD. All participants in the ASD group (Table 1), and two of the controls, participated in Experiment 1. As in the first experiment, the groups were matched for the proportion of females, age [$t(22) = .99$, $p = .33$] and IQ [$t(22) = 1.03$, $p = .31$]. The presence of autism, as indexed by AQ scores, was significantly greater [$t(22) = 6.49$, $p < .001$] in the ASD group ($M = 34.3$, $SD = 7.0$) than in controls ($M = 16.5$, $SD = 6.4$).

Stimuli & Procedure

Two alterations were made to the procedure used in the first experiment. First, in Experiment 1, the adapting and test stimuli were presented at different locations and at different scales. In Experiment 2, however, the adapting and test stimuli were both presented at the same central location, and both stimuli subtended 8° × 6°. Second, the fixation cross, present during the adaptation period in Experiment 1, was removed. It was reasoned that the absence of a fixation cross in previous studies (Pellicano et al., 2007; Rutherford et al., 2011; Ewing et al., 2013) may accentuate group differences in retinotopic adaptation due to atypicalities in fixation behavior in the autistic sample (e.g., Senju & Johnson, 2009). In all other respects the procedure of Experiment 2 was identical to that used in Experiment 1.

Results and discussion

The adaptation-induced shifts in identity and expression attributions seen in Experiment 2 are shown in right-hand panel of Figure 2c. Both the ASD [$t(11) = 5.09$, $p < .001$] and neurotypical [$t(11) = 10.66$, $p < .001$] groups again demonstrated robust identity aftereffects, $M = 17.6\%$ ($SD = 11.9\%$) and $M = 15.4\%$ ($SD = 5.1\%$), respectively. The magnitude of the identity aftereffects did not differ between the groups [$t(22) = 0.54$, $p = .59$], or correlate with autism severity, as indexed by either AQ [$r(22) = .01$, $p = .98$] or ADOS score [$r(10) = .06$, $p = .86$]. The ASD [$t(11) = 5.53$, $p < .001$] and neurotypical [$t(11) = 2.79$, $p = .018$] groups also demonstrated substantial expression aftereffects ($M = 16.4\%$, $SD = 10.4\%$ and $M = 10.8\%$, $SD = 13.5\%$, respectively), but no group difference emerged [$t(22) = 1.20$, $p = .25$]. Again, no

significant correlations were observed between the magnitude of the expression aftereffects and either AQ [$r(22) = .29$, $p = .17$] or ADOS [$r(10) = .48$, $p = .11$] scores[2]. These results mirror closely those seen in Experiment 1, and suggest comparable levels of adaptation in autistic adults and neurotypical controls.

Collapsing across groups, the adaptation effects observed in Experiment 2 for both identity [$t(54) = 2.67$, $p = .010$] and expression [$t(54) = 2.81$, $p = .007$] were significantly greater (13.6% and 16.5%, respectively) than those seen in Experiment 1 (6.4% and 11.1%). This reflects the greater contribution of retinotopic adaptation in Experiment 2, thereby confirming the effectiveness of the scale and position manipulations employed in Experiment 1.

GENERAL DISCUSSION

Previous studies of facial adaptation in samples of children with ASD have reported evidence of reduced aftereffects for facial identity (Pellicano et al., 2007; Ewing et al., 2013). The aim of the present study was to determine whether adults with autism also show diminished aftereffects for facial identity and expression. Contrary to findings with samples of children with ASD, both of the experiments conducted revealed robust adaptation in adults with autism. In Experiment 1, using a procedure that minimized the contribution of low-level retinotopic adaptation, we observed substantial aftereffects, indistinguishable from those seen in matched controls, for both facial identity and expression. A similar pattern of results emerged in Experiment 2 using a revised procedure that increased the contribution of retinotopic adaptation to the facial aftereffects observed.

It has been suggested previously that reduced adaptation may be a stable feature of the autism phenotype (e.g., Simmons et al., 2009; Pellicano & Burr, 2012). However, the present findings raise the possibility that the diminished aftereffects seen in ASD children may reflect delayed or atypical developmental trajectories, whereby individuals take longer to achieve typical

[2] As in Experiment 1, the weak trend observed was for a positive correlation between ADOS and aftereffect size; greater autism severity was associated with increased adaptation. Again, this non-significant trend was not predicted and we note the direction for clarification only.

levels of facial adaptation. Many features of adult face perception are thought to develop incrementally over many years (e.g., Carey et al., 1980; Mondloch et al., 2002; Germine et al., 2011). Facial aftereffects are widely attributed to the way faces are represented within a multidimensional face space, by opponent cortical populations (Webster & Macleod, 2011). Importantly, the constituent dimensions of this representation space are thought to develop over time, shaped by our visual experience of faces (Valentine, 1991; Cohen Kadosh, Johnson, Henson, Dick, & Blakemore, 2013). Young infants who show reduced interest in social stimuli, such as those who may later receive an ASD diagnosis (Osterling & Dawson, 1994; Swettenham et al., 1998; Maestro et al., 2002), might therefore take longer to develop adaptive mechanisms.

The conclusion that adults with ASD show typical facial adaptation at first appears inconsistent with a recent report that family-members, including siblings (aged 7-16 years) and parents (aged 34-52 years) of children with ASD also show atypical adaptation (Fiorentini, Gray, Rhodes, Jeffery, & Pellicano, 2012). Of particular interest, the authors observed broadly similar effects for the siblings and parents of the ASD children. However, two factors make this study hard to interpret. First, the analyses reported are not conducted on the parameters of psychometric functions (e.g., PSEs), but on the total number of responses associated with each half of the morph continua. This is a less sensitive measure of adaptation than those used elsewhere (e.g., Pellicano et al., 2007; Ewing et al., 2013). Second, the ages of the siblings and parents varied considerably. Each category is likely to be heterogeneous, including individuals at different stages of development, with varying face perception abilities. Together, the results of the present study, and those of Fiorentini et al. (2012), Pellicano et al. (2007), and Ewing et al. (2013), highlight the need for a systematic investigation – possibly utilizing a longitudinal approach - of the relationship between age and facial adaptation in the ASD population.

In addition to age, an appreciation of the individual differences attributable to IQ and gender may also afford a richer understanding of the facial aftereffects seen in this population. The autistic adults tested in the present experiments were high functioning, with a mean IQ of 115. It remains to be seen whether lower-functioning adults with autism also demonstrate typical levels of adaptation. Moreover, recent work examining the sub-clinical population, suggests

that higher autistic traits may predict weaker adaptation in adult males, but *stronger* adaptation in adult females (Rhodes, Jeffery, Taylor, & Ewing, 2013). Consistent with the present results, putative interactions between gender and autistic traits suggests that the prevailing view – that diminished adaptation is a common, stable feature of autism – may be overly simplistic, and that a more nuanced interpretation is required.

We have suggested that our failure to replicate reports of diminished facial adaptation seen in children with ASD, using an adult sample, may reflect a developmental delay in the acquisition of typical face space representation. However, several alternative accounts deserve consideration. First, it is possible that children with ASD simply spend less time than older ASD participants looking at adapting stimuli (Simmons et al., 2009). A related possibility is that different patterns of stimulus fixations, or differential fixation stability, disproportionately affect the retinotopic adaptation of children with ASD. The absence of accompanying eye-tracking data makes it hard to evaluate these accounts where group differences have been observed (Pellicano et al., 2007; Fiorentini et al., 2012; Ewing et al., 2013). The similar aftereffects seen in the present experiments provide a strong indication of similar fixation behavior in our control and ASD groups. It is also noteworthy that Rutherford et al. (2011) found no differences in the fixation behavior of adults with ASD and matched controls, having used an adaptation interval of 45 seconds. Excluding the possibility that differences in fixation behavior are responsible for reduced aftereffects in ASD children remains a priority for future research.

A second possibility worthy of discussion is that individuals with autism may require increased exposure to faces to achieve typical levels of recalibration. The 8-second adapting intervals employed in the present study are considerably shorter than those used by many authors (e.g., Rutherford, Chattha, & Krysko, 2008; Rhodes, Jeffery, Clifford & Leopald, 2007; Rhodes et al., 2010; Skinner & Benton, 2010; Rutherford et al., 2011). Nevertheless, if adults with autism achieve typical recalibration, but do so less efficiently, group differences may only be evident following shorter periods of adaptation (e.g., 5 seconds; Pellicano et al., 2007; Fiorentini et al., 2012). Future research addressing slower adaptation in ASD should seek to exclude the

possibility that participants with autism are simply slower to adopt stable fixations during the adaptation phase, and thus accumulate less retinotopic adaptation.

Finally, in the experiments described, we took advantage of the fact that emotional facial expressions contain both an identity and an expression signal. This allowed direct comparison of the identity and expression aftereffects elicited by the same stimuli. While this feature of the design has certain advantages, it is also a salient departure from the methods used previously (Pellicano et al., 2007; Ewing et al., 2013). Crucially, emotion cues could not be used to attribute identity, and vice-versa: participants had to judge the identity of stimuli drawn from an emotion-invariant Harold-Felix continuum; and judge the expression of stimuli drawn from an identity-invariant disgust-anger continuum. Any attempt to attribute identity by looking for emotion cues would result in chance performance. Because the paradigm used measured the strength of the perceptual shifts in orthogonal identity and expression planes, the aftereffects seen are 'pure' measures of identity and expression adaptation. Nevertheless, it is important that future research addresses how the presence of facial emotion on the adapting face affects perceptual recalibration processes in typically developing participants and in individuals with ASD. For example, an interesting possibility is that the presence of emotion helps participants with ASD maintain fixations, thereby yielding typical facial aftereffects.

The faces we encounter everyday are constantly changing, a result of the deformations associated with communicative gestures, facial speech, and emotional expression. If facial identity adaptation serves any useful function – and the process of adaptive recalibration is widely thought to play a crucial role in both identity and expression recognition (Dennett et al., 2012; Rhodes et al., 2010; Webster & Macleod, 2011) – it has to be able to operate across a vast range of expressions and viewpoints. Observing substantial identity adaptation, despite the presence of salient emotion cues in the adapting stimuli, is a striking feature of the present results that further confirms the robustness of this phenomenon.

In summary, the present findings challenge the view that individuals with autism cannot achieve typical levels of facial adaptation. Rather, these results indicate that adults with autism can, under some conditions, demonstrate entirely normal adaptation for facial expression and

identity. Whereas previous reports of diminished adaptation have been reported with samples of children with ASD, the present results were obtained with adult participants, raising the possibility that previously reported group differences reflect delayed or atypical developmental trajectories. However, the present findings highlight the need for future research addressing how facial adaptation in ASD behaves as a function of i) the IQ of ASD samples; ii) the adaptation interval employed; iii) the emotional content of the adapting face. It also remains to be seen whether reports of atypical adaptation effects in samples of children with ASD reflect reduced retinotopic adaptation, reduced face-specific adaptation, or atypical fixation behavior. However, irrespective of the mechanism responsible, diminished facial aftereffects do not appear to be a stable, universal feature of the condition.

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FIGURES

Figure 1:

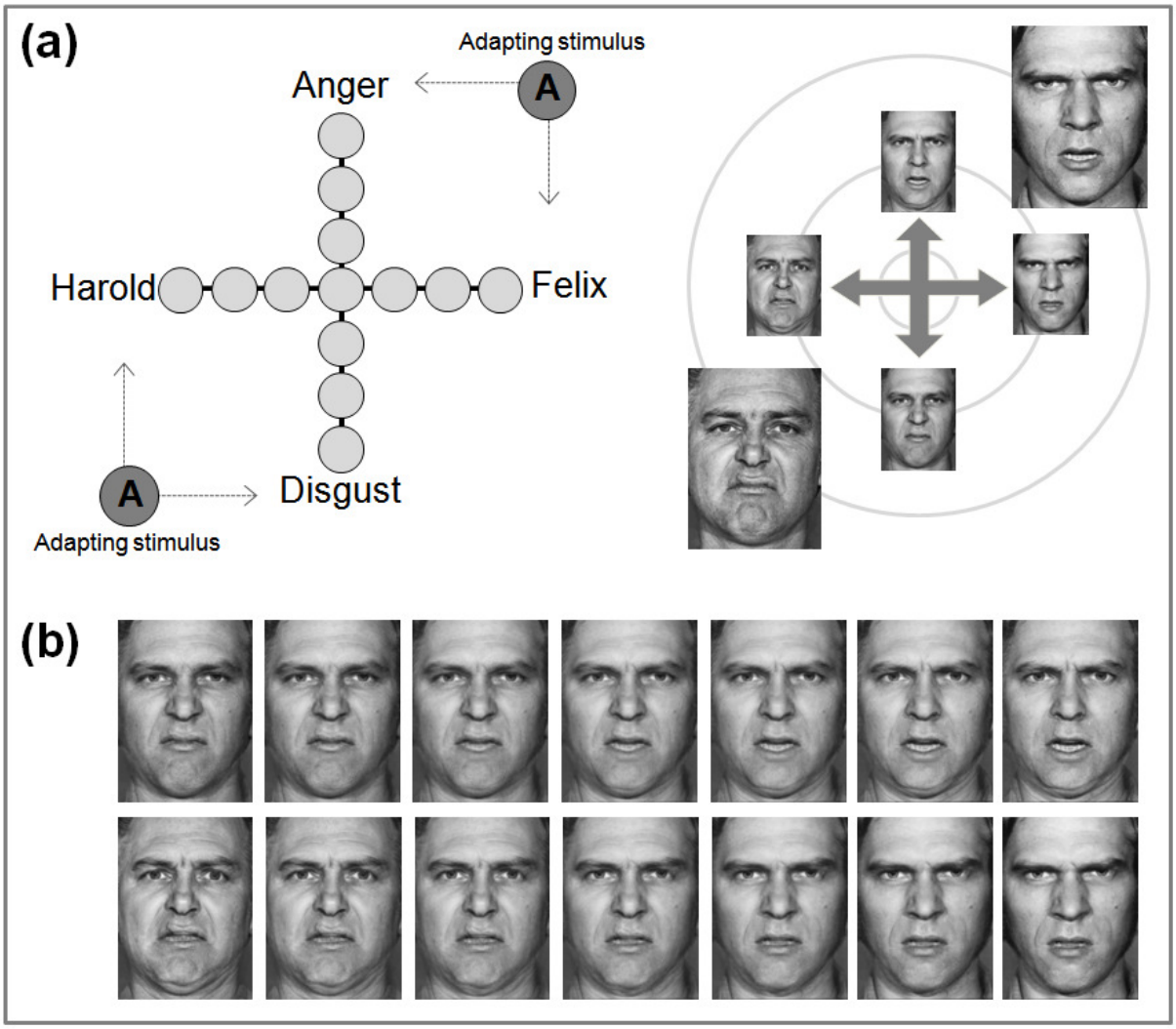

Figure 1: (a) Two morph continua were produced; an emotion continuum blending disgust and anger expressed by an average identity, and an identity continuum blending 'Harold' and 'Felix' identities expressing an average facial emotion. Participants completed four blocks of attribution trials: Judging identity having adapted to Harold expressing disgust; judging identity having adapted to Felix expressing anger; judging emotion having adapted to Harold expressing disgust; judging identity having adapted to Felix expressing anger. (b) The disgust-anger emotion continuum (top row); the Harold-Felix identity continuum (bottom row).

Figure 2:

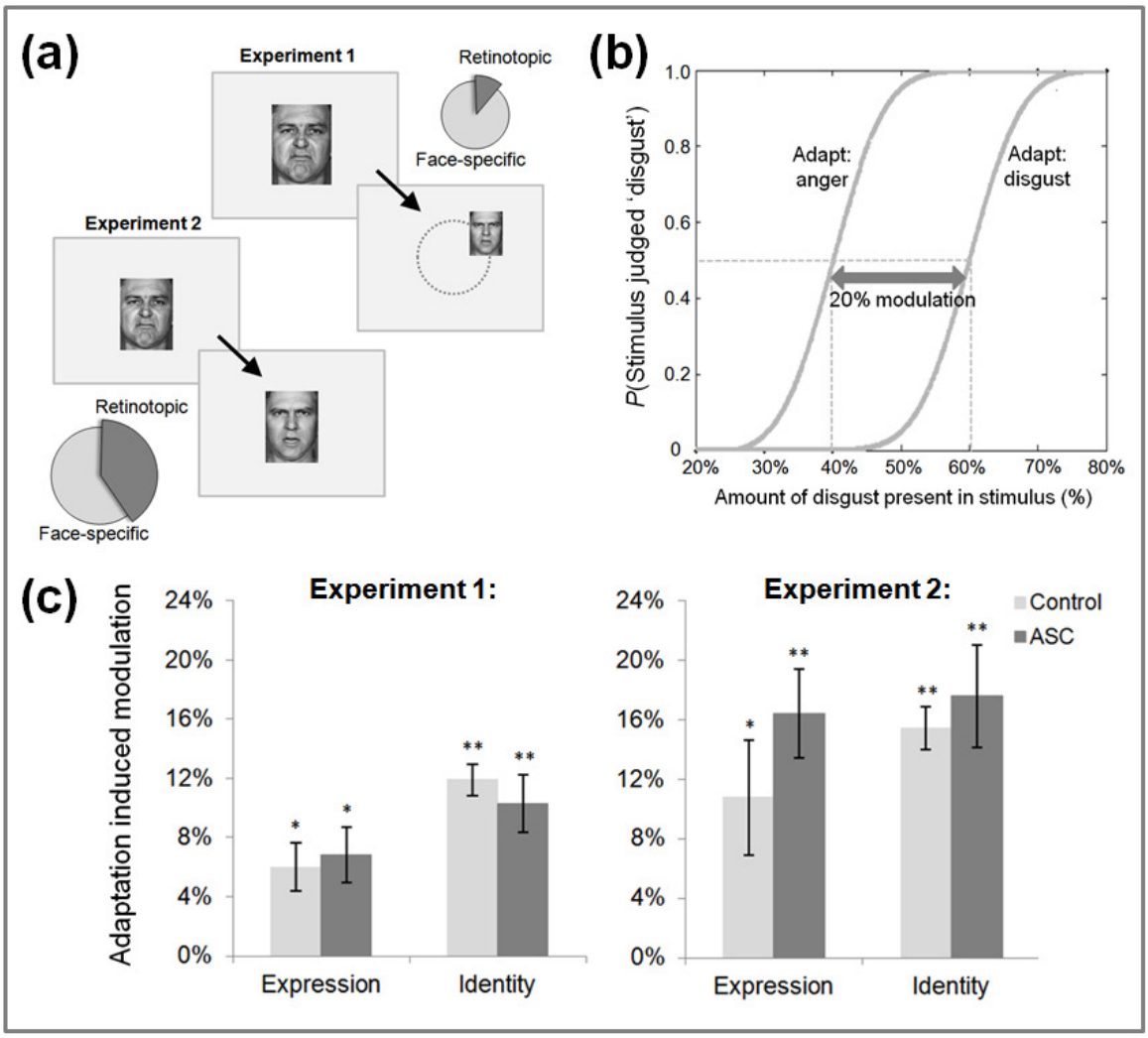

Figure 2: (a) In Experiment 1 adapting and test images were presented at different scales and locations, thereby minimizing the contribution of retinotopic adaptation. In Experiment 2 adapting and test stimuli were shown at identical scales and locations to maximize both retinotopic and face-specific adaptation. (b) The magnitude of participants' identity and expression aftereffects was inferred from the difference between the points of subjective equality (PSEs) estimated for the two identity and two expression functions. (c) In both experiments the ASD group demonstrated robust adaptation, for facial identity and expression, indistinguishable from that seen in neurotypical controls. * indicates significance at $p < .025$; ** indicates significance at $p < .001$.

TABLES:

Table 1: ADOS classification, Autism Diagnostic Observational Schedule (ADOS), Autism Spectrum Quotient (ASQ), and IQ scores, assessed with the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1997). All participants from Experiment 1 were invited to participate in Experiment 2. Twelve members of the original ASC group and two of the controls were willing and available to participate.

Participant	ADOS Classification	ADOS Total	ASQ	Full-scale IQ	Experiment
1	Autism	10	28	125	1
2	Autism Spectrum	7	46	132	1
3	Autism Spectrum	9	41	103	1 & 2
4	Autism Spectrum	8	31	108	1 & 2
5	Autism	10	38	124	1 & 2
6	Autism	7	27	125	1
7	Autism	10	26	102	1 & 2
8	Autism Spectrum	7	42	116	1 & 2
9	None	6	37	127	1 & 2
10	Autism	11	37	118	1 & 2
11	Autism	15	29	118	1 & 2
12	Autism	10	42	112	1 & 2
13	Autism Spectrum	7	36	124	1 & 2
14	Autism	11	19	105	1 & 2
15	Autism	15	18	97	1
16	Autism	11	33	117	1 & 2