Face processing in Autism Spectrum Conditions: the role of alexithymia, mood disorder symptoms and gaze behaviour

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Abstract

Autism Spectrum Conditions (ASC) are often associated with atypical gaze behaviour and face processing. Facial emotion recognition (FER) specifically appears to be impaired in ASC, although alexithymia has been argued to actually be the factor driving atypical FER. Additionally, anxiety and depression are prevalent co-occurring conditions alongside ASC, and thus may impact socio-emotional abilities. Whether it is autism, alexithymia, anxiety, or depression that is driving atypical emotion processing and gaze behaviour is the focus of this thesis. In a review of the literature, Chapter Two indicated that anxiety and depression are associated with some socio-emotional abilities in autism, but not all; however, there was a notable lack of research on the role of depression in autistic socioemotional abilities. Chapter Three demonstrated that neither autism or alexithymia, affected face processing. However, gender (specifically those identifying as neither male nor female) was significantly associated with emotion recognition, suggesting a potential link between gender diversity and emotion perception. Chapter Four indicated that face masks covering the mouth region hindered both emotion and identity recognition (more so emotion recognition) for both autistic and non-autistic participants, but that the autistic group was more affected by masks, suggesting that autistic people may draw more information from the mouth region when processing faces. Chapter Five found that autistic traits were associated with reduced gaze to the eye region of emotional face stimuli, and that alexithymia was associated with emotion recognition abilities. Anxiety and depression were not associated with face processing or gaze behaviour. Overall, the findings of this thesis indicate a connection between autism and gaze behaviour, and alexithymia and emotion recognition, but that these relationships are not consistent. Future research should focus on what other factors may be driving gaze and face processing, and which atypical socioemotional behaviours pattern with autism versus alexithymia.

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Chapter One: Introduction and Literature review

Introduction

A core feature of Autism Spectrum Conditions (ASC)[1] are impairments in social communication. Recognising facial expressions is an important aspect of socialising with others, and deficits in recognising other individuals' emotions from their facial expressions can cause difficulties in social communication, social interaction, and empathy (Loth et al., 2018). Previous research indicates that facial emotion recognition in autistic individuals differs to that of neurotypical individuals (e.g. Ashwin et al. 2006; Nomi & Uddin, 2015; Uljarevic & Hamilton, 2013), although whether individuals with ASC have a specific impairment in detecting emotional expressions in the facial region or whether another factor is responsible for these socio-emotional deficits requires further research (Sato et al., 2017).

The causes of social impairments in autism, particularly in the area of facial and emotional processing, remains debated (Tanaka & Sung, 2016). It has been argued that autistic people may not process faces holistically which leads to disadvantaged facial processing and recognition; however, other research indicates this is not accurate and that autistic people can utilise holistic strategies in facial processing (Lopez, Donnelly, Hadwin, & Leekam, 2004). Alternatively, there is evidence from eye tracking studies that suggest that atypical eye contact may hinder face and emotion recognition (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001). Research has identified several potential causes for atypical eye contact in autism; for example, some studies demonstrate that autistic people focus more on the mouth region and therefore miss social cues from the eyes (Kliemann et al., 2010; Klin et al., 2002; Pelphrey et al., 2002). In comparison, some studies suggest that individuals with ASC actively avoid direct eye contact, which may be due to anxiety or feeling uncomfortable with social interactions (Dalton et al., 2005; Kliemann et al., 2010; Kylliainen & Hietanen, 2006).

The role of eye gaze behaviour in autism and emotion recognition is particularly interesting given the recent Covid-19 pandemic. During the height of the pandemic, the wearing of face masks became widespread and face masks have been shown to hinder facial identity and emotional expression recognition (Marini et al., 2021). Face masks have also been shown to specifically negatively affect emotion recognition abilities in participants with high autistic

[1] Note: Throughout the thesis, the term "autism" and "autism spectrum condition" are used interchangeably, and "autistic person/people" is used as opposed to "person/people with autism". This is based on common clinical parlance (which still commonly refers to autism spectrum conditions/disorders) and documented preferences of autistic people (Keating et al., 2023).

traits compared to participants with low autistic traits (Pazhoohi, Forby, & Kingstone, 2021). This suggests that while face masks hinder face processing for most people, they potentially have a greater detrimental effect for those with high autistic traits or diagnoses of ASC. This could be due to autistic individuals' tendencies to avoid eye contact and look more at the mouth, a region that is naturally covered by face masks.

However, it appears that eye contact differences and face processing difficulties in autism are not universal; some autistic individuals appear to show atypical eye gaze behaviour and difficulties with emotion recognition, while others do not (Harms, Martin, & Wallace, 2010; Uljarevic & Hamilton, 2013). This suggests that there may be an important factor that determines whether emotion or eye contact problems are seen in autism.

One recent suggestion that attempts to explain this heterogeneity in autism is alexithymia. Alexithymia is characterised by the inability to identify emotions experienced by the self (Sifneos, 1973). It is frequently co-occurring with ASC (Oakley et al., 2020). Alexithymia appears to play a role in reduced eye fixation in ASC, which may be related to the social impairments associated with autism (Bird, Press, & Richardson, 2011). Cook, Brewer, Shah and Bird (2013) have suggested co-occurring alexithymia may be accountable for face perception deficits in autism, particularly poor facial emotional expression recognition.

In addition to explaining behavioural responses to emotional stimuli, alexithymia also appears to predict neural responses during emotion processing. Specifically, alexithymia has been linked to atypical activation of the anterior insula and in fMRI studies investigating empathy in ASC, hypo-activation of the anterior insula was associated with difficulties in emotional awareness (Silani et al., 2008). This suggests there may even be differences at the neural level between neurotypical individuals, those with ASC, and those with ASC and alexithymia. These findings hint further that co-occurring alexithymia in ASC may be accountable for emotion processing deficits, and that the role of alexithymia in emotion processing in ASC warrants further research as it may potentially play a causal role in emotion processing deficits in autism, and both may contribute to the social impairments associated with autism.

However, alexithymia is not the only candidate factor that could help explain heterogeneity in emotion processing and eye gaze behaviour in autism. Research has found that anxiety and depression are also associated with impaired ability to recognise facial emotional expressions in typically developed adults (Demenescu et al., 2010; Skokaukas & Gallagher, 2010;

Stewart et al., 2006), as well as being associated with reduced direct eye contact (Schneier et al., 2011; Waxer, 1974). However, there appears to be less research into the effects of depression and anxiety on facial emotion recognition and eye-gaze with autistic participants, despite these conditions being very common in autism (Honkalampi et al., 2000; Marchesi, Brusamonti, & Maggini, 2000). Importantly, alexithymia has been demonstrated to have a close relationship with anxiety and depression: it is possible that previous demonstrations that linked alexithymia to the socio-emotional difficulties in autism were confounded by the presence of depression and anxiety.

The current thesis will bring together the topics of face processing, emotion recognition, autism, alexithymia and mood disorders. The overarching aim of this thesis is to investigate the roles of alexithymia and mood disorder symptomatology in face processing and eye gaze behaviour differences in autism. Prior to main chapters, relevant background theories are presented in the literature review below. This introductory chapter ends with a detailed outline of the structure of the remaining thesis chapters.

Emotion recognition and ASC

Facial emotion recognition has been shown to be impaired, at a group-level, in ASC. Uljarevic and Hamilton (2013) conducted a meta-analysis of 48 studies and found a large effect size for studies investigating emotion recognition on tasks using both static and dynamic facial stimuli with participants with ASC ranging from 6 to 41 years of age (although it should be noted that after correcting for publication bias, this effect size became a moderate effect size rather than a large effect size). This effect size indicated poorer performance in facial emotional expression recognition in comparison to typically developed participants. Yeung (2022) more recently conducted a meta-analysis of 148 studies focusing on facial emotion recognition in ASC, and similarly to Uljarevic and Hamilton (2013), they also found a significant impairment in facial emotion recognition in autism. They found that emotion recognition was impaired across all basic emotions and that this impairment was present across modalities (e.g. voice, body), as well as this presentation of impairment being unique to ASC compared to other clinical conditions (e.g. Williams Syndrome, conduct disorder).

Uljarevic and Hamilton (2013) also investigated the effect of IQ and age on effect size and found no significant effect, suggesting facial emotion recognition deficits in ASC occur across a range of ages and intelligence. This has been supported by research by Rump et al.

(2009) who conducted research using a dynamic facial emotion recognition task (short clips showing models with emotional facial expressions) with participants aged from 8 to 53 years old with the aim of investigating emotion recognition in ASC across different ages. They found that in the control group, performance increased with age but this was not true for the ASC group as the adults in the ASC group performed similarly to the autistic children. This suggests that individuals with autism do not continue to develop and refine their facial emotion recognition abilities through to adulthood, and that facial emotion recognition can be impaired across the lifespan of individuals with ASC.

However, while much research indicates atypical emotion recognition in ASC, there have been inconsistent results. Some studies have found that in groups of individuals with ASC without intellectual disability, facial emotion recognition does not seem to be impaired (Adolphs et al. 2001; Loveland et al. 2008; Neumann et al. 2006; Ogai et al. 2003; Rutherford & Towns 2008). This appears to be especially true for autistic individuals without intellectual disabilities as evidence indicates that facial emotion recognition is not as poor as other research suggests or is comparable to neurotypicals (Baron-Cohen et al. 1997; Teunisse & de Gelder 2001). The inconsistent results are likely due to the heterogeneity amongst autistic individuals as well as differences between the methodology and tests used within studies (Loth et al., 2018). For example, emotion recognition difficulties may not be even across all emotion categories: many studies have found that adults with ASC have a reduced accuracy in identifying emotions, particularly negative emotions (Ashwin et al. 2006; Bal et al. 2010; Corden et al. 2008; Howard et al. 2000; Wallace et al. 2008). This is supported by the meta-analysis by Uljarevic and Hamilton (2013) which found that recognition of happiness was less impaired than the recognition of fear in autistic individuals.

Additionally, some autistic individuals may be able to compensate for any deficits in emotion recognition and face processing in certain situations. Harms, Martin, and Wallace (2010) suggest that compensatory mechanisms might aid certain autistic individuals with facial emotion recognition but that the processing of the facial emotions may remain atypical and that the individuals may only be able to use these compensatory mechanisms on certain tasks and that real-world emotion recognition may still be impaired.

While research indicates that the majority of individuals with ASC have deficits in facial expression recognition, further research is required to establish the differences between those individuals with ASC displaying facial emotion recognition impairments and those who do

not display these impairments. Establishing factors that may differentiate these groups may reveal factors that cause these impairments, such as atypical neurological processing, other symptoms of autism, or other factors such as alexithymia or mood disorders.

Face processing in neurotypicals and ASC: identity and familiarity

When we come into contact with another person, a set of interrelated mechanisms are used to process the person's identity and emotional expression, and how we process their face may depend on whether the person is familiar or unfamiliar to us. It is important to consider the extent to which problems with emotion recognition in autism are reflective of a specific difficulty with emotions versus being situated in a more general problem with face processing. This is because factors that attempt to explain heterogeneity in emotion processing (e.g. alexithymia, mood disorder symptomatology) may need to consider whether relationships to predictive factors are specific to emotion processing or extend to identity processing as well. A brief summary of theories regarding identity versus emotion recognition is thus made here, with a consideration of the role of familiarity in face processing (note: all stimuli used in the experimental chapters utilise faces that would have been unfamiliar to the participants). I then consider the evidence with regards to identity processing and the effect of familiarity in autism.

Face recognition (both identity and expression) are both vital components of social interaction. Theories of face perception suggest that face identity and facial expression are processed by distinct mechanisms (Calder & Young, 2005). Furthermore, the processing of unfamiliar and familiar faces also appears to have different mechanisms which suggests that familiar and unfamiliar faces are processed differently. Natu and O'Toole (2011) state that familiar faces are represented with 'rich visual, semantic, and emotional codes' that are efficiently and quickly perceived and recognised, while unfamiliar faces challenge memory systems and therefore take up different processes and are less effortless. The notion that unfamiliar and familiar face recognition may have different neural processes is also supported by neuroscientific research indicating different areas of brain activation for processing familiar and unfamiliar faces. For example, a PET study by Rossion et al. (2001) found different activation patterns when perceiving unfamiliar and familiar faces in the right middle occipital gyrus, the right inferotemporal cortex, and the right posterior fusiform gyrus. Additionally, research has identified that specific neural areas associated with face processing

are involved in discriminating unfamiliar and familiar faces (Johnston & Edmonds, 2009; Rossion, Schiltz, & Crommelinck, 2003).

The extent to which deficits in emotion or identity processing are observed in autism may also depend on the familiarity of the faces. Research has indicated that individuals with autism, especially children, may have particular deficits in recognising unfamiliar faces (Boucher & Lewis, 1992). Neuroimaging evidence shows that the fusiform gyrus activates when shown a familiar face in a more similar pattern to neurotypicals but shows a different pattern of activation when viewing unfamiliar faces (Aylward et al. 2004; Pierce et al. 2004; Pierce & Redcay, 2008). However, a lack of fusiform gyrus activation for unfamiliar faces may be the result of the lack of familiarity with novel stimuli used in experimental tasks as the fusiform gyrus has been shown to specialise in the processing of areas of expertise, not just face expertise (Gauthier et al. 1999; Marcus & Nelson 2001; Nelson, 2001).

With regards to identity processing in autism, the evidence base suggests that autistic individuals, as a group, have poorer identity processing abilities. Griffin, Bauer, and Scherf (2021) conducted a meta-analysis of 112 studies regarding face recognition issues in autism and found deficits for face recognition and discrimination, and that these were present across tasks, IQ, age, and gender. This suggests a face recognition impairment in autism. However, Weigelt, Koldewyn, and Kanwisher (2012) reviewed behavioural studies and found that there was no evidence for differences in how facial identity is processed for autistic individuals, but there appears to be differences in how well faces are remembered, suggesting the impairment may be memory-based rather than recognition/process-based. Furthermore, Cook et al. (2013) investigated identity recognition (alongside emotion recognition) and found that neither autism nor alexithymia predicted identity recognition abilities in autistic participants. Potentially, other factors than autism may be driving differences in face recognition, and further research could aid in establishing the exact nature of these observed differences in face recognition, memory, and processing.

Atypical Eye Gaze in ASC

One proposal that attempts to explain atypical face processing (of both emotion and identity) is that autistic people show differences in where they look when processing faces. Abnormal eye gaze direction and duration is associated with autism, such as a shorter duration of eye gaze towards the eye region of faces (Papagiannopoulou et al., 2014), and abnormal eye contact has been associated with emotion recognition impairments in autism (Cuve, Gao &

Fuse, 2018). Studies have shown that individuals with autism attend less to the eye area of the face and that even when the eyes are attended to, the information is not used as effectively for emotion recognition in comparison to neurotypical controls (Corden et al. 2008; Baron-Cohen et al. 1997; Gross 2008; Spezio et al., 2007a; Spezio et al., 2007b b; Pelphrey et al. 2002). Reduced eye contact likely affects social communication and facial processing, including facial emotion recognition, contributing to the social impairments associated with ASC (Davies et al., 2011; Kuusikko et al., 2009; Neumann, Spezio, Piven & Adolphs, 2006).

In addition to reduced looking to the eyes, researchers have posited that increased looking at the mouth region could also impact emotion recognition in autism. Wieckowski and White (2017) conducted an eye tracking study on youth with ASC investigating eye gaze and emotion recognition in social scenes. They found that the participants both were worse at recognising negative emotions (specifically disgust and sadness) and showed less fixation to the eye region than controls. The autistic participants also showed more fixation to the mouth region, which Wieckowski and White (2017) suggest may be the marker for poorer emotion recognition. Similarly, research by Black et al. (2020) found that longer fixation to the mouth region was associated with impaired facial emotion recognition ability in autistic adults. However, Black et al. (2020) noted that while atypical eye gaze behaviour is associated with poorer facial recognition in ASC, previous research has provided inconsistent findings to whether it is altered eye gaze behaviour to the eye or mouth region that affects emotion recognition (or whether it is a combination of both) or if impaired facial emotion recognition and abnormal eye gaze is only associated with the processing of basic emotions (commonly used as stimuli in research) and that eye gaze behaviour differs when viewing more complex emotions in everyday social settings.

Notably, these looking differences appear to expand to both familiar and unfamiliar faces. Sterling et al. (2008) conducted an eye tracking study and found that individuals with ASC do not use ‘normative’ gaze patterns when shown familiar faces. For both familiar and unfamiliar faces, participants with ASC paid less attention to the eyes and spent less time viewing them compared to typically developed participants. This suggests autistic people show atypical eye gaze and facial processing for both unfamiliar and familiar faces.

Alexithymia in emotion recognition and eye gaze behaviour in ASC

As described above, while meta-analyses suggest differences in emotion recognition abilities between autistic and non-autistic people at group level, there seems to be a wide variety of

emotion recognition ability amongst autistic people. Recent research suggests that alexithymia may play a role in the heterogeneity of facial emotion recognition in ASC.

Alexithymia is characterised by the inability to identify emotions experienced by the self (Sifneos, 1973), and encompasses difficulties distinguishing between emotions one might be experiencing (e.g. struggling to tell whether one is feeling angry or sad), trouble describing emotions and putting one's feelings into words, and tending to avoid thinking about one's emotions and emotional material. Alexithymia is elevated in a number of different psychiatric and neuropsychological populations and has been described as a transdiagnostic risk factor for poor mental health, potentially via alexithymia's effects on emotion regulation (Preece et al., 2022).

Alexithymia is frequently co-occurring with ASC. Oakley et al. (2020) report that many individuals with ASC report higher levels of alexithymia, with 47.3% of autistic females and 21.0% of autistic males meeting the cut-off for clinically relevant alexithymia. In particular, difficulties in describing feelings were associated with self-reported social communication difficulties, and difficulty identifying feelings were associated with anxiety symptom severity. Emotional processing deficits in ASC may reflect co-occurring alexithymia (Bird & Cook, 2013; Kinnaird et al., 2019). Indeed, while alexithymia is common in ASC, it is not universal: the heterogeneity of alexithymia in autism could account for the heterogeneity of emotional abilities in the autistic population.

Indeed, research by Cook et al. (2013) investigated the role of alexithymia in facial emotion recognition in adults with ASC. They used static facial stimuli in a task designed to measure both emotion recognition and identity abilities, which meant that participants would be unaware whether they would be attributing emotion or identity to the stimuli during the task, reflecting face processing outside of experimental conditions where attention to both identity and emotion recognition is required. They found that autism severity was unrelated to expression-recognition ability while alexithymia predicted facial emotion recognition abilities (though not identity processing abilities). This suggests co-occurring alexithymia may be responsible for impaired facial emotional expression recognition in ASC.

Further evidence shows that alexithymia negatively affects emotion recognition. Donges and Suslow (2015) conducted a facial emotion recognition and memory task using unfamiliar faces with a range of negative, positive, and neutral expressions. They found that the difficulty describing feelings module of the TAS-20 (Toronto Alexithymia Scale, Bagby,

Parker, & Taylor, 1994) was inversely correlated with correct recognition of negative emotions and that participants performance on the recognition and memory task was associated with their alexithymia scores. While this demonstrates that alexithymia may affect both memory and facial emotion recognition, this study was conducted on neurotypical participants. It may well be likely that alexithymia affects emotion recognition similarly in clinical populations, such as those with ASC, but further research is required to establish this.

However, there is some evidence that alexithymia may not be solely responsible for atypical emotion recognition. Keating et al. (2022) examined emotion recognition and autism using a paradigm that manipulated spatial and kinematic aspects of the stimuli. The findings indicated that alexithymia was not associated with emotion recognition accuracy, and that the autistic participants demonstrated reduced recognition of angry stimuli (and autistic traits were a significant predictor of anger recognition accuracy). Alexithymia was associated with higher ratings (based on intensity of emotions the participants thought the stimuli were feeling) of incorrect and correct emotions, indicating that alexithymia does play a role in atypical emotion processing, but that potentially autistic traits may be driving reduced recognition abilities (at least for anger). Again, this highlights the need for further research in order to establish the nature of the relationships between alexithymia, autism, and emotion recognition.

The role of alexithymia on eye gaze and emotion recognition in ASC has also been investigated. ASC is associated with reduced eye contact and reduced fixation on faces, particularly the eye area. However, eye tracking studies have found mixed results for reduced eye fixation in autistic individuals when looking at social scenes, which Bird, Press, and Richardson (2011) suggest may be due to co-occurring alexithymia. They found that the degree of alexithymia, not autism severity, predicted eye gaze and eye fixation when autistic adults were watching video clips of faces. They suggest that individuals with both ASC and alexithymia may have a unique set of emotional and social impairments in comparison to individuals with just ASC.

This is supported by more recent research by Cuve, Gao, and Fuse (2018), who conducted a systematic review investigating emotion recognition and eye-tracking in young adults with autism spectrum conditions. They also determined that evidence was inconsistent, with some studies reporting overall reduced attention to the eyes but not always being associated with poorer facial emotion recognition, while other studies reported emotion recognition deficits

without atypical eye gaze and fixation. Cuve, Gao, and Fuse (2018) also concluded that alexithymia may modulate eye gaze and emotion recognition in ASC.

More research is needed to understand co-occurring alexithymia and ASC, and the implications alexithymia may have on emotion recognition, emotion awareness, and social communication abilities. Indeed, better understanding of alexithymia in autism has implications for supporting autistic people: Oakley et al. (2020) suggest that psychological therapies regarding emotional awareness may improve social communication and symptoms of anxiety in individuals with ASC. This is supported by Kinnaird et al. (2019) who suggest that autistic individuals with alexithymia may benefit from interventions tailored to their emotional difficulties. For such interventions to be effective, more evidence is needed regarding the impact of alexithymia on emotional processing.

Mood disorders and emotion recognition in ASC

Mental health conditions such as depression and anxiety are common in autism, the lifetime prevalence being estimated at 42% for anxiety and 37% for depression (Hollocks et al., 2019; Skokauskas & Gallagher, 2010). With mood disorders such as anxiety and depression being highly prevalent in ASC, it is likely they have an effect on behaviour, social and communication abilities, and life experiences.

For example, in neurotypical individuals, mood disorders have also been shown to have an effect on facial emotion recognition. Demenescu et al. (2010) found that adults with anxiety displayed a significant impairment in emotional expression recognition (Cohen's $d = -0.35$) and that depression was associated with a larger impairment ($d = -0.58$). Additionally, Torro-Alves et al. (2016) found that individuals with high social anxiety had poorer facial emotion recognition particularly in ambiguous and low intensity presentations of facial emotional stimuli. If depression and anxiety are associated with impairments in facial emotion recognition in typically developed individuals, it likely plays a role in these deficits in ASC.

White et al. (2018) conducted a review on psycho-social treatments for children and adolescents with ASC, anxiety and depression. They discussed a link between social communication, social interest, and facial emotion recognition impairments to anxiety and depression in ASC. They found that psychosocial treatments such as mindfulness-based treatments (e.g. including hobbies of special interest to increase social interest) treating symptoms of anxiety and depression were found to improve social behaviour and emotional

awareness. This suggests that anxiety and depression may be an underlying cause of social communication deficits in ASC including facial emotion recognition and that adapted interventions for anxiety and depression for use with autistic individuals may alleviate some social impairments.

The cognitive mechanisms behind the relationship between mood disorders and emotion recognition has been investigated. Atypical emotion processing in mood disorders, such as anxiety and depression, has been shown to be related to attentional biases towards negative stimuli (Armstrong & Olatunji, 2012). Vazquez et al. (2016) conducted an eye tracking study with depressed participants and found a sustained attention to negative information when shown emotional stimuli and faster disengagement from positive stimuli. This demonstrates that depression may cause atypical processing of emotional stimuli, and this likely includes emotional faces.

Negative attentional biases have also been identified in ASC. Zhao, Zhang, and Maes (2016) investigated attentional biases to disgusted faces with children with ASC. They found that the children with ASC attended to the disgusted faces quicker compared to neutral and happy faces, and that they also displayed a tendency to avoid looking at where the face stimuli where presented after initial engagement. This indicates that negative emotional faces are processed differently to neutral and positive emotions, but that for all facial expressions there was a tendency to avoid looking at the stimuli, which may relate to the atypical eye contact observed in individuals with ASC.

Research has also indicated biases to negative emotional stimuli in adults with ASC. Unruh, Bodfish, and Gotham (2020) compared adults with ASC, typically developed adults with depression, and never-depressed controls and found that both the clinical groups oriented quicker to negative socio-emotional stimuli. This supports previous research that depression causes atypical processing of emotional stimuli (particularly negative stimuli) but also demonstrates that ASC may have the same effect. This is supported by previous research in facial emotion recognition that suggest that while facial emotion recognition is poorer for all emotions, negative emotions such as disgust and sadness seem to be particularly affected with poorer recognition (Ashwin et al. 2006; Bal et al. 2010; Corden et al. 2008; Howard et al. 2000; Wallace et al. 2008; Wieckowski & White, 2017; Zhao, Zhang, and Maes, 2016). However, it should be noted that depression has a high co-morbidity rate with ASC and there are common symptoms between ASC and depression, such as poor sleep, repetitive cognition

and decreased sociability, so it is possible that co-morbid depression may create these biases to negative emotions in autistic individuals.

While there is research establishing the prevalence of anxiety and depression, there is less research specifically investigating the effect of both depression and anxiety on facial emotion recognition abilities in autistic individuals. Potentially, depression and anxiety will affect facial emotion recognition in a similar way to non-autistic individuals but whether they have an independent effect or exacerbate existing facial emotion recognition deficits in ASC requires investigation (Oakley et al., 2020).

Are mood disorders or alexithymia responsible for emotion recognition deficits in ASC?

Whether alexithymia or mood disorders are responsible for atypical facial emotion recognition in autism is yet to be established. There is evidence that non-autistic individuals with alexithymia and co-occurring depression or anxiety have lower emotional intelligence, which may link to impaired facial emotion recognition abilities (Onur et al. 2013). Also, research indicates that alexithymia and mood disorders may have a close relationship as alexithymia may be related to difficulties with emotion regulation (Morie et al., 2019; Venta et al. 2013). In particular, alexithymia has been shown to be high in those with ASC and anxiety and/or depression (Honkalampi et al. 2000; Karukivi et al. 2010), and the relationship between alexithymia and mood disorders has also been shown in neurotypical populations (Morie et al., 2019).

As research demonstrates that ASC, alexithymia, and mood disorders have overlapping symptomology/characteristics and are often co-occurring, the distinctiveness of the traits of these conditions and how they can be effectively measured is questioned. For example, Stewart et al. (2006) noted that depression can be difficult to diagnose in ASC due to some overlapping characteristics, such as social withdrawal and sleep problems. This highlights the need to adapt diagnostic measures for use with autistic patients (Gotham, Unruh, & Lord, 2015; Cassidy et al., 2018). In contrast, research by Marchesi, Brusamonti, and Maggini (2000) has clarified that alexithymia, anxiety and depression are separate constructs through a factor analysis of the TAS-20 (Toronto Alexithymia Scale; Bagby, Parker, & Taylor, 1994) and the HADS (the Hospital Anxiety and Depression scale, Zigmond & Snaith, 1983). They concluded that while they are closely related, they are distinct and that the independent effects of alexithymia, anxiety, and depression on emotion recognition in autism requires further research. Similar recent factor analytic approaches have supported the same result for

autistic traits and alexithymia: while they are highly correlated, they are argued to be conceptually distinct (Cuve et al, 2021).

While evidence suggests alexithymia and mood disorders are related to impairments in facial emotion recognition, more research is needed to establish the independent effects alexithymia, mood disorders, and traits of ASC have on facial emotion recognition as currently the independent effects have not been thoroughly established. Additionally, research into alexithymia and mood disorders in autism will provide insight on the implication's alexithymia, anxiety, and depression may have on individuals with ASC and whether they may benefit from tailored interventions (Kinnaird et al., 2019). Furthermore, if research indicates that co-morbid depression and/or anxiety modulates poorer facial emotion recognition in autistic individuals, then interventions and therapies targeting anxiety and depression such as CBT may benefit the individual and aid in addressing social impairments resulting from symptoms of mood disorders.

While the notion that alexithymia may explain atypical facial emotion processing in autism has begun to be explored, the effect of co-morbid depression and anxiety in ASC on emotion recognition must be considered as there are high prevalence rates of these co-morbid conditions in ASC and further research is needed to establish the effects that depression and anxiety have on emotion processing in ASC (Oakley et al., 2020). Therefore, examining whether it is co-occurring alexithymia or a co-morbid mood disorder that plays a role in atypical facial emotion recognition and eye gaze behaviour in ASC warrants further investigation.

Mask wearing and impaired emotion recognition

As a result of the Covid-19 pandemic, mask coverings became common place in everyday life in order to reduce the transmission of the coronavirus. Whether it be a medical mask or personalised cloth covering, it was commonplace to interact with people whilst wearing face masks covering the lower part of the face. Covering parts of the face, particularly the mouth, hinders the recognition of facial expressions as you are having to solely rely on information from the eyes and the social context.

Previous pre-pandemic research highlighted the importance of the mouth in recognising emotions, finding that when stimuli are presented missing the mouth region, reactions times are significantly longer than when the full face is presented (Koch, 2005). Indeed, the

'Bubbles' technique (originally developed by Gosselin and Schyns, 2001) has been used to identify regions of the face that are most frequently used when recognising emotional expressions, and have indicated that the mouth region was the dominant facial feature that was attended to by participants (Blais et al., 2012). Additionally, evidence from research using more ecologically valid stimuli has been conducted. Fischer et al. (2012) investigated facial emotion recognition with faces wearing a niqab. They found that the perception of emotions was affected by the absence of facial information (as only the eyes were visible) and that in particular there was less perception of happiness which they suggested was due to the fact that happiness is most commonly expressed by a smile, which was not visible. This highlights the importance of the mouth region in recognising specific emotions, such as happiness. These studies indicate the importance of the mouth region and suggest that mask wearing will likely hinder emotion recognition significantly.

Research conducted with stimuli wearing face masks bears out these predictions. More recently, Marini et al. (2021) investigated the effect of masks on emotion and identity recognition, and found that emotion attribution and identity recognition (particularly re-identification of a masked individual) are compromised by mask wearing. Carbon (2020) investigated whether wearing face masks affects emotion recognition. Their participants reported lower confidence in their assessment of the face mask wearing stimuli in comparison to the full-face stimuli, and were less accurate in recognising the masked stimuli. Carbon (2020) states that as the mask covers an area of the face that is highly relevant for effective non-verbal emotional expression communication and recognition, emotion recognition and emotion differentiation can be heavily compromised.

Mask wearing and impaired emotion recognition in ASC

It has been demonstrated that mask wearing and covering the lower half of faces affects face processing in neurotypical populations, but face masks likely have even more implications for facial emotion recognition in clinical populations such as autism. As mask wearing was part of everyday social interactions, it is likely that is has had a negative effect on social communication in autistic individuals, who may already experience impairments in social abilities such as communication, empathy, and facial emotion recognition (Loth et al., 2018).

However, specific research into the effects of the pandemic and mask wearing on clinical populations is ongoing and there are some preliminary findings. Gehdu, et al. (2023) investigated emotion recognition, mask wearing, and alexithymia in autism, and found that

alexithymia, not autism, was associated with poorer emotion recognition when presented with whole or partial face stimuli (occluded by masks).

However, these results differ to previous research suggests that autism may also play a role when the face is occluded. Using the 'Bubbles' technique to only show parts of the face, it has been shown that participants with ASC focused less on the eyes region and relied on the mouth region for identifying emotions (Spezio, Adolphs, Hurley & Piven, 2007a, 2007b). Results showed that participants were comparable to controls in terms of emotion recognition accuracy but used different strategies in facial processing by avoiding the eyes and focusing on the mouth. This suggests that some autistic individuals can make use of information from the mouth in emotion recognition and while it may be atypical, it is not necessarily impaired. However, when it comes to wearing face masks and covering the mouth region, emotion recognition may be impaired in autistic individuals because of the reliance on the mouth region in their face processing strategies. Alternatively, the role that alexithymia plays in atypical facial emotion recognition cannot be dismissed, and thus research should focus on determining casual relationships between facial emotion recognition, autism, and alexithymia when face masks are present, as potentially the observed effects of alexithymia may differ when participants face processing is disrupted (for example, by face masks).

However, there is limited research on the extent to which alexithymia rather than autism may explain particular detrimental effects of face masks: if alexithymia rather than autism drives atypical looking patterns to the eyes versus mouth, then face masks may only exert additional problems for autistic people when alexithymia is present. Thus, further research is needed on the effects of mask wearing on emotion recognition and social impairments in ASC, and the role of alexithymia.

Goals of the Current Thesis

The foremost aim of the current thesis was to investigate the impact of alexithymia, autism/autistic traits, and mood disorders (namely anxiety and depression) on the face processing abilities of those with and without autism, specifically their facial emotion recognition abilities. Whilst facial emotion recognition was the main focus of our investigation due to previous literature indicating atypical facial emotion processing in autism, facial identity recognition abilities were also examined in every study to assess whether differences and/or deficits in face processing or associations with alexithymia/mood disorder symptomatology were driven by a general face processing issue in

autism/alexithymia (so involving both emotion and identity recognition), or was domain-specific to either emotion or identity recognition. Alongside this main area of inquiry, the effect of these alexithymic and autistic traits on gaze behaviour was also examined, and in a separate study the impact of face masks on face processing was explored.

Chapter Two aimed to specifically review the literature surrounding the impact of anxiety and depression on socio-emotional abilities in ASC. This chapter was conducted as a narrative review examining empathy, theory of mind, emotion recognition, and emotion regulation, and how they are (or are not) associated with autism, mood disorders, and alexithymia. Anxiety and depression are commonly co-occurring alongside ASC, and socio-emotional abilities are often atypical or impaired in ASC. There is evidence to suggest that anxiety and depression may be associated with atypical socio-emotional abilities in autism, but whether they play a causal or mediating role is of interest as this could indicate areas where interventions and support may be useful. Additionally, while there is a lot of research regarding anxiety and autism, more research is needed for depression and its potential impact on the socio-emotional cognition and wellbeing of autistic individuals. Outlining areas for future research is important, as this can aid in establishing causal relationships, the effectiveness of current support, and the design of new interventions.

Chapter Three aimed to examine facial emotion and identity recognition and the effects of autism, alexithymia and mood disorders, via a registered report. Emotion recognition appears to be atypical for some of the autistic population, and it was previously thought that emotion recognition deficits were influenced by ASC, but recent evidence suggests that alexithymia may be the factor driving this atypical emotion processing. It is important to investigate the role of alexithymia as while it is not classified as a condition, it does appear to occur alongside autism and other mental health conditions; indeed, our registered report examined the potential role of anxiety and depression symptoms in face processing abilities. Identifying the potential effects of alexithymia and/or mood disorder symptoms on socio-emotional cognition may help with the design and tailoring of appropriate interventions for autistic people.

Chapter Four was conducted within the context arising from the Covid-19 pandemic. Face masks and coverings had previously been more common in medical and dental settings, and the addition to the general population having to use them on a daily basis was quite novel, and the impact unknown. As face masks were part of daily life when the study was devised,

the impact of using face masks during social situations was of interest, particularly for clinical populations. Thus, the study in Chapter Four aimed to examine the impact of face masks on facial emotion and identity recognition, in particular whether autism and/or alexithymia had an amplifying effect on the detrimental impacts of face masks. Assessing the impact of face masks on clinical populations is important in informing future practise, as identifying areas that some may find difficult to wear face masks may help in designing alternatives, for example the use of clear face masks in medical settings have been shown to improve patient understanding and comfort, and so this may also benefit other situations. It should be noted that the roles of anxiety and depression were not examined in this study.

Chapter Five aimed to examine the roles of autistic traits, alexithymia, and mood disorder symptoms on eye gaze behaviour during face processing. Atypical gaze behaviour has often been observed in autistic individuals, and it is suggested that some autistic individuals may look more to the mouth region during face processing as they can avoid the 'social pressure' of the eyes and still gain information regarding the emotional state of the individual from the mouth, e.g. smiling. Recently, alexithymia has been identified as a potential factor behind atypical gaze behaviour in facial emotion processing, indicating that alexithymia does not just affect emotion recognition, but potentially also the cognitive processes driving emotion processing and socio-emotional behaviour. Atypical gaze behaviour, autism, and alexithymia is an important route of investigation as unravelling the intricacies of these factors and their relationships may aid in indicating whether it is alexithymia or autism (or mood disorders) driving the atypical gaze and face processing. Identifying the factors behind differences in gaze and emotion processing may then help establish and tailor interventions and support for individuals, for example targeting alexithymia or mood disorders may ameliorate other symptoms and difficulties. It should be noted that a general population sample was used, and so autistic traits were measured, rather than examining groups with/without an autism diagnosis.

The thesis concludes with a general discussion drawing together the findings and conclusions of the empirical and theoretical chapters, with my reflections and thoughts of future directions for research.

Chapter Two: The impact of depression and anxiety on socio-emotional abilities in autism spectrum conditions - a narrative review.

Declarations

Competing interests

The authors have no competing interests to declare.

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Author's contributions

Natasha Baxter made substantial contributions to the conception and design of the review, and drafted the review. Aneta Kruczek and Emily Sullivan were volunteer research assistants who assisted with searching and screening the literature. Hannah Hobson made substantial contributions to the conception and design of the review, searching and screening the literature, and provided feedback/edited the paper. Dr Felicity Sedgewick made contributions to the conception and design of the review. Both authors (NB and HH) approved the submitted manuscript.

Abstract

Anxiety and depression are common co-morbid conditions alongside autism spectrum conditions (ASC). In non-autistic individuals, there is some evidence that anxiety and depression is associated with atypical socio-emotional abilities, such as emotion regulation, emotion recognition, empathy, and theory of mind. This narrative review aimed to examine these socio-emotional abilities and their relationships with anxiety and depression in autistic individuals. However, alexithymia is often associated with atypical emotion processing in ASC, and so the influence of alexithymia is also considered. This review indicates that anxiety is associated with emotion regulation, empathy, and theory of mind. Depression appears to have associations with theory of mind and empathy. However, depression, ASC, and their relationships with anxiety and depression appears to be an area requiring more direct investigation, especially into the effectiveness of therapies. Alexithymia seems to play a role in atypical empathy, theory of mind, and emotion recognition, but further research would be useful in clarifying whether alexithymia plays a causal, mediating, or other role in conjunction with ASC, anxiety, and or depression. Future research should aim to ascertain the nature of the relationships between ASC, socio-emotional abilities, alexithymia, anxiety, and depression which will benefit the design of effective and tailored interventions and support for individuals with ASC and anxiety and/or depression.

Introduction

Depression and anxiety greatly impact a person's quality of life (Hansson, 2002). In addition to the direct impact of the symptoms of depression and anxiety themselves, these conditions can affect multiple areas of cognitive and emotional functioning (Langarita-Llorente and Gracia-Garcia, 2019; Stuhrmann, Suslow, and Dannlowski, 2011). Autism spectrum conditions (ASC) have high co-morbid rates of anxiety and depression, with prevalence rates being estimated across the lifespan at 42% for anxiety and 37% for depression (Hollocks et al., 2019; Skokauskas, & Gallagher, 2010). While differences in various aspects of socio-cognitive processing are considered typical and even diagnostic of autism (e.g. differences in emotion recognition, difficulties with theory of mind), the role of mood disorders in socioemotional abilities in autism is unclear. Potentially, in addition to the impact that mood disorders have autistic individuals' wellbeing, these conditions could also have a causal or at least amplifying role in the social and emotional difficulties many autistic people experience.

If depression and anxiety could be contributing significantly to the socioemotional profile we associate with autism, then these conditions would need to be highly prevalent across the lifespan, as socio-emotional difficulties are reported amongst autistic people of all ages, and autism is typically diagnosed in childhood (though adult diagnoses are increasing; see Huang, Arnold, Foley, & Trollor, 2020). Indeed, anxiety and depression appear to be highly prevalent in both autistic adults and children. Current prevalence rates for autistic adults suggest that approximately 23% have depression and 27% have anxiety disorders (Hollocks et al., 2019). In autistic children, several studies have identified high rates of anxious and depressive characteristics. Mayes et al. (2011a) found high levels of parent-reported symptoms of depression and anxiety in their autistic children, with 62% of 1-5 year olds being described as anxious and 28% being described as depressed, 76% of 6-10 year olds being described as anxious and 48% as depressed, and 89% of 11-18 year olds being described as anxious and 72% being described as depressed. Children with 'high-functioning autism' (term used within the study) were found to have significantly higher rates of anxiety and depression than those with 'low-functioning' autism (term used within the study), although percentages were still relatively high with 88% of 'low-functioning' 11-18 year olds being anxious and 58% being depressed. Vasa et al. (2013) also found that levels of autistic children and adolescents with anxiety was higher than non-autistic peers, with 26% and 41% of autistic school age and adolescents showing anxious symptoms compared to up to 8.6% in non-autistic equivalents. Depression was also significantly associated with anxiety in each age group, indicating that

many autistic youths have high levels of both anxiety and depression symptoms. Overall, this highlights the high prevalence of depression and anxiety in ASC across the lifespan, and the potential impacts of these high levels on the broader socio-cognitive profile of autism warrants investigation.

A range of social and emotional abilities appear to be affected in autism. Theory of Mind (ToM) is one of these abilities, defined as the ability to understand other's beliefs, emotions and actions, an important skill for communication and socialising with others (Frith & Happé, 1994). Research does indicate ToM abilities differ between autistic and non-autistic individuals, with autistic people often having poorer ToM abilities (Brewer, Young, & Barnett, 2017). Both depression and anxiety have been linked to poorer ToM abilities, particularly there appears to be a relationship between impaired affective ToM and depression (Lei and Ventola, 2018; Mattern et al., 2015). Affective ToM refers to the ability to represent the affective states of yourself and others, and cognitive ToM refers to the ability to mentalise the cognitive states of yourself and others (Gabriel et al., 2021). Both appear to be potentially impaired in autism, although there is more evidence regarding specifically impaired affective ToM (Altschuler et al., 2021).

Empathy is another important ability for social interactions and can be defined as the ability to recognise, understand and feel what another person may be feeling or thinking (Cuff et al., 2016).). Mennin et al., 2005). Cognitive empathy can be defined as the ability to know what other people are feeling, while affective empathy is the ability to feel what another person is feeling (Smith, 2006). Empathy does appear to be atypical in autism, with poorer empathic abilities being shown in some autistic individuals (Pepper et al., 2019). Anxiety and depression are also associated with empathic abilities, anxiety (especially social anxiety), which may account for the poorer social skills often associated with anxiety and autism (Brett and Maybery, 2022). In terms of depression, in non-autistic individuals there is evidence that cognitive empathy can be affected in depressed individuals; whether this is also the case in autistic people is of interest.

There is substantial conceptual overlap between empathy and theory of mind, particularly the cognitive components. Bensalah, Caillies, and Anduze (2016) investigated empathy and theory of mind in children aged 4-6 years, and found that cognitive empathy was significantly correlated with ToM, but that affective empathy was not. They concluded that cognitive empathy may employ mentalising processes similar to that of theory of mind (e.g. false-belief

understanding), but that affective empathy may employ more unique mechanisms. Furthermore, Mathersul, McDonald, and Rushby (2013) found similar results in autistic adults, as cognitive empathy was associated with ToM, but affective empathy was not. Whilst empathy and ToM appear highly associated, potentially with overlapping mechanisms, there is neuropsychological evidence that affective empathy and theory of mind are separate constructs due to the activation of different neural areas such as the amygdala and insula, often associated with emotions (Dvash & Shamay-Tsoory, 2014; Preckel, Kanske & Singer, 2018). Cognitive empathy and theory of mind appear to be highly related in the sense that similar neurological pathways are activated, e.g. Dvash & Shamay-Tsoory (2014) state the medial prefrontal cortex, superior temporal sulcus activate for cognitive ToM, and the ventromedial prefrontal cortex for affective ToM. Despite close links and potential overlap, this review will discuss ToM and empathy separately, with empathy referring to the sharing and resonating with others emotions, and theory of mind referring to the 'cognitive understanding' of others thoughts, mental states and intentions (Preckel, Kanske and Singer, 2018).

Additionally, emotion recognition is another socio-emotional ability that is often atypical with autistic individuals, particularly the recognition of facial emotion expressions. A meta-analysis covering research from both autistic adults and children, indicated poorer emotion recognition when compared to non-autistic controls (Uljaveric and Hamilton, 2013). There are numerous studies indicating poorer emotion recognition in autistic individuals, using both static and dynamic stimuli, with a range of different emotions, and emotion recognition tasks. The causal factor behind this atypical emotion recognition is of interest, as there is research indicating that it may be atypical face processing in general or that reduced activation in the amygdala that may be impacting emotion recognition (Corbett et al., 2009; Riby, Doherty-Sneddon, and Bruce, 2009). Additionally, anxiety and depression have been of interest in emotion recognition, as in non-autistic individuals it has been found that anxiety is associated with poorer facial emotion recognition, and it is likely they may also impact autistic individuals due to the high prevalence of co-morbid mood disorders in autism (Demenescu et al., 2010; Torro-Alves et al., 2016).

The final socio-emotional ability this review will be focusing on is emotion regulation. Emotion regulation can be defined as the ability to assert control over your emotional state, for both positive and negative emotions, and a range of strategies (such as cognitive appraisal) may be used to do so (McCrae and Gross, 2020). Emotion regulation, particularly

maladaptive regulatory strategies, are often associated with ASC, particularly in autistic children, adolescents, and those with intellectual disability (Mazefsky et al., 2013; Sáez-Suanes et al. 2020). Goldsmith and Kelley (2018) investigated regulatory strategies and social impairment in autistic youths, and found that more effective strategies (such as re-appraisal) were associated with less severe autistic symptomatology and lesser social impairment. These findings highlight the potential importance of emotion regulation in social functioning and personal wellbeing. In non-autistic and samples with general anxiety disorder (GAD), it has been shown that anxiety is associated with poorer ability to self-soothe negative emotions and decreased ability to understand and manage their own emotions (Mennin et al., 2005). This suggests a link between anxiety and poorer emotion regulation. Similarly, depression has been linked to poorer emotion regulation. Joormann and Gotlib (2010) found that depressed participants showed increased levels of rumination after viewing negative material, and in formally depressed participants, higher levels of depressive symptoms were associated with reduced use of reappraisal, rumination, and expressive suppression. These results indicate that maladaptive emotion regulation strategies may be associated with increased level of depressive symptoms in non-autistic, depressed participants. Overall, whether we see the same pattern of emotion regulation strategies in depressed and anxious autistic individuals is of interest.

Thus, there exists evidence that depression and/or anxiety impact ToM, empathy, emotion recognition and emotion regulation in non-autistic people, and it is thus of interest whether mood disorders significantly contribute to the problems seen in socio-cognitive abilities in autism. However, there is another factor that may be responsible for differences in socio-emotional abilities present in autism other than anxiety and depression. The “alexithymia hypothesis” argues that many of the emotional problems reported in autism can actually be accounted by high rates of alexithymia (Bird & Cook, 2013). Alexithymia can be defined as the inability to recognise emotions within the self, and has high prevalence rates in ASC, with 47.3% of autistic females and 21.0% of autistic males being reported as having high levels of alexithymia (Oakley et al., 2020; Sifneos, 1973). The impact of alexithymia on some socio-emotional abilities in autism has been the subject of research, indicating that alexithymia may play a role in atypical emotion recognition, empathy and emotion regulation, although the exact nature of these relationships is yet to be established – are they causal, mediatory, or otherwise (Cook et al., 2013; Morie et al., 2019; Mul et al., 2018). Kinnaird et al. (2019) suggests that in particular, atypical emotion processing in ASC may reflect the effects of co-

occurring alexithymia in some autistic people, and so how alexithymia, autism, and mood disorders interact and consequently impact socio-emotional abilities is intriguing, particularly in regards to which factors are playing a causal role in atypical socio-emotional abilities, and which are mediating relationships, for example does alexithymia mediate the relationships between autism and emotional abilities?

This review aims to examine the effects of depression and anxiety on socio-emotional abilities in autism. Specifically, this review will consider the evidence that emotion recognition, ToM, empathy, and emotion regulation are significantly impacted by depression and anxiety in autism. The alternative explanation, that alexithymia underpins the socioemotional problems seen in autism, is also considered. The review begins with a consideration of anxiety's impacts, followed by depression, and then alexithymia.

The impact of anxiety on socio-emotional abilities in autism

Anxiety is common amongst autistic individuals, and there are a range of anxiety disorders that may present, ranging from general anxiety disorder (with 15-35% prevalence rates) to separation anxiety disorder, with prevalence rates of 9-38% (van Steensel, Bögels & Perrin, 2011; White et al., 2009). In particular, social anxiety (or social phobia) is a common form of anxiety in autistic individuals, with one out of four autistic individuals being diagnosed (Bejerot, Eriksson, and Mörtberg, 2014; White et al., 2014).

Anxiety impacts on an individual's social wellbeing and social skills, and many autistic people do report feeling anxious in daily social situations (Maddox and White, 2015). Syu and Lin (2018) found a relationship between anxiety, loneliness, and sensory sensitivity in autistic adults, and concluded that higher levels of sensory sensitivity may result in avoiding social situations as they are overstimulating, resulting in social anxiety and social withdrawal, which may result in individuals having less practise/experience in social situations and thus less practise/experience applying their socio-emotional cognition. These findings highlight the relationship between anxiety, autism, and socio-emotional wellbeing, suggesting that anxiety and autism can both affect how an individual navigates the social world. Similar findings have also been found in autistic children. Sukhodolsky et al. (2020) found that 67% of their autistic sample (N = 180) had characteristics of anxiety (rated by parent-report), and that higher levels of anxiety were associated with poorer sociability, separation anxiety, and social withdrawal. Furthermore, research indicates that autistic individuals with greater social impairments report higher levels of anxiety (Cox, 2020; Riesker et al., 2012). Thus, there

appears to be a relationship between anxiety and sociability in autistic individuals. The following sections consider the impact of anxiety on more specific socio-emotional abilities, namely ToM, empathy, emotion recognition and emotion regulation.

Anxiety and Theory of Mind

ToM is an important skill for socialising, understanding and communicating with others. Impairments and delays in ToM development are associated with ASC, and impaired or delayed development of ToM is associated with more broadly impaired social abilities, particularly in autistic children (Mao et al., 2023). However, not all research has documented ToM differences in autistic people: Scheeran et al. (2012) examined ToM abilities in children and adolescents with and without ASC, and found that the autistic participants performed similarly to non-autistic participants. They also found that ToM skills improved with age, and that adolescents of both groups were capable of completing advanced ToM tasks. This suggests that autistic individuals do have ToM: could varying levels of anxiety help to explain when research does and does not document ToM differences in autism?

Indeed, where ToM delays are present in autism, these delays do appear to be associated with anxiety: Lei and Ventola (2018) examined ToM skills and their relationship with anxiety in autistic children. The findings indicated that poorer ToM skills were linked with deficits in social communication and higher levels of parent-reported symptoms of anxiety. Delayed ToM skills may negatively impact social interactions, leading to higher levels of anxiety, particularly social anxiety.

The link between ToM and anxiety could be driven by maladaptive cognitive appraisals. Negative cognitive appraisals are a characteristic of anxiety, and are often linked with attentional bias in cognitive systems of anxiety (Liu & Li, 2019). For example, Kim, Kim, and Kim (2016) found attentional bias to negative emotion stimuli could be decreased when changing the cognitive appraisal of that stimuli, and vice versa. This highlights that cognitive processes may influence emotion and behaviour, and research has identified that cognitive appraisals can influence physiological processes and produce feelings of anxiety (Mauss et al., 2007). Maladaptive cognitive appraisals have also been associated with anxiety and ToM in autistic individuals. Sharma, Woolfson, and Hunter (2014) assessed negative appraisals, ToM, and anxiety in autistic children and found that maladaptive cognitive appraisals, such as future expectancy (whether a future outcome will be positive or negative), were associated with anxiety in the autistic sample and not in the non-autistic controls. These maladaptive

cognitive appraisals were also significant predictors of ToM ability in the autistic group. Potentially reduced abilities to deal with negative situations (and the possibility of negative future outcomes) may be associated with lower ToM abilities and may increase levels of anxiety. These findings highlight the relationships between ToM, negative appraisals, and anxiety and the causal direction of the relationships may help indicate areas where interventions and support may be useful for autistic individuals, for example could targeting the maladaptive negative appraisals reduce symptoms of anxiety and improve ToM abilities?

Anxiety and empathy

Atypical empathy has been identified as a characteristic of autism, particularly atypical empathetic response, for example an inappropriate response to another's emotional state (Harmsen, 2019). Atypical empathy is not unique to autism; Pepper et al. (2019) investigated empathy in individuals with ASC, early psychosis, and social anxiety disorder, and found that all three groups reported lower levels of empathy and lower social abilities when compared to non-clinical controls. However, out of the three clinical groups, the autistic group had the lowest levels of self-reported empathy. Thus, while empathy and social difficulties are not specific to autism, they are perhaps more impacted in autism than in other clinical groups, and for that reason empathy differences have been considered a "hallmark" of autism (Baron-Cohen, 2009; but see Fletcher-Watson & Bird, 2020).

Empathy has been argued to have multiple facets and subtypes, and plausibly different components of empathy might be differently related to anxiety. Brett and Maybery (2022) examined autistic traits (such as social difficulties and restricted/repetitive behaviours), anxiety, cognitive and affective empathy in the general population. They used a serial mediation analysis and found that higher levels of social difficulties were associated with higher levels of alexithymia and trait anxiety. Trait anxiety did not appear to be directly linked to cognitive empathy, though higher levels of alexithymia were associated with increased anxiety and less cognitive empathy (alexithymia will be discussed in more detail in a later section). For affective empathy, social difficulties and restricted behaviours indirectly *increased* affective empathy via trait anxiety and alexithymia, however the direct effect of social difficulties was to *decrease* affective empathy. Brett and Maybery (2022) state that these opposing effects reflect an 'inconsistent mediation', with one effect arising from an 'internal' process where anxious individuals fear negative evaluation and consequently are more aware of others, and another effect arising from an 'external' process where individuals

with social difficulties engage less in social actives and thus have less experience with affective empathy.

Potentially uneven empathy profiles could have a role in anxiety. Shalev et al. (2022) investigated the notion of empathic disequilibrium (non-equal levels of cognitive and affective empathy) and autism and found that higher empathic disequilibrium towards affective empathy (higher levels of affective than cognitive empathy) and lower total empathy predicted a greater likelihood of ASC diagnosis and autistic traits in both autistic and non-autistic participants. Higher empathic disequilibrium towards affective empathy was also associated to the social domain of autistic traits, whilst cognitive empathy was associated to the non-social domain. The findings of Brett and Maybery (2022) suggest that different internal and external processes may be influencing empathy, particularly affective empathy, and that there is an inconsistency in the effects of anxiety on empathy. Potentially higher empathic disequilibrium towards affective empathy may influence social difficulties, and when combined with the influence of alexithymia and autism, we see these social difficulties and the internal and external processes influencing levels of anxiety and empathy.

Not only may different components of empathy be important, but so might different aspects of anxiety. Sönmez and Jordan (2022) assessed different types of empathy and anxiety in adolescents with and without ASC. The findings indicated that, for the autistic group, lower levels of social anxiety were correlated with higher levels of cognitive empathy, and higher levels of affective empathy were correlated with higher levels of general anxiety. They suggested that higher affective empathy may lead to some autistic individuals feeling distressed by other's emotions, with the opposite being true for cognitive empathy. As cognitive and affective empathy were associated with different types of anxiety (social anxiety and general anxiety, respectively), this may reflect specific empathy and anxiety type difficulties, rather than general empathy and anxiety impairments. Furthermore, there were no significant correlations between anxiety and empathy for the non-autistic group, which suggests these relationships between empathy and anxiety are specific to autism.

Some of the relationships with anxiety described in the empathy literature may reflect close or shared mechanisms reported in studies of ToM. Indeed, cognitive empathy and ToM appear to use similar neural pathways, with affective empathy using different areas of the brain during activation (Preckel, Kanske and Singer, 2018). In the study by Mathersul, McDonald, and Rushby (2013), autistic participants showed lower levels of both cognitive

and affective empathy compared to non-autistic controls, with cognitive empathy also being associated with ToM abilities (and affective empathy not). In non-autistic participants, social anxiety has been demonstrated to be negatively correlated with ToM and 'empathic accuracy' for positive emotional stimuli (Alvi, et al., 2020). Social anxiety may affect ToM and empathy in a similar manner, but these findings need to be explored with autistic participants in order to establish these relationships, as potentially different effects of social anxiety on empathy (and ToM) may be observed in the autistic population (in comparison to the non-autistic population).

Anxiety and emotion recognition

Emotion recognition has been shown to be atypical in some autistic individuals, with often worse performance being shown on emotion recognition tasks when compared to non-autistic controls, in both children and adults (see Uljaveric and Hamilton, 2013, for a meta-analysis). Thus far, there has been limited evidence to suggest that anxiety plays a role in emotion recognition abilities in autism. Wong et al. (2012) examined facial emotion recognition in children with ASC and children with social phobia, with both groups completing measures of social skills and anxiety. They found that the autistic children were less accurate than non-clinical controls on emotion recognition, the social phobia group did not perform significantly different from the other groups, and that social anxiety and social skills were not correlated with performance on the emotion recognition task. While the differences in emotion recognition abilities were identified for the autistic participants, anxiety appeared to not be a factor in the relationship between emotion recognition and autism. This is also supported by research by Folz et al. (2022), who investigated emotion recognition, social anxiety, and autistic traits in a non-clinical sample. They found that poorer emotion recognition performance was associated with high autistic traits, but that emotion recognition performance was not associated with social anxiety.

While emotion recognition abilities appear not to be associated with anxiety in autism, there is some neurophysiological evidence that anxiety and autism may impact on emotional face processing. Charidza and Gillmeister (2022) investigated the mirror neuron system and its role in the differentiation and recognition of emotional faces, as well as the roles of trait anxiety and trait autism in a non-clinical sample. They found that positive facial expressions elicited greater central beta event-related desynchronization (ERD; beta ERD having been argued to reflect mirror neuron activity) compared to negative facial expressions.

Additionally, individuals with higher trait anxiety showed less ERD differentiation between happy and sad faces, whilst individuals with higher autistic traits showed less ERD differentiation happy and fearful faces. Using an implicit rather than a behavioural measure, these results indicate differences between non-clinical, high anxiety traits, and high autism traits individuals in the processing of certain emotional expressions. During behavioural tasks, autistic participants may be able to compensate for their atypical emotion processing (Harms, Martin, and Wallace, 2010), but with more implicit measures we can still see the differing cognitive processes. Whilst this study has some benefits with its methodology, the use of non-autistic and non-anxious participants (as in not clinically diagnosed participants) limits the generalisability of these findings to the anxious and autistic populations. Whilst there is evidence that anxiety may affect emotion recognition similarly to autistic traits, it appears that it is not a causal factor in the impairments observed in emotion recognition in ASC.

Anxiety and emotion regulation

Being able to regulate one's emotions appropriately in social situations is an important aspect to positive emotion well-being and effective social interactions. Poor emotion regulation, such as poor emotional control and heightened emotional response, are characteristics associated with ASC, particularly in autistic children, adolescents, and those with intellectual disability (Mazefsky et al., 2013; Sáez-Suanes et al. 2020). Perhaps because of this, unlike much of the research discussed so far, for emotion regulation, there is more research that helps to show to what extent the role of anxiety in socioemotional abilities differs for autistic people with and without intellectual disability.

Anxiety has also been linked to emotion regulation in autism in both samples with and without intellectual disability. Connor et al. (2020) investigated emotion regulation, anxiety, and autism in a sample of autistic youth with and without intellectual disability and found that higher levels of parent-reported anxiety were associated with impaired emotion regulation abilities. Interestingly, emotion regulation abilities and autism symptom severity were not associated with each other, suggesting that anxiety may be influencing impaired emotion regulation in some autistic individuals. Similar findings have been found by Sáez-Suanes et al. (2020), who also found a relationship between emotion regulation and anxiety in autistic adults with intellectual disability. Swain et al. (2015) found a relationship between emotion dysregulation and social anxiety in autistic adults without intellectual disability.

These findings support the link between anxiety and emotion regulation, across autistic subgroups.

There are some suggestions for why there is a relationship between anxiety and emotion regulation in autism. White et al. (2014) proposes that one of the reasons for increased anxiety in autistic individuals is atypical emotion regulation or impaired emotion regulation abilities. They also propose that these differences in emotion regulation may stem from issues with wider regulation and cognitive processes, physiological processes (e.g. sensory sensitivity, arousal), and other socio-emotional abilities. Alternatively, other research has more specifically suggested that emotion regulation may be associated with anxiety in autism via maladaptive strategies. Samson et al. (2015) investigated emotion regulation strategies in autistic individuals and found that higher levels of anxiety were associated with maladaptive emotion regulation strategies (such as repetitive behaviour). Pickard et al. (2020) found similar results in autistic adolescents, finding that maladaptive emotion regulation strategies were associated with social anxiety. These findings continue to support the link between anxiety and emotion regulation and suggest that having poor emotion regulation strategies may result in higher anxiety levels and poorer overall emotion regulation, poorer social skills and social interactions.

Anxiety, autism, and socio-emotional abilities

Together, research demonstrates that anxiety in autism is associated with several socioemotional abilities, although some have demonstrated stronger or more consistent relationships to anxiety than others. While emotion recognition seems to show limited associations to anxiety in autism, theory of mind, empathy and emotion regulation are associated with anxiety in autism. Considering the causal mechanisms that link these constructs, problems with emotion regulation could be considered causal to anxiety. Indeed, perhaps a difficulty regulating emotions, or employing suitable cognitive strategies in social situations, could also underly problems with ToM and empathy: ToM difficulties have been linked to maladaptive cognitive appraisals (Sharma et al. 2014), and autistic people may experience higher levels of personal distress when faced with others' negative emotions, which could feasibly be caused by emotion regulation difficulties, or lead to issues with emotion regulation (Sönmez & Jordan, 2022).

The impact of depression on socio-emotional abilities in autism

Depression, like anxiety, appears to be quite common in individuals with ASC. Prevalence rates vary, with the lifetime prevalence ranging from 12.3% to 37% (Hollocks et al., 2019; Hudson, Hall, and Harkness, 2019). Thus, there is a large portion of autistic individuals who comorbid depressive disorders. These may affect autistic individuals at any age as higher prevalence rates and symptom presentation occur in childhood and adulthood (Bitsika and Sharpley, 2015; Smith and White, 2020). The variability in prevalence rates may be due to the heterogeneity of depression (thus making depression difficult to diagnose in some cases), the tools for diagnosing depression (are they suitable to be used with autistic individuals?), and the heterogeneity of diagnostic tools used in research (DeFilippis, 2018; Hollocks et al., 2019; Lynch, Gunning & Liston, 2020). Some aspects of depression in autistic individuals are similar to the presentation in non-autistic people, including low mood, suicidal thoughts and self-harm, lack of energy, and avoiding social contact (Stewart, et al., 2006). However, depression can present differently in autistic individuals: differences in presentation may include social withdrawal (such as avoiding social contact), obsessive behaviours, stimming, and sleep disturbance (Chandrasekhar & Sikich, 2022; Pezzimenti et al., 2019).

Like anxiety, depression likely impacts socio-emotional abilities, such as ToM and empathy. Indeed, depressive symptomatology itself taps into socioemotional differences, as social withdrawal, loneliness, and low social motivation can be characteristics of depression in autistic individuals (Smith & White, 2020; Stewart et al., 2006). Depressive symptoms may to arise from social problems but may in turn may lead to social withdrawal and exacerbation of the original social issues. Indeed, poorer social skills have been associated with loneliness in some autistic individuals, and this experience of loneliness and poorer social interactions may contribute to low mood and social withdrawal (Han, Tomarken, & Gotham, 2019). Interventions aimed at improving social skills have been shown to improve mood: Rumney and MacMahon (2017) theorised that poorer social skills may lead to the development of mood disorders in autistic individuals, and conducted a systematic review to examine whether social skills interventions can positively affect the mood of autistic children and adolescents. They found that the majority of reviewed studies indicated improvements in mood, suggesting there may be an association between poorer social skills and depression in autism.

Overall, it appears there is a relationship between depression and social capacity in autistic individuals, and the impact of depression on more specific socio-emotional abilities, such as ToM and empathy, is of interest.

Depression and Theory of Mind

ToM appears to be negatively associated with anxiety, and it seems that depression may also affect ToM abilities. Research indicates differences, usually deficits, in ToM in depressed individuals compared to non-clinical individuals. Wolkenstein, Schönenberg, Schirm, and Hautzinger (2011) investigated social functioning and ToM in a depressed sample and found that there was some impairment of ToM. They concluded that there may be a link between depression and ToM, which may impact an individual's social capabilities. This is supported by research by Mattern et al. (2015), who investigated whether it was a general ToM deficit or specifically a deficit in affective ToM that is associated with depression. The findings indicated that there was a general ToM impairment, but also a significant impairment of affective ToM when compared to cognitive ToM. These results suggest that ToM is negatively affected by depression, particularly when mentalizing other's emotional states and using emotional context. These findings regarding affective ToM may be due to affective symptoms in depression, such as low mood and anhedonia (lack of interest/pleasure), as potentially disordered emotional states in the individual may affect their perception of others emotional states.

Furthermore, if an individual has atypical ToM, they may struggle to identify and mentalise their own mental states, as well as others (Williams, 2010). With regards to evidence that depression and ToM are linked in autism, there are some clues, but generally limited research that has directly examined this relationship. McCauley et al. (2019) investigated self-esteem, depression, and ToM in autistic children and youth (aged 9 to 17 years), with a primary aim of understanding whether self-esteem predicted depression and/or ToM abilities. Results indicated that the autistic participants rated their self-esteem lower than the non-autistic controls, that self-esteem was strongly related to depression for both groups, and that self-esteem was negatively related to theory of mind only for the autistic participants. Greater ToM abilities were associated with lower self-esteem: the authors speculate that autistic youth with the poorest ToM skills may lack the social abilities to pick up on negative evaluations from their peers, while those with slightly better ToM may recognise (or at least perceive) negative evaluations, and thus suffer reduced self-esteem as a consequence.

Potentially, depression and poorer mental health may be the result of reduced self-esteem as a consequence of having enough ToM ability to recognise negative evaluations from others. Self-esteem was strongly related to depression for both groups, but self-esteem was only related to ToM for the autistic group, this may be reflecting differences in ToM abilities between autistic and non-autistic individuals, potentially poor self-esteem can lead to depression for both groups, but generally non-autistic individuals have capable ToM abilities and the issues with self-esteem do not arise from their ToM. While these results highlight that the relationships between self-esteem and ToM may not be so straight forward as worse socioemotional abilities leading to universally worse mental health, the relationship between ToM and depression was not directly examined. This is also the case for other research that has examined ToM and depression: Crane, Goddard, and Pring (2013) found that autistic adults had higher levels of depression and lower scores on ToM tasks compared to non-autistic controls, but the relationship between depression and ToM abilities was not reported.

Furthermore, Block et al. (2021) found that autistic adults differed to non-autistic controls on measures of depression and ToM, generally reporting higher levels of depression and performing poorer on ToM tasks. These findings in both support to the notion that ToM is negatively impacted in autism, and that individuals with autism and co-morbid depression have may have specific deficits in ToM, particularly affective ToM. However, the direct correlations between ToM were not the direct focus of the study (face-memory was) and so this highlights the need for research directly examining depression and ToM in ASC. Whether depression is the factor behind ToM deficits, or whether it is autism, or a combination of both, requires direct examination in future research.

Depression and empathy

As previously noted, empathy appears to be impacted for some autistic individuals, and difficulty mentalising and resonating with other's negative states may impact an individual's self-esteem and social skills (as they struggle to understand other's emotions and how to react appropriately), which may lead to social withdrawal and depressive symptomology.

This is supported by research conducted with depressed non-autistic adults. A systematic review by Schreiter, Pijnenborg and Aan Het Rot (2013) demonstrated that there was a relationship between depression and impaired cognitive empathy, and that depression was particularly related to affective empathy, especially 'empathic stress' (their term for personal distress, the common term used in empathy measures to index stress experiences at others'

emotions due to emotion contagion). Depression was also linked to deficits in ToM and poor empathic accuracy.

Empathy, ASC, and depression appear to be an under-researched area but there is some preliminary evidence with autistic traits in a non-clinical population. Watanabe et al. (2021) investigated autistic traits, ADHD traits, depression, and empathy in medical students. Higher degree of ADHD and autistic traits were associated with depression, and independently autistic traits were associated with lower empathy. Again, the specific relationship between depression and empathy was not directly examined; potentially as depression and empathy were associated with autistic traits, they may also interconnect with or mediate each other. Lu et al. (2023) examined camouflaging, empathy, depression, autistic traits in a non-clinical sample of Chinese students (camouflaging refers to behaviours that autistic individuals may use, consciously or unconsciously, to mask autistic characteristics). The results demonstrated that depression, autistic traits, and camouflaging were intercorrelated. Empathy was not associated with depression but was correlated with autistic traits and camouflaging. Mediation analysis indicated that autistic traits and depression were mediated by camouflaging, and that higher levels of empathy were associated with autistic traits and camouflaging. They concluded that being higher in empathy may aid in maintaining camouflaging of autistic traits, and that sustained camouflaging may be mentally taxing and lead to 'fragile self-coherence', resulting in depressive symptoms. Thus, higher levels of empathy may be associated with depression because these empathy abilities afford some autistic people the opportunity to engage in coping strategies such as camouflaging, which may have negative long-term consequences.

The review of empathy and depression in non-autistic adults by Schreiter, Pijnenborg and Aan Het Rot (2013) also outlined several limitations with research in this area. Firstly, the heterogeneity of mood disorders and the variation within the sample may only partially reflect the complexity of the relationship between mood disorders and empathy (and ToM), and it is difficult to ascertain causality. Secondly, gender is often not considered as a factor and there is evidence that depressive symptoms can differ between the genders (Smith et al., 2008). A study about camouflaging in male and female autistic people found that females showed more camouflaging behaviours than men, but that higher levels of camouflaging in men were associated with higher levels of depression (Lai, 2017). Potentially there may be some gender differences in autism when experiencing depressive symptoms, and further

research may be able to create a more detailed profile on gender differences in autism and co-morbid depression, and their associations with socio-emotional abilities (Hedley et al., 2018).

Depression and emotion recognition

Similarly, to research on other areas of socioemotional abilities, there is some evidence that depression may affect emotion recognition, however evidence appears to be mixed and there appears to be a lack of research into depression and emotion recognition in autism specifically. A meta-analysis by Dalili et al. (2015) examining studies of emotion recognition and depression (studies comparing depressed patients and controls) found evidence of impaired emotion recognition, particularly for anger, disgust, fear, happiness and surprise amongst patients with depression. However, there was a lack of evidence for impaired recognition of sadness. The findings suggest that emotion recognition can be impaired in depression, but the effect size was small, and they concluded that some of the studies could have been underpowered. They also found that the depressed participants reported lower levels of confidence in their responses compared to the controls. These findings demonstrate the heterogeneity of facial emotion recognition abilities in depressed patients and highlight the need for further research with high power and larger samples. While these findings are based on non-autistic participants, it is plausible that depression may affect autistic participants in a similar manner.

Indeed, neuroimaging research provides some clues that depression similarly affects emotion recognition in autism. Ohtani et al. (2021) investigated brain activation on an emotion recognition task and measured frontotemporal hemodynamic responses using functional near-infrared spectroscopy (fNIRS) with a sample of young autistic adults with and without depression. They found that both autistic groups showed reduced activation compared to controls in the right ventrolateral prefrontal cortex, the autistic and depressed group showed reduced activation compared to the autistic not depressed group in the right anterior temporal cortex, and the depressed autistic group also showed reduced activation compared to the controls in the left ventrolateral prefrontal cortex. These findings suggest that there may be differences in regional brain activation during emotion recognition between depressed autistics, non-depressed autistics, and non-autistics. These results may reflect atypicalities in face processing between autistic and non-autistics, demonstrating potentially different cognitive processes for face and emotion processing (Riby, Doherty-Sneddon, and Bruce, 2009). While there is evidence that emotion recognition is atypical autism, the role of

depression warrants further investigation to ascertain whether depression represents a substantial causal factor. Currently, this is an under-researched area and future research should aim to examine the role of depression in emotion recognition in ASC.

Depression and emotion regulation

As previously described poor emotion regulation or maladaptive regulatory strategies appear to be associated with autism (Mazefsky et al., 2013; Sáez-Suanes et al. 2020). Poor emotion regulation strategies have also been associated with depressive symptoms in autism.

Similar to work on anxiety, maladaptive strategies represent an important link between emotion regulation and depression, and this appears to hold true for both autistic and non-autistic people. Rieffe, De Bruine, De Rooij and Stockmann (2014) examined emotion regulatory strategies and levels of depression in autistic children and non-autistic controls. In terms of regulatory strategies, the study focused on approach, avoidance and rumination. The findings indicated that rumination appeared to increase depressive symptoms in both the autistic and non-autistic (control) group, and that approach and avoidance aided in preventing depressive symptoms. These results suggest that poorer emotion regulation, specifically poor regulatory strategies such as rumination, may affect the mood and social abilities of an autistic individual. Interestingly, the non-autistic control group seemed similarly affected by poorer emotion regulation strategies, suggesting that non-autistic people may be susceptible to depressive symptoms in a similar manner as autistic people. This notion has support from a study conducted by Pouw, Rieffe, Stockmann, and Gadow (2013), who also investigated emotion regulation strategies and depression in autistic children. They found that those with avoidant strategies had less symptoms of depression, maladaptive coping strategies were positively associated with depressive symptoms, and negative friendship interactions were associated with depressive symptoms in the autistic participants. Additionally, the non-autistic controls who used maladaptive strategies had more symptoms of depression, similar to autistic children using maladaptive strategies. These findings indicate a relationship between specific emotion regulation strategies and depression, particularly maladaptive strategies and higher levels of depressive symptomatology, and that this relationship may be present in both autistic and non-autistic individuals.

The findings of Rieffe, De Bruine, De Rooij and Stockmann (2014) also suggest that it is poorer emotion regulation that may cause depressive symptoms, rather than depression affecting emotion regulation. Potentially, levels of depression may be affected by poor or

maladaptive emotion regulation due to the introspective nature of emotion regulation, if an individual struggles to regulate their own emotions, they may struggle to regulate their mood, increasing experience of low mood and other depressive symptoms.

There has been some support for the potential causal relationship between emotion regulation and depression in autism. Sáez-Suanes et al. (2020) investigated emotion regulation and depression in a sample of autistic people with intellectual disabilities. Using hierarchical linear regression, they found that emotion regulation was a predictor of depression in this sample. Further analysis established that emotion regulation played a mediating role between autism and depression. These findings add support to the notion that emotion regulation may be associated with depression, particularly playing a role in depressive symptoms in autistic individuals. There are some limitations with this study however, as the findings in a sample of participants with intellectual disabilities may not be generalisable to other autistic (and non-autistic) populations. Potentially, those with ASC without intellectual disability may have more cognitive capacity/processes in order to deal with emotion regulation than those with intellectual disability, and so those with intellectual disability may employ different emotion regulation strategies or display reduced capability in emotion regulation.

Depression, autism, and socio-emotional abilities

In comparison to anxiety, there is less direct evidence on the association between depression and socioemotional abilities in autism. In non-autistic samples, ToM and empathy appear to be affected by depression, particularly affective ToM and cognitive empathy. This may reflect difficulties mentalizing others' emotional and cognitive states and may affect social interactions and social experience. Preliminary evidence with autistic traits in general population samples indicates that depression and empathy may be independently associated with autistic traits, but not having a strong relationship with each other. More research is needed to examine the role of depression in emotion recognition. Poorer emotion regulation may cause depressive symptoms, and similar to research on anxiety increased use of maladaptive strategies has been noted to be important in autism.

As yet, more research in needed to understand the predictive power of depression symptoms in socioemotional abilities in autism, although we may make some predictions based on the research on anxiety. For example, there is evidence that affective empathy in particular may be associated with anxiety in autism (Sönmez & Jordan, 2022). Perhaps poorer affective may be associated with poorer mental health more generally, for both autistic and non-autistic

individuals, increasing the likelihood of both anxiety and depression symptoms. These effects may be amplified for autistic individuals due to problems with as emotion regulation, as they may struggle to manage their own emotions when empathising with others, especially for intense, negative emotions.

Alexithymia and socio-emotional abilities

So far, we have considered the evidence for the role of depression and anxiety in the socioemotional abilities of autistic people, but we now consider the possible role of another construct that correlates highly with depression and anxiety symptoms and is highly prevalent in autistic people: alexithymia. Alexithymia appears to affect emotional intelligence generally (e.g. see Davidson & Morales, 2022), but also the specific socioemotional abilities of interest to the present review. Indeed, alexithymia has been put forward as an explanation for many of the emotional differences reported in autism (Bird & Cook, 2013).

Alexithymia appears to account for significant variation in both autistic and non-autistic children's social abilities. Scheerer, Boucher, and Iarocci (2021) investigated social competence and alexithymia in autistic and non-autistic children, finding that scores on the alexithymia measure accounted for 16.2% of the variance in autistic children and 17.4% of the variance in the non-autistic children's parent-reported social competence. This indicates that poorer levels of parent-reported social competence may be similarly associated with higher levels of alexithymia for both autistic and non-autistic children.

In the field of empathy research, evidence suggests that alexithymia appears to play a role in lower levels of empathy (Komeda et al., 2015). Komeda et al. (2019) conducted research examining whether autistic adults would show empathy towards other autistic people and non-autistic people. To measure empathy, they used 24 stories, half of which included autistic protagonists, and the other half non-autistic protagonists. After reading the stories, participants were asked about the characters' emotional and mental states, and whether they would help the characters. Results showed that the autistic participants would show empathy to both other autistic people and non-autistic people, but that this was significantly less in participants with high levels of alexithymia. Mul et al. (2018) found that lower levels of both cognitive and affective empathy were related to alexithymia in a sample of autistic adults. They concluded that alexithymia may play a mediating role regarding empathy in autism, leading to impairments in both cognitive and affective empathy. In a further study with autistic youth, alexithymia was associated with higher levels of personal distress and lower

levels of empathic concern towards others (Butera et al., 2022). Together, these findings suggest that empathy differences may not be present in all autistic people, and that alexithymia may pose a viable explanation for the previously documented relationships between autism and lower levels of empathy.

Similar to empathy, an alternative explanation for atypical emotion recognition in autism is the effect of alexithymia. Cook et al. (2013) examined the role of alexithymia on facial emotion recognition in a sample of autistic adults and found that autism was not associated with lower facial emotional expression recognition ability, but that alexithymia was. Similarly, Ola and Gullon-Scott (2020) found that alexithymia was related to less accurate emotion recognition performance, rather than autism symptom severity, in a female autistic sample. Similar findings have been found with non-autistic participants: Donges and Suslow (2015) found that poorer performance on a facial emotion recognition task was associated with higher alexithymia scores. These findings suggest that alexithymia may play a role in atypical, generally impaired, facial emotion recognition abilities.

Empathy and ToM are often conflated, or at least considered to overlap considerably, but recent consideration of the research suggests that autism is related to atypical ToM, but that it is alexithymia driving atypical empathy (Brewer, Happé, Cook, & Bird, 2015; Fletcher-Watson & Bird, 2020). A review by Pisani et al. (2021) examined 63 studies, to exolore the mixed results regarding the role of alexithymia in atypical ToM. They found that alexithymia was associated with poorer ToM abilities when the tasks used to measure ToM required emotion recognition. A relationship between atypical emotion recognition and alexithymia has been established (Cook et al., 2013), and thus it appears that some findings examining alexithymia and ToM may be reflecting the alexithymia-emotion recognition relationship instead. Future research should utilise more valid measures of ToM in order to avoid conflation between ToM and other socio-emotional abilities.

Not only is alexithymia associated with socioemotional abilities, but alexithymia and depression, and alexithymia and anxiety, are also associated. Greater anxiety and depression have been reported to be associated with two of the three common subcomponents of alexithymia, greater difficulty identifying and describing feelings (Liss, Mailloux, & Erchull, 2008). Bloch et al., (2021) examined depression and alexithymia in autistic adults and non-autistic clinical controls. One of the alexithymia subscales (difficulty in identifying feelings) was the strongest predictor for depressive symptoms in both groups. While highly correlated,

anxiety, depression and alexithymia have been argued to reflect different constructs; that is, it is not the case that alexithymia measures are simply measuring symptoms of anxiety and depression (Marchesi, Brusamonti & Maggini, 2000). Given the correlations between alexithymia, depression and anxiety, perhaps alexithymia rather than depression or anxiety per se drives the profile of socioemotional problems reported in autism?

Interestingly, in a sample of non-autistic depressed patients, alexithymia not depression was associated with decreased empathy, indicating that depression may not be the factor behind impaired empathy (Hoffman, 2016). However, in a study examining both cognitive and affective empathy (in non-autistic, depressed patients), it was found that alexithymia affected cognitive empathy, but that both alexithymia and depression affected affective empathy, particularly personal distress (Banzhaf et al., 2018). Thus, it appears that empathy can be affected by alexithymia *and* depression, but the nature of their relationship is complex. Further investigation into whether depression or alexithymia best predict empathy in autism would be beneficial.

Alexithymia may also partly explain the link between autism and increased rates of depression and anxiety. Indeed, Morie et al. (2019) found that emotion regulation and alexithymia both mediated the relationship between depression and autism, providing further support that emotion regulation may lead to depression and suggesting that alexithymia may contribute to this relationship. This evidence posits questions about the role of alexithymia in socio-emotional abilities and suggests that alexithymia may interact with emotion regulation to contribute to the development of depressive symptoms. In addition, Brett and Maybery (2022) investigated autistic traits, anxiety, empathy, and alexithymia, using series mediation analyses. Results indicated that alexithymia alone was not a significant mediator of social difficulties and empathy and was not by itself an indirect mediator (i.e. the pathway from autistic traits to alexithymic traits to affective empathy was not significant). However, when combined with anxiety, alexithymia had an indirect mediating effect on social difficulties and affective empathy: autistic traits were associated with increased alexithymia, which was associated with increased anxiety, and anxiety was what was associated with affective empathy.

At present, there is limited research that measures depression, anxiety, alexithymia and the socioemotional abilities of interest to this review, especially in autistic samples. Alexithymia may not affect all socio-emotional abilities, or at least not in the same manner. Further

research could aid in establishing whether alexithymia causes (or mediates) general socio-emotional deficits or whether they are specific. Indeed, some have argued that alexithymia is *not* related to impaired ToM, once the reliance of certain ToM measures on emotion recognition is considered (Pisani et al.2021).

Furthermore, there is heterogeneity amongst the autistic population, including their emotional abilities and social competence (Bird and Cook, 2013; Scheerer, Boucher, and Iarocci, 2021), Therefore, the evidence that alexithymia may be deficit-specific (rather than a general emotion processing impairment) may be reflecting this heterogeneity, with some individuals having more or less identified atypical socio-emotional abilities.

One issue with research investigating mood disorders and alexithymia in autism is the similarity and even often overlapping symptomatology of these conditions, especially as they are commonly co-occurring. Another issue arising from this is the potential difficulty in measuring these conditions as unique conditions. Depression and anxiety are often hard to diagnose in autistic individuals due to overlapping symptoms with autism and with each other (Cath et al., 2008; Stewart et al., 2006). However, research has provided some clarification that whilst they are closely related, alexithymia, anxiety, and depression are separate constructs and can be measured individually effectively (Marchesi, Brusamonti, & Maggini, 2000).

It should be noted that the roles of alexithymia and depression or anxiety are not necessarily mutually exclusive. For example, alexithymia and depression/anxiety may both contribute to reduced social competence (for example social withdrawal and lack of social motivation are often associated with depression) and may therefore both have important roles in hindering social skills in autistic individuals.

Overall, the role of alexithymia is an interesting aspect to socio-emotional research, but due to the complex nature of the interactions with autism and mood disorders, the exact nature of the relationships between alexithymia and socio-emotional abilities is not yet fully understood.

Discussion

Anxiety and depression are common co-occurring conditions in ASC. Understanding how these common mental health problems impact the socioemotional profile of autism is crucial,

especially given recent arguments that socioemotional abilities vary greatly in autism and may not be best explained by autism per se (Bird & Cook, 2013).

From the evidence reviewed, anxiety appears to be associated with empathy, emotion regulation, and ToM although there is a need to further establish the specific roles anxiety plays in these abilities. Anxiety appears to not be associated with impaired emotion recognition in autism, and it appears that autism itself or perhaps alexithymia may be the modulating factor in this case. For depression, there is less research evidence on the role of depression in specific socioemotional abilities in autism as compared to anxiety. As with anxiety, depression is associated with emotion regulation, as maladaptive regulatory strategies are associated with depressive symptoms. Further research is needed to examine the impact of depression on emotion recognition in autism as there is some preliminary evidence that depression can affect neural signatures during emotion recognition in autistic individuals (Ohtani et al., 2021).

The importance of understanding the unique contributions of depression to the specific socioemotional abilities reviews here is highlighted by some evidence that suggest depression may outperform anxiety in predicting social competence. Johnston and Iarocci (2017) investigated anxiety and depression symptoms in autistic children to ascertain whether one or the other was more associated with social competence. The results indicated that anxiety and depression both contribute to variance in social competence for autistic children, but only depression accounted for a significant amount. This suggests that depression may have a greater impact on social competence and wider social abilities than anxiety. Depression also appears to impact the affective side of some socio-emotional abilities in non-autistic participants (e.g. affective ToM and affective empathy), and this may be due to symptoms such as low mood and emotional numbness, as these symptoms may hinder general emotional processing, including emotional cognitive processes for understanding others emotions. Indeed, apathy and anhedonia have been linked to facial emotion recognition deficits in Parkinson's disease and emotional word memory problems in depressed patients, (Liu, et al., 2012; Martínez-Corral, et al., 2010).

Additionally, there is some evidence that depression alters emotional processing, such as bias towards negative emotions and impaired facial emotion recognition (Demenescu et al., 2010; Vazquez et al., 2016). Poor emotional cognition may negatively affect social interactions, which may lead to other depressive symptoms such as social withdrawal, and these overall

may further affect social competence, leading to a negative feedback loop. Whilst depression may have wider implications for social abilities and interactions, as this review demonstrates, anxiety has vital implications for empathy, emotion regulation, and ToM, and should not be dismissed.

It appears that anxiety and depression affect a range of socio-emotional abilities to some extent, some more than others, and it begs the question whether these are a series of specific impairments or whether anxious and depressive symptoms affect social and emotional cognition and abilities in general. Nuske, Vivanti & Dissanayake (2013) conducted a review examining whether emotion impairments are unique to, universal, or specific in ASC. To summarise, they argue that atypical emotion processing is not universal across the autistic population, atypical emotion processing is not unique to autism, but that the specific profile of atypical emotion processing seen in autism is specific to autism. Nuske et al. (2013) suggest the specific profile of atypical emotion processing in autism appears to be unique due to a specific pattern of 'strengths and weaknesses' observed in emotion processing in ASC, including difficulties processing social stimuli (but not non-social), 'complex' emotions (in contrast to basic), negative emotions (compared to positive). Additionally, more demanding tasks appear to hinder emotion processing (e.g. stimuli with occluded parts of the face or blended emotional face stimuli).

The role of alexithymia in social and emotional impairments in autism must also be considered, particularly, as in some cases it appears that alexithymia, not autism or mood disorders, is the factor behind some of the atypical social-emotional abilities we find in autism. For example, alexithymia appears to impact emotion recognition, empathy, and emotion regulation. For emotion recognition, anxiety does not appear to impact recognition abilities and there is some evidence that depression may affect recognition although this needs further experimental evidence. There is evidence, however, that alexithymia does play a role in emotion recognition, with research indicating that alexithymia modulates impairments in facial emotion recognition in autistic individuals (Cook et al., 2013). For empathy, it appears alexithymia can affect both cognitive and affective empathy, but that depression can also impact them as well. The nature of the relationship between empathy, alexithymia, and depression appears to be complex, with evidence suggesting that alexithymia may play a mediating role (Banzhaf et al., 2018). For emotion regulation, both alexithymia and difficulties with emotion regulation may contribute to the relationship between autism and depression (Morie et al., 2019). However, the relationships between

alexithymia, anxiety, depression, and socio-emotional abilities are complex, with evidence suggesting that alexithymia might be a mediator or even play a causal role. Future research is needed to examine these complex relationships and ascertain their individual and combined contributions to atypical socio-emotional abilities in ASC.

There are some limitations within the research discussed that should be noted. Firstly, as already noted, the relative lack of research in the impact of depression in autism greatly limits the strength of conclusions we can draw about the role of depression in the socioemotional profile typically seen in autism. It has been argued that current research has tended to focus on the causes of ASC and biomedical (e.g. genetics) aspects to ASC: Pellicano, Dinsmore, and Charman (2014) conducted a study where they discussed current and future areas of autism research with stakeholders, researchers and autistic individuals. Autistic individuals were reported to be dissatisfied with the current focuses of autism research, preferring autism research to focus more on aspects of their daily lives, including creating evidence-based interventions and support groups. Increased research into depression's impact on autistic people's social abilities would seem in keeping with this goal.

Indeed, more work is needed to inform how we can aid autistic people with anxiety and depression. At present, there is a lack of research regarding the treatment of depression in autism, including a lack of pharmacological studies, and what research there is focuses on adults (DeFilippis, 2018). White et al. (2018) identified that there is little research on how to effectively treat depression in autistic people, particularly the effectiveness of CBT, whilst there is a plethora of research regarding the treatment of anxiety. Identifying methods that are beneficial and alleviate symptoms of depression for autistic individuals would be a useful area for further research, whether that is examining CBT or alternative therapies, as it will help with the design of treatment programs for depressed autistic people and aid in informing future care. Furthermore, in particular relevance to the aims of the present review, interventions can help provide evidence for causal relationships (Eberhardt & Scheines, 2007): for example, treatments for depression in autism would provide an opportunity to observe whether socio-emotional abilities improve after an intervention for anxiety/depression, which could help indicate that anxious/depressive symptoms were causing/exacerbating the socio-emotional difficulties. Although, as Eronen (2020) writes, even with using interventions, it is hard to establish true causality as it is difficult to reliably assess what the intervention actually accomplished and causal assumptions may not consider individual causal relationships.

Another challenge in this area is the measurement and detection of depression and anxiety in autism. For example, DeFilippis (2018) outlined some major issues within research on depression for autistic individuals, stating that there is a lack of reliable diagnostic scales, particularly for those with verbal deficits or intellectual disability. The lack of appropriate diagnostic tools for both research and clinical use may affect the reliability and validity of diagnoses, as well as research findings, as has perhaps contributed to the relative lack of research in depression's role in socioemotional difficulties in autism.

Aside from the measurement of depression and anxiety symptoms, another issue within research is heterogeneity of methods used between studies, as there tends not to be standardised measures/tasks of socio-emotional abilities and mood disorders (Hollocks et al., 2019). Additionally, studies may use different diagnostic tools of measures of anxiety and depression, with some studies recruiting clinically diagnosed autistic/depressed/anxious individuals from medical/support centres, and some studies using self-report measures of anxious/depressive symptoms, autistic traits, and social/emotional difficulties. The variety of measures and designs used likely contributes to the inconsistent and variability of findings sometimes observed in autism research (Loth et al., 2013).

Furthermore, the conclusions regarding the role of depression, anxiety or alexithymia in socioemotional difficulties may be invalid if the evidence draws from only a subset of the autistic population. Autism research is often conducted on individuals with autism and without intellectual disability, even though it is estimated that the population of autistic people with intellectual disability is about 50% of the autistic population (Russell et al., 2019). Russell et al. (2019) conducted a meta-analysis investigating intellectual disability and research bias and found that 94% of autistic participants did not have intellectual disability, an estimate that is clearly very discrepant with the proportion of intellectual disability in the autistic population. They concluded that selection bias was present across autism research and that this creates issues such as limiting the generalisability of research findings across the autistic population. While there was some notable exception within this review (e.g., Sáez-Suanes et al., 2020), in general the field suffers from the wider selection bias problems documented by Russell et al (2019), and future research could include more work with those with autism and intellectual disability to help provide more knowledge regarding their experience of mental health conditions.

Future research

The current review identified several areas that currently are under-researched and more empirical evidence is required, in particular the impact of depression on social and emotional processing in autism is under-researched. Whilst there is evidence that depression may affect emotion recognition and empathy in non-autistic individuals, these findings may not be generalisable to the autistic population, and thus examining depression and these abilities in autistic individuals is vital (Dalili et al., 2015; Mazza et al., 2014).

In addition, future research should investigate whether anxiety and depression play a causal or mediating role in atypical socio-emotional abilities, or alternatively anxiety and depression may arise from the atypical socio-emotional abilities themselves; for example, there is evidence that poorer emotion regulation strategies are associated with depressive symptoms (Saez-Suanes et al., 2020). The current evidence base makes it difficult to untangle whether poorer emotion processing and social skills leads to symptoms of anxiety and depression, or vice versa. Understanding these relationships may aid in informing the design of interventions to help those with anxiety/depression, autism, and social impairments.

Whether alexithymia is a mediator or causal factor for atypical socio-emotional abilities warrants further examination. There is evidence for both, and it is likely that alexithymia can influence socio-emotional abilities, as well as being a mediating factor between autism, mood disorders, and social impairments (Cook et al., 2013, Brett & Maybery, 2022). Potentially alexithymia may exacerbate autistic characteristics and depressive/anxious symptoms (Barros, Figueiredo, & Soares, 2022; Bloch et al., 2021). Further research into these complex relationships would aid in increasing understanding of alexithymia and its interactions with anxiety, depression, and socio-emotional abilities in ASC. Furthermore, insight into alexithymia and mood disorders may be useful in designing interventions and support programmes.

Also, further research is required to investigate affective ToM, and how it may affect other emotional abilities. Additionally, more research is needed to investigate the relationship between autism, ToM, and anxiety. The current research suggests that anxiety does impact ToM in autistic individuals, and that negative cognitive appraisals may play a role, but overall, the area needs further research to ascertain the specific cognitive mechanisms and factors behind the relationship between anxiety and ToM. Furthermore, there has been some attempt at creating ToM interventions for autistic individuals, and Fletcher-Watson et al. (2014) conducted a review of the efficacy of these interventions. They found that there was

some evidence that ToM skills could be attained, though participants struggled to adapt these skills across different contexts. Evidence that some ToM interventions are effective allows future research to use these interventions to assess whether anxiety and depression symptoms improve as well, which will aid in establishing a causal model between ToM, autism, and anxiety and depression.

Conclusion

To conclude, anxiety and depression are associated with socio-emotional abilities in autism, though direct mechanisms that link these variables, and directions of influence, are yet to be understood: further examination has implications both for basic understanding of the root of socio-emotional differences in autism, and also implications for future clinical practice.

Chapter Three: The role of emotional factors in face processing abilities in Autism Spectrum Conditions

Declarations

Chapter Three has been submitted for publication at Research in Autism Spectrum Disorders (RASD) and is currently under-review. Chapter Three was submitted as a stage two registered report, after receiving in principle acceptance, and so the stage one protocol has already been submitted and reviewed. Please note that "Supplementary Materials" for this journal article are included in Appendix 1 in this PhD thesis.

Ethics Approval

Informed consent was obtained from participants before taking part in the study. Ethics approval was obtained from the ethics committee of the Psychology department, University of York, UK (ID: 920).

Availability of data

This study was pre-registered on the Open Science Framework and data used in analysis is available here: https://osf.io/rhykm/

Competing interests

The authors have no competing interests to declare.

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Author's contributions

Natasha Baxter made substantial contributions to the design of the study, data collection, data analysis and interpretation, and drafted the paper. Hannah Hobson made substantial contributions to the design of the study, data analysis and interpretation, and provided feedback/edited the paper. Both authors approved the submitted manuscript.

Abstract

Facial emotion recognition is considered atypical in individuals with autism spectrum conditions (ASC), but emotion recognition abilities vary widely in autistic people, and there are inconsistent findings on the causes of these differences. Research indicates alexithymia may result in facial emotion recognition differences in ASC. Alternatively, mood disorders have been linked to atypical facial emotional expression recognition abilities in neurotypical adults. Research investigating both the effects of alexithymia and mood disorders (depression and anxiety) is necessary to establish which of these factors may cause atypical facial emotion recognition in ASC. This study aimed to examine whether alexithymia or mood disorder symptomology is a predictor of atypical facial emotion recognition in individuals with ASC. Ninety-eight non-autistic adults and 80 autistic adults were recruited. Participants completed an online facial processing task to examine emotion and identity recognition abilities, the AQ-28, the TAS-20, and the HADS to measure autism severity, alexithymia symptoms, and depression and anxiety symptoms. Regression-based analyses found that autistic traits and autistic group membership did not predict face processing abilities after accounting for demographic variables, alexithymia and mood disorder symptomatology: however, neither alexithymia nor mood disorder symptoms predicted variance in face processing abilities either. Analyses using a newly proposed reduced TAS measure did not change these findings, neither did analyses that considered the role of self-identification of autism versus formal diagnosis. Our results concur with previous meta-analyses of face processing in autism spectrum disorder which report that studies do not always report deficits in face processing in autism: our findings are also not supportive of the model that argues that alexithymia explains face processing difficulties in autism.

Introduction

One of the core features of Autism Spectrum Conditions (hereafter ASC) is atypical social communication. The nature of these differences has been argued to include problems with facial processing and decreased ability in recognising other individuals' emotions from their facial expressions, which can cause difficulties in socio-emotional abilities (Ko, 2018; Loth et al., 2018). Indeed, previous research suggests that facial emotion recognition in autistic individuals may be reduced or atypical, relative to neurotypical individuals (Nomi and Uddin, 2015).

However, research on facial emotion recognition in autism has provided inconsistent results. Some studies have found that there is a reduced accuracy in identifying emotions, particularly negative emotions (Ashwin et al. 2006; Bal et al. 2010; Corden et al. 2008; Howard et al. 2000; Wallace et al. 2008; Uljarevic and Hamilton, 2013). Meanwhile, other research has shown that in groups of individuals with ASC without intellectual disability, facial emotion recognition does not seem to be impaired (Adolphs et al. 2001; Baron-Cohen et al. 1997; Loveland et al. 2008; Neumann et al. 2006; Ogai et al. 2003; Rutherford and Towns, 2008; Teunisse and de Gelder, 2001). Meta-analyses have however suggested that sample IQ does not determine whether an emotion recognition deficit is observed (Uljarevic and Hamilton, 2013); this same analysis concluded that while there was evidence for decreased emotion recognition abilities in ASC, there was evidence of publication bias and substantial heterogeneity.

This heterogeneity of research findings may reflect heterogeneity within the autistic population with regards to emotion processing. While a study by Loth et al. (2018) indicated that the majority of individuals with ASC have atypical facial expression recognition, some do not display these differences. Therefore, further research is required to establish the differences between those individuals with ASC displaying facial emotion recognition impairments and those who do not display atypical emotion recognition (Sato et al., 2017). Candidate factors that could differentiate those with autism and emotion recognition differences from autistic individuals without these problems include alexithymia or mood disorders.

Alexithymia, mood disorders and emotion recognition in ASC

Recent research suggests that alexithymia may play a role in facial emotion recognition in ASC. Alexithymia is characterised by the inability to identify emotions experienced by the

self and others (Sifneos, 1973), and frequently co-occurs with ASC. Oakley et al. (2020) report that many individuals with ASC report higher levels of alexithymia, with 47.3% of autistic females and 21.0% of autistic males meeting the cut-off for clinically relevant alexithymia. Kinnaird et al. (2019) suggest that emotional processing differences in ASC may reflect co-occurring alexithymia and that while alexithymia is common in ASC, it is not universal, which reflects the heterogenous nature of ASC. Alexithymia has been linked to atypical activation of the anterior insula, and in fMRI studies investigating empathy in ASC, hypo-activation of the anterior insula was associated with difficulties in emotional awareness (Silani et al., 2008). This suggests there may even be differences at the neural level between neurotypical individuals, and those with ASC, which may lead to variation in their ability to perform emotion-related tasks.

The effect of alexithymia on emotion recognition has been investigated but mixed results have been found. Cook et al. (2013) found that autism severity was unrelated to expression-recognition ability, while alexithymia predicted facial emotion recognition abilities. This suggests co-occurring alexithymia may be responsible for atypical facial emotional expression recognition in ASC. However, Shah et al. (2019) challenges whether alexithymia plays a role in atypical emotional processing in autism. They found that autistic traits were more of a predictor than alexithymia of atypical empathy. However, given that they looked at empathy, rather than emotional processing, this point of difference between these two papers could be due the focus on different socio-emotional abilities: future research is needed to clarify the effect of alexithymia on emotional face processing and empathy.

Additionally, the conflicting findings may be explained by the different research designs employed by the researchers. Shah et al. (2019) argues that a matched approach, such as that used by Cook et al. (2013), can suffer from biased sampling. In the case of alexithymia research, a biased sample can occur when using a matched design as alexithymia is more present in autistic populations compared to non-autistic populations (50% versus 5%, Kinnaird et al., 2019). As the prevalence of alexithymia in the autistic population is considerably higher than in the typically developed population, Cook et al.'s control sample does not match the proportion of alexithymia in the population, and can be considered to have a high number of people with high alexithymia traits in the non-clinical sample, and these high levels of alexithymia may be linked to other factors that influence the research such as interpersonal issues and mental health conditions. Shah et al. (2019) states that matched groups are difficult to be truly representative of their respective populations and that this can

result in inaccurate statistical comparisons and population-level inferences. Furthermore, Shah et al. (2019) noted issues within this field of research such as small sample sizes lacking statistical power. They conclude that larger samples with sufficient statistical power examining the potential predictors of atypical emotional abilities in autism would be beneficial to future research.

There are additional issues with alexithymia and autism research, regarding the measures used to measure these constructs. As discussed in Ruzich et al (2015), while the AQ measures are commonly used in research and in practice as screening instruments, arguably this measure does not have suitable sensitivity and specificity for population screening of autism per se. Thus, those who score "above threshold" on the AQ may not be considered "autistic". Furthermore, there have been issues with incorrect cut-off values being used for the AQ-10, and this has had implications for research, as it significantly affected the effect sizes reported by research into the relationship between autism and behaviours of interest (Waldren et al., 2022). This suggests that researchers must be aware that thresholds may be too lenient (or stringent) and ensure that the correct threshold is used appropriately. In addition, the psychometric properties of the original TAS-20 alexithymia measure have been questioned: while the TAS-20 has been argued to be a reliable measure of alexithymia in typically developed and clinical populations (Bagby, Parker, & Taylor, 1994; Bagby, Taylor, & Parker, 1994; Cook et al., 2013), others have argued the original proposed TAS-20 model was not adequate for either autistic or typically developed populations (Williams & Gotham, 2021). Williams and Gotham (2021) used the factor analysis to reduce the items of the TAS-20 to eight items (the 8-item general alexithymia factor score, the GAFS-8), and found sound psychometric properties and that the GAFS-8 did predict alexithymia traits without any deficiencies in validity. This suggests that the GAFS-8 may be a more suitable measure to be used instead of the TAS-20, but there has yet to be investigation into how these measures compare in their prediction of socioemotional abilities, such as emotional face recognition.

There are other factors that may be responsible for emotion recognition differences in ASC such as mood disorders including anxiety and depression. Mental health conditions such as depression and anxiety are common in autism, the lifetime prevalence being estimated at 42% for anxiety and 37% for depression (Hollocks et al., 2019; Skokauskas, & Gallagher, 2010). Research has shown that depression and anxiety are associated with atypical facial emotional expression recognition abilities in non-autistic adults (Demenescu et al., 2010; Skokaukas & Gallagher, 2010; Stewart et al., 2006). Demenescu et al. (2010) found that adults with anxiety

displayed significant decreased abilities in emotional expression recognition (Cohen's *d* =−0.35) and that depression was associated with a larger display of decreased ability in emotion recognition (*d* =−0.58). Additionally, Torro-Alves et al. (2016) found that individuals with high social anxiety had poorer facial emotion recognition particularly in ambiguous and low intensity presentations of facial emotional stimuli. While there is research establishing the prevalence of anxiety and depression, there is a paucity of research specifically investigating the effect of depression and anxiety on facial emotion recognition abilities in autistic individuals. Potentially, depression and anxiety will affect facial emotion recognition in a similar way to non-autistic individuals, but whether they have an independent effect or exacerbate existing atypical facial emotion recognition in ASC requires investigation (Oakley et al., 2020).

Mood disorders and alexithymia may also interact in ASC, and there is existing evidence to suggest relationships between depression, anxiety and alexithymia. Alexithymia and mood disorders have a close relationship, as alexithymia may be related to atypical emotion regulation, and evidence suggests alexithymia may mediate the relationship between anxiety, depression, and autism (Morie et al., 2019; Venta et al. 2013). Indeed, as research demonstrates that both alexithymia and mood disorders have overlapping symptomology and are often co-occurring, the distinctness of the traits of these conditions and how they can be effectively measures is questioned. However, research by Marchesi, Brusamonti, & Maggini (2000) has clarified that alexithymia, anxiety and depression are separate constructs through a factor analysis of the TAS-20 (Toronto Alexithymia Scale, Bagby, Parker, & Taylor, 1994) and the HADS (the Hospital Anxiety and Depression scale, Zigmond & Snaith, 1983).

Alexithymia has been shown to be high in autistic individuals but is also associated with mood disorders such as anxiety and depression in those without ASC (Honkalampi et al. 2000; Karukivi et al. 2010). The relationship between alexithymia and co-occurring depression or anxiety may be explained by evidence suggesting individuals with these co-occurring conditions have lower emotional intelligence, which may also have implications for differences in facial emotion recognition abilities (Onur et al. 2013). Morie et al., (2019) suggest that individuals with ASC are more likely than the neurotypical population to have alexithymia, and that alexithymia may affect emotional processes (such as regulation), leading to increased possibility of mood disorders. Whether alexithymia or a mood disorder then contributes to atypical emotion recognition in ASC is of interest to the current study.

While evidence suggests alexithymia and mood disorders are related to atypical facial emotion recognition, more research is needed to establish the independent effects alexithymia, mood disorders, and traits of ASC have on facial emotion recognition. Additionally, research into alexithymia and mood disorders in autism will provide insights that could help tailor interventions (Kinnaird et al., 2019).

Berggren et al., (2018) conducted a systematic review investigating emotion recognition training for autistic individuals. They found that while emotion recognition training improved emotion recognition in some of the studies, the generalisability of the training to normal daily life and social activities was unclear. While emotion recognition training likely benefits some individuals, perhaps targeting differences in emotion recognition is not effective for all. Indeed, if mood disorder symptomatology is highly predictive of atypical emotion recognition in autism, this would question the approach of interventions that train emotion recognition, instead suggesting that interventions targeting depression or anxiety would be perhaps doubly effective, in both relieving the mood disorder symptoms, and having positive effects on social emotional cognition.

Aims of this study

The aim of this study is to investigate the extent to which co-occurring alexithymia and/or a mood disorder symptomatology predict atypical facial emotion recognition in individuals with autism. Previous research indicates that alexithymia has an effect on facial emotion recognition abilities in autism (Cook et al., 2013). However, research investigating the effects of mood disorders has mainly focused on typically developed samples. Therefore, for this study we aim to use both a sample of adults with ASC and a sample of non-autistic adults as our sample to establish the effects of anxiety and depression on facial emotion recognition abilities.

Our research is based on a study by Cook et al. (2013), in which they investigated co-occurring alexithymia and facial emotion recognition ability in individuals with autism, finding that autism severity was not related to expression recognition while alexithymia correlated strongly. We aim to replicate their findings with alexithymia but also investigate whether co-morbid mood disorders such as anxiety and depression may account for the differences in facial emotion expression recognition, specifically the ability to distinguish disgust from anger. Additionally, as alexithymia, anxiety and depression have overlapping traits (one can also argue that ASC has some overlapping symptomology as well), whether a

combination of both alexithymia and mood disorders further exacerbates atypical emotion recognition is of interest.

Method

Pre-existing non-autistic data

Our project includes data from both non-autistic and autistic individuals. In order to establish the feasibility of the task and procedure, the non-autistic data were collected in accordance with a preregistered protocol (available to view here: https://osf.io/wjvne), prior to registration with the journal. Our methodological details here encompass both what was collected for non-autistic and autistic participant group. While we used comparable tasks and measures to those of Cook et al. (2013), as noted by Shah et al. (2019) there are problems with a matched group approach to alexithymia research, and thus we opted to recruit a large sample and use regression-based analyses to ensure we have sufficient power and can examine the predictors of atypical facial emotion processing.

Participants

The non-autistic group was recruited via social media, 'word of mouth', and via Prolific. We aimed to collect 100 non-autistic participants and had 119 complete datasets. However, 21 were removed due to being incomplete, or failing an attention check measure. This means that data from a sample of 98 non-autistic adults has been collected in a previous study using the same methods, materials and experimental design. This sample serves as a comparison group to the autistic sample. For the preliminary results drawn from the non-autistic group only (and which featured in the Stage 1 version of this report) see "*Results from the non-autistic sample – Preregistered analyses*" in Appendix 1.

To be comparable to the data collected from this control group, we aimed to collect complete datasets from at least 100 autistic people (see our Power analysis section for considerations on potential data loss). During data collection, it was discovered that a number of recruited individuals were suspected to be fraudulent participants: this was due to an autism organization sharing the online link to take part in the study directly without having to contact the research team. Data gathered during this period of recruitment is not included in the study as a high number of participants' emails and addresses (collected for reimbursement purposes) were deemed to be suspicious. This unfortunately had a knock-on effect on our ability to meet our intended recruitment target, due to resource constraints. In the end, we

successfully collected 80 autistic data sets. Even when outliers were removed, we achieved our minimum power requirements for 73 autistic datasets for each regression conducted.

Similar methods were used to recruit our autistic sample, with the exception of Prolific, plus via the Autistica research network. Participants needed to be aged 18 years and over. Both participants with clinically diagnosed ASC or those who self-identify as autistic were welcomed into the study: we recognise that for certain individuals a formal diagnosis can be hard to obtain, especially in adults and females, as females tend to present differently to males, and ASC may be 'missed' in adults who were not identified as children (Gould & Ashton-Smith, 2011; Murphy et al., 2016). Therefore, we invited invite both individuals who self-identify as autistic, and those who have received a formal diagnosis to participate. Participants were asked whether they have received a formal diagnosis or self-identify as autistic, to establish whether self-identification is associated with our variables of interest. Table 3.1 summarises the participants' characteristics.

Tasks and materials

Face processing task

Many forms of facial recognition tests and stimuli have been developed and used in previous research to measure facial emotion recognition. Our stimuli have been selected from previous research by Cook et al. (2013), a method that allows us to establish whether emotion recognition problems are occurring in the context of more general face processing difficulties, using identical stimuli for both emotion and identity recognition tasks.

The stimuli consisted of 14 images of two morphed faces displaying anger and disgust, a technique originally developed by Calder et al. (1996). The two cross-morph sets comprised of 'Harold' displaying anger to 'Felix' displaying disgust and 'Harold' displaying disgust to 'Felix' showing anger. The set began with 80% intensity of 'Harold' displaying anger with 20% intensity of 'Felix' showing disgust. The next image would then be 70% intensity of 'Harold' displaying anger with 30% intensity of 'Felix' showing disgust, and then increasing and decreasing respectively until 80% intensity for Felix showing disgust and 20% intensity for 'Harold' displaying anger. This was repeated for the other set consisting of 'Harold' showing disgust and 'Felix' showing anger'. A strength of these stimuli sets is that they can be used to easily control for general face processing problems by checking identity recognition, using the same stimuli for both emotion recognition and identity recognition trials. The cross-morphed stimuli also reflect real-life face and emotion processing as

typically both identity and expression are attended to when encountering faces. Measuring identity recognition allowed us to determine whether alexithymia or mood disorders were having an effect on facial processing overall or just facial emotion recognition. Indeed, it has been proposed that there is a link between autism and atypical face processing as research suggests a 'diminished level of expertise' for processing faces (Gauthier, Klaiman, & Schultz, 2008). Of course, only including a disgust-anger morph stimuli set limits the generalizability of the findings to a wider range of emotions; because of the testing time required to gather enough data for our dependent variables, it was thought that including another contrast (e.g. fear-surprise) would likely impact on recruitment and attrition. It was thus decided to examine disgust-anger only.

Participants first completed a training and practice phase. They were presented with 4 images in succession, Harold being angry, Harold being disgusted, Felix being angry, and Felix being disgusted, with the identity and emotion presented stated underneath (e.g. "This is Harold angry"). In order to test that participants can recognise Harold and Felix, and recognise their emotions when presented at 80% (the maximum intensity for one emotion and identity, e.g. 80% Harold and anger, and 20% Felix and disgust), they were then presented with the same images as before, but with the addition of the keyboard presses that participants use to respond in the experimental trials. These trials gave feedback to the participants, either a red cross indicating they were wrong or a green tick mark to indicate they were correct. They completed eight of these feedback trials. There were then 24 practise trials in the style of the experimental phase (without feedback).

Following the training and practice phase, stimuli were presented in 10 blocks comprising of 28 experimental trials each. The 14 stimuli were presented twice within each block in random order. Trials start with a fixation cross lasting 1500ms. Stimuli are presented for 800ms and are then replaced by a prompt stating either 'Harold or Felix?' or 'Disgust or Anger'? Between each block, during which participants can take a break, participants were presented with a screen showing the stimuli for the two emotions at 80% intensity for the two individuals with clear labels for identity and expression.

Attention check measure

Given that this study was conducted online away from experimenter supervision, we are including a task to check participants were attending to the screen. A grey star is presented briefly (for 800ms) in certain experimental blocks (specifically blocks 2, 6, and 9).

After every block, participants are asked if they saw the star. Only participants who correctly report seeing all grey stars were retained for analyses.

Questionnaire measures

Self-report measures were used for capturing autistic traits, alexithymia traits and anxiety and depression symptoms.

The Hospital Anxiety and Depression scale (HADS, Zigmond & Snaith, 1983) consists of seven questions for anxiety and seven questions for depression. Depression and anxiety scales are scored separately. Scoring for each item is on a 0-3 Likert scale, with 3 being the highest for anxiety/depression. For the HADS depression and anxiety scores (scored separately, resulting in an anxiety and a depression score for each individual), 0-7 indicates a 'normal' (phrase used in the measure) non-anxious/depressed score, 8-10 is considered borderline/mildly anxious/depressed, and 11-21 is considered 'abnormal' or a clinical case (phrase used in the measure). Scores can range from 0 to 21. The HADS has been shown to perform well in measuring anxiety and depression symptoms in non-clinical samples as well as clinical samples (Bjelland, Dahl, Haug, & Neckelmann, 2002), including the identification of anxiety and depression in autistic individuals, with good internal consistency and convergent validity for both the anxiety and depression scales (Uljarevic et al, 2018). The means, standard deviations and distribution of the HADS data for our data from typically developed adults confirm the measure is normally distributed, with suitable variation in scores that will allow for the exploration of the impacts of anxiety and depression on our variables of interest.

The Toronto Alexithymia Scale (TAS-20, Bagby, Parker, & Taylor, 1994) consists of three subscales: difficulty describing feelings (reflected in 5 items), difficulty identifying feeling (7 items), and externally-oriented thinking (8 items). Items are scored on a 1-5 Likert scale, with five of the items being reverse scored (specifically items 4, 5, 10 18 19). This involves reversing the score the participant answered, e.g. if a participant answered 4 on the scale, then it would be reversed to 2 when totalling the items/sub-scale items. A total score is produced, alongside a score for each of the three subscales. A total score of 51 below indicates no alexithymia, whereas a score of 52-60 indicates possible alexithymia, and a score of 61 or over indicates the individual is alexithymic. Total scores can range from 20-100 (Seo et al., 2009). Research has shown that the TAS-20 has construct validity and provided evidence of the TAS-20 as a reliable measure of alexithymia (Bagby, Parker, & Taylor, 1994;

Bagby, Taylor, & Parker, 1994). Research also indicates it is suitable for use with individuals with ASC, showing string test re-test reliability and convergent validity with other measures such as the Bermond-Vorst Alexithymia Questionnaire (Berthoz & Hill, 2005; Zech et al., 1999). The TAS-20 has also been previously used in autism and emotion recognition research (e.g. Cook et al., 2013).

The abridged Autism Quotient (AQ-28, Hoekstra et al., 2011) consists of 28 items taken from the AQ-50 (Baron-Cohen et al., 2001). These 28 items reflect five factors: social skills, routine, switching, imagination, and recognising numbers/patterns. Items are scored on a 1-4 Likert scale, with 13 items having reverse scoring, similarly, to as described in the TAS-20. A score of 65 and above indicates high autistic traits. Scores range from 28-112 (Sizoo et al., 2015). The abridged AQ version still retains high validity as a measure of autism symptomology (Hoekstra et al., 2011), and we determined that a shorter version of the AQ would be suitable for our experimental design in order to decrease fatigue effects and attrition rates.

It should be noted we are not including an IQ measure in this study. Cook et al. (2013) found no effect of IQ, and neither did the meta-analysis performed by Uljarevic and Hamilton (2013); thus, it was decided not to include an IQ measure.

Procedure

Participants read information about the study and only proceeded once consent was given. Participants who took part in the non-autistic study received the same information and consent forms. Ethical approval was given by the ethics board of the Psychology department of the University of York.

Testing was done online using the experiment builder software 'Gorilla' (www.gorilla.sc). After reading the information sheet and completing the consent form, participants completed the facial emotion and identity recognition task. Participants first completed the training and feedback phase, to teach the participants what the identity and emotions shown are ('Harold' and 'Felix', disgust and anger), then 24 practise trials before beginning the main task. The training and practise trials served only to familiarise participants with the task, and as there are only a small number of practise trials we could not generate the required variables to assess participant performance. Therefore, all participants progressed to the main task after training. After completing the main task, the participants then completed a demographics questionnaire, AQ-28, TAS and HADs. They were also asked if they have been diagnosed

with anxiety or depression, and whether they had been diagnosed with ASC or are self-identified. After completing these measures, the participants were debriefed and thanked for their participation. The task and measures lasted approximately 30 to 45 minutes.

Analysis plan

Analyses were conducted using MATLAB, and SPSS statistics. The dependent variables for this experiment are the point of subjective equivalence (PSE) which is a measure of bias and details the point on the expression or identity dimension at which participants are equally likely to make either attribution (i.e. either "Harold" or "Felix", or "Anger" or "Disgust"), and the attribution threshold which is an index of attribution precision. A lower PSE would indicate the participants are seeing faces as angry or as Felix earlier on the emotion/identity continuum, and a higher PSE would indicate seeing anger/Felix later. The attribution data was modelled by fitting cumulative Gaussian functions to estimate the psychometric functions, function fitting will be completed in MATLAB using the Palamedes toolbox (Prins & Kingdom, 2018), and separate functions for the expression and identity dimensions were modelled for each participant. The attribution threshold was based on the standard deviation of the Gaussian distribution that best fits the data. Lower attribution thresholds suggest better performance.

Pre-registered statistical tests

To test the contribution of autistic traits, alexithymic traits and mood disorder symptomatology to predicting emotion and identity recognition, we conducted a series of hierarchical regressions. These are summarised in Figure 3.1 (see appendix 1).

In regression series 1, we entered age, gender, and ethnicity as predictors in step 1 to control for these broad demographic effects on performance. In step 2, we entered our alexithymia measure (the TAS-20), and then step three included our anxiety and depression measure (the HADS), and step 4 included our autism trait measure (the AQ-28). All self-report measures were entered as continuous variables.

While autistic traits have been conceptualised as a continuous trait, along which both autistic and non-autistic individuals vary, it is of course possible that the extent of one's autistic traits may not be as predictive as whether or not one has a clinically significant level of autistic traits. Regression series 2 will test this possibility, taking a categorical approach to

the autism variable. In this regression, and we compared the neurotypical comparison group and the autism group in step 4.

The third regression also took a categorical approach to check if the presence of self-identified autistics influenced the model-fit. In this regression series, we included three levels to our group variable: non-autistics, self-identifying autistic participants, and autistic participants who stated that they have a formal diagnosis.

We planned to include some simple correlational analyses, descriptive statistics, and t-tests to further clarify any group differences, and directions of effect. We also examined the group differences of participants who meet the commonly used threshold for alexithymia (a score of 61 of above on the TAS-20), and those who do not, as previous research indicates we will also find a difference between these groups (Cook et al., 2013). This was done via a series of non-parametric tests, to explore whether those who are above the threshold are associated with higher levels of depression, anxiety, autistic traits, and poor facial emotion and identity recognition.

Additional pre-registered analyses, which are reported in the Supplementary Materials for brevity, were also run. In a further set of regressions, the AQ-28 threshold was used to create a group of autistic participants above the threshold, and also a non-autistic group below the threshold. It seemed possible that including those who self-identify as autistic, while being inclusive of certain groups who may struggle to receive a formal diagnosis, this could affect the results, possibly reducing our effect sizes, if these individuals had reduced autistic characteristics. Therefore, using the AQ-28 threshold, we removed those with high AQ scores in the non-autistic group and those with low AQ scores in the autistic group, and thus created more differentiation between our two samples. This further allowed us to see if including the self-identifying individuals affected the results. We also ran a supplementary analysis using the TAS-8, to determine whether using this shorter alexithymia measure, which has been argued to have improved psychometric properties, can establish the role of alexithymia. Finally, we performed an additional regression with the identified outliers included, to assess whether by removing outliers, we are also removing theoretically interesting cases as those individuals may have high traits of alexithymia and show the effect of alexithymia on emotional face processing.

Power analysis

A previous meta-analysis by Uljarevic and Hamilton (2012) examined emotion recognition in ASC compared to neurotypicals. This analysis reported a large effect size before adjusting for publications bias (which then decreased to a moderate effect size). Additionally, a meta-analysis investigating alexithymia and autism found large to medium effect sizes for between groups differences (neurotypicals and autistics), suggesting a higher prevalence of alexithymia in autistic individuals (Kinnaird, Stewart, and Tchnturia, 2020). Therefore, previous research indicates that we can expect at least a medium effect size, with regard to emotion recognition and alexithymia comparison between autistic and non-autistic groups. However, a key target for our own study is the extent to which independent predictors add to a model explaining emotion recognition ability, rather than straight forward group comparisons of alexithymia and emotion recognition. Previous research investigating whether alexithymia explains emotion recognition deficits in autistic and non-autistic participants reported a large effect size ($f^2 = 0.364$), this being the effect size for the variance explained by alexithymia when added to the regression model (Cook et al., 2013). However, given reported concerns over publication bias in emotion recognition and autism research where effects may have been inflated (Uljarevic and Hamilton, 2013), we based our power analysis on a more moderate medium effect size.

A priori analyses were carried out to establish the required sample size for a regression analysis, which will be our core hypothesis-testing statistical procedure. The power analysis was carried out using the software G*Power (Faul et al., 2009), with 1 number of tested predictors and 6 total predictors, and an effect size (f^2) was 0.15. This power analyses thus tests the power for a predictor to reveal a significant independent contribution to a model, over and above previously entered predictors. Results showed that a sample size of 73 participants would be needed for a significance level of 0.05 and the level of power at 0.90. Also, power analyses states that a sample size of 67 would be required to establish a correlation between emotion recognition and the TAS-20 (the alexithymia measure).

Our first sample of non-autistic participants was a sample of 98 participants, (7 of which were considered outliers in our preliminary analyses). We intended to collect at least 100 complete datasets from our autistic sample, and at least 90 datasets that survive removal due to attention check fails, being an outlier, or poor function fit. As noted in our Participants section above, we were unable to achieve this, but our final sample was 178 participants, after removing incomplete datasets and individuals who failed the attention check. This sample size should still negate any issues with multi-collinearity, and inflation of previous estimate

due to publication bias. As previously noted, we included those who self-identify as autistic in our autistic sample in order to aid in recruiting our goal of at least 100 participants. Also, we did not formally check participants for their clinical diagnosis: as we cannot verify who has a formal diagnosis of autism, we felt it practical to allow people who self-identify as autistic to take part and just ask them if they are or are not formally diagnosed. See Table 3.1 for participant characteristics.

Results

As per our pre-registered analysis plan, we conducted all analyses with outliers excluded and included. In the below sections are the results when outliers were excluded (using a Cook's Distance threshold of 0.022, a threshold applied for each regression independently, as Cook's Distances are calculated for each regression separately). Please see the Supplementary Materials (Appendix 1 of PhD thesis) for results with outliers included, but in summary when all participants were included no regression model was significant, at any step or for any dependent variable. While some predictors were correlated with each other (see Table 3.2), all variance inflation factors were below a conservative threshold of 2.5, meaning we had no indications of multicollinearity within the data.

The role of alexithymia and autistic traits in predicting face processing

As per our analysis plan, we conducted a series of hierarchical regressions to test the roles of alexithymia versus autistic traits on face processing abilities. Figure 3.1 summarises the steps of our regressions. Table 3.2 includes the correlations between the face processing variables, depression and anxiety scores, autistic traits and alexithymic traits.

In our first series of regressions, a continuous measure of autistic traits, the AQ-28, was entered in the final step. Table 3.3 summarises the results of this regression series. For emotion attribution thresholds, identity attribution thresholds, and identity PSE, no model was significant at any step. For emotion PSE, the regression model was significant at steps 1s and 2 (although the addition of the TAS in step 2 did not lead to a significant increase in the variance explained). A significant predictor throughout the steps was gender, specifically the dummy variable indicating "other" gender. Age was also significant in step 4. See sub-section "The role of gender" for follow up tests on gender effects on emotion PSE. Table 3.4 summarises the coefficients for the regressions with significant models.

Together, these results suggest that neither autistic nor alexithymic traits explain a significant amount of variance in face processing abilities after demographic variables are accounted for.

The role of autism status and autism self-identification in predicting face processing

Further regression series were conducted, to test a) whether autism status (autistic versus non-autistic) rather than continuous autistic traits contributed to face processing ability after controlling for alexithymia, anxiety and depression and b) whether self-identification versus clinical diagnosis of autism affected the results.

Table 3.5 summarises the results of the regression series that considered the role of autistic status. For emotion attribution thresholds, identity attribution thresholds, and identity PSE, no model was significant at any step. For emotion PSE, the regression model was significant at steps 1, 2 and 4, although beyond model 1 no additional steps added significant explained variance. Table 3.6 summarises the coefficients for the regressions with significant models. Again, 'other' gender was significant at every step, with age being significant in step 4. After accounting for demographic variables, autistic group status, alexithymia, nor anxiety or depression accounted for variance.

Table 3.7 summarises the results of the regression series that considered the role of self-identification. Similar to the regression series reported above, emotion attribution thresholds, identity attribution thresholds, and identity PSE, no model was significant at any step. For emotion PSE, the regression model was significant at steps 1, 2 and 4, although beyond model 1 no additional steps added significant explained variance. Table 3.8 summarises the coefficients for the regressions with significant models. Once more, 'other' gender was significant at every step, with age being significant in step 4. After accounting for demographic variables, autistic group status vs. self-identifying status, alexithymia, nor anxiety or depression accounted for variance.

In addition, we used the AQ-28 scores to remove non-autistic individuals who scored above the AQ-28 threshold, and autistic individuals who scored below the AQ-28 threshold. This resulted in 52 non-autistic participants and 78 autistic participants (interestingly the two participants removed from the autistic group were both clinically diagnosed, not self-identifying). Regression analyses with participants who had AQ-28 scores incongruent with their group removed did not yield any significant models at any step, with any dependent variable, or any significant changes in variance explained between steps. See Supplementary Materials, in Appendix 1, for the statistics from these regressions.

Effects of high versus low alexithymic traits

We compared participants who scored above the standard threshold for "high" alexithymic traits to those who scored below this threshold on our face processing measures, autistic traits, and anxiety and depression scores. We again removed outliers: this was done using the thresholds used for regression series 1 (see Supplementary Materials, in Appendix 1 for results when all participants including outliers are included). Given that the face processing variables, AQ scores and HADS scores were not normally distributed, these comparisons were conducted using the non-parametric Mann-Whitney U tests. Table 3.9 includes the statistics for these comparisons.

Comparing participants who scored above and below the high alexithymia cut-offs on the face processing variables, no comparisons reached significance (see Table 3.9). Highly alexithymic participants had higher autistic traits, and lower anxiety scores. Depression scores were not significantly different between the group.

Findings with reduced TAS-8 alexithymia measure

The regression series were also run with the standard TAS total score replaced with a revised TAS-8 measure. The results were identical to those with the long-form TAS measure. There were no significant models or changes in explained variance for analyses with emotion attribution thresholds, identity attribution thresholds, or identity PSE, for all version of regression (i.e. when AQ scores, autism group status, and self-identification status were entered in step 4). For emotion PSE scores, the regression using AQ scores was significant at steps 1 and 2, but no significant change in the amount of variance explained was found. The regressions using autism group (autism versus non-autism) and self-identification status (clinical diagnosis vs self-identification vs non-autistic controls) were significant at steps 1, 2 and 4, but there was no significant change in variance explained beyond the variables entered in step 1.

The statistics for the regressions with the TAS-8 measure are reported in the Supplementary Analyses, in Appendix 1. Note that these analyses were run using the same Cook's Distance threshold rule, calculated for each regression separately.

Unregistered analyses: The role of gender identity in emotion perception

While neither alexithymia nor autistic traits/status were significant predictors of our face processing variables in our analyses, gender did emerge as a significant predictor of emotion

PSE scores in the above analyses. Follow-up comparisons comparing emotion PSE between participants who we male, female or selected "other" as their gender showed that the other group had significantly lower emotion PSE scores (N = 16, Mdn = .47) compared to both males (N = 74, Mdn = .49) and females (N = 88, Mdn = .50): Kruskal-Wallis H (2) = 9.52, *p* = .009.

Unregistered interaction analysesAlthough unregistered, whether there were interactions between variables was investigated due to the overlapping nature of some of the variables (such as ASC and alexithymia), and potential mediation relationships between predictors. For the regression series that used the AQ-28 in Model 4, we added an interaction term for the AQ*TAS, which was calculated by first centring the AQ and TAS scores, then multiplying them together. The VIF scores indicated no issues with multicollinearity. The regression series was completed for each of the dependent variables, and the interaction term was non-significant in all.

Discussion

This study aimed to investigate the extent to which co-occurring alexithymia and/or mood disorder symptomatology predicted atypical facial emotion recognition in autistic and non-autistic individuals. It was predicted that alexithymia would be associated with poorer emotion recognition (rather than autism). We also predicted that anxiety and depression may also contribute to differences seen in emotion recognition abilities between autistic and non-autistic individuals. While we did find the expected correlation between alexithymia and autistic traits, neither alexithymia nor autism (or autistic traits) contributed significantly to emotion or identity recognition abilities in any of the analyses conducted (including analyses using newly proposed alexithymia measures, with outliers included, etc). This was also true for anxiety and depression. The only significant predictor was gender: gender was a significant predictor for emotion PSE scores, and follow-up comparisons indicated that the 'other' gender group had significantly lower emotion PSE scores compared to the male and female groups.

While autistic traits did show a small zero order correlation with emotion attribution scores, this association did not survive controlling for demographic variables in our regression analyses. Autistic group membership also did not predict face processing abilities in our regression analyses. Previous literature has documented emotion processing difficulties in autism (Uljaveric & Hamilton, 2013), but noted that studies are far from consistent: not all

autistic individuals experience differences or impairments in their socio-emotional abilities, reflecting the heterogeneity of the condition (Baron-Cohen et al., 1997). Indeed, one motivation for the present study was to consider the argument that previously reported effects of autism were actually driven by alexithymia (Cook et al., 2013). However, no effect on, or association with, our face processing task was found with alexithymia either.

Plausibly, the relationship between alexithymia and face processing may be more complex than a simple linear effect: some research into alexithymia and other socio-emotional abilities indicates that the role of alexithymia may be more mediatory or may not influence atypical socio-emotional abilities at all (Brett & Maybery, 2022; Morie et al., 2019; Shah et al., 2019). Furthermore, other authors have posited that proposed explanatory power of alexithymia in socioemotional problems in autism have been overstated (Shah et al., 2019). While alexithymia and emotion recognition abilities have been the topic of numerous studies, with both clinical and non-clinical populations, to the best of our knowledge there has yet to be a meta-analysis that has established the current evidence base on the relationship between alexithymia and emotion recognition, and, importantly, assessed the degree to which publication bias may be present in this literature. This need was discussed in a systematic review of the role of alexithymia in emotional face processing (Grynberg et al., 2012), but this research need appears to have been as yet unmet. It is a particular strength of the registered report format that studies are assessed and accepted in principle prior to data collection and before results are known: we contemplate that there may be other unpublished studies similar to ours that failed to find effects of alexithymia on emotion processing.

Mood disorder symptomatology did not predict emotion processing either. Previous research with non-autistic individuals indicated that anxiety and depression may affect emotion recognition abilities (Charidza & Gillmeister, 2022, Dalili et al., 2015), and so with the relatively high prevalence rates of both anxiety and depression in autism, it was thought that mood disorders would contribute to some degree in this study. However, other studies have indicated that females with and without depression perform similarly on facial emotion recognition tasks (Fieker, Moritz, Köther & Jelinek, 2016). Some research indicates that anxiety has little effect on emotion recognition in autism: Wong et al. (2012) investigated social anxiety and emotion recognition in a sample of autistic children and found that autistic children performed poorer than non-autistic controls on an emotion recognition task, but that anxiety was not a contributing factor. Plausibly, our measures of depression and anxiety may have lacked sufficiently specificity to uncover more nuanced relationships: for example,

social worries and fear of negative evaluation has been shown to impact eye gaze behaviour in autism (White, Maddox & Panneton, 2015). Of course, given that we did not find strong effects of autism in face processing abilities, there is not an autism effect for anxiety or depression to explain in the current data.

One factor that did predict some of our face processing measures was gender. The effect of gender was not predicted, although previous research into alexithymia, autistic traits and empathy did highlight that gender may have an important predictive role in empathy abilities (Shah et al., 2019). Possibly, effects of gender diversity could be to do with neurodivergence. An association between gender diversity and neurodiversity has been observed (van Vlerken, Fuchs, & van der Miesen, 2020), and in the present study, we did note that the "other" gender group had the highest autistic traits of all three gender groups, and our autistic sample had twice as many people who considered themselves to have an "other gender", compared to the non-autistic group. The measure on which gender was predictive was emotion PSE: specifically, it indicated that those who described their gender as other perceived the morphed faces to be angry earlier in the morph spectrum than males or females, indicating a bias (relative to males and females) to see anger rather than disgust. While interesting, we would emphasise that this does not mean that face processing is deficient in those who do not identify as male or female, and rather than necessarily due to associations with neurodivergence, could be a product of these individuals' social experiences. We could speculate that individuals who do not identify as male or female may be more likely to have been in threatening situations, been the subject of bullying or violence: such experiences could be expected to lead to a heightened sensitivity to anger in others. Indeed, transgender people are at an increased likelihood of being victims of multiple episodes of violence (Messinger, Guadalupe-Diaz, & Kurdyla, 2022). However, our gender category of "other" is very vague and limits our ability to draw form conclusions between individuals' gender identities and emotion processing abilities.

Age was also a significant predictor for the emotion PSE variable step 4 of the regression, suggesting that age accounted for some of the variance. This may be due to the range of ages used between samples, with the average age of the non-autistic sample being in their mid-20s and the autistic sample having an average age of 41 years old. The range of ages may be explained by recruitment processes, with the autistic sample coming from an established research network, and the non-autistic sample being recruited from university students and Prolific. Age has been shown to have an effect of emotion recognition, with often older

individuals demonstrating decreased performance compared to young adults, although this effect tends to have been shown in participants over 60 (West et al., 2012). Potentially, the current study reflects some age differences, although this is difficult to determine due to age not being a consistent significant predictor in the same way that the 'other' gender variable was.

Overall, the implications of the current study indicate that neither alexithymia, autism, nor mood disorders significantly impacted the facial emotion recognition abilities of autistic individuals. While this differs to previous research, and opens up avenues for further examination, it also begs the question whether there are other factors contributing to atypical facial emotion recognition abilities. Gender appeared to have an effect on emotion recognition abilities in this sample, specifically individuals who identified as neither male nor female, and so future research could focus on gender diversity, autism, and social-emotional cognition. Given the expansive research that has taken place on alexithymia and emotion processing abilities, we argue that a meta-analysis and assessment of previous publication bias would be timely.

One consideration is that this study was conducted online. While the effectiveness of collecting data online and the quality of the data has been questioned, it appears that there is little to no statistical difference between collecting data online or in-person (Sauter, Stefani, & Mack, 2022; Uittenhove, Jeanneret, & Vergauwe, 2022). Therefore, it is unlikely that using an online design would have reduced the quality of the current experiment. However, our online approach does raise two issues for reflection.

Firstly, unfortunately in our recruitment of the autistic sample, it came apparent that there were fraudulent participants attempting to take part multiple times. Thankfully, this was noticed and none of this sample were used in the final data set. While the current study was able to rectify issues with fraudulent participants, it does appear that issue is happening increasingly with research. Pellicano et al. (2023) was also affected by 'scammer participants' pretending to be autistic individuals, or parents of autistic individuals, during online interviews. We echo the warnings of Pellicano et al (2023) that autism researchers need to be alert to the real threat of imposter participants, especially in online research.

Secondly, it is plausible that effects of autism and alexithymia seen in previous face-to-face research may not have been observed in our online study because of delivering the experiment in this modality. Plausibly, the ability to participate from their own homes in a

more comfortable environment relative to most research laboratories, to self-pace (to some extent), and without perceptions of judgement from any research staff, autistic individuals may perform quite well on emotion processing tasks such as ours. Sucksmith et al. (2013) asked autistic adults without intellectual disability to complete an online facial emotion task and suggested that the participants may have felt more comfortable at home compared to a lab environment, and that this increases the validity of the results, as it is more reflective of the participants natural abilities. We concur that being able to take part at home, away from the lab, may have reduced apparent deficits in face processing, although how "valid" a reflection of ability depends on under what circumstances individuals are usually processing faces in their daily lives. Certainly, understanding the role of general environment factors and experiment modality is important for the field to connect research findings to real world difficulties, as online research may diverge from participant reports or clinical observations of finding face processing hard "in real life".

As part of conducting our study online, we opted in to include individuals who self-identify as autistic, given the practical barriers verifying formal diagnoses even if we insisted on all participants having a clinical diagnosis before taking part. There was no "penalty" to participants if they reported that they self-identified as autistic, so we consider it unlikely that participants would have been motivated to be dishonest about their autism diagnostic status. None of our analyses suggested that including self-identifying individuals reduced our effects: indeed, in one analysis where we removed autistic adults who did not score above the AQ threshold, the participants who did not meet this threshold all reported to hold formal diagnoses. Broadly our experience would support the future inclusion of self-identifying autistic adults in research studies, particularly as this may make research more inclusive for subgroups who struggle to attain a formal diagnosis (e.g. women).

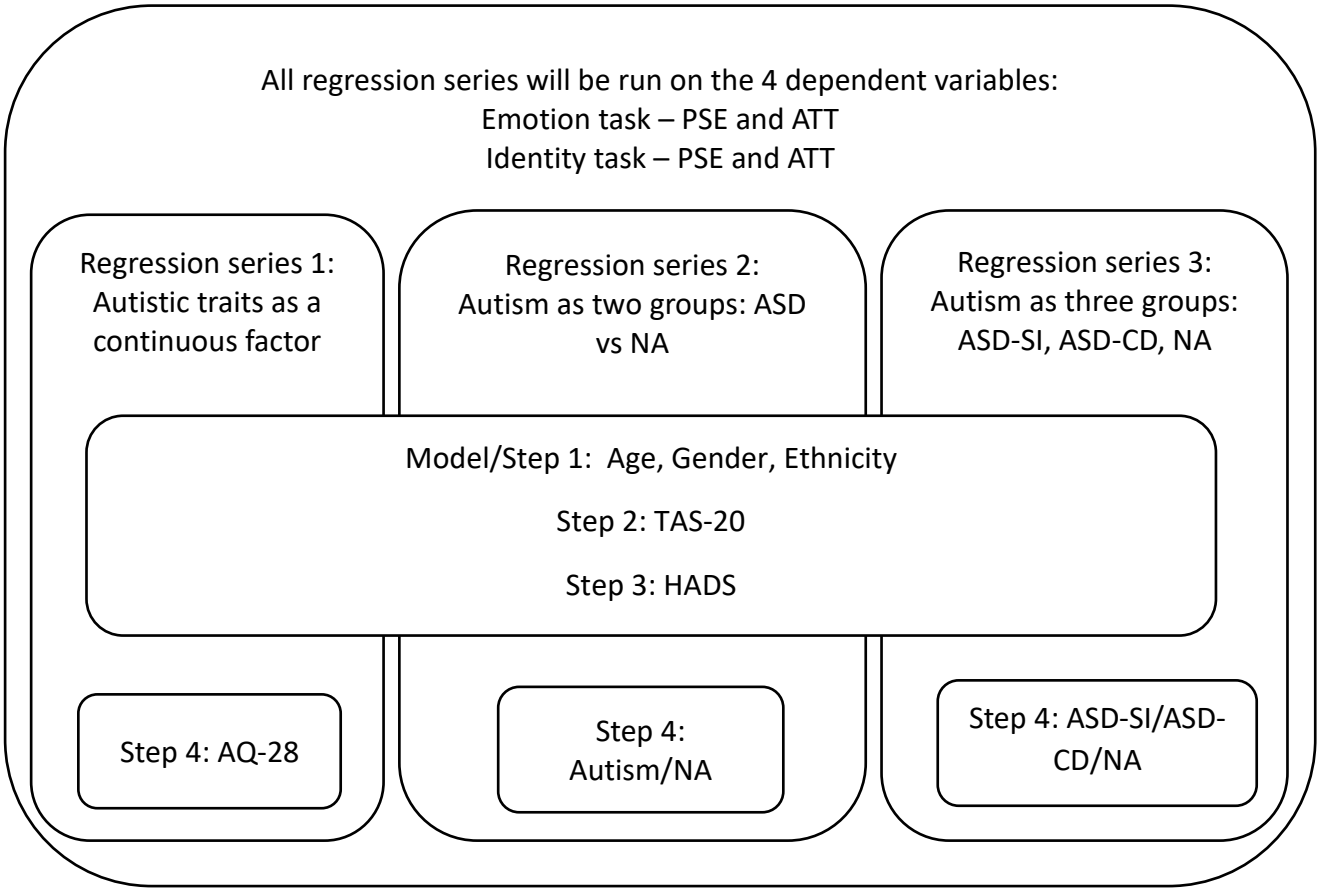
One limitation of the present study is that the non-autistic group showed surprisingly high levels of autistic and alexithymic traits. 46.9%% and 23.5%% of our "non-autistic" sample scored above threshold on the AQ and TAS-20 respectively (by comparison, 97.5 % and 52.5% of the autistic group scored above threshold on these measures, which is in line with previous reports; Hoekstra et al, 2011; Kinnaird et al., 2019). While it suggests our non-autism sample may have been unusual in some way, our regression-based analyses should still have been able to uncover relationships between alexithymia, autistic traits and our face processing measures as there should still have been sufficient variation at the lower end of

these scales for associations to be able to show themselves. Thus, we do not believe atypicalities in the non-autistic sample can account for the lack of effects.

In sum, our present study did not find the expected effects of alexithymia on emotion face recognition. Weak associations between autistic traits and emotion recognition did not survive controlling for other variables in our analyses. This adds to a growing picture of heterogenous results regarding autism and emotion recognition but does not support alexithymia as a strong candidate for explaining this heterogeneity. Future meta-analyses and a review of the evidence base for a relationship between alexithymia and emotion recognition is warranted. We also did not observe associations with mood disorder symptomatology and posit that future research may need more specific measures of specific aspects of mood disorders such as social anxiety to evaluate the role of different forms of mood disorders in emotion recognition. Future research should focus on establishing the influence of gender diversity on socio-emotional abilities in autistic spectrum conditions though we speculate that social experience rather than neurodiversity per se may explain why gender non-conforming individuals showed differences in their perceptions of anger and disgust.

Appendix one

Figure 3.1. Diagram showing the hierarchical regression series



ASD-SI = self-identifying autistics, ASD-CD = clinically diagnosed autistics, NA = non-autistics, AQ-28 = Autism Quotient, TAS-20 = Toronto Alexithymia Scale, HADS = Hospital Anxiety and Depression Scale, PSE = Point of subjective equivalence, ATT = Attribution Threshold.

Tables 3.1-3.9

TABLE 3.1: PARTICIPANT CHARACTERISTICS

	Full Sample (*N* = 178)	Autistic Sample (*N* = 80)	Non-autistic Sample (*N* = 98)
GENDER	*N* Female = 88 *N* Male = 74 *N* Other = 16	N Female = 48 N Male = 21 N Other = 11	N Female = 40 N Male = 53 N Other = 5
AGE	33.14 (13.06)	41.02 (13.07)	26.70 (8.88)
ETHNICITY	*N* Asian = 10 *N* Black = 1 *N* Mixed = 4 *N* White = 163	*N* Asian = 3 *N* Black = 1 *N* Mixed = 3 *N* White = 73	*N* Asian = 7 *N* Mixed = 1 *N* White = 90
HADS-DEPRESSION	8.94 (2.10)	9.25 (2.12)	8.68 (2.06)

HADS-ANXIETY	9.93 (2.99)	8.91 (2.76)	10.77 (2.93)
AQ-28	74.73 (15.31)	86.96 (11.06)	64.74 (10.21)
TAS-20	55.68 (12.86)	60.88 (12.50)	51.44 (11.58)

For non-category data, statistics reported are means with standard deviations in parentheses.

TABLE 3.2: CORRELATIONS BETWEEN ALEXITHYMIA TRAITS, AUTISTIC TRAITS, DEPRESSION AND ANXIETY SYMPTOMS AND FACE PROCESSING VARIABLES

	TAS-TOTAL	AQ	HADS Depression	HADS Anxiety	Emotion PSE (N = 174)	Identity PSE (N = 176)	Emotion attribution (N = 174)	Identity attribution (N = 170)
TAS-TOTAL		.617, p <.001	.076, p =.155	-.308, p <.001	0.023, p = .380	0.115, p =.064	0.093, p =.112	0.042, p = .292
AQ	.617, p<.001		.149, p = .023	-.326, p<.001	0.033, p = 0.334	0.049, p = 0.258	.137, p = 0.036	0.106, p = 0.085
HADS Depression	.076, p =.155	.149, p = .023		-.024, p =.374	.132, p =.041	.079, p =.148	-.080, p =.146	-.142, p =.032
HADS Anxiety	-.308, p <.001	-.326, p <.001	-.024, p =.374		0.024, p = 0.375	0.004, p = 0.480	-0.032, p = 0.336	-0.019, p = 0.402

All correlations are Spearman's Rank and one-tailed. Note that correlations with the face processing variables are with outliers removed, as per Cook's Distance threshold rules, generated for regression series 1 (AQ in final step).

TABLE 3.3: REGRESSION SERIES 1 (AQ ENTERED IN STEP 4)

	Emotion Attribution Total N (ASC, TD) = 174 (77, 97)		Identity Attribution Total N (ASC, TD) = 170 (75, 95)		Emotion PSE Total N (ASC, TD) = 174 (77, 97)		Identity PSE Total N (ASC, TD) = 176 (78, 98)	
	F (Df), p-value	**F change, p-value**	**F (Df), p-value**	**F change, p-value**	**F (Df), p-value**	**F change, p-value**	**F (Df), p-value**	**F change, p-value**
MODEL 1	1.63 (5,168), .156	1.63, 0.156	0.19 (5,164), .968	0.19, 0.968	2.40 (5,168), .039	2.40, 0.039	1.46 (5,170), .207	1.46, 0.207
MODEL 2	1.98 (6,167), .071	3.63, 0.059	0.57 (6,163), .753	2.49, 0.116	2.34 (6,167), .034	1.96, 0.163	1.69 (6,169), .126	2.79, 0.097
MODEL 3	1.57 (8,165), .136	0.39, 0.676	0.93 (8,161), .490	2.00, 0.139	1.96 (8,165), .055	0.83, 0.439	1.26 (8,167), .270	0.01, 0.988
MODEL 4	1.46 (9,164), .165	0.62, 0.432	0.93 (9,160), .505	0.86, 0.354	1.81 (9,164), .070	0.67, 0.415	1.11 (9,166), .356	0.03, 0.868

Model 1 included demographic variables: Age, Gender, and Ethnicity. Model 2 includes demographic variable and the TAS-20 alexithymia scale. Model 3 included these variables, as well as the HADS. Model 4 included the previous variables as well as the AQ-28.

TABLE 3.4: COEFFICIENTS FOR EMOTION PSE REGRESSION SERIES 1 (AQ ENTERED IN STEP 4)

MODEL		Beta	*t*	p-value
1	(Constant)		39.929	<0.001
	Age	-0.119	-1.588	0.114
	Male gender	-0.134	-1.717	0.088
	Other gender	-0.218	-2.792	0.006
	Asian ethnicity	0.053	0.712	0.477
	Mixed ethnicity	0.018	0.239	0.812
2	(Constant)		23.210	0.000
	Age	-0.136	-1.796	0.074
	Male gender	-0.141	-1.803	0.073
	Other gender	-0.225	-2.887	0.004
	Asian ethnicity	0.061	0.809	0.419
	Mixed ethnicity	0.010	0.130	0.896
	TAS-TOTAL	0.106	1.402	0.163
3	(Constant)		13.604	0.000
	Age	-0.138	-1.808	0.072
	Male gender	-0.150	-1.861	0.065
	Other gender	-0.225	-2.880	0.005
	Asian ethnicity	0.049	0.652	0.515
	Mixed ethnicity	0.001	0.008	0.993
	TAS-TOTAL	0.108	1.347	0.180
	HADS Anxiety	0.026	0.322	0.748
	HADS Depression	0.093	1.230	0.220
4	(Constant)		12.636	0.000
	Age	-0.160	-1.977	0.050
	Male gender	-0.133	-1.588	0.114
	Other gender	-0.232	-2.945	0.004
	Asian ethnicity	0.050	0.659	0.511
	Mixed ethnicity	0.004	0.055	0.956
	TAS-TOTAL	0.057	0.552	0.581
	HADS Anxiety	0.035	0.424	0.672
	HADS Depression	0.083	1.084	0.280
	AQ	0.091	0.817	0.415

TABLE 3.5: REGRESSION SERIES 2 (AUTISTIC VS NON-AUTISTIC GROUP STATUS ENTERED IN STEP 4)

	Emotion Attribution Total N (ASC, TD) = 174 (77, 97)		Identity Attribution Total N (ASC, TD) = 170 (75, 95)		Emotion PSE Total N (ASC, TD) =174 (77, 97)		Identity PSE Total N (ASC, TD) = 176 (78, 98)	
	F (Df), p-value	**F change, p-value**	**F (Df), p-value**	**F change, p-value**	**F (Df), p-value**	**F change, p-value**	**F (Df), p-value**	**F change, p-value**
MODEL 1	1.63 (5,168), .156	1.63, 0.156	0.19 (5,164), .968	0.19, 0.968	2.40 (5,168), .039	2.40, 0.039	1.46(5,170), .207	1.46, 0.207
MODEL 2	1.98 (6,167), .071	3.629, 0.059	0.57 (6,163), .753	2.49, 0.116	2.34 (6,167), .034	1.96, 0.163	1.69 (6,169), .126	2.80, 0.097
MODEL 3	1.57 (8,165), .136	0.392, 0.676	0.93 (8,161), .490	2.00, 0.139	1.96 (8,165), .055	0.83, 0.439	1.26 (8,167), .270	0.01, 0.988
MODEL 4	1.59 (9,164), .122	1.66, 0.199	1.07 (9,160), .386	2.125, 0.147	2.11 (9,164), .031	3.15, 0.078	1.14 (9,166), .337	0.26, 0.609

Model 1 included demographic variables: Age, Gender, and Ethnicity. Model 2 includes demographic variable and the TAS-20 alexithymia scale. Model 3 included these variables, as well as the HADS. Model 4 included the previous variables as well autistic vs. non-autistic group status.

TABLE 3.6: COEFFICIENTS FOR EMOTION PSE REGRESSION SERIES 2

MODEL		Beta	t	p value
1	(Constant)		39.929	0.000
	Age	-0.119	-1.588	0.114
	Male gender	-0.134	-1.717	0.088
	Other gender	-0.218	-2.792	0.006
	Asian ethnicity	0.053	0.712	0.477
	Mixed ethnicity	0.018	0.239	0.812
2	(Constant)		23.210	0.000
	Age	-0.136	-1.796	0.074
	Male gender	-0.141	-1.803	0.073
	Other gender	-0.225	-2.887	0.004
	Asian ethnicity	0.061	0.809	0.419
	Mixed ethnicity	0.010	0.130	0.896
	TAS-TOTAL	0.106	1.402	0.163
3	(Constant)		13.604	0.000
	Age	-0.138	-1.808	0.072
	Male gender	-0.150	-1.861	0.065
	Other gender	-0.225	-2.880	0.005
	Asian ethnicity	0.049	0.652	0.515
	Mixed ethnicity	0.001	0.008	0.993
	TAS-TOTAL	0.108	1.347	0.180
	HADS Anxiety	0.026	0.322	0.748
	HADS Depression	0.093	1.230	0.220
4	(Constant)		13.757	0.000
	Age	-0.229	-2.502	0.013
	Male gender	-0.109	-1.297	0.196
	Other gender	-0.247	-3.141	0.002
	Asian ethnicity	0.051	0.675	0.500
	Mixed ethnicity	-0.005	-0.063	0.950
	TAS-TOTAL	0.066	0.792	0.429
	HADS Anxiety	0.048	0.585	0.559
	HADS Depression	0.075	0.987	0.325
	Autism Group (autistic or non-autistic)	0.183	1.774	0.078

TABLE 3.7: REGRESSION SERIES 3 (AUTISTIC VS NON-AUTISTIC VS SELF-IDENTIFYING STATUS ENTERED IN STEP 4)

	Emotion Attribution Total N (ASC, NT) = 174 (77, 97)		Identity Attribution Total N (ASC, NT) = 170 (75, 95)		Emotion PSE Total N (ASC, NT) = 74 (77, 97)		Identity PSE Total N (ASC, NT) = 176 (78, 98)	
	F (Df), p-value	**F change, p-value**	**F (Df), p-value**	**F change, p-value**	**F (Df), p-value**	**F change, p-value**	**F (Df), p-value**	**F change, p-value**
MODEL 1	1.63 (5,168), .156	1.63, 0.156	0.19 (5,164), .968	0.19, 0.968	2.40 (5,168), .039	2.40, 0.039	1.46 (5,170), .207	1.46, 0.207
MODEL 2	1.98 (6,167), .071	3.63, 0.059	0.57 (6,163), .753	2.49, 0.116	2.34 (6,167), .034	1.96, 0.163	1.69 (6,169), .126	2.79, 0.097
MODEL 3	1.57 (8,165), .136	0.39, 0.676	0.93 (8,161), .490	2.00, 0.139	1.96 (8,165), .055	0.83, 0.439	1.26 (8,167), .270	0.01, 0.988
MODEL 4	1.50 (10,163), .145	1.17, 0.312	0.98 (10,159), .461	1.17, 0.314	1.91 (10,163), .047	1.66, 0.193	1.02 (10,165), .427	0.14, 0.871

Model 1 included demographic variables: Age, Gender, and Ethnicity. Model 2 includes demographic variable and the TAS-20 alexithymia scale. Model 3 included these variables, as well as the HADS. Model 4 included the previous variables as well as autistic vs. non-autistic vs. self-identifying group status.

TABLE 3.8: COEFFICIENTS FOR EMOTION PSE REGRESSION SERIES 3

MODEL		Beta	t	p value
1	(Constant)		39.929	0.000
	Age	-0.119	-1.588	0.114
	Male gender	-0.134	-1.717	0.088
	Other gender	-0.218	-2.792	0.006
	Asian ethnicity	0.053	0.712	0.477
	Mixed ethnicity	0.018	0.239	0.812
2	(Constant)		23.210	0.000
	Age	-0.136	-1.796	0.074
	Male gender	-0.141	-1.803	0.073
	Other gender	-0.225	-2.887	0.004
	Asian ethnicity	0.061	0.809	0.419
	Mixed ethnicity	0.010	0.130	0.896
	TAS-TOTAL	0.106	1.402	0.163
3	(Constant)		13.604	0.000
	Age	-0.138	-1.808	0.072
	Male gender	-0.150	-1.861	0.065
	Other gender	-0.225	-2.880	0.005
	Asian ethnicity	0.049	0.652	0.515
	Mixed ethnicity	0.001	0.008	0.993
	TAS-TOTAL	0.108	1.347	0.180
	HADS Anxiety	0.026	0.322	0.748
	HADS Depression	0.093	1.230	0.220
4	(Constant)		13.719	0.000
	Age	-0.237	-2.532	0.012
	Male gender	-0.108	-1.285	0.201
	Other gender	-0.250	-3.160	0.002
	Asian ethnicity	0.048	0.638	0.524
	Mixed ethnicity	-0.003	-0.039	0.969
	TAS-TOTAL	0.065	0.780	0.436
	HADS Anxiety	0.048	0.580	0.563
	HADS Depression	0.076	0.996	0.321
	Formal Diagnosis (vs non-autistic)	0.173	1.710	0.089
	Self-diagnosis (vs non-autistic)	0.122	1.360	0.176

TABLE 3.9: COMPARING FACE PROCESSING VARIABLES, AUTISTIC TRAITS AND ANXIETY AND DEPRESSION SYMPTOMS BETWEEN HIGH AND LOW ALEXITHYMIA GROUPS

	High Alexithymia Group (*Mdn*)	N	Low Alexithymia Group (*Mdn*)	N	Mann Whittney U, Z, p-value (two-sided)
EMOTION ATTRIBUTION	0.1608	63	0.1411	111	3082, -1.298, 0.194
IDENTITY ATTRIBUTION	0.0986	63	0.0958	107	3052.5, -1.026, .305
EMOTION PSE	0.4970	63	0.4932	111	3151.5, -1.080, .280
IDENTITY PSE	0.5284	65	0.5287	111	3348, -.795, .426
AQ	88.00	65	67.00	113	1390.5, -6.90, <.001
HADS ANXIETY	9.00	65	11.00	113	2533.5, -3.46, <.001
HADS DEPRESSION	9.00	65	9.00	113	3163.5, -1.56, .120

Supplementary materials from Chapter Three.

Results from the non-autistic sample: Preregistered analyses conducted before the registered report.

The non-autistic sample had already been collected prior to registration with the journal, and these data were analysed according to our preregistered analysis plan. Originally, data was collected from 119 participants; however, some data sets were incomplete (7 data sets), and some failed the attention check (14 data sets), leaving 98 complete data sets (before checking for outliers; see below). To examine the effect that alexithymia, autism traits, and depression and anxiety have on emotion and identity recognition, hierarchical regressions were conducted.

Before the regressions were performed, checks for collinearity were made. For each regression, collinearity tolerance and the variance inflation factor were all under the appropriate thresholds (the threshold of <10 for the variance inflation factor, and a threshold of >.10 for collinearity). Additionally, checks for outliers were conducted using Cook's distance, with a threshold of 4/n. Seven outliers were identified, leaving data for 91 participants.

Summaries of the regression analyses can be found in Tables S3.1-3.4. The regressions showed that none of the models were significant, at any step, for any of the dependent variables. None of the independent variables contributed significantly to the models. The results suggest that in the neurotypical population, alexithymia, mood disorders, and autism traits do not predict facial emotion and identity recognition. The R^2, R^2 change, and p-values associated with *F* change and overall models for the emotion and identity conditions, PSE and attribution threshold variables, are reported in Tables S3.1-3.4. The unstandardized

regression coefficients, the standardized regression coefficients, and *p*-values for the individual predictors are reported in the Supplementary Materials.

Descriptive statistics for the dependent variables were also examined (see Table S3.5).

Demographic variables (age, gender, ethnicity) were entered in Model 1. The TAS-20 was entered in Model 2. The HADS was entered in Model 3. The AQ-28 was entered in Model 4.

TABLE S3.1: **REGRESSION RESULTS FOR EMOTION RECOGNITION, PSE**

	R^2	**R^2 change**	***p*-value for *F* change**	***p*-value for model**
MODEL 1	.016	.016	.921	.921
MODEL 2	.017	.001	.811	.961
MODEL 3	.021	.004	.846	.986
MODEL 4	.022	.000	.849	.994

***TABLE S3.2*: REGRESSION RESULTS FOR EMOTION RECOGNITION, ATTRIBUTION THRESHOLD**

	R^2	**R^2 change**	***p*-value for *F* change**	***p*-value for model**
MODEL 1	.014	.014	.946	.946
MODEL 2	.016	.002	.646	.966
MODEL 3	.024	.008	.712	.978
MODEL 4	.046	.022	.179	.914

TABLE S3.3. REGRESSION RESULTS FOR IDENTITY RECOGNITION, PSE.

	R^2	R^2 change	*p*-value for *F* change	*p*-value for model
MODEL 1	.041	.041	.600	.600
MODEL 2	.049	.008	.411	.632
MODEL 3	.078	.029	.280	.546
MODEL 4	.093	.015	.257	.512

TABLE S3.4. REGRESSION RESULTS FOR IDENTITY RECOGNITION, ATTRIBUTION THRESHOLD.

	R^2	R^2 change	*p*-value for *F* change	*p*-value for model
MODEL 1	.030	.030	.759	.759
MODEL 2	.030	.000	.890	.855
MODEL 3	.070	.040	.177	.628
MODEL 4	.107	.037	.073	.391

TABLE S3.5: MEAN AND STANDARD DEVIATIONS FOR THE EMOTION AND IDENTITY TASK PSE AND ATTRIBUTION THRESHOLDS

	Emotion task PSE	Emotion task attribution threshold	Identity task PSE	Identity task attribution threshold
MEAN	0.52	0.21	0.53	0.11
STANDARD DEVIATION	0.29	0.47	0.05	0.15

Results of regression analyses with no outliers removed

Below are the correlations and regressions when no outliers are removed. All regressions and correlations are conducted with N = 178. Note that steps 1-3 will be identical for all regression series, as only the predictor in step 4 will change.

TABLE S3.6: CORRELATIONS BETWEEN FACE PROCESSING VARIABLES, ALEXITHYMIC TRAITS, AUTISTIC TRAITS, DEPRESSION AND ANXIETY SYMPTOMS

	TAS-TOTAL	AQ	HADS Anxiety	HADS Depression	EMO PSE	ID PSE	EMO ATT	ID ATT
TAS-TOTAL		.617**, *p* <.001	-.308**, *p* <.001	0.076, *p* = 0.155	0.0112, *p* = 0.437	0.119, *p* = 0.057	0.099, *p* = 0.095	0.033, *p* = 0.33
AQ	.617**, <.001		-.326**, <.001	.149*, *p* = 0.023	0.04, *p* = 0.297	0.034, *p* = 0.328	.160*, *p* = 0.017	0.071, *p* = 0.172
HADS ANXIETY	-.308**, <.001	-.326**, <.001		-0.024, *p* = 0.374	0.014, *p* = 0.425	0.002, *p* = 0.487	-0.075, *p* = 0.159	-0.043, *p* = 0.285
HADS DEPRESSION	0.076, *p* = 0.155	.149*, *p* = 0.023	-0.024, *p* = 0.374		.138*, *p* = 0.033	0.071, *p* = 0.171	-0.051, *p* = 0.249	-.132*, *p* = 0.039
EMO PSE	0.012, *p* = 0.437	0.04, *p* = 0.297	0.014, *p* = 0.425	.138*, *p* = 0.033		0.097, *p* = 0.098	.248**, <.001	.141*, *p* = 0.03
ID PSE	0.119, *p* = 0.057	0.034, *p* = 0.328	0.002, *p* = 0.487	0.071, *p* = 0.172	0.097, *p* = 0.098		-0.04, *p* = 0.299	-.139*, *p* = 0.032
EMO ATT	0.099, *p* = 0.095	.160*, *p* = 0.017	-0.075, *p* = 0.159	-0.051, *p* = 0.249	.248**, <.001	-0.04, *p* = 0.299		.361**, <.001
ID ATT	0.033, *p* = 0.33	0.071, *p* = 0.172	-0.043, *p* = 0.285	-.132*, *p* = 0.039	.141*, *p* = 0.03	-.139*, *p* = 0.032	.361**, <.001	

TABLE S3.7: REGRESSION SERIES 1-3

	EMO ATT		**ID ATT**		**EMO PSE**		**ID PSE**	
MODEL STEP (DFS)	F, p-value	F change, p-value	F, p-value	F change, p-value	F, p-value	F change, p-value	F, p-value	F change, p-value
MODEL 1 (6, 171)	.31, .929	.31, .929	1.74, .114	1.74, .114	.80, .572	.80, .572	.59, .736	.59, .736
MODEL 2 (7,170)	.35, .929	.58, .449	1.65, .124	1.10, .297	.69, .683	.05, .826	.51, .830	.002, .962
MODEL 3 (9, 168)	.90, .526	2.80, .063	1.43, .179	.674, .511	.97, .467	1.93, .148	.58, .810	.86, .425
MODEL 4 - AQ (10, 167)	.95 , .489	1.37, .243	1.30, .236	.159, .690	1.05, .401	1.77, .185	.63, .784	1.08, .300
MODEL 4 – ASC VS NT (10, 167)	.81, .615	.08, .781	1.29, .238	.141, .708	.93, .507	.59, .443	.59, .819	.68, .412
MODEL 4 – ASC VS NT VS SI (11, 166)	.78, .657	.29, .753	1.20, .288	.25, .780	.85, .593	.33, .719	.58, .841	.59, .553

Model 1 included demographic variables: Age, Gender, and Ethnicity. Model 2 includes demographic variables and the TAS-20 alexithymia scale. Model 3 included these variables, as well as the HADS. The regression series were ran 3 times with different Model 4 variables. Firstly, Model 4 included the previous variables as well as the AQ-28. Secondly, with autistic vs. non-autistic group status. Thirdly, with autistic vs. non-autistic vs. self-identifying group status.

Results of TAS Group analyses with no outliers removed

TABLE S3.8: COMPARING FACE PROCESSING VARIABLES BETWEEN HIGH AND LOW ALEXITHYMIA GROUPS

	High Alexithymia Group (Mdn) (N=113)	Low Alexithymia Group (Mdn) (N = 65)	Mann Whittney U, Z, p-value (two-sided)
EMO ATT	0.14	0.16	3197, -1.44, .151
ID ATT	0.09	0.10	3371, -.91, .362
EMO PSE	0.49	0.50	3379, -.885, .376
ID PSE	0.53	0.53	3413, -.784, .433

Results of regression analyses with only AQ congruent participants

Below are the regression results when only autistic participants who score above the AQ28 cut off, and non-autistic participants who score below the AQ28 cut off, are included. All regressions conducted with N =130. Note that steps 1-3 will be identical for all regression series, as only the predictor in step 4 will change.

TABLE S3.9: REGRESSION SERIES 1-3 (AQ CONGRUENT PARTICIPANTS ONLY)

	EMO ATT		**ID ATT**		**EMO PSE**		**ID PSE**	
MODEL STEP (DFS)	**F, p-value**	**F change, p-value**	**F, p-value**	**F change, p-value**	**F, p-value**	**F change, p-value**	**F, p-value**	**F change, p-value**
MODEL 1 (6, 123)	.46, .840	.46, .840	.31, .932	.31, .932	.96, .456	.96, .456	.60, .734	.60, .73
MODEL 2 (7, 122)	.77, .615	2.61, .109	.59, .764	2.26, .135	1.03, .417	1.41, .238	.51, .826	.02, .879
MODEL 3 (9, 120)	.96, .473	1.62, .202	.75, .667	1.29, .281	1.25, .272	1.97, .144	.58, .814	.82, .445
MODEL 4 - AQ (10, 119)	.94, .502	.71, .401	.71, .711	.45, .504	1.34, .217	2.07, .153	.58, .825	.67, .416
MODEL 4 – ASC VS NT (10, 119)	.87, .563	.09, .771	.80, .631	1.25, .265	1.22, .284	.983, .324	.59, .819	.73, .396
MODEL 4 – ASC VS NT VS SI (11, 118)	.87, .572	.48, .620	.77, .672	.87, .422	1.10, .366	.49, .613	.57, .853	.54, .585

Model 1 included demographic variables: Age, Gender, and Ethnicity. Model 2 includes demographic variables and the TAS-20 alexithymia scale. Model 3 included these variables, as well as the HADS. The regression series was ran 3 times with different Model 4 variables. Firstly, Model 4 included the previous variables as well as the AQ-28. Secondly, with autistic vs. non-autistic group status. Thirdly, with autistic vs. non-autistic vs. self-identifying group status.

Results of regressions using TAS-8 alexithymia measure

Tables S3.10-3.12 summarises the results of the regressions conducted using the reduced TAS-8 alexithymia measure. Note that these regressions used the Cook's Distance outlier rule: this means that each regression may have different outliers removed, as Cook's Distance is calculated for specific regressions. Demographic variables (age, gender, ethnicity) were entered in Model 1. The TAS-8 was entered in Model 2. The HADS was entered in Model 3. The regression series was repeated three times with a different Model 4. Firstly, Model 4 included the previous variables as well as the AQ-28. Secondly, with autistic vs. non-autistic group status. Thirdly, with autistic vs. non-autistic vs. self-identifying group status.

TABLE S3.10: REGRESSION SERIES 1 (AQ ENTERED IN STEP 4) USING TAS8

	EMO ATT n = 174 (77, 97)		ID ATT 169 (75, 94)		EMO PSE (174 (77, 97))		ID PSE 176 (78, 98)	
	F (Df), p-value	**F change**	**F (Df), p-value**	**F change**	**F (Df), p-value**	**F change**	**F (Df), p-value**	**F change**
MODEL 1	1.63 (5,168), .156	1.63, 0.156	0.19(5,163), .966	0.19, 0.966	2.40 (5,168), .039	2.40, 0.039	1.46 (5,170), .207	1.46, 0.21
MODEL 2	1.77 (6,167), .109	2.40, 0.123	0.33(6,162), .919	1.05, 0.306	2.34 (6,167), .034	1.97, 0.162	1.68 (6,169), .129	2.73, 0.101
MODEL 3	1.40(8,165), .202	0.33, 0.723	0.73 (8,160), .663	1.92, 0.150	1.99 (8,165), .051	0.94, 0.392	1.25(8,167), .274	0.01, 0.987
MODEL 4	1.38(9,164), .202	1.22, 0.271	0.88 (9,159), .548	1.99, 0.160	1.83 (9,164), .066	0.63, 0.428	1.10 (9,166), .363	0.006, 0.940

TABLE S3.11: REGRESSION SERIES 2 (AUTISM GROUP ENTERED IN STEP 4) USING TAS8

	EMO ATT 174 (77, 97)		ID ATT 169 (75, 94)		EMO PSE 174 (77, 97)		ID PSE176 (78, 98)	
	F (Df), p-value	**F change**	**F (Df), p-value**	**F change**	**F (Df), p-value**	**F change**	**F (Df), p-value**	**F change**
MODEL 1	1.63 (5,168), .156	1.63, 0.156	0.19 (5,163), .966	0.19, 0.966	2.40 (5,168), .039	2.40, 0.039	1.46 (5,170), .207	1.46, 0.207
MODEL 2	1.77 (6,167), .109	2.40, 0.123	0.33 (6,162), .919	1.05, 0.306	2.34 (6,167), .034	1.97, 0.162	1.68 (6,169), .129	2.73, 0.101
MODEL 3	1.40(8,165), .202	0.33, 0.723	0.73(8,160), .663	1.92, 0.150	1.99 (8,165), .051	0.94, 0.392	1.25 (8,167), .274	0.01 0.987
MODEL 4	1.46 (9,164), .169	1.87 0.173	0.96 (9,159), .475	2.72, 0.101	2.12 (9,164), .030	3.01, 0.084	1.14 (9,166), .341	0.28, 0.600

TABLE S3.12: REGRESSION SERIES 3 (SELF-IDENTIFICATION ENTERED IN STEP 4) USING TAS8

	EMO ATT 174 (77, 97)		ID ATT 169 (75, 94)		EMO PSE 174 (77, 97)		ID PSE 176 (78, 98)	
	F (Df), p-value	**F change**	**F (Df), p-value**	**F change**	**F (Df), p-value**	**F change**	**F (Df), p-value**	**F change**
MODEL 1	1.63(5,168), .156	1.63, 0.156	0.19(5,163), .966	0.19, 0.966	2.40 (5,168), .039	2.40, 0.039	1.46 (5,170), .207	1.46, 0.207
MODEL 2	1.77 (6,167), .109	2.40, 0.123	0.33(6,162), .919	1.05, 0.306	2.34 (6,167), .034	1.97, 0.162	1.678(6,169), .129	2.73, 0.101
MODEL 3	1.40(8,165), .202	0.33, 0.723	0.73 (8,160), .663	1.92, 0.150	1.99 (8,165), .051	0.94, 0.392	1.25 (8,167), .274	0.01, 0.987
MODEL 4	1.37 (10,163), .197	1.26, 0.285	0.88 (10,158), .553	1.45, 0.237	1.92(10,163), .046	1.60, 0.206	1.02 (10,165), .431	0.15, 0.865

Chapter Four: The impact of face masks on autistic and non-autistic adults' face processing abilities

Declarations

Chapter Four has been submitted for publication at Research in Autism Spectrum Disorders (RASD) and is currently under review. A preprint is available to view: Hobson, Hannah Madaleine and Baxter, Natasha and Harlow, Lucy and Harrison, Ebony and Smith, Caitlin, The Impact of Face masks on Autistic and Non-Autistic Adults' Face Processing Abilities. Available at:
SSRN: https://ssrn.com/abstract=4467011 or http://dx.doi.org/10.2139/ssrn.4467011

Note that supplementary material for this chapter can be found in Appendix 2.

Ethics Approval

Informed consent was obtained from participants before taking part in the study. Ethics approval was obtained from the ethics committee of the Psychology department, University of York, UK (ID: 112).

Availability of data

This study was pre-registered on the Open Science Framework, see here: https://osf.io/vsgfw/

Competing interests

The authors have no competing interests to declare.

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Author's contributions

Natasha Baxter made substantial contributions to the conception and design of the study, data collection, data analysis and interpretation, and drafted the paper. Hannah Hobson made substantial contributions to the design of the study, data analysis and interpretation, and provided feedback/edited the paper. Lucy Harlow, Caitlin Smith, and Ebony Harrison

contributed to data collection of the non-autistic sample. All authors approved the submitted manuscript.

Abstract

Atypical emotion recognition is argued to be characteristic of autism spectrum conditions (ASC), underpinned in part by atypical eye gaze, with less eye contact and more gaze direction towards the mouth. Given the widespread adoption of face masks due to the Covid-19 pandemic, exploring the effect of face masks on face processing in autism, as they occlude the mouth region, is of interest. This study investigated the impact of face masks on emotion and identity recognition, with a non-autistic (N = 38) and autistic sample (N = 25). Participants also completed the Toronto Alexithymia Scale, and the abridged Autism Quotient, to investigate the roles of alexithymia and autistic traits in any impacts of face masks on face processing abilities. Masks were detrimental to performance for both autistic and non-autistic groups, and masks affected participants' performance on the emotion recognition task more than the identity recognition task. Autistic participants' performance was more negatively affected by the presence of masks, for both identity and emotion recognition. The impact of face masks was not predicted by alexithymia or autistic traits, after gender, ethnicity and age were considered. Overall, the results suggest that masks negatively affect face processing for both non-autistic and autistic groups, particularly emotion recognition, however the impact of masks is amplified for autistic people. These results supplement reports of autistic people that masks interfere with social interactions.

Introduction

As a consequence of the Covid-19 pandemic, for many across the globe wearing a face mask became, (and in some parts of the world remains) part of daily life. While an important public health measure to help protect individuals from the spread of Covid-19, face masks may negatively affect social interactions. Indeed, multiple studies have reported detrimental effects of masks on identity recognition and emotion recognition, and on participants' confidence in emotion recognition (Carbon 2020; Marini et al, 2021; Parhoozi, Forby, & Kingstone 2021). Some populations with pre-existing atypical face processing abilities could potentially be more adversely affected by face masks than others: the focus of the current paper is the impact of face masks on the face processing abilities of autistic people.

Prior to the pandemic and the rise in the use of face masks, research already indicated that obscuring facial regions impacts face processing, including emotion recognition. For example, participants are less able to recognise emotions in faces wearing a niqab, with happiness recognition particularly affected, arguably due to the loss of information about the mouth region (Fischer et al., 2012). Indeed, the mouth region is most frequently attended to when looking at faces for emotion recognition (Blais et al., 2012). Face masks, which cover up the mouth region, would thus be expected to impact people's abilities to recognise others' emotions.

Some populations may be more adversely affected by the disrupting influence of covering regions of the face, such as by face masks, than others, especially if face processing was atypical to begin with. Previous research has identified atypical facial emotion recognition in autistic individuals (Riby, Doherty-Sneddon, & Bruce, 2009; Uljarevic & Hamilton, 2013). A meta-analysis conducted by Uljarevic and Hamilton (2013) reported poorer emotion recognition (of a moderate effect size) in autistic samples compared to typically developed samples. One factor in reduced emotion recognition could be atypical eye gaze and contact. Wieckowsi and White (2017) investigated emotion recognition with an eye tracking study including dynamic stimuli and autistic youths. They found that autistic participants showed less fixation to the eye region and were poorer at recognising negative emotions. Results also demonstrated that participants fixated more to the mouth region, which the researchers concluded was the cause of the impaired emotion recognition. Indeed, in addition to increased fixations to the mouth region, research indicates that autistic individuals may pay

less attention to the eyes (Bal et al, 2010). Cuve, Gao and Fuse (2018) conducted a systematic review examining eye-tracking studies and emotion recognition in autism. They found less eye contact in autistic individuals compared to non-autistic individuals (characterised by reduced preference to the eye region, and fixations away from the eyes), that longer fixation time to the eyes was associated with better performance, and that fixation to the mouth region was not associated with more accurate emotion recognition.

Given atypical emotion processing in autism, and given evidence suggests autistic people look more at the mouth region (which is naturally obscured by face masks), it could be predicted that face masks would perhaps further exacerbate emotion processing difficulties in autistic individuals. Evidence on this is at present mixed. Pazhoohi, Forby, and Kingstone (2021) used samples from students and the general population, but they also included a measure of autism traits (the AQ; Autism Quotient 10 item version). They found that participants with high AQ scores were less accurate and less confident on emotion recognition, compared to the low AQ participants. However, while they found interaction effects between mask condition and AQ group for confidence ratings, they did not find these for accuracy: this suggests that while people who have high autistic traits show greater reductions in confidence when masks are introduced, masks do not adversely affect their actual accuracy over and above how much masks affect people low in autistic traits. This research is limited in that it lacks a clinical autism sample, relying instead on individuals who are high in autistic traits. Research with face masks has been conducted using a clinical autistic population, but with a focus on identity recognition. Autistic participants were poorer compared to a typically developed group at face recognition when presented with face mask stimuli after learning the faces un-obscured (Tso, Chui, Hang & Hsiao, 2021).

Furthermore, Wallace, Coleman, and Bailey (2008) presented autistic participants with stimuli just showing either the eyes or the mouth. Results demonstrated that participants were impaired when recognising fearful eyes and disgusted mouths, and they confused fearful eyes with being angry. These findings suggest that emotion recognition is affected when not all areas of the face are available, suggesting that face masks would compound emotion recognition abilities in autism. Aside from these behavioural studies, recent research into autistic people's experiences with face masks (both wearing them and being around others who wear them) highlights that face masks have detrimental effects on understanding others

and on social interactions (Clegg, Wood, Hobson & Sedgewick, 2023). These effects are in part due to the loss of information that helps autistic people gauge others' reactions, but also interferes with their ability to lip read.

It is important to note that emotion recognition ability is highly heterogeneous in autism, and that autism per se may not be the source of emotion recognition problems; indeed, some research has suggested that alexithymia, not autism, is the cause of atypical emotion recognition and eye gaze behaviour in some populations (Bird & Cook, 2013). Alexithymia is the inability to recognise and describe one's emotions (Sifneos, 1973). It is more common in autism than in the general population, with 47% of females and 21% of males having co-occurring alexithymia, relative to 10% of the non-autistic population (Oakley et al., 2020), but while typically correlated, alexithymic and autistic traits are separate constructs (Cuve et al., 2021). Alexithymia is associated with poorer socioemotional abilities, and differences in eye gaze during face processing. Fujiwara (2018) investigated alexithymia and emotion recognition in high and low alexithymic non-autistic participants using a behavioural task and eye-tracking. Results indicated that, compared to participants low in alexithymic traits, highly alexithymic participants had less accurate emotion recognition and showed a reduced preference to the eye region (as measured by total dwell time). Intriguingly, this is a similar pattern of results to those found in studies with autistic participants. If alexithymia negatively affects emotion recognition in typically developed participants, could it also be the factor driving atypical emotion recognition and eye gaze behaviour in autism?

Evidence thus far supports the idea that alexithymia may underpin atypical emotion processing in autism. The systematic review conducted by Cuve et al (2018) argued that alexithymia has been associated with atypical eye gaze in some emotion processing studies (Bird, Press & Richardson, 2011). Other research has found that alexithymia accounts for emotion processing difficulties in autism, rather than autism per se. Ola and Gullon-Scott (2020) reported that higher levels of alexithymia are associated with poorer emotion recognition in autistic females. High levels of alexithymia have also been found to impact autistic people's abilities to recognise lower intensity emotions (Ketelaars et al., 2016); this may suggest that high intense emotional expressions are recognisable even by highly alexithymic individuals, but alexithymia reduces individuals' abilities to distinguish between more subtle emotional presentations.

If it is alexithymia that modulates emotion recognition, then perhaps the detrimental effects of face masks may compound difficulties associated with alexithymia rather than autism. Recent research has indeed sought to answer whether alexithymia may exacerbate difficulties in emotion recognition caused by covering parts of the face. Maiorana et al. (2022) conducted a facial emotion recognition task with typically developed participants, with stimuli wearing face masks, or without face masks and showing the eyes or mouth only. Participants' alexithymia scores correlated with their reaction times, with those high in alexithymia taking longer to process faces; interestingly, when masks were present, there was no correlation between reaction time and alexithymia scores. This suggests that the impact of alexithymia on face processing is affected by the presence or absence of masks.

This study aimed to investigate the impact of face masks on facial emotion and identity recognition in autistic and typically developed adults, and the role of alexithymia in the impact of face masks. We firstly hypothesise that the presence of face masks will lead to decreased emotion recognition abilities. Secondly, we hypothesise that autism and higher autistic traits will predict decreased emotion recognition abilities. Thirdly, we predict higher alexithymic traits will detrimentally affect emotional recognition abilities. Finally, we will investigate whether alexithymia rather than autistic traits predicts the impact of face masks on face processing abilities.

Methods

Preregistered design

Our design, methodological approach and main analyses were all preregistered prior to the start of data collection. The pre-registration can be accessed via the Open Science Framework: https://osf.io/n8cd2

Our power analyses were guided by the means and standard deviations for attribution thresholds, for autistic and non-autistic participants, as reported in Cook et al (2013) and Brewer et al (2015). Given that our key hypothesis was that there would be a three-way interaction between group, mask condition and emotion-identity task, such that autistic participants would be particularly negatively affected by face masks when performing emotion recognition, we used the Shiny app to perform Monte Carlo simulations of our factorial experimental design and plan a suitable sample size. Simulations indicated that

having 25 participants per group, assuming a large interactive effect, would provide 88.9% power. Our other planned analyses were to perform regressions to examine the predictive power of alexithymia versus autistic traits for the impact of face masks on emotion versus identity recognition. We planned thus to recruit more than 25 per group in order to perform these regressions: we planned to stop collecting data once 100 participants had been recruited due to time and budget constraints. As described below, the nature of the dependent variables meant that not all complete datasets could be included in the analyses.

Participants

A total of 93 participants were recruited, but only 63 complete datasets were obtained, comprising 38 typically developed and 25 autistic participants. For the typically developed group, 11 participants could not have their dependent variables generated (see Design for more detail), and 1 participant had incomplete data. For the autistic group, 18 participants could not have their dependent variables generated. In total, 30 participants could not be included in data analysis. Participants were recruited via social media, through the University of York Psychology department (control sample), and through the Autistica research network and local autism support services (autistic sample). All participants were aged 18 years or over and were fluent in English. The autistic sample were asked if they were clinically diagnosed or self-identify as autistic. Self-identifiers were included in this research because a formal autism diagnosis can be hard to attain as an adult, and as a female, and the practise of self-identification is becoming more common within the autistic community (Cook, Hull, Crane & Mandy, 2021; Murphy et al., 2016; Sarrett, 2016). Three self-identifiers took part in the study, and 22 clinically diagnosed autistic participants (and thus their inclusion is unlikely to have biased our results, given their small number). Table 4.1 summarises the participant groups' key characteristics (see Appendix 2). The autistic group scored significantly higher on the autistic and alexithymic trait measures (described below), and were significantly older, but did not differ in terms of their ethnicity or gender breakdowns.

Materials

Identity and emotion recognition task

Face identity and emotion recognition tasks were based on Cook et al (2013), with some additions by the current research team. The stimuli consist of 28 images, 14 of which are

from the original task, and 14 which are the same original images but with a face mask edited to cover the mouth and nose (see *Figure 4.1 in Appendix 2*).

The 14 images consist of two cross-morphed images of Ekman faces (Calder et al., 1996), on an identity and emotion continuum (see *Figure 4.2*). 14 total images of "Harold" morphing to "Felix", and anger morphing to disgust were created. Each point represents an image, starting at 80% intensity for one emotion and identity, and 20% the other identity and emotion, with percentages of identity and emotion increasing in increments of 10%. For example, the first image would show 80% Harold and anger, with 20% Felix showing disgust.

Questionnaire measures

Two self-report questionnaires were used to measure alexithymia and autism traits. Alexithymia was measured by the Toronto Alexithymia Scale (TAS-20, Bagby, Parker, and Taylor, 1994), which consists of three subscales: externally-oriented thinking (8 items), difficulty identifying feeling (7 items), and difficulty describing feelings (5 items). The TAS-20 has been used previously in autism and alexithymia research and has been shown to be appropriate to be used with both general and autistic populations (Bagby, Parker and Taylor, 2020; Schroeders, Kubera and Gnambs, 2021). The TAS-20 has also been shown to have strong test-retest reliability and validity (Berthoz and Hill, 2005).

Autistic traits were measured by the abridged Autism Quotient (AQ-28, Hoekstra et al., 2011), consisting of 28 items which are taken from the AQ-50 (Baron-Cohen et al., 2001). Five aspects of autistic traits are reflected in the items: routine, imagination, switching, social skills, and recognising numbers and patterns. The AQ-28 has been argued to have acceptable internal consistency (Ashouri, Asgharzade, Ebrahimi, Ghojazadeh, & Akbarzadeh, 2020).

Design

This study used a mixed design: all participants experienced both the mask and no-mask conditions, and emotion and identity trial types, while participants were either in our autistic or non-autistic group. The task generates two dependent variables: the point of subjective equivalence (PSE) and attribution threshold, for both emotion and identity recognition. Attribution threshold is a measure of precision, and the PSE is a measure of bias describing

the point on the identity or emotion recognition dimension at which participants are of equal likelihood to make either attribution (Cook et al., 2013).

To generate our key variables, MATLAB (specifically the PALAMEDES toolbox, Prins & Kingdom, 2009) was used to fit cumulative Gaussian functions in order to provide estimates of psychometric functions. This was done to extract the PSE and attribution threshold, for both emotion and identity, for each participant. Note that for some participants, the distribution of their responses would not allow a function to be fitted, and thus dependent variables could not be generated.

Procedure

Ethics approval was given by the University of York's Psychology department ethics board. All participants gave informed consent before participating. This study took place online, using the experiment software 'Gorilla' (Anwyl-Irvine, Massonié, Flitton, Kirkham, & Evershed, 2019; www.gorilla.sc).

The task began with a learning and feedback phase, where participants were shown the stimuli with identity and emotion labels, for a block of 12 trials: this was included because the identities of Harold and Felix would be unfamiliar to our participants, and they would need to have seen then before being tested on the cross-morphed stimuli. Participants then completed a practice phase, simulating the main task, lasting for 8 trials. Responses in the learning and feedback and practice phase did not feed into the dependent variable scores and were not used to screen participants (i.e. participants did not have to perform to a certain level to enter the main experimental task). The participants then completed the main task, comprising 560 trials. Stimuli were presented in 20 blocks of 28 trials. On a given trial, face stimuli were presented for 800 milliseconds, and participants were then asked either whether the image showed disgust or anger, or Harold or Felix. Trial types (mask or no mask, identity or emotion question) were randomised across all blocks. Between each block, participants took a short break, and were shown a screen with the four images at 80% intensity with labels for participants to continue to familiarise themselves with the emotions and identities. Participants then completed a demographics questionnaire, the TAS-20, and the AQ-28. The study overall took approximately 1.5 hours.

Results

Our data was found to be non-normal using the Shapiro-Wilks test, on all dependent variables except for the non-autistic group on the identity condition (for both PSE and attribution threshold). Given this, for our analyses, we conducted both parametric and non-parametric tests. We did not stipulate a means to reject outliers in our pre-registration, and given minimum power requirements, outliers have been retained in our analyses.

A three-way mixed ANOVA was conducted, with factors mask condition (mask versus no mask), task (identity versus emotion recognition) and participant group (autistic versus non-autistic). Sphericity was assumed and Levene's test was non-significant for each variable. Note that summaries of main effects and interaction effects are supplemented in places with estimated marginal means and standard errors rather than conventional true arithmetic means and standard deviations in places: this is because of the nature of the dependent variables. Calculating a true arithmetic average for all masked stimuli, regardless of whether they were seen in the emotion or the identity condition, would be problematic, as the attribution thresholds are calculated specific to the task in which the stimuli were seen.

Mean PSE scores across conditions and groups are summarised in Table 4.2. For the PSE, there were no significant main effects or interaction effects.

Mean attribution thresholds across conditions are shown in Table 4.3; note that lower values correspond to better performance. There was a significant effect of mask condition: $F(1, 61) = 28.13$, $p < .001$. Performance was worse in the mask condition (Estimated Marginal Mean = .52, SE = .07) compared to the no mask condition (Estimated Marginal Mean = .21, SE = .04). A significant effect of task was also found: $F(1,61) = 24.18$, $p < .001$. Performance was worse in the emotion task (Estimated Marginal Mean = .60, SE = .10) compared to the identity task (Estimated Marginal Mean = .12, SE = .01).

Additionally, there was a significant interaction effect between mask condition and participant group: $F(1,61) = 4.29$, $p = .043$. While both participant groups performed more poorly in the mask compared to no mask condition, the effect of masks was greater for the autistic group than the non-autistic group (see *Figure 4.3*; see also, non-parametric analyses below). To examine this interaction effect further, independent t-tests were conducted to compare the performance of the autistic and non-autistic groups on the mask and no mask

conditions, for emotion and identity tasks. None of the tests were significant, suggesting that the groups performed similarly when specific mask and task conditions were compared.

There was also a significant interaction between mask condition and task: $F(1,61) = 23.70$, $p < .001$. Both emotion and identity tasks were negatively by the presence of masks, but emotion trials showed a greater negative effect of masks (See *Figure 4.4*).

No significant interaction was found between task and participant group. There was also no significant three-way interaction between condition, task, and group. There was also no significant overall main effect of participant group.

Given the non-normality of the data, and potential impacts on our ANOVA, Kruskal-Wallis tests and Wilcoxon tests were also conducted, in addition to our preregistered parametric analyses. These non-parametric tests largely confirmed the results of the ANOVA. Wilcoxon tests were used to compare emotion and identity recognition performance, and the impact of masks, in each participant group separately. For the non-autistic group, a significant effect of masks was found for both emotion ($Z = -4.76$, $p < .001$) and identity recognition ($Z = -2.75$, $p = .006$). For the autistic group, a significant effect of masks on both emotion ($Z = -4.37$, $p < .001$) and identity ($Z = -3.67$, $p < .001$) recognition were also found. Thus, both groups were affected by masks for both emotion and identity recognition.

To test for an interactive effect of participant groups and mask condition, we first calculated difference scores for the attribution scores, which were to be used in our preregistered regressions: the attribution scores from the mask condition were subtracted from the scores from the no-mask condition. These scores were then compared between the non-autistic and autistic groups. Table 2.4 summarises the difference scores calculated, including the difference scores used in our regressions (described below). The Kruskal-Wallis test indicated an effect of participant group on mask-no mask difference scores, both for the emotion task ($H(1) = 5.52$, $p = .019$) and the identity task ($H(1) = 4.81$, $p = .02$). This indicates that there was a greater effect of masks for the autistic group, in either task (see also Table 4. 3).

The role of continuous autistic and alexithymic traits

As per our pre-registered analyses, correlations and regressions were conducted to investigate a) the predictive power of alexithymia and autistic traits on facial emotion and identity

recognition, and b) the predictive power of alexithymia and autistic traits on *the impact of masks* on facial emotion and identity recognition. While our previous analyses compared autistic versus non-autistic participants, these analyses allowed for the investigation of the role of continuous autistic traits, using AQ scores, in addition to alexithymia via TAS-20 scores.

Table 4.5 summarises the correlations between emotion and identity attribution scores, PSE scores, and AQ and TAS scores. Neither autistic traits nor alexithymic traits correlated significantly with any of the attribution threshold or PSE scores.

With regards to whether autistic traits or alexithymia predicted emotion or identity recognition, four hierarchical regressions were conducted, on the attribution threshold and PSE scores for the identity and emotion tasks in the no mask condition. Each regression had the same three steps: firstly, demographic variables were entered (age, gender, and ethnicity), secondly the TAS-20, then thirdly the AQ-28. This order allowed us to examine whether autistic traits explained any additional variance once alexithymia was accounted for. For brevity, the statistics for these regressions are reported in Appendix 2.

For the regressions using the emotion and identity attribution thresholds, models were non-significant but certain steps and predictors did account for some variance. Ethnicity was a significant predictor for the identity attribution threshold (see Table S4.12 in Appendix 2), although the model was overall non-significant. Additionally step 1 and 3 of our regression for the emotion attribution threshold was significant, although none of the predictor variables were significant (see Table S4.10 in Appendix 2). Our regression models were not significant at any stage, except for the regression model for the PSE scores from the emotion task which was significant: our model including our demographic variables significantly predicted PSE scores. In terms of individual predictors, “other” gender predicted PSE scores, as did ethnicity, at all three steps of the model (see Table S4.13 in Appendix 2). Subsequent steps added to the model did not explain a significant amount of additional variance. Regression models for PSE scores for our identity task were not significant: adding AQ scores at step 3 did add significant explained variance to the model (though the model overall was not significant). In terms of individual predictors, “other” gender and AQ scores were significant (though again in the context of an overall non-significant model; see Tables S4.15 and S4.16 in Appendix 2).

With regards to examining the role of autistic versus alexithymic traits on the impact of face masks, there were four further regressions conducted, on our four dependent variables difference scores: firstly, the difference scores between the mask and no mask conditions for attribution in the emotion task; secondly, the difference scores between the mask and no mask conditions for the identity task; thirdly, the difference scores between the mask and no mask conditions for PSE for the emotion task; fourthly, the difference scores between the mask and no mask conditions for PSE for the identity task. Each regression had the same three steps as described above: firstly, demographic variables, secondly the TAS-20, then thirdly the AQ-28.

Both AQ and TAS scores were significantly correlated with the difference score for emotion attribution (see Table 4.6), specifically higher autistic or alexithymic traits patterned with a more negative impact of masks on emotion recognition. However, for our regressions, none of the models were significant, at any step, for any of the dependent variables. For brevity, the statistics for these regressions are reported in Appendix 2. None of the predictors contributed significantly to the models, at any step. Thus, neither alexithymia nor autistic traits predicted the impact of face masks on facial emotion and identity recognition, once demographic variables had been considered.

Unregistered interaction analyses

Whilst unregistered, it was noted that some of our variables may interact due to the close nature of the relationships between (often co-occurring, with overlapping symptomology, and may mediate each other). We examined the interaction between alexithymia and autistic traits by adding an alexithymia group factor to our ANOVA (Task x Mask x Autism Group x TAS Group). There are no additional interaction effects when TAS Group is added, but the addition of TAS Group does seem to remove the previous interactions with autism group * mask condition.

To get an understanding of the effects of alexithymia separate from autism groups, a further ANOVA was run, with Autism Group removed and replaced by TAS Group *Task x Mask x TAS Group). In this ANOVA, TAS Group interacts with mask condition ($F = 4.022$, $p = .049$). There was no significant three-way interaction between Mask condition * Task * TAS

Group. There is no main effect of TAS Group. The Mask and Task effects are as detailed previously.

Discussion

Previous theories concerning face processing and emotion recognition in autism have argued that autistic individuals have atypical gaze patterns to the eyes versus mouth (Bal et al., 2010; Cuve et al, 2018; Wieckowsi & White, 2017). Face masks, which cover up the mouth region purportedly fixated to more by autistic people, have become widespread; yet whether they disproportionately affect the face processing abilities of autistic people is unclear. The aim of the present study was to examine the impact of face masks on the face processing abilities of autistic and non-autistic adults. In addition, given speculation that alexithymia rather than autism per se underpins atypical emotion processing in autism, this study also sought to examine the role of alexithymia on face mask effects. The results indicate that performance on both the identity and emotion recognition task was negatively affected by the presence of masks, for both autistic and non-autistic participants. However, masks had a significantly greater effect on emotion recognition than identity recognition.

Additionally, the effect of masks was greater for the autistic group, suggesting that while face masks have a negative impact on face processing for both autistic and non-autistic individuals, and may hinder social interactions for both populations, the negative impact is greater for autistic individuals. While autistic traits and alexithymic traits correlated significantly with participants' mask versus no mask difference scores (an index of how greatly individuals' performance was affected by masks) for emotion recognition, neither continuous alexithymic or autistic traits significantly predicted face mask effects once demographic variables were controlled for. Together, these results suggest that autistic people are more negatively affected by face masks than non-autistic people, but that this group difference is not explained by alexithymia, and the extent of one's autistic traits also does not predict face mask effects. Emotion and identity recognition themselves (as opposed to face mask effects) were not predicted by alexithymic traits, and only identity recognition, specifically PSE scores, were predicted by autistic traits (and it should be noted this was in the context of an overall non-significant regression model). This suggests limited predictive power of continuous measures of alexithymic or autistic traits for face processing abilities in the present study.

Furthermore, the unregistered interaction analyses must be considered, as it was found that the addition of a TAS group removed previous interactions between autism group and mask condition. And in a separate ANOVA, TAS group interacts with mask condition. This may suggest that it is not autism driving poorer performance in the mask conditions, but alexithymia. However, there is no main effect of TAS group, and it did not alter all the findings regarding autism, and due to their correlated nature, it may be more prompt to deduce that both alexithymia and autism are playing some role here, and that it is difficult to disentangle their exact effects with this paradigm and sample. This does posit an interesting quandary for future research however, can co-occurring alexithymia and autism both influence participant performance and thus are both influencing emotion recognition abilities? There is certainly evidence that separately both can play a role (see Cook et al., 2013; Uljaveric and Hamilton, 2013), but in certain samples with highly correlated, potentially co-occurring alexithymia and autism, maybe it is a unique combination of both driving atypical emotion recognition.

These findings support previous literature investigating the impact of obscuring facial regions on emotion recognition. The mouth region is an important source of information during face processing, and obscuring it, such as covering it with a face mask, negatively affects the accuracy, confidence and speed of facial emotion recognition (Carbon, 2020; Marini et al., 2021). Our results demonstrate the negative impact of face masks on both autistic and non-autistic participants' face processing abilities, but also suggest that this impact is worse for emotion versus identity recognition.

It is noteworthy that we did not find an overall effect of autism status on emotion recognition. There is an inconsistency of findings in autism and emotion recognition research, as some studies have not identified atypical emotion recognition in autistic individuals (Adolphs et al. 2001; Loth et al., 2018; Rutherford and Towns 2008), and some have indicated that some individuals may compensate for poorer emotion recognition abilities (Harms, Martin, and Wallace, 2010). Plausibly, some inconsistency may be accounted for by the heterogeneity of the autistic population. Indeed, the present study considered the role of alexithymia, a factor that has been argued to underpin some of the inconsistency of findings regarding emotion processing in autism (Bird & Cook, 2013), as alexithymia varies across the autistic population. While alexithymia correlated with the impact of face masks on emotion

recognition, it did not predict face mask difference scores after demographic variables were controlled. Furthermore, our analyses also did not find that alexithymia predicted emotion or identity recognition, as indexed by ether attribution threshold or PSE scores. Our autistic sample were also significantly more alexithymic than our non-autistic sample, and far more scores above the TAS-20 cut off in the autism group (15/25) than in the non-autistic group (1/38); and yet, no main effect of autism group was found. Plausibly, the lack of findings for alexithymia could be related to the intensity of the stimuli used, as previous research has suggested that alexithymia reduces individuals' abilities to distinguish between low intensity emotional presentations (Ketelaars et al., 2016); if our stimuli were too emotionally intense, even participants high in alexithymia would be able to distinguish them.

Autistic samples are also heterogenous with regards to other traits and abilities which could explain why some studies report emotion recognition deficits and some do not. In particular, a considerable amount of autism and emotion recognition research use participants without intellectual disability, and these participants have been found to be able to use high level cognitive compensatory mechanisms as they age, whereas those with intellectual disability do not (Mazzoni et al., 2020). However, it should be noted that a meta-analysis of 48 studies of autism and emotion recognition found no meta-analytic effect of participant IQ (Uljarevic and Hamilton, 2013).

Another potential source of inconsistency across studies of emotion recognition in autism could be derived from participant gender: Ketelaars et al. (2016) used a female autistic sample and found no impaired emotion recognition. Female autistics have also been shown to 'mask' or 'camouflage' themselves, in order to 'fit in' socially and hide any differences they may perceive (Hull, Petrides, and Mandy, 2020): potentially, female autistics may be more predisposed to compensatory mechanisms used in social interactions, and so can compensate for atypical emotion recognition. The sample of autistic participants in the current study was predominantly female (although this actually meant the samples of autistic and non-autistic participants were relatively well matched in terms of their genders): this could also contribute to the lack of autism group effect on emotion recognition.

While no overall group effect was found, autistic participants' face processing was more disrupted by the masks than the non-autistic participants. This resonates with reports from autistic people that face masks do cause problems for daily social interactions and

communication: Clegg et al. (in prep) analysed social media posts by autistic people about face masks and conducted a survey of autistic adults about their experiences with face masks (both wearing them themselves and interacting with others wearing them). Autistic people reported that face masks led to some interactions feeling effortful, negative and anxiety-inducing, as they interfered with their abilities to monitor others' emotional reactions (though it should be noted that autistic people did not have wholly negative experiences with face masks, as they also offered means to camouflage some of their autistic behaviours in public). The extra disruption experienced by autistic people by others' face masks could plausibly arise due to atypical looking patterns during face processing: if autistic people tend to fixate more at the mouth region, its occlusion may mean that the cues they are used to using to support their emotion recognition are removed, meaning that autistic participants are more disrupted by masks than non-autistic participants, who tend to look more at the eye region. However, this disruption occurred across both emotion and identity processing, as opposed to the specific impact we hypothesised on emotion recognition.

There are limitations for the current study that must be considered. Firstly, the current study is limited in examining only emotion recognition abilities for two negative emotions (anger versus disgust). In order to have a sufficient number of trials to generate our dependent variables for both mask and no mask conditions, the testing procedure was already over one hour: testing more emotions was thus impractical in the current study. Previous research indicates that recognition of different emotions is differently affected in autism, with negative emotions being more likely to induce a group effect than positive emotions (Enticott et al., 2013; Shanok, Jones and Lucas, 2019; Uljarevic and Hamilton, 2013; Wallace, Coleman, and Bailey, 2008). Different emotions are also differently affected by occlusion (Fischer et al., 2012). Thus, further research on the impact of face masks in autism with a wider array of emotions is warranted.

In addition, a more diverse sample of participants would help to illuminate the role of ethnicity and gender in face processing abilities. Our current study found some significant effects of participant gender and ethnicity: specifically, participants who selected "other" or "prefer not to say" when asked about their gender showed different PSE scores for both identity and emotion trials, and ethnicity was a significant predictor of emotion PSE and identity attribution thresholds. Only a small minority of our participants were not white, and

all of our stimuli were of white men, so our present data can speak little to the role of ethnicity in face processing. Nonetheless, this finding is in keeping with wider literature on the effects of ethnicity on face processing, notably "own race" effects, whereby faces that match one's own ethnicity have a perceptual advantage, due to generally higher familiarity during development with faces of one's own race (e.g. Bar-Haim, Ziv, Lamy, & Hodes, 2006; Walker & Tanaka, 2003). Few participants did not classify themselves as male or female, and our present study did not distinguish between participants who described themselves as a gender "other" than male or female, and participants who did not wish to disclose their gender (but maybe indeed describe themselves as male or female). There has been limited investigation into the emotion and identity recognition abilities of people of other genders, though some have speculated that autistic traits and rates of autism are higher amongst transgender people (Warrier et al., 2020). In any case, the significance of these demographic variables in the present study highlights the importance of controlling for them in investigations concerning face processing, autism and alexithymia. Our autistic and non-autistic samples did not differ in their rates of different ethnicities or genders, and thus it seems unlikely to have impacted our findings.

There are also some issues with the reliability and validity of the measures used in the current study. For the TAS-20, whether it accurately measures alexithymia has been questioned. While there is research supporting its use in research, with both general and clinical populations, other research has found deficiencies. Leising, Grande and Faber (2009) conducted a factor analysis and concluded that the TAS-20 assesses 'general psychological distress', rather than alexithymia. We did not take measures of depression, anxiety or other mental health factors; future research may benefit from including such measures to establish whether any alexithymia effects are independent of other mental health effects. Additionally, the psychometric properties of the AQ-28 have been examined. Sizoo et al. (2015) found that the AQ-28 did not have sufficient validity to predict diagnoses of ASC. This could perhaps pose an explanation for why our autistic group showed greater mask effects, but continuous AQ score did not predict mask effects.

In summary, the impact of face masks on face processing autism represents an interesting area of study, both as a naturalistic means to test predictions made by theories concerning eye gaze and emotion processing in autism and alexithymia, and to provide further understanding

about how autistic people may be differently affected by face masks in their daily lives. Our study does suggest that autistic people's face processing is more adversely affected by face masks than non-autistic people's face processing, though this effect is not specific to emotion recognition but impacts identity recognition as well. The extent of participants' autistic and alexithymic traits did not predict the impact of masks after age, gender and ethnicity were accounted for. Our results can be combined with research that considers autistic people's experiences with face masks to highlight that important public health measures like face masks may have unequal effects on populations with pre-existing atypical face processing abilities.

Appendix Two

TABLE 4.1: PARTICIPANT CHARACTERISTICS

	TD (*N* = 38)	ASC (*N* = 25)
GENDER	Male = 13, Female = 24 Other/Prefer not to say = 1	Male = 9 Female = 13 Other/Prefer not to say = 3
MEAN AGE (SD)	29.71 (15.21)	40.32 (15.26)
ETHNICITY	White = 36 Asian = 2	White = 24 Asian = 1
MEAN AQ (SD)	57.84 (11.68)	90.68 (15.05)
MEAN TAS (SD)	42.61 (10.87)	62.40 (14.88)

TD = typically developed group. ASC = autistic group. AQ = Autism Quotient-28. TAS = Toronto Alexithymia Scale-20.

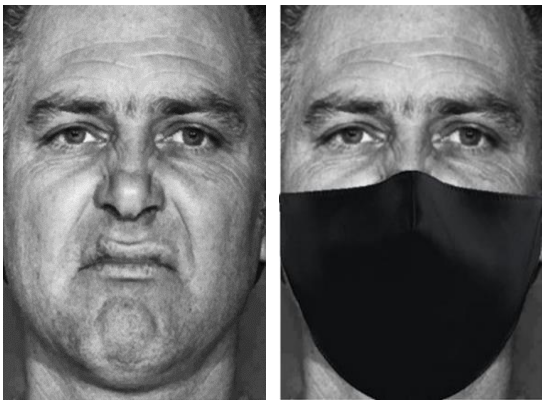


Figure 4.1. Example Stimuli. The left images show the original photo used in no mask condition, and right image shows the edited version used in mask condition.

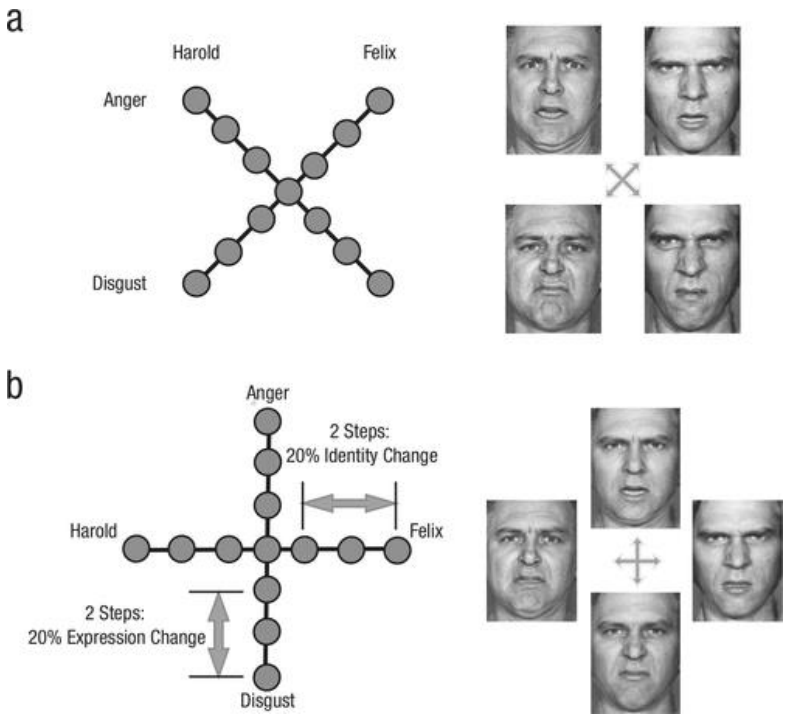


Figure 4.2. Diagram of the identity and emotion continuum for Harold/Felix, disgust/anger, from Cook, R., Brewer, R., Shah, P., & Bird, G. (2013).

TABLE 4.2: DESCRIPTIVE STATISTICS FOR PSE SCORES FOR MASK CONDITIONS, EMOTION VERSUS IDENTITY TASK, AND PARTICIPANT GROUP

CONDITION	*Task*	*Group*	*Mean*	*Std. Deviation*
MASK	Emotion	TD	.50	.46
		ASC	.55	.39
		Overall	.51	.43
	Identity	TD	.54	.05
		ASC	.54	.06
		Overall	.54	.05
NO MASK	Emotion	TD	.53	.25
		ASC	.52	.07
		Overall	.53	.20
	Identity	TD	.52	.04
		ASC	.50	.04
		Overall	.51	.05

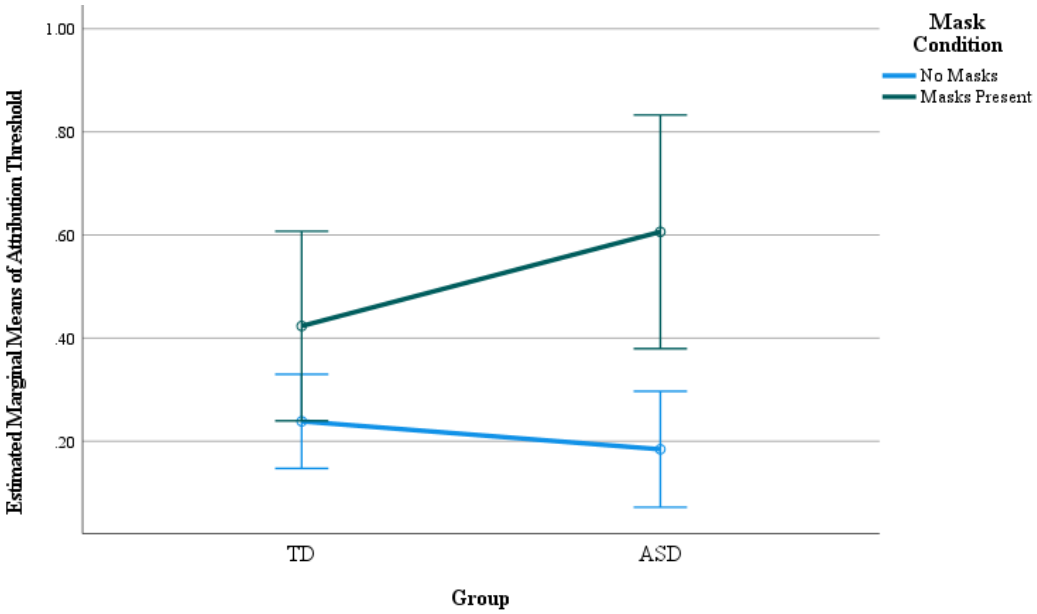
Note that these are true arithmetic means. TD = typically developed group. ASC = autistic group.

TABLE 4.3: DESCRIPTIVE STATISTICS FOR MASK CONDITIONS, EMOTION VERSUS IDENTITY TASK, AND PARTICIPANT GROUP

CONDITION	*Task*	*Group*	*Mean*	*Std. Deviation*
MASK	Emotion	TD	0.72	0.95
		ASC	1.05	1.32
		Overall	0.85	1.12
	Identity	TD	0.13	0.05
		ASC	0.16	0.09
		Overall	0.14	0.07
NO MASK	Emotion	TD	0.37	0.67
		ASC	0.26	0.31
		Overall	0.33	0.55
	Identity	TD	0.10	0.04
		ASC	0.11	0.05
		Overall	0.11	0.04

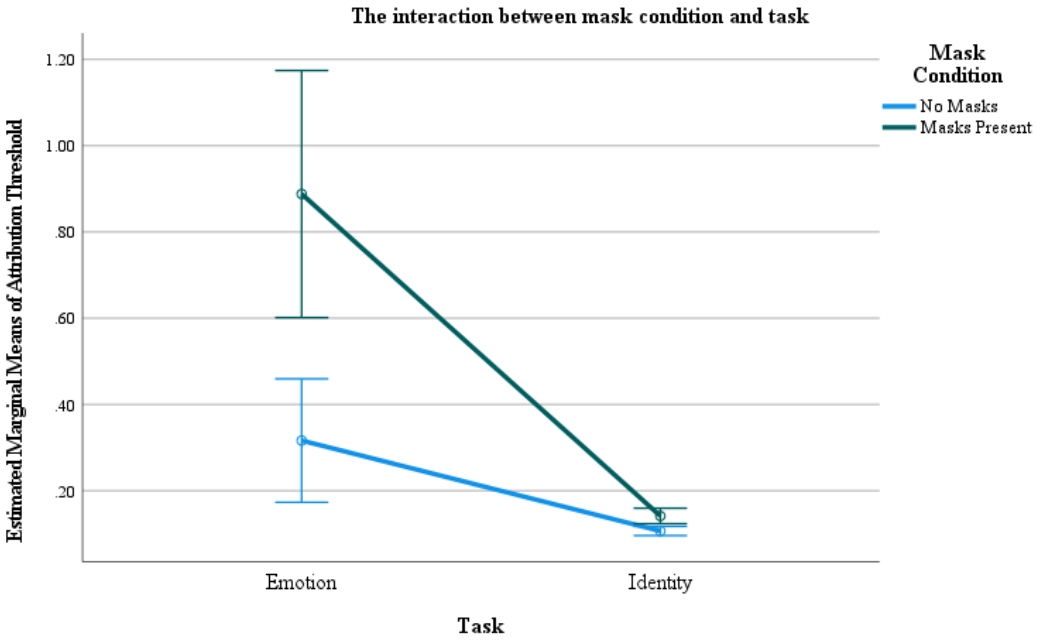
Note that these are true arithmetic means. For the attribution threshold, lower thresholds indicate better performance. TD = typically developed group. ASC = autistic group.

Figure 4.3: The interaction between autism group status and mask condition.



Error bars represent 95% confidence intervals.

Figure 4.4: The interaction between mask condition and task.



Error bars represent 95% confidence intervals.

TABLE 4.4: AVERAGE MASK-NO MASK DIFFERENCE SCORES FOR AUTISTIC VERSUS NON AUTISTIC PARTICIPANTS

GROUP	Task	Difference scores – attribution (SD)	Difference scores – PSE (SD)
TD	Emotion	-.35 (.54)	.03 (.24)
	Identity	-.02 (.05)	-.02 (.05)
ASD	Emotion	-.80 (1.22)	-.02 (.39)
	Identity	-.05 (.06)	-.04 (.05)

TD = typically developed group. ASC = autistic group.

Table 4.5: Correlations between attribution threshold, PSE scores and autistic and alexithymic traits

	Emotion attribution	*Identity attribution*	*Emotion PSE*	*Identity PSE*	*AQ-28*	*TAS-TOTAL*
Emotion attribution		.319*	-0.033	.259*	-0.063	0.154
Identity attribution	.319*		0.030	0.199	-0.059	0.048
Emotion PSE	-0.033	0.030		0.022	0.122	0.066
Identity PSE	.259*	0.199	0.022		-0.185	-0.052
AQ-28	-0.063	-0.059	0.122	-0.185		.771**
TAS-TOTAL	0.154	0.048	0.066	-0.052	.771**	

N = 63. All significance values are two-tailed, all coefficients are Spearman's Rank. * denotes <.05, and ** denotes <.001.

Table 4.6: Correlations between difference scores and autistic and alexithymic traits

		Difference scores					
		Emotion attribution	*Identity attribution*	*Emotion PSE*	*Identity PSE*	*AQ-28*	*TAS-TOTAL*
Difference scores	*Emotion attribution*		.604**	-0.180	.252*	-.361**	-.293*
	Identity attribution	.604**		-0.208	.373**	-0.243	-0.164
	Emotion PSE	-0.180	-0.208		0.038	-0.068	-0.096
	Identity PSE	.252*	.373**	0.038		-0.157	-0.152
	AQ-28	-.361**	-0.243	-0.068	-0.157		.771**
	TAS-TOTAL	-.293*	-0.164	-0.096	-0.152	.771**	

N = 63. All significance values are two-tailed, all coefficients are Spearman's Rank. * denotes <.05, and ** denotes <.001.

Supplementary materials from Chapter Four.

Regression analyses on impact of masks

The following Tables (Tables S4.1 to S4.8) report the statistics for a series of regressions in which the dependent variable was a difference score, calculated by subtracted attribution scores (Tables S4.1 to S4.4) or PSE scores (Tables S4.5 to S4.8) obtained in the mask condition from the no mask condition. For each regression, model 1 was entered with demographic variables (age, gender, ethnicity). The TAS-20 was entered alongside demographic variables in Model 2. And Model 3 added the AQ-28.

Table S4.1: Hierarchical regression results for impact of masks on emotion attribution thresholds

	R^2	R^2 change	*p*-value for *F* change	*p*-value for model
Model 1	.114	.114	.129	.129
Model 2	.160	.046	.082	.070
Model 3	.168	.008	.461	.099

TABLE S4.2: PREDICTORS FOR IMPACT OF MASKS ON EMOTION ATTRIBUTION THRESHOLDS

VARIABLES	B	β	t-value	p-value
MODEL 1 FEMALE GENDER	.480	.267	1.973	.053
OTHER (GENDER)	-.195	-.054	-.406	.686
ETHNICITY	-.189	-.046	-.341	.743
AGE	-.006	-.113	-.879	.383
MODEL 2 TAS	-.013	-.230	-1.769	.082
MODEL 3 AQ	-.006	-.151	-.742	.461

Table S4.3: Hierarchical regression results for impact of masks on identity task attribution thresholds

	R^2	R^2 change	*p*-value for *F* change	*p*-value for model
Model 1	.046	.046	.590	.590
Model 2	.062	.015	.340	.590
Model 3	.065	.004	.637	.687

TABLE S4.4: PREDICTORS FOR IMPACT OF MASKS ON IDENTITY ATTRIBUTION THRESHOLDS

VARIABLES	B	β	t-value	p-value
MODEL 1 FEMALE GENDER	.012	.106	.755	.453
OTHER (GENDER)	.005	.021	.154	.878
ETHNICITY	-.013	-.053	-.383	.703
AGE	-.001	-.169	-1.270	.209
MODEL 2 TAS	.000	-.132	-.961	.340
MODEL 3 AQ	.000	-.103	-.474	.637

Table S4.5: Hierarchical regression results for impact of masks on emotion PSE scores

	R^2	R^2 change	*p*-value for *F* change	*p*-value for model
Model 1	.109	.109	.148	.148
Model 2	.133	.024	.213	.139
Model 3	.133	.000	.904	.219

TABLE S4.6: PREDICTORS FOR IMPACT OF MASKS ON EMOTION PSE SCORES

VARIABLES	B	β	t-value	p-value
MODEL 1 FEMALE GENDER	.064	.104	.766	.447
OTHER (GENDER)	-.138	-.111	-.839	.405
ETHNICITY	-.312	-.220	-1.639	.107
AGE	.001	.061	.475	.636
MODEL 2 TAS	-.003	-.167	-1.261	.213
MODEL 3 AQ	.000	-.025	-.121	-.904

Table S4.7: Hierarchical regression results for impact of masks on identity PSE scores

	R^2	R^2 change	*p*-value for *F* change	*p*-value for model
Model 1	.052	.052	.538	.538
Model 2	.062	.011	.426	.586
Model 3	.067	.005	.593	.674

TABLE S4.8: PREDICTORS FOR IMPACT OF MASKS ON IDENTITY PSE SCORES

VARIABLES	B	β	t-value	p-value
MODEL 1 FEMALE GENDER	-.004	-.037	-.262	.794
OTHER (GENDER)	-.048	-.241	-1.760	.084
ETHNICITY	.008	.036	.264	.793
AGE	6.671	.022	.163	.871
MODEL 2 TAS	.000	-.110	-.802	.426
MODEL 3 AQ	.000	-.116	-.537	.593

Regressions for emotion and identity recognition – Attribution scores

The following Tables (S4.9 to S4.12) report the statistics for a series of regressions in which the dependent variable was the attribution scores obtained from the no mask conditions, for either emotion (Tables S4.9 and S4.10) or identity attribution (Tables S4.11 and S4.12). For each regression, model 1 was entered with demographic variables (age, gender, ethnicity). The TAS-20 was entered alongside demographic variables in Model 2. And Model 3 added the AQ-28.

Table S4.9: Hierarchical regression results for emotion attribution thresholds

	R^2	R^2 change	*p*-value for *F* change	*p*-value for model
Model 1	.165	.165	.031	.031
Model 2	.166	.000	.961	.060
Model 3	.196	.030	.151	.049

TABLE S4.10: PREDICTORS FOR EMOTION ATTRIBUTION THRESHOLDS

VARIABLES	B	β	t-value	p-value
MODEL 1 FEMALE GENDER	-.069	-.061	-.467	.642
OTHER (GENDER)	.382	.170	1.321	.192
ETHNICITY	.664	.257	1.985	.052
AGE	.007	.201	1.615	.112
MODEL 2 TAS	.000	-.006	-0.049	.961
MODEL 3 AQ	-.008	-.292	-1.456	.151

Table S4.11: Hierarchical regression results for identity attribution scores				
	R^2	**R^2 change**	***p*-value for *F* change**	***p*-value for model**
Model 1	.092	.092	.224	.224
Model 2	.097	.005	.588	.312
Model 3	.106	.010	.443	.370

TABLE S4.12: PREDICTORS FOR IDENTITY ATTRIBUTION SCORES

VARIABLES	**B**	**β**	**t-value**	**p-value**
MODEL 1 FEMALE GENDER	.001	.015	0.108	.915
OTHER (GENDER)	-.012	-.075	-0.557	.580
ETHNICITY	.057	.300	2.217	.031
AGE	.000	-.055	-0.421	.675
MODEL 2 TAS	.000	.073	0.545	.588
MODEL 3 AQ	.000	-.163	-0.773	.443

Regressions for emotion and identity recognition – PSE scores

The following Tables (S4.13 to S4.16) report the statistics for a series of regressions in which the dependent variable was the PSE scores obtained from the no mask conditions, for either emotion (Tables S4.13 and S4.14) or identity attribution (Tables S4.15 and S4.16). For each regression, model 1 was entered with demographic variables (age, gender, ethnicity). The TAS-20 was entered alongside demographic variables in Model 2. And Model 3 added the AQ-28.

Table S4.13: Hierarchical regression results for emotion PSE scores

	R^2	R^2 change	*p*-value for *F* change	*p*-value for model
Model 1	.391	.391	<.001	<.001
Model 2	.397	.006	.458	<.001
Model 3	.400	.003	.614	<.001

TABLE S4.14: PREDICTORS FOR EMOTION PSE SCORES

VARIABLES	B	β	t-value	p-value
MODEL1 FEMALE GENDER	.053	.134	1.189	.239
OTHER (GENDER)	.262	.328	2.988	.004
ETHNICITY	.430	.469	4.240	<.001
AGE	-.001	-.109	-1.029	.308
MODEL 2 TAS	-.001	-.082	-0.747	.458
MODEL 3 AQ	.001	.088	0.508	.614

Table S4.15: Hierarchical regression results for identity PSE scores

	R^2	R^2 change	*p*-value for *F* change	*p*-value for model
Model 1	.107	.107	.155	.155
Model 2	.107	.000	.893	.250
Model 3	.171	.064	.042	.093

TABLE S4.16: PREDICTORS FOR IDENTITY PSE SCORES

VARIABLES	B	β	t-value	p-value
MODEL 1 FEMALE GENDER	-.004	-.050	-0.371	.712
OTHER (GENDER)	-.053	-.337	-2.539	.014
ETHNICITY	.010	.054	0.400	.690
AGE	.000	-.063	-0.491	.625
MODEL 2 TAS	.000	.018	0.135	.893
MODEL 3 AQ	-.001	-.423	-2.077	.042

Chapter Five: Examining the interrelationships between autistic traits, eye gaze behaviour, and alexithymia.

Declarations

Chapter Five has been submitted for publication in the special collection 'Social perception and cognition in autism' in Scientific Reports.

Ethics Approval

Informed consent was obtained from participants before taking part in the study. Ethics approval was obtained from the ethics committee of the Psychology department, University of York, UK (ID: 120).

Availability of data

This study is part of a larger, ongoing study and data is not yet available for open access.

Competing interests

The authors have no competing interests to declare.

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Author's contributions

Natasha Baxter made substantial contributions to the design of the study, data collection, data analysis and interpretation, and drafted the paper. Hannah Hobson made substantial contributions to the design of the study, data analysis and interpretation, and provided feedback/edited the paper. Trish Chinzara made substantial contributions to the data collection. This study was part of a larger study of which Natasha Baxter collaborated with Trish Chinzara. Naomi Walker was a volunteer research assistant who assisted in preparing data for analysis. All authors approved the submitted manuscript.

Abstract

Atypical eye gaze is associated with autism, often characterised with less looking at the eyes and potentially looking more at the mouth. Atypical facial emotion recognition is also associated with autism, although previous research indicates alexithymia may actually be responsible for emotion recognition differences in autism. The current study aims to investigate whether autistic traits are associated with less fixation to the eye region and more to the mouth region, and whether alexithymia influences these eye gaze behaviours. Participants from a student population (N = 42) completed self-report autistic traits, alexithymia, and anxiety and depression measures (given the potential for mood disorder symptoms to confound our relationships of interest). Participants also completed a facial emotion and identity recognition task whilst being observed by an eye tracker. Results showed a negative correlation between autistic traits and the average dwell time to the eye region. Greater alexithymic traits were associated with poorer performance on the emotion recognition task. However, alexithymia was not associated with any eye gaze measure tested. This suggests that autistic traits are associated with less total looking at the eyes, and alexithymia is associated with emotion recognition abilities, both findings that have been reported previous literature: however, our results do not support a model in which alexithymia can explain the atypical eye gaze behaviours reported in autism, and therefore the emotion recognition difficulties. Future research is needed to establish the relationships between eye gaze, alexithymia and autistic traits, in clinical samples.

Introduction

Atypical eye gaze has long been associated with autism; specifically, autistic people have been reported to avoid eye contact with others (Baron-Cohen et al. 1997; Papagiannopoulou et al., 2014). The 'eye avoidance' hypothesis suggests that this is due to the eye region's perception of being 'socially threatening' (Tanaka and Sung, 2016). This hypothesis proposes that eye avoidance is partially adaptative, decreasing social stress. However, as a result, facial processing may be affected, leading to reduced abilities in processing facial emotions, identities, and facial cues (Tanaka and Sung, 2016).

The 'eye avoidance' hypothesis has support from empirical evidence, including evidence that social anxiety may contribute to atypical eye gaze behaviours in some autistic individuals. Hessels et al. (2018) found that participants who scored highly either for autistic traits or social anxiety symptoms both showed less total dwell time to the eye region compared to participants who did not score highly for either. Additionally, Ni et al. (2022) found an interaction between autistic traits and social anxiety, in that social anxiety was associated with early avoidance of the eyes for individuals scoring highly for both autistic traits and social anxiety. Furthermore, Kleberg et al. (2017) found that individuals with social anxiety disorder and high autistic traits displayed delayed orientation to the eye region. These studies highlight the relationship between autistic traits, social anxiety, and eye avoidance, and suggest that for autistic people and people high in autistic traits, increased levels of social anxiety predict avoiding the eyes of others.

Specific aspects of social anxiety and their contribution to eye avoidance have also been investigated and highlight that the relationships between these variables may be more complex than previously assumed. White, Maddox, and Panneton (2015) investigated eye gaze and social anxiety in autistic adolescents, specifically social worries and fear of negative evaluation. In comparison to non-autistic controls, the autistic adolescents demonstrated lower fixation duration towards social stimuli (in this instance, static images of emotional faces). However, for the autistic participants, fear of negative evaluation actually predicted greater gaze duration to faces showing negative emotions (anger and disgust). These findings support the 'eye avoidance' hypothesis to an extent, as autistic individuals fixated less to faces overall, but for more 'socially threatening' emotions, fixation was actually increased.

Increased fixation may be due to the salience of these negative emotions and while it increases social concerns, it is adaptive as to protect the individual.

Of course, the eyes are not the only region of a face that contain important information during face processing. The mouth region is also thought to contribute important information during face processing, including emotion recognition (Blais et al., 2012): indeed, Roberson et al. (2012) investigated face processing when the eyes or mouth were obscured either by sunglasses or a mask, and found that participants performed worse at recognising facial expressions when either of these areas were obscured.

While evidence suggests that autistic individuals may look less at the eyes, there is also evidence that autistic individuals may look at the mouth region more in face processing. In an eye tracking study by Neumann, Spezio, Piven, and Adolphs (2006), autistic participants fixated more to the mouth region compared to the non-autistic control group, suggesting that autistic individuals may use information from the mouth region when processing faces. However, not all evidence agrees that autistic individuals attend more to the mouth region relative to the eye region. In fact, there is evidence that autistic individuals attend to the eyes and mouth less in a similar manner (Spezio et al., 2007). Furthermore, Jónsdóttir et al. (2023) found that autistic children looked away from both eyes and the mouth faster than controls. Anxiety was also not found to have an effect. It was concluded that avoidance of looking at face regions in autism may not be specific to the eye region. Potentially, gaze avoidance may include all salient areas of the face, although the role of anxiety needs further clarification to assess whether it is influencing atypical gaze. Whether autistic individuals fixate more to the mouth in face processing is of interest, as it may be an alternative strategy to gaining social information from the face, whilst still avoiding the eyes. The mixed research evidence indicates an area of interest for future research, as further examination of face processing in autism may help provide clarity on atypical eye (and mouth) gaze.

Atypical looking behaviour to different regions of the face, be that the eyes or mouth, may have implications for face processing abilities, in particular emotion processing. Uljarevic and Hamilton (2013) conducted a meta-analysis regarding emotion recognition in autism and concluded that there was evidence of emotion recognition difficulties, particularly for negative emotions such as fear. Some studies have linked atypical emotion recognition abilities to differences in eye gaze behaviour. Wagner et al. (2013) examined emotional face

processing in adolescents with autism spectrum conditions and compared them to non-autistic controls, using both eye tracking and event related potential (ERP, derived from the use of electroencephalography) paradigms. Overall, there were not many significant differences between the groups (with them often performing similarly), but there were subtle differences such as the non-autistic group showed faster ERP responses to the face stimuli when spending more time looking at the eyes, and the autistic group appearing to spend more time looking towards the mouth, which was associated with slower ERP responses to the face stimuli. Thus, when processing emotional faces, it appears that where individuals look may affect the processing of these faces, and thus mechanisms such as facial emotion recognition may be affected by atypical gaze behaviour, such as looking towards the mouth more rather than the eyes.

Together, broadly research suggests that autistic people look less at the eye region, more at the mouth region, and these looking behaviours may be driven at least in part by anxiety-related factors and may in part explain findings that autistic people have difficulties with emotion recognition. However, recent research has identified that alexithymia may be the factor contributing to atypical facial emotion recognition. Alexithymia is the inability to identify emotions experienced by the self and others, and is frequently co-occurring with autism (Oakley et al., 2020; Sifneos, 1973). Cook et al. (2013) investigated facial processing in autism, and the potential influence of alexithymia. Results indicated that alexithymia was significantly correlated with emotion attribution precision for the emotion recognition task, but autism was not. Neither alexithymia nor autism were significantly correlated with any of the identity recognition variables, indicating a specific relationship between facial emotion recognition and alexithymia, as opposed to relationships with more general face processing abilities.

There is wider evidence that co-occurring alexithymia in some autistic individuals may be responsible for deficits seen in facial emotion recognition. Similarly, Ketelaars, et al. (2016) also found that alexithymia was associated with poorer facial emotion recognition abilities for lower intensity emotions. More recently, using an all-female sample, Ola and Gullon-Scott (2020) found that higher levels of alexithymia were associated with less accurate facial emotion recognition, and that autistic traits did not associate with recognition abilities. Also, Ola and Gullon-Scott (2020) showed that neither alexithymia nor autism was associated with

the speed of processing, suggesting that it may be purely a recognition deficit, not an issue with face processing itself. This adds support to the notion that alexithymia (a condition characterised by not being able to recognise one's own emotions) may be affecting the ability to recognise emotions in others.

Intriguingly, a link between alexithymia, eye gaze, and emotional face processing has been identified. A review by Cuve, Gao, and Fuse (2018) reported that the research evidence with inconsistent with regards to atypical gaze and emotion recognition in autism, and that alexithymia may modulate eye gaze and emotion recognition in autism spectrum conditions. Subsequently, Cuve et al. (2021) used an eye tracking paradigm to investigate whether atypical emotion recognition in autism is due to atypical visual exploration of faces, and whether alexithymia is actually responsible for atypical eye gaze, rather than autism per se. The results indicated that atypical gaze to the eyes is predicted best by alexithymia, not autistic traits. Additionally, this effect was found in both the autistic and non-autistic participants, indicating further that autism is not modulating this relationship. However, as noted by Cuve et al (2021), there has been very limited empirical research that combines measures of eye gaze, emotion recognition, alexithymia and autism/autistic traits.

The current study aimed to investigate whether higher levels of autistic traits or alexithymia traits are associated with less fixation towards the eyes. The current study builds upon previous research by investigating the relationship between autistic traits, alexithymia, and what regions of the face are fixated on during an emotion and identity recognition task, using static face stimuli. Anxiety and depression measures were also included in the current study, to assess any effects of anxiety and depression of eye gaze measures. Whilst anxiety (particularly social anxiety) may play a role in atypical gaze behaviour, it was thought that it would be appropriate to also include a depression measure as depression is often highly correlated with anxiety, alexithymia, and autism, and so including a depression measure may help parse out any effects that may be caused by depression (Hollocks et al., 2019; Morie et al., 2019; Skokauskas, & Gallagher, 2010). It was hypothesised that individuals with higher levels of autistic traits will spend less time looking at the eyes. Also, it was hypothesised that higher autistic traits would be associated with looking more at the mouth region of the stimuli. Additionally, it was hypothesised that higher levels of alexithymia will be associated with less gaze to the eye region and poorer emotion recognition.

Methods

Participants

Forty-five participants were initially recruited from the Psychology department at the University of York. Three participants did not complete the eye tracking component and emotion and identity recognition task so 42 participants were used for data analysis (mean age = 19.55, SD = 1.611). The majority of the sample identified as female (83.3%). All participants were aged 18 years and over and were fluent in English. The majority of participants took part to be rewarded with credits for their research methods modules, others took part voluntarily.

Materials

Emotion and identity recognition task

Participants completed this task whilst having their eye movements tracked by the eye tracker. Face identity and emotion recognition tasks were based on Cook et al (2013). The stimuli consisted of 14 images, from the original task. The 14 images consist of two cross-morphed images of Ekman faces (Calder et al., 1996), on an identity and emotion continuum (see *Figure 5.1 in Appendix 3*). There were 14 images of Harold morphing to Felix, and anger morphing to disgust. Each point in the figure represents an image, starting at 80% intensity for one emotion and identity, and 20% the other identity and emotion, with percentages of identity and emotion increasing in increments of 10. For example, the first image would show 80% Harold and anger, with 20% Felix showing disgust. See *figure 5.1* for morph diagram.

Measures

Three self-report questionnaires were included to measure alexithymia, autistic traits, and anxiety and depression symptoms.

Alexithymia was measured by the Toronto Alexithymia Scale (TAS-20, Bagby, Parker, and Taylor, 1994). The TAS-20 has been used previously in autism and alexithymia research and has been shown to be appropriate to be used with both general and autistic populations (Bagby, Parker and Taylor, 2020; Schroeders, Kubera and Gnambs, 2021). Additionally, the TAS-20 has been found to be reliable across cultures, and has internal reliability and re-test reliability (Bagby, Parker, and Taylor, 2020).

Anxiety and depression symptomology were measured by the Hospital Anxiety and Depression scale (HADS, Zigmond and Snaith, 1983). The HADS has been found to have good internal consistency and convergent validity for both the anxiety and depression scales (Uljarevic et al, 2018). In addition, a review concluded that the HADS was effective in assessing symptom severity in clinical and general populations (Bjelland, Dahl, Haug, and Neckelmann, 2002).

Autism severity was measured by the abridged Autism Quotient (AQ-28, Hoekstra et al., 2011), consisting of 28 items which are taken from the AQ-50 (Baron-Cohen et al., 2001). The AQ-28 is a reliable alternative to the AQ-50, as it has been found to display validity across cultures, consistently measure similar traits in both autistic and general populations, and have good internal consistency (Ashouri, Asgharzade, Ebrahimi, Ghojazadeh and Akbarzadeh, 2020; Chee, Scheeren, and De Vries, 2023; Murray et al., 2014). Additionally, the AQ-28 has benefits of being shorter than the AQ-50, alleviating participant fatigue effects and being easier to implement in larger studies (Hoekstra et al., 2011). Furthermore, the AQ-28 and the AQ-50 are highly correlated, indicating the AQ-28 is an appropriate and effective alternative (Hoekstra et al., 2011).

We note here that this study was completed in collaboration with another researcher who also collected data on participants' language and communication abilities. These variables were not the focus of this particular set of analyses and are not reported here.

Eye tracking equipment

The eye tracking system used in the present study was the EyeLink Portable Duo System from SR Research. The tripod mount was used in the study, with the tracker positioned in front of the participant and below the computer screen on which the participant completed the emotion and identity recognition task.

Design

The experiment used a mixed design. The within-subjects factor was that all participants took part in all task conditions (emotion recognition and identity recognition). The between-subjects factors were that the sample was split for analysis into high and low AQ-28 score groups and high and low TAS-20 score groups. The emotion and identity recognition task generates two dependent variables: the point of subjective equivalence (PSE) and attribution

threshold, for both emotion and identity recognition. Attribution threshold is a measure of precision, and the PSE is a measure of bias describing the point on the identity or emotion recognition dimension at which participants are of equal likelihood to make either attribution (Cook et al., 2013). Our eye gaze behaviour dependent variables included average number of fixations for all regions of interest (mouth, eyes, nose, overall face), and average dwell time (i.e. total amount of time spent looking at a given region across a given trial) for all regions of interest. Regions of interest were determined manually for each stimuli image using SR Research Data Viewer software. See *figure 5.2* for an example of the manually determined regions of interest (with an example of participant fixations), specifically the separate eye regions (averaged to create one eye variable for analysis), the nose, the mouth, and whole face regions.

Procedure

Ethical approval was given by the ethics committee at the University of York Psychology department. Participants gave informed consent before taking part in the study. The majority also completed a set of questionnaires (the AQ-28, the HADS, the TAS-20, and the language measures for the larger project) at home using the experimental software Gorilla. Those who did not complete the questionnaires at home did so before taking part in the study in the lab. The eye tracking and emotion and identity recognition task took place in the Psychology department at the University of York. On arrival, participants were asked if they had read the information sheet, completed the consent form and the questionnaires, and were given the opportunity to ask questions. Participants then completed a language task for the larger project, lasting approximately 5-10 minutes. Participants were then briefed on the eye tracking component and positioned themselves in the chair in front of the computer and eye tracker, and the height of the chair and position of the eye tracker were altered accordingly with participant height. The distance from the eye tracker to the participants eyes was approximately 45cm, and the distance between the participants eyes and the computer screen was approximately between 80-90cm (variation due to participant height differences). Participants were asked to keep still and avoid re-adjusting their head and seating position during the task but were told that they could take a break at any time by alerting the researchers, who could pause the experiment and eye tracker. Before the task began, the participants completed the eye tracker calibration. Once calibration was completed, the

emotion and identity recognition task began. Participants were shown information and instructions for the task on the computer screen, and the researcher also verbally explained the task and how to use the keyboard to respond. The researcher was also in the room, so that participants could ask any questions/alert the researcher to wanting to take a break or withdraw. The participants then completed 14 practise trials and given the opportunity to ask any questions and view the instructions again. The main task then consisted of 280 trials and took approximately 30-45 minutes. Participants were then verbally debriefed, given the opportunity to ask questions, and were also sent a written debrief via email.

Results

For data analysis, MATLAB (specifically the PALAMEDES toolbox, Prins & Kingdom, 2009) was used to fit cumulative Gaussian functions in order to provide estimates of psychometric functions. This was done for the PSE and attribution threshold, for both emotion and identity, and for each participant. Eye tracking data was computed using the SR research Data viewer software. Data was analysed using SPSS. Our data was found to be non-normal for several variables using the Shapiro-Wilks test. The HADS depression scale, the emotion PSE, the average dwell times for the mouth, the average fixations for the mouth, the average fixations for the eyes, and the emotion attribution threshold variables were found to be non-normally distributed. The AQ-28, TAS-20, HADS anxiety scale, identity PSE, average dwell time for the eyes, and identity attribution threshold variables were normally distributed. Thus, both parametric and non-parametric tests were conducted. For the parametric tests, independent sample t-tests were conducted. The Mann-Whitney U test was the non-parametric test used in conjunction with the t-tests to compare the groups on the non-normally distributed variables.

For analysis, the sample were split into two groups. Firstly, they were split into high and low AQ groups using the median (60.5) as a cut-off. The median was used to split groups due to it being a non-clinical sample and there may not be enough participants meeting the AQ-28 threshold to conduct analysis. Secondly, the sample was split by TAS-20 scores, also using the median method (TAS-20 median = 47.5). Analyses run with the AQ-28 split and the TAS-20 split were run separately.

Comparing those with high vs low alexithymic traits, there was a significant difference on the HADS anxiety measure between the high TAS-20 group *(M* = 12.10, *SD* =2.30) and the low TAS-20 group (*M* = 14, *SD* =2.02)*; t*(40) = -2.85, *p* = .003 (one-sided). There was also a significant difference on the AQ-28 measure between the high TAS-20 group (*M* =63.52, *SD* =9.14) and the low TAS-20 group (*M*=58.29, *SD* =8.59); *t*(40) = 1.91, *p* = .031 (one-sided), indicating higher autistic traits amongst those with higher alexithymic traits. Emotion recognition attribution thresholds also differed between the high (median = .15, interquartile range = .091) and low alexithymic groups (median = .11, interquartile range =.041); *z* = -2.20, *p* = .028. It should be noted that higher scores on the attribution threshold indicate worse performance, thus those with higher alexithymic traits showed recued emotion recognition precision. No significant results were found for the rest of the variables, or for any measures of eye gaze behaviour.

Comparing those with high versus low autistic traits, a significant difference in TAS-20 scores was found between the high AQ-28 group (*M* =53.29, *SD* =13.34) and the low AQ-28 group (*M* =43.19, *SD* =11.91); *t*(40) = 2.59, *p* = .007 (one-sided). There were no other significant differences between the high and low autistic traits groups.

To further examine relationships between our variables, one-tailed parametric and non-parametric correlations were conducted, across the whole sample. Pearson correlations were used in cases where both variables were normally distributed, and Spearman's rank used when both or either variable was not normally distributed. A positive correlation was found between AQ-28 and TAS-20 scores; *r*(40) = .26, *p* = .034. A negative correlation was also found between AQ-28 scores and the average dwell time for the eye region; *r*(40) = -.38, *p* = .006. In addition, a negative correlation was found between the TAS-20 and the anxiety scale of the HADS; *r*(40) = -.32, *p* = .020. A positive correlation was found between the emotion attribution threshold and the TAS-20; r_s(40) = .38, *p* = .009. Also, a positive correlation was found between the average fixations for the eyes and the average fixations for the mouth; r_s(40) = .54, *p* <.001. See appendix 3 for correlation matrix.

Interaction analyses

To assess for any interactions, we ran additional 2-way ANOVAs (High-Low TAS Group x High-Low AQ Group) to examine potential interaction effects on eye gaze behaviour and

face processing abilities. We found no significant interaction effects for the dwell time to eyes, whilst there was a significant interaction effect for dwell time to mouth, plus main effects and AQ and TAS group. However, the dwell time to mouth variable is not normally distributed, which may explain why these effects were not present in our non-parametric tests. Thus, any results from the parametric test must be considered due to the inappropriate application of a parametric test, however, a suitable non-parametric alternative is not currently known. Whilst these interactions may be considered, conclusions cannot be drawn.

Discussion

This study aimed to investigate whether high autistic traits or high levels of alexithymia were associated with atypical fixation to the eyes and mouths of face stimuli. Based on previous literature, it was predicted that participants with higher autistic traits would look less at the eyes and more at the mouth. Additionally, it was predicted that alexithymia would negatively affect emotion recognition abilities and predict less gaze towards the eyes.

Our findings partially support previous literature. There was a positive correlation between the autistic and alexithymic traits, which is in keeping with the high prevalence rates of co-occurring alexithymia and autism (Oakley et al., 2020). Higher levels of autistic traits were associated with lower dwell time to the eye region. This supports our hypothesis and previous literature indicating that autistic individuals do demonstrate lower levels of eye contact and fixation to the eye region in face processing. Additionally, the performance on the emotion recognition task was correlated with the alexithymia measure, and there was a significant difference between the high and low alexithymia groups, with those with higher alexithymic traits showing less precision in their emotion attribution scores. Thus, higher levels of alexithymia are associated with poorer facial emotion recognition abilities, which is supported by previous literature which suggests that it is alexithymia, not autism, that negatively affects facial emotion recognition (Cook et al., 2013).

However, in contrast to previous literature, there was no relationship found between alexithymia and eye gaze (Cuve et al., 2021). Neither anxiety or depression were associated with any of the face processing measures, but there was a significant difference between the high and low alexithymia groups for anxiety, although contrary to usual patterns, anxiety and the alexithymia were negatively correlated.

Overall, the results indicate autistic traits were associated with less looking to the eyes. However, the 'eye avoidance' hypothesis posits that social anxiety may be the factor around reduced eye contact in autism spectrums conditions, and in the current study, anxiety was not associated with gaze behaviour. It is possible that reduced eye fixation seen in some autistic individuals may be independent of anxiety. Indeed, in children with Fragile X syndrome, social avoidance was associated with autism symptoms but not anxiety symptoms (Roberts et al., 2019). Alternatively, the lack of effects of anxiety on eye gaze behaviour could be because a general anxiety measure was used, and perhaps a more specific social anxiety measure may provide more insight on the gaze avoidance – social anxiety relationship. Furthermore, as the study took part in controlled, laboratory conditions using a face processing task which may not replicate social situations, potentially, the study and task did not elicit specific feelings of social anxiety and social stress that some individuals may experience. Finally, it may be the case that anxiety (or social anxiety) is only predictive of eye gaze behaviour amongst those with autism or high autistic traits: we lacked the statistical power to meaningfully compare the correlations between anxiety and eye gaze behaviour in our high and low autistic traits groups, and our sample did not include clinical cases of autism.

In addition, it was predicted that individuals with high autistic traits would look more at the mouth region, however the current study provided no evidence for this (Neumann, Spezio, Piven, and Adolphs, 2006). There are mixed results regarding autism and fixating to the mouth in face processing: Bar-Haim, Shulman, Lamy and Reuveni (2006) found that both autistic and non-autistic boys both attended to the eyes more than the mouth, and in a similar manner. Additionally, Vettori et al., (2020) also found that both autistic and non-autistic boys looked longer at the eyes than the mouth. They also found that the autistic boys switched more often between facial features, indicating 'feature-based' face processing. In a systematic review, Papagiannopoulou et al. (2014) concluded that there was evidence of reduced gaze fixation to the eyes in autism, but no evidence for increased gaze fixation to the mouth. However, due to inconsistent results for gaze fixation and the mouth, future research is needed to establish a relationship due to the heterogeneity in mouth-focused studies. The current study contributes to the mixed results regarding the mouth region in face processing, highlighting the need for further research.

The current study also identified an association between alexithymia and poorer emotion recognition. This relationship has been identified in previous literature, but interestingly it was also predicted that alexithymia would affect gaze behaviour. Studies have indicated that alexithymia may modulate the relationship between autism and poorer emotion recognition, though only a few have directly explored the relationships between autism, gaze behaviour, and atypical emotional face processing (Cook et al., 2013, Cuve et al., 2021). Bird, Press, and Richardson (2011) demonstrated that alexithymia, not autism, predicted eye fixation in a sample of autistic adults. The current study did not find evidence of this relationship, which may be due to the heterogeneity seen in autism and alexithymia. Mixed results between studies is common within autism research, particularly emotion recognition and face processing research. There is even evidence to suggest that autistic individuals do not always display impaired face processing (Jemel, Mottron, and Dawson, 2006). Bird, Press, and Richardson (2011) even commented that there is mixed evidence for reduced eye contact in individuals with autism spectrum conditions. While they also suggested alexithymia may be the causal factor for these mixed results, evidently more research is needed into the seemingly complex and heterogenous nature of the relationships between alexithymia, autism, gaze behaviour, and facial emotion recognition.

There are several limitations to the current study. Firstly, a non-autistic sample was used, drawing upon the local student population, which naturally limits the generalisability of the results to the autistic population. On the other hand, the fact that a relationship between higher autistic traits and reduced fixation to the eyes was found does indicate that we can demonstrate comparable effects in non-clinical samples.

Secondly, the static face stimuli used in the current study may not accurately reflect real-world social situations. Chevallier et al. (2015) investigated which stimulus types are the most effective in autism social attention and motivation research. They compared ‘Static Visual Exploration’ (static images of objects and people), ‘Dynamic Visual Exploration’ (videos of faces and objects), and ‘Interactive Visual Exploration’ (videos of people interacting with objects in a naturalistic environment). Eye tracking was used to assess participant gaze during the tasks, and participants were children with and without autism spectrum conditions. Their findings indicate that only the interactive task was sensitive to group differences, and they concluded that the ‘ecological relevance’ of social stimuli should

be considered in task design. However, Yeung (2022) conducted a systematic review and meta-analysis of the tasks used in emotion recognition research and found that there were no significant differences in terms of effect sizes between the static and dynamic stimuli used in the tasks, which suggests that static stimuli are just as effective and ecological valid as more 'realistic' dynamic stimuli. Thus, it is unlikely that the static stimuli would have had a negative effect on the results and methodology of the current study. Furthermore, area of interests used in eye tracking research tend to be subjective and vary greatly between studies and stimuli sets. Hessels, Kemner, van den Boomen, and Hooge (2016) comment that due to this variation, it is difficult to compare results between studies. In addition, stimuli sets vary greatly between each other, with numerous ones being available and/or designed for specific paradigms. Potentially, the use of open science practises may help alleviate some of these issues, with the accessibility of open data and experimental materials leading to better replication and the sharing of materials in order to create consistency between studies.

Future research would benefit from replicating this study with autistic sample, in order to establish the relationships between autistic traits and eye gaze, and emotion recognition and alexithymia. Additionally, previous models also indicate effects of autism on emotion recognition, and alexithymia on eye gaze, and the expected model would be that alexithymia or autism would impact eye gaze and thereby impact emotion recognition. Instead, specific independent effects of autism and alexithymia were found, rather than either trait explaining variation in both eye gaze behaviour and emotion recognition. Future research could investigate this 'decoupling' of autistic traits and alexithymia in the behaviours they predict. For example, compensatory mechanisms may play a role in certain atypical cognitive processes in autism, and potentially disrupting these compensations may demonstrate atypical abilities such as emotion recognition. A task investigating gaze behaviour could attempt to constrain eye gaze to regions such as the eyes (e.g. via use of fixation crosses to direct and constrain participant gaze) and observe the effects on emotion recognition abilities. Indeed, previous research using face masks has provided evidence that covering certain regions of the face (i.e. the mouth) disrupts emotion recognition in autistic people, and so constraining gaze away the mouth region may disrupt face processing as it appears some autistic people are actively using the mouth more in face processing (Hobson et al., 2023). Furthermore, ascertaining causal relationships are difficult to achieve in cross-sectional research (are

alexithymic traits driving reduced emotion recognition abilities or vice versa?), but future research should aim to create paradigms or observe natural causal relationships between autism, alexithymia, gaze, and emotion recognition. For instance, acquired alexithymia following brain injury is common (Fynn, Gignac, Becerra, Pestell & Weinborn, 2021) and these cases could be used in alexithymia and emotion recognition research.

To conclude, the current study provides evidence for reduced eye fixation in face processing for individuals with autistic traits, and that alexithymia may negatively influence facial emotion recognition abilities for individuals with high alexithymic traits. However, given a lack of associations between alexithymia and eye gaze behaviour, the present study would suggest that alexithymia's impact on emotion processing appears not to be driven by alexithymia's impacts on eye gaze behaviour. Whether these findings are consistent with a clinical autistic sample is of interest for future research.

Appendix Three

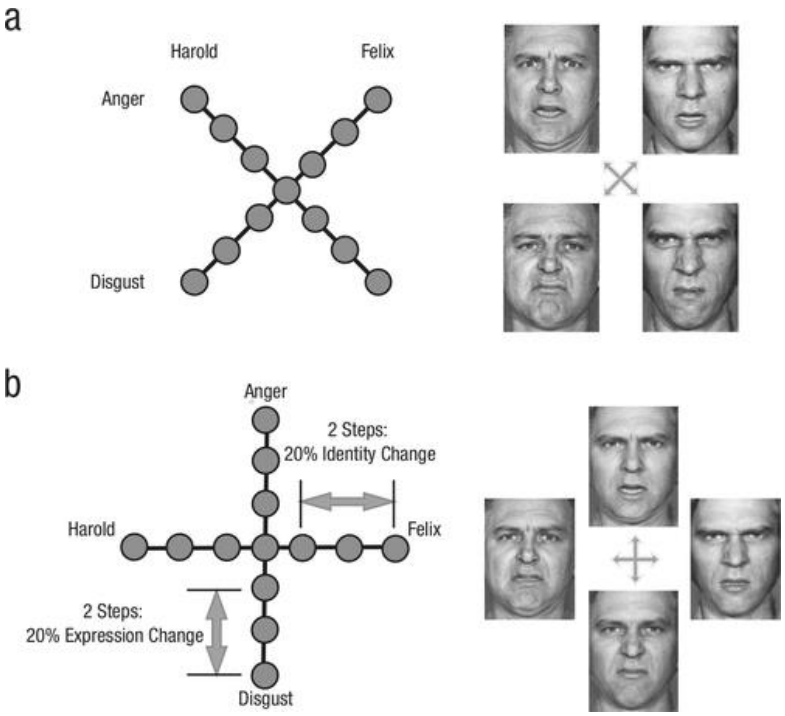

Figure 5.1. Diagram of the identity and emotion continuum for Harold/Felix, disgust/anger, from Cook, R., Brewer, R., Shah, P., & Bird, G. (2013).

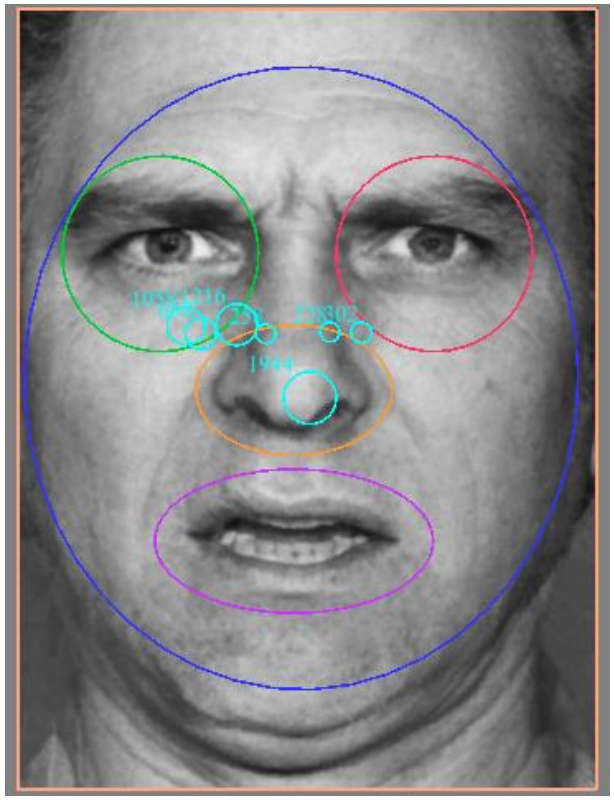

Figure 5.2. Figure showing an example of the regions of interest and example participant fixation data. Whilst manually determined for each stimulus image, the regions of interest remained the same: left eye, right eye (averaged with the left eye to create a total eye region), nose, mouth, and whole face.

Table S5.1 - Correlation matrix for Chapter Five

	TAS-20	AQ-28	HADS-A	HADS-D	Emotion ATT	Emotion PSE	Identity ATT	Identity PSE	Eye dwell time	Mouth dwell time	Eye Fixations	Mouth fixations
TAS-20	x	r = .26, p = .034	r = .32 p = .020	r_s = .231 p = .070	r_s = .38 p = .009	r_s = .241 p = .067	r = .083 p = .312	r = -.022 p = .446	r = -.170 p = .141	r_s = -.045 p = .388	r_s = -.127 p = .211	r_s = -.156 p = .162
AQ-28	r = .26, p = .034	x	r = -.151 p = .170	r_s = .181 p = .126	r_s = -.091 p = .288	r_s = .002 p = .495	r = -.143 p = .200	r = -.189 p = .115	r = .38 p = .006	r_s = 152 p = .169	r_s = -.233 p = .069	r_s = .056 p = .362
HADS-A	r = .32 p = .020	r = -.151 p = .170	x	r_s = .147 p = .177	r_s = -.072 p = .330	r_s = -.205 p = .102	r = .008 p = .481	r = -.075 p = .317	r = .131 p = .205	r_s = .057 p = .361	r_s = .118 p = .229	r_s = .145 p = .180
HADS-D	r_s = .231 p = .070	r_s = .181 p = .126	r_s = .147 p = .177	x	r_s = .214 p = .087	r_s = .307 p = .027	r_s = -.145 p = .196	r_s = -.141 p = .186	r_s = -.134 p = .198	r_s = -.032 p = .420	r_s = -.185 p = .120	r_s = -.024 p = .441
Emotion ATT	r_s = .38 p = .009	r_s = -.091 p = .288	r_s = -.072 p = .330	r_s = .214 p = .087	x	r_s = .314 p = .024	r_s = .231 p = .088	r_s = -.028 p = .432	r_s = .062 p = .351	r_s = -.213 p = .094	r_s = .043 p = .396	r_s = -.189 p = .121
Emotion PSE	r_s = .241 p = .067	r_s = .002 p = .495	r_s = -.205 p = .102	r_s = .307 p = .027	r_s = .314 p = .024	x	r_s = .079 p = .324	r_s = .179 p = .134	r_s = .078 p = .316	r_s = .137 p = .200	r_s = -.240 p = .068	r_s = -.025 p = .438
Identity ATT	r = .083 p = .312	r = -.143 p = .200	r = .008 p = .481	r_s = -.145 p = .196	r_s = .231 p = .088	r_s = .079 p = .324	x	r_s = -.217 p = .099	r_s = .141 p = .202	r_s = .048 p = .389	r_s = .032 p = .426	r_s = -.082 p = .315
Identity PSE	r = -.022 p = .446	r = -.189 p = .115	r = -.075 p = .317	r_s = -.141 p = .186	r_s = -.028 p = .432	r_s = .179 p = .134	r_s = -.217 p = .099	x	r_s = -.113 p = .238	r_s = -.094 p = .276	r_s = -.210 p = .091	r_s = -.117 p = .230
Eye dwell time	r = -.170 p = .141	r = .38 p = .006	r = .131 p = .205	r_s = -.134 p = .198	r_s = .062 p = .351	r_s = .078 p = .316	r_s = .141 p = .202	r_s = -.113 p = .238	x	r_s = -.035 p = .414	r_s = .263 p = .046	r_s = -.182 p = .124
Mouth dwell time	r_s = -.045 p = .388	r_s = 152 p = .169	r_s = .057 p = .361	r_s = -.032 p = .420	r_s = -.213 p = .094	r_s = .137 p = .200	r_s = .048 p = .389	r_s = -.094 p = .276	r_s = -.035 p = .414	x	r_s = .046 p = .386	r_s = .712 p < .001

Eye Fixations	r_s = -.127 p = .211	r_s = -.233 p = .069	r_s = .118 p = .229	r_s = -.185 p = .120	r_s = .043 p = .396	r_s = -.240 p = .068	r_s = .032 p = .426	r_s = -.210 p = .091	r_s = .263 p = .046	r_s = .046 p = .386	x	r_s = .54 p < .001
Mouth fixations	r_s = -.156 p = .162	r_s = .056 p = .362	r_s = .145 p = .180	r_s = -.024 p = .441	r_s = -.189 p = .121	r_s = -.025 p = .438	r_s = -.082 p = .315	r_s = -.117 p = .230	r_s = -.182 p = .124	r_s = .712 p < .001	r_s = .54 p < .001	x

Chapter Six: General Discussion

Summary of findings

The foremost aim of this research was to investigate the impact of alexithymia, autism/autistic traits, and mood disorders (namely anxiety and depression) on the face processing abilities of those with autism or high autistic traits, particularly their facial emotion recognition abilities. An additional line of inquiry was to examine these factors alongside gaze behaviour and the use of obscured stimuli (wearing face masks covering the mouth and nose region). The findings from my research indicate areas for future research and have interesting theoretical implications.

In Chapter Two, the narrative review aimed to explore the literature regarding the impact of anxiety and depression on socio-emotional abilities in ASC, specifically ToM, empathy, emotion recognition, and emotion regulation. The literature indicates that socio-emotional abilities may be atypical or impaired in some autistic individuals, and that these differences may be linked to anxiety or depression (Kasari & Patterson, 2012; Langarita-Llorente & Gracia-Garcia, 2019; Stuhrmann, Suslow, & Dannlowski, 2011). Overall, it appears that anxiety and depression have associations with empathy, ToM, and emotion regulation, but less so emotion recognition. The evidence on relationships with depression however was very limited, drawing largely on evidence from non-autistic samples. Anxiety and depression appear to be related to some, but not all, socio-emotional abilities, however the exact nature of these relationships is yet to be seen, whether they are causal, mediating, or a result of poorer socio-emotional abilities.

In Chapter Three, this study aimed to examine the extent to which alexithymia and/or anxiety and depression predicted atypical facial emotion recognition in autistic individuals (with non-autistic controls from the general population as a comparison). Previous research indicated that alexithymia was more likely to be the significant predictor of poorer emotion recognition abilities rather than autism, and that anxiety and depression may also contribute to atypical or impaired emotion recognition, which has been observed in non-autistic samples (Cook et al, 2013; Dalili et al., 2015); Demenescu et al., 2010). The results indicated that neither alexithymia, autism (both autism versus non-autism group and continuous autism traits), anxiety or depression contributed significantly to face processing abilities (both emotion and

identity recognition), although gender was a significant predictor for one of the emotion recognition variables. The effect of gender was not predicted and may reflect an association between gender diversity and neurodiversity (van Vlerken, Fuchs & van der Miesen, 2020).

The study in Chapter Four aimed to investigate the impact of face masks on face processing abilities for autistic individuals (and non-autistic controls), as well as the contributions of autism and alexithymia. Preceding evidence suggests that face masks hinder both emotion and identity recognition (Carbon, 2020; Marini et al., 2021), and so it was predicted that face masks would impair facial emotion recognition, and that alexithymia and autism would also contribute to reduced emotion recognition capabilities. Given that alexithymia and autism had been shown to impact eye gaze behaviour, it was also expected that either of these factors could predict increased detrimental effects from face masks (as those with high alexithymic traits/autism may look more the mouth than the eyes, but information from the mouth region would be occluded by the face masks). The findings demonstrated that face masks hindered performance on both the emotion and identity recognition tasks, though more so for emotion recognition, for both the autistic and non-autistic groups. However, the effect of face masks on face processing was greater for the autistic group. Neither alexithymia nor continuously measured autistic traits contributed significantly to predicting face processing abilities, nor the individually indexed impacts of face masks.

In Chapter Five, this study aimed to explore whether high autistic traits or high levels of alexithymia were associated with atypical fixation to the eyes and mouths of emotional face stimuli. The contributions of anxiety and depression were also assessed. Previous literature indicates that autistic individuals may have reduced eye contact (which may reflect social anxiety) and may rely more on social information from the mouth region when face processing (Hessels et al., 2018; Kleberg et al., 2017; Neumann, Spezio, Piven, & Adolphs, 2006). However, atypical gaze behaviour has also been associated with alexithymia (Cuve et al., 2021). It was predicted that the associations between gaze behaviour, alexithymia, and autism would be observed in those with high autistic traits or high alexithymic traits. This study was conducted with participants from a student population, and so autistic traits were measured rather than autism diagnosis. The findings indicated that higher autistic traits were associated with lower looking time to the eye region, but that higher levels of alexithymia were only associated with poorer performance on the emotion recognition task. Thus, the

predictors of interest showing decoupled relationships to our different behaviours of interest (eye gaze behaviour vs emotion recognition). Neither anxiety nor depression were associated with gaze behaviour or face processing.

Detailed discussions specific to the narrative review and empirical studies are included in Chapters Two to Five. Here, I present an analysis of the overall findings and implications of the whole thesis, as well as areas for future research.

The role of autism and autistic traits

Autism has been identified as a factor in atypical emotion recognition, and atypical gaze behaviour often characterised by less looking towards the eyes (Tanaka & Sung, 2016; Uljaveric & Hamilton, 2013). However, the findings of the current thesis are not consistent with previous literature, with the exception of Chapter Five. Chapter Five found that higher autistic traits were associated with lower looking time to the eye region, consistent with the notion that individuals with high autistic traits display less eye contact. These results suggest that individuals with high autistic traits may use the eyes less when processing faces, giving credence to holistic face processing mechanisms or using the mouth more in face processing, both of which have been theorised as potential explanations as to reduced eye contact (Joseph & Tanaka, 2003; Tanaka & Sung, 2016; Neumann, Spezio, Piven, & Adolphs, 2006). Additionally, the results also provide some credibility to the 'eye avoidance' hypothesis, as those with high autistic traits did spend less looking towards the eyes, however the 'eye avoidance' hypothesis posits that social anxiety may be a factor in reduced looking time to the eye region, and in this particular study relationships between anxiety, gaze behaviour, and autistic traits were not found. However, it should be noted that a more general anxiety measure was used, and thus a specific social anxiety measure may find this relationship. In Chapters Three and Four, no effect of autism diagnosis or autistic traits was found on emotion recognition abilities. This is in contrast to the literature indicates that autism is associated with atypical (often impaired) emotion recognition abilities (Uljaveric & Hamilton, 2013).

Several factors may be responsible for these divergencies. As noted throughout the thesis, emotion processing appears to be heterogeneous in autism, with many autistic people not displaying deficits or differences in emotion recognition. A key question for the present thesis

was what factors could help explain this heterogeneity, in particular the role of alexithymia and mood disorder symptomatology. I return to the notions that alexithymia or depression/anxiety explains variability in emotional abilities in subsequent sections, and first consider other potential reasons for this set of findings.

As discussed in the chapter discussions, the lack of autism effect may have been due to the study being completed by participants online. While evidence suggests that differences between face-to-face and online studies are negligible (and both are effective), participants (in particular autistic participants) may have felt more comfortable and less stressed when completing the tasks, potentially leading to better performance and increased validity (Sucksmith et al., 2013). Furthermore, when conducting behavioural experiments online, it is hard to standardise stimuli size and participant viewing distance (Brascamp, 2021). There are methods for avoiding variation, such as instructing participants to stay at arm's length from the screen, or estimating distance based on screen size and participant height (Brascamp, 2021). In these Chapters, participants were asked to take part using a computer or laptop, and the Gorilla experimental software only allowed these devices. While this was predominantly done as a keyboard was needed for the task, it also prevented participants taking part on mobiles or tablets, which have a much smaller screen than laptops/computers. Although laptop/computers screen vary in size, this would have helped in keeping the experiment and stimuli in the format as designed, and potentially avoided major variations. While the effectiveness of collecting data online and the quality of the data has been questioned, it appears that there is little to no statistical difference between collecting data online or in-person (Sauter, Stefani, & Mack, 2022; Uittenhove, Jeanneret, & Vergauwe, 2022). With consideration to autism research specifically, online research has been used effectively with autistic participants (without intellectual disability): for example, Griffiths et al. (2019) produced results using an online protocol that were comparable to those found in a meta-analysis of lab-based emotion recognition studies (Uljaveric & Hamilton, 2013), suggesting that online autism research (particularly emotion recognition tasks) can be compared to lab-based evidence and contribute to the literature (Ashworth et al., 2021).Therefore, it is unlikely that using an online design would have affected the outcomes of the experiments in Chapters Three and Four.

Another consideration is the role of gender. In Chapter Three, gender was associated with one of the emotion recognition variables, specifically those identifying as a gender 'other' than male or female perceived the morphed stimuli as angry earlier in the morph continua than the male and female groups. Considering the potential context of gender diverse people having experienced more aggression (Messinger, Guadalupe-Diaz, & Kurdyla, 2022), it may be that a bias towards the detection of anger is present in some gender diverse individuals due to previous experiences of bullying or being in threatening situations: victims of bullying have been shown to show differences in their emotion recognition abilities, including a higher rate of mistaking neutral faces for angry faces (Franzen, de Jong, Veling, & Aan Het Rot, 2021). Additionally, the 'other' gender group had the highest autistic traits, providing credence for a potential relationship between neurodiversity and gender diversity (van Vlerken, Fuchs, & van der Miesen, 2020). The study on the impact of face masks in Chapter Four also reported an effect of gender on emotion and identity PSE. However, the emotion PSE was in the opposite direction to that documented in Chapter Three: that is, those who selected "other/prefer not to say" ($N = 4$) when describing their gender perceived anger *later* in the morph spectrum compared to males and females (rather than earlier, as was the case in Chapter Three). In both of these chapters, the number of participants not selecting male or female as their gender was very small, so these findings require further research to explore the role of gender divergence on face processing abilities.

Aside from effects of gender divergence, high rates of females in our sample may also have impacted the studies' findings. For Chapter Four, the autistic sample was predominantly female, and 'masking' or 'camouflaging' is often observed in females with ASC, and thus potentially the participants may have employed compensatory mechanisms during the emotion recognition task, diminishing between group effects. Compensation for emotion recognition has been observed in research (Harms, Martin, & Wallace, 2010). Interestingly, research indicates that emotion perception is comparable between autistic males and females (Lai et al., 2012), and whilst 'camouflaging' is more predominant in those who are autistic and assigned female at birth, 'camouflaging' can also present in autistic males, and thus potentially autistic males and females can 'camouflage' and utilise compensatory processes for emotion processing (Lai et al., 2017).

Additionally, there is a debate over the type of stimuli used in emotion recognition tasks, whether static or dynamic stimuli are more effective in reflecting real-life social situations and facial expressions. Chapters Three and Four (and Five) used static stimuli, which may not be ecologically valid due to not being naturalistic enough (Chevallier et al., 2015). However, a systematic review and meta-analysis of emotion recognition studies and tasks found that there were no significant differences between static and dynamic tasks for their effect sizes (Yeung, 2022). If designed appropriately, it is likely that both static and dynamic stimuli have their merits and are suitable for appropriate experiments. Thus, it seems unlikely the lack of autism group effects was driven by the use of static stimuli. Furthermore, previous research indicates that face processing for unfamiliar and familiar faces may rely on different mechanisms, and thus the use of unfamiliar face stimuli in the empirical chapters may have affected the results, as unfamiliar face processing requires more effort and use of neural systems such as recognition and memory (Natu & O'Toole, 2011). However, while processing unfamiliar faces may have been more difficult compared to familiar faces for the participants, using unfamiliar face stimuli would have been more reflective of 'real-life' face processing as we often have social interactions with people we have not met before. This provides some ecological validity for the stimuli used in the empirical chapters.

Overall, the data from the present thesis indicates that appears that autistic traits are associated with reduced gaze to the eye region, but not emotion recognition abilities, and the present studies found limited evidence of poorer emotion recognition amongst autistic people: they were however more impacted in their face processing abilities by the presence of face masks, which is in keeping with the notion that autistic people look more at the mouth relative to the eyes, and rely more on information in the mouth region when processing faces. The discrepancies regarding the current results of emotion recognition and the wider literature may be due to several factors, and so future research should investigate the influence of gender on emotion recognition abilities. Furthermore, building on the results from Chapters Four and Five, future research could examine gaze behaviour when faces are occluded (such as with face masks), to investigate what processes autistic individuals employ when they cannot use information from the mouth region, potentially constraining gaze away from the mouth may lead to autistic individuals relying on the eyes or other facial regions in order for emotion recognition. Whether it is reduced eye contact due to social anxiety or

differences in face processing that modulate reduced eye fixations will help inform avenues for interventions and support plans; for example, if social anxiety is a factor, treating the anxiety may aid in ameliorating any social difficulties an individual may be experiencing.

The impact of alexithymia

Recent research has identified alexithymia as a potential factor in atypical emotion recognition and gaze behaviour in emotion processing. The literature suggests that in fact it may be alexithymia, not autism, influencing these differences (Bird, Press, & Richardson, 2011; Cook et al., 2013; Cuve, Gao, & Fuse, 2018; Donges & Suslow, 2015). Similar to our findings regarding autism, no significant results were found for the role of alexithymia except for Chapter Five. Chapter Five found that higher levels of alexithymia were associated with poorer performance on one of the emotion recognition variables. This supports previous literature such as Cook et al. (2013) and Donges and Suslow (2015), who found an association between alexithymia and atypical facial emotion recognition. However, Chapter Five also predicted that an effect of alexithymia on gaze behaviour would be observed, but this was not the case. This contradicts the theory that alexithymia, rather than autism (traits of which were observed to have an association with gaze), may influence gaze behaviour during facial emotion processing (Bird, Press, & Richardson, 2011; Cuve, Gao, & Fuse, 2018).

The modality of these studies and specific aspects of the task design could have impacted on the study's ability to demonstrate effects of alexithymia. For Chapters Three and Four, no significant results may have occurred to due to the task being completed online rather than face-to-face, although as previously discussed emotion recognition research has been successfully implemented online and it seems an unlikely explanation for these null effects. As discussed in relation to autism above, it is possible that participants with high alexithymic traits felt more comfortable completing the task at home and less comfortable in the lab, leading to alexithymic individuals only showing deficits in face processing in a face-to-face study. However, the results indicated a specific relationship between their emotion recognition performance and their alexithymia scores: if their performance was poor because of the environment, then we would potentially see significantly worse performance on the identity recognition task as well, but this was not observed, indicating a specific association between higher alexithymia scores and poorer performance on the emotion recognition task.

The stimuli used in both studies may have been a factor in the lack of findings for alexithymia. Both studies used the same stimuli, which are cross-morphed images of faces moving from 'disgust' to 'anger' and 'Harold' to 'Felix' on two continuums. At points the emotion intensity may be 20% and previous research has indicated that alexithymia may hinder the specific recognition of lower intensity emotions, therefore some participants may have struggled with some of the stimuli (Ketelaars et al., 2016). On the other hand, stimuli emotion presentations include both anger and disgust, for example 20% anger and 80% disgust, so participants should have no issue identifying the high intensity emotion. It may be the case, that even with cross-morphed emotional facial stimuli, the participants were still able to accurately recognise the more prominent emotion and alexithymia did not hinder their abilities.

Rather than null effects of alexithymia in our studies being unusual, a different possibility is that other null effects remain unpublished, and that our findings are reflective of wider academic activities on the topic of alexithymia. Publication bias remains an important problem within the field of psychology (Kühberger, Fritz, & Scherndl, 2014): when non-significant results remain unpublished, the research literature becomes representative, and in the context of this thesis' current topics this has important implications for the evidence base for supporting autistic people (see Hobson, Poole, Pearson, & Fletcher-Watson, 2022). At present, there has not been a meta-analysis of alexithymia and emotion recognition studies, and as such there has been no formal examination of the potential impact of publication bias. This is different to the autism and emotion recognition literature: indeed, both meta-analyses by Trevisan and Birmingham (2016) and Uljaveric and Hamilton (2013) correct for publication bias and still find an effect of autism on emotion recognition abilities. Additionally, Pisani et al. (2021) conducted a systematic review of alexithymia and theory of mind studies and found significant publication bias that they concluded likely contributed to the inconsistent findings across the literature.

Despite the inconsistencies of the results from Chapters Three and Four, one of the strengths of the empirical studies was the use of experimental task from Cook et al. (2013). The experimental task was closely copied and used in an online paradigm (compared to in-person as in Cook et al., 2013), with the addition of edited stimuli (to have face masks) in Chapter Four. Similar measures were also used, for example the TAS-20 alexithymia scale.

Additional measures were the AQ-28 (rather than the Autism Diagnostic Observation Schedule or AQ-50), and the HADS anxiety and depression scale. Replicating the task and some of the measures adds some validity to the empirical chapters of this thesis, suggesting that potentially Chapters Three and Four genuinely found no significant results for the impact of alexithymia (and autism) on emotion recognition. The samples used in Chapters Three and Four also met pre-determined power requirements, suggesting that the samples used would have been adequate to establish an effect if one was present.

As discussed in the discussion of Chapter Three, to ascertain whether other factors may be driving atypical emotion recognition abilities in neuro-diverse populations, rather than alexithymia and autism, a meta-analysis of the current expanse of research is warranted with particular focus on whether there is a consistent effect between alexithymia and emotion processing, and what other factors may be playing a role in this relationship or independently driving atypical emotion recognition. With the non-significant results from Chapters Three and Four, and the significant results from Chapter Five, future research could also examine whether the effect of alexithymia on emotion recognition is something seen in a lab environment, and whether it can be seen in more naturalistic environments. As no effect was seen in the studies conducted at home (online), future research could specifically examine emotion recognition in real-life, either between participants in a social setting or via more naturalistic stimuli such as videos or VR (virtual reality) sessions of emotional social interactions.

The effects of anxiety and depression

Anxiety and depression do not appear to influence atypical emotion recognition in autistic individuals. In the first empirical study (Chapter Three), no effect of anxiety and depression was found. Similar results were found in the third empirical study (Chapter Five). This differs to previous literature which indicated that an effect of anxiety and depression may be observed in autistic individuals as this has been demonstrated in non-autistic participants. Both anxiety and depression have been associated with impaired emotion recognition, with depression being linked to a larger impairment (Demenescu et al., 2010). Social anxiety, common in autistic individuals (with prevalence rates of about 50%, Maddox & White, 2015), has been shown to be linked to poorer recognition of low intensity emotion stimuli

presentations (Torro-Alves et al., 2016). However, these relationships were not found in the empirical studies of this thesis, in contradiction of predications.

In contrast to the predictions in the empirical studies and literature discussed in Chapter One, Chapter Two reviewed the literature surrounding anxiety, depression, and socio-emotional abilities in autistic individuals, and identified that emotion recognition in autism does not appear to be greatly affected by anxiety and depression. Anxiety does not appear to be associated with atypical emotion recognition for autistic individuals (Folz et al., 2022; Wong et al., 2012), whereas there is some neurological evidence that depression may affect emotion recognition in autistic adults (Ohtani et al., 2021). However, the evidence surrounding depression and emotion recognition is mixed, and specifically the associations between depression, emotion recognition, and autism appear to be an under-researched topic, and so is difficult to determine relationships. Similar to the empirical Chapters, Chapter Two concludes that there is little evidence for the influence of depression and anxiety on facial emotion recognition in ASC, despite evidence in non-autistic populations. One could argue that the lack of findings may be due to the methodology used, in particular the methods used to measure mood disorders in autism; however, the anxiety and depression measures used in the present thesis have been shown to reliable and effective in previous research with both autistic and non-autistic samples (Bjelland, Dahl, Haug, & Neckelmann, 2002; Uljarevic et al, 2018), and were used in both an online and in-person study (and so criticisms regarding online studies are negated), and thus the findings (or lack of) appear to be credible.

From the review in Chapter Two, it appears anxiety and depression do affect some socio-emotional abilities in ASC. Anxiety is associated with empathy, emotion regulation, and theory of mind, although these relationships appear to be complex and the exact nature, whether causal, mediating or otherwise, should be further investigated as there may be positive implications for the design of effective interventions and support plans. Depression is associated with theory of mind and empathy, particularly the affective elements of these abilities. Depression may also be related to poorer emotion regulation, but again, the nature of these relationships is complex and would benefit from further research, as similar to anxiety, research could help inform interventions and clinical practise.

Chapter Two appears to support the results regarding anxiety and depression from the empirical Chapters (Three to Five); there does not appear to be a relationship between anxiety

and depression and emotion recognition in ASC. However, due to common co-occurrence of anxiety and depression symptoms in some autistic individuals, it is likely that anxiety and depression do have an effect on some socio-emotional abilities in ASC, which is observed in the narrative review (Chapter Two). Future research should focus on the relationships between anxious and depressive symptoms and socio-emotional abilities (such as theory of mind, emotion regulation, and empathy) as potentially parsing out the cause-and-effect components may aid in the design of more effective interventions. For example, if depression impacts theory of mind and empathy abilities, leading to increasing social problems and social withdrawal, treatment of depression could help improve social outcomes in autism; while conversely, if theory of mind and empathy problems lead to social problems and withdrawal which contribute to depression, then targeting theory of mind and empathy abilities could help prevent the development of depression in autistic people. In Chapter Two, it was also discussed that interventions for depression in autistic individuals was an under-researched area (DeFilippis, 2018; White et al., 2018), and thus future research should also focus here, as some interventions may not be as effective for autistic individuals and the development of tailored interventions for autistic and other neuro-diverse populations would be beneficial.

Gaze behaviour, occluding the mouth, and face processing

Atypical gaze behaviour has been widely reported within autism, specifically reduced eye contact during social situations. However, recent literature indicates that it may be alexithymia driving this behaviour (Bird, Press, & Richardson, 2011). Individuals with autism may avoid the eyes due to social anxiety or it may reflect differences in face processing mechanisms: in any case, reduced looking to the eye region would indicate that autistic people are not using the eyes to gain information about other's emotional states and thus may rely on information from the mouth region more (Neumann, Spezio, Piven, & Adolphs, 2006). Chapter Four found that obscuring the mouth region with face masks did disrupt face processing for autistic individuals, particularly emotion recognition. This was not unique to the autistic sample, with the non-autistic sample also being negatively affected; however autistic people were more adversely affected by the presence of face masks, when processing both identity and emotion. Paired with the results of Chapter Five, which found that reduced gaze to the eye region was associated with autistic traits, potentially some

autistic individuals are using the mouth region rather than the eyes more for facial emotion processing and thus obscuring the mouth can have negative consequences. These results mirror those found across the literature, with evidence suggesting that face masks have a detrimental effect on face processing (Carbon 2020; Marini et al, 2021; Parhoozi, Forby, & Kingstone 2021), and that reduced eye contact is often observed in autistic individuals (Hessels et al., 2018; Kleberg et al., 2017; Papagiannopoulou et al., 2014).

However, in Chapter Five, it was predicted that alexithymia may also influence gaze behaviour, but no relationship was found. This differs from current literature which suggests that alexithymia may be driving atypical (typically reduced) gaze behaviour towards the eyes (rather autism), with this effect even being observed in non-autistic participants (Bird, Press, & Richardson, 2011; Cuve, Gao, & Fuse, 2018).

Potential factors that may explain the discrepancies between the literature and the lack of findings regarding gaze and alexithymia may lie with some methodological considerations. Firstly, a student sample was used, not a clinical autism/alexithymia group, and thus perhaps it was more difficult to see some of the effects of alexithymia (and autism). However, as autistic traits were found to be associated with gaze, and alexithymia being associated with emotion recognition, evidently the sample was suitable for finding relationships between some variables, and there must have been sufficient variation in alexithymia and autistic trait scores for these relationships to be evident. In addition, in Chapter Five, alexithymia was found to impact emotion recognition abilities, indicating that there are likely no general issues with the alexithymia measure used (the TAS-20 alexithymia scale). Potentially, with a clinical sample, effects may be larger, and more relationships can be established. Additionally, the sample included 42 participants, and potentially with a larger sample more effects of alexithymia may be observed.

Future research should focus on establishing the impact of alexithymia on gaze behaviour in clinical populations (such as ASC), or non-clinical high alexithymic trait individuals as wider research seems to indicate some effect on atypical gaze towards the eyes. In addition, Chapter Four provides insight into some of the detrimental effects of using face masks for autistic individuals. During the Covid-19 pandemic, face masks became part of our daily lives, and it appears for some autistic individuals, wearing face masks during social interactions may have hindered face processing, especially facial emotion recognition. The implications of this

research posit that covering the mouth region can negatively affect face processing, and thus further investigation into alternative face coverings may be useful for any future need for widespread use of face masks. Currently, as the need for widespread public use of face mask to prevent the spread of Covid-19 has diminished, it is often medical and dental settings that face coverings are used. Research has identified clear face masks help patient understanding and patient-clinician trust (Kratzke, et al., 2021; Marini et al., 2021), and so future research could examine this within clinical populations, potentially using experimental paradigms as well as asking clinical populations for recommendations that they would prefer in the future.

Conclusion

To summarise, the current thesis has demonstrated that the roles of autism and alexithymia in facial emotion processing are yet to be fully understood, and future research should focus on why we such mixed results throughout the literature, whether it be due to the highly heterogenous nature of autism, publication bias or other factors. Additionally, the current thesis also re-affirms the importance of the mouth region in face processing, and that the use of face masks during the Covid-19 pandemic likely had a detrimental impact on both autistic and non-autistic individuals, and that potential alternatives should be considered in settings where face coverings are still prevalent. Furthermore, the importance of creating effective interventions and support plans for autistic individuals experiencing anxiety and depression has been highlighted, as while anxiety and depression do not appear to greatly impact face processing, evidence does suggest that they do affect other components of social and emotional cognition for autistic individuals. This research is important in developing our understanding of co-occurring conditions alongside autism and understanding the effects of these conditions on cognitive processes as well as the daily lives of people with ASC. Increasing our understanding of the relationships between autism, alexithymia, and mood disorders has positive implications, such as the design of appropriate, tailor-made support and interventions for neuro-diverse populations.

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