Visual and Physiological Mechanisms Underlying Emotion Processing in Autism and Alexithymia

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St John's College
University of Oxford

A thesis submitted for the degree of
Doctor of Philosophy
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Supervisor: Geoffrey Bird, PhD
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ABSTRACT

Impairments in emotion processing are one of the key diagnostic social symptoms of autism. Proposed explanatory mechanisms include atypical visual perceptual processes (e.g., social attention) and physiological responses to socioemotional stimuli (e.g., other people's faces). However, experimental evidence for these processes has been highly equivocal, and prevailing models have been unable to account for the documented individual differences observed in these processes.

Chapter 1 reviews the current conceptualisation of autism and outlines the theoretical context of my research, highlighting key hypotheses and mechanisms underlying emotion processing in autism which will be explored throughout the thesis. In particular, the potential explanatory role of the "*alexithymia hypothesis"* is discussed. Alexithymia is a condition frequently co-occurring with (but not unique to) autism and is linked to individual differences in socioemotional processes that have been implicated in autism (e.g., social attention, emotion processing, physiological responses). This thesis will therefore investigate the contribution of alexithymia to several aspects of socioemotional functioning thought to be linked to autism.

Chapter 2 investigated the spatiotemporal dynamics of gaze allocation to faces during emotion processing tasks in autism and alexithymia, and contrasted traditional social attention hypotheses to recent applications of Bayesian accounts of autism to social attention processes. Evidence from traditional and novel methods revealed that atypical eye gaze during emotion processing is best predicted by alexithymia in both autistic and non-autistic individuals. Specifically, alexithymia affected the non-linear temporal patterns of gaze allocation to faces and predicted reduced modulation of eye gaze to cued visual exploration

of emotions. The alexithymia effect is argued to reflect imprecise emotion concepts and priors for spatiotemporal dynamics of facial expressions. This proposal was supported by three additional experiments in **Chapter 3** linking alexithymia to atypical modulation of social attention and emotion processing by stimulus dynamics, and by task- and emotion-relevant priors.

Chapter 4 focused on exploring the potential role of alexithymia in disrupting links between physiological responding and emotion processing proposed by arousal-based models of autism and alexithymia. A novel approach capitalising on multimodal recording of physiological responses, was used to characterise multivariate profiles of physiological mobilisation to emotions. This approach provided evidence for alexithymia, not autism, driven effects on atypical physiological mobilisation to emotions, and puts forth a novel hypothesis with implications for autonomic and interoceptive accounts of alexithymia, autism and emotion.

One remaining challenge to the alexithymia hypothesis has been the possibility that alexithymia may simply reflect expected variation within the already multifaceted autism phenotype. In **Chapter 5,** two large-scale studies of clinical and non-clinical samples used confirmatory factor analysis and network psychometric modelling to address this remaining question. Results suggested separate underlying latent factors for alexithymia and autism, as well as separate clusters of symptoms which highlighted structural and dimensional distinctions of the two conditions.

In **Chapter 6,** I discuss the implications of this body of work for theory and practice and outline novel directions for research on autism, alexithymia and socioemotional processes. Combined, this body of work provides the most extensive test of the alexithymia hypothesis and highlights the specific mechanisms through which alexithymia affects socio-emotional

processes in autism. This work also offers a model for studying typical and atypical socioemotional processes and several methodological and conceptual advances with implications for basic and applied research on socioemotional processes.

Dedicated to my late grandfather, vovó Jaime Cuve (1927 - 2021).

I wish you could live forever so that we could have spent a little more time together.

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DECLARATION

This thesis comprises work conducted by Hélio Clemente José Cuve for the degree of Doctor of Philosophy from the University of Oxford. Hélio Clemente José Cuve led the design, conceptualisation, recruitment, data collection, analyses, data visualisation, interpretation and writing of all the work that contributed to this thesis. The role of collaborators is specified below.

Several chapters contain content that has been published (Experiment 1 in Chapter 2, Experiment 4 and 5 in Chapter 4, and Chapter 5) or currently under review in peer-reviewed journals (Chapter 3 and Chapter 4). But all these experiments and chapters have been edited and formatted specifically for this thesis. Hélio Cuve authored and produced all the text, figures and tables that are reused in relevant sections and was the first author of all the relevant publications. Supervisors and collaborators agreed for the respective materials to be included in the thesis and acknowledge the primary and significant contribution of Hélio Cuve to all the co-authored papers.

The published work which in which **Experiment 1 in Chapter 2** involved 5 collaborators**.** Hélio Cuve led the conception and design, data acquisition, analysis and interpretation, visualisation, drafting, and revising of the manuscript. Santiago Castiello and Dr Brook Shiferaw contributed to the implementation of entropy analyses and revising the published manuscript. Eri Ichijo, a member of the lab group, was responsible for conducting formal diagnostic interviews with autistic participants as a pre-requisite for the various studies within the research group. Dr Caroline Catmur contributed to revising the published manuscript and lab discussions. Professor Geoff Bird contributed as the main supervisor and commented on all aspects of my work.

For the published work in which **Experiments 4 and 5** in **Chapter 4** is based on, Hélio Cuve worked with 4 collaborators. Hélio Cuve led the conception and design, data acquisition, data processing, analysis and interpretation, visualisation, drafting, and revising the published manuscript of both experiments. Jelka Stojanov, Xavier Miklos Gall and Lucy Johnson Perret contributed part of the data acquisition and data processing. Both Dr Caroline Catmur and Professor Geoffrey Bird revised the published manuscript.

For **Chapter 5,** Hélio Cuve collaborated with Dr Jennifer Murphy and Dr Hanna Hobson who contributed data for the published paper. Hélio Cuve led all aspects of conception, data acquisition, processing, analysis and interpretation, visualisation, drafting, and revising the manuscript. Dr Jennifer Murphy and Dr Hanna Hobson contributed to the revision of the manuscript for publication. Dr Caroline Catmur contributed to discussions and revisions of the published manuscript. Professor Geoff Bird contributed to the supervision, interpretation, and revision of the published manuscript.

Publications

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Note to Reader

To optimise this document for online and electronic use, clickable hyperlinks and cross references are provided where relevant in the electronic version (e.g., mention to figures, tables, appendices, list of contents, etc.) and will typically be marked with different formatting relative to the surrounding text.

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ABBREVIATIONS

ACC – Anterior Cingulate Cortex

ADOS – Autism Diagnostic Observation Schedule.

APA – American Psychological Associations (also used for American Psychiatric Associations)

AQ-50 or 28 – Autism Spectrum Quotient Questionnaire (50 or 28 items)

ASD or ASC – Autism Spectrum Disorder / Autism Spectrum Condition

AUS – Action Units

AIC – Akaike Information Criterion

BADIA/BADA – Barycentric Discriminant Analysis

BIC – Bayesian Information Criterion

BVAQ – Bermond-Vorst Alexithymia Questionnaire

CFA – Confirmatory Factor Analysys

DASS – Depression Anxiety and Stress Scale

DDF – Difficulties describing feelings

DIF – Difficulties identifying feelings

DSM – Diagnostic and Statistical Manual of Mental Disorders

EDA – Electrodermal activity

EEG – Electroencephalography

EFA – Exploratory Factor Analysis

EGA – Exploratory Graphical Analysis

EOT – Externally oriented thinking

EXP - Experiment

FACS – Facial action coding system

FMRI – Functional magnetic resonance imaging

GGM – Gaussian Graphical Models

HR – Heart Rate

HRV – Heart Rate Variability

IQ – Intelligence Quotient

mPFC – Medial Pre-frontal Cortex

PCA – Principal Component Analysis

PTSD – Post traumatic stress disorder

RSA – Respiratory Sinus Arrhythmia

SCL – Skin Conductance Level

SCR – Skin Conductance Response

SK – Spatial-kinematics

SM – Supplementary materials.

SVD – Singular value decomposition

TAS-20 – Toronto Alexithymia Scale

TDCS – Transcranial direct current stimulation

TMS – Transcranial magnetic stimulation

WASI – Weschler Abbreviated Scale of Intelligence

1 CHAPTER 1. INTRODUCTION

This thesis presents a series of studies exploring socioemotional functioning in Autism Spectrum Disorder (henceforth autism). With the application of recent methodological advances, prevailing theoretical models are contrasted with emerging hypotheses to identify and describe the mechanisms implicated in typical and atypical socioemotional functioning in autism.

In this section, I begin with an overview of what autism is. I will review the current formal conceptualisation of autism, views on aetiology, diagnosis, and assessment. I will highlight specific areas of the condition relating to socioemotional functioning which this thesis will focus on and introduce the theoretical frameworks which will guide most of the experimental work discussed throughout the thesis.

1.1 What is Autism?

The first modern descriptions of autism are linked to Leo Kanner (Kanner, 1943) and Hans Asperger(Asperger, 1938). Kanner's description of autism focused on emotional deficits characteristic of both the patients and their families (Kanner, 1943), whereas Asperger's descriptions were of intellectual gifts coupled with psychopathic-like characteristics (Asperger, 1938; Wolff, 2004). In the most recent version of the DSM-5 (APA, 2013), autism is classified as a neurodevelopmental disorder defined by two groups or clusters of symptoms:

A. Persistent deficits in social communication and social interaction. These may include deficits in socioemotional reciprocity in conversational interactions, deficits in nonverbal

communicative behaviours (producing and processing facial expressions, eye-contact), and difficulties with developing and maintaining relationships.

B. Restricted, repetitive patterns of behaviour, interests, or activities. This may include stereotyped or repetitive interests and patterns of behaviour (including motor behaviour stereotypy), insistence on sameness, hyper- or hypo-reactivity to sensory input, or unusual interest in sensory aspects of the environment.

The **prevalence** of autism is estimated to be around 2.2% with more than 24.8 million people globally affected (Vos, Allen, & Arora, 2016). However, autism has seen one of the largest increases in prevalence of any neuropsychiatric disorder, with a 787% increase over just 20 years, particularly for women (Matson & Kozlowski, 2011; Russell et al., 2021). Rather than a true increase in prevalence, this likely reflects increased reporting and changes in practices and assessment, as well as increased public awareness.

The **aetiology** of autism remains largely unknown and is a topic of ongoing research. Most research to date has suggested risk factors including genetics and pathophysiology of the brain. A genetic component to autism, in particular, appears certain, as evidenced by the increased familiar prevalence (in parents and offspring of diagnosed individuals) and from twin studies (Ronald, Happé, Price, Baron-Cohen, & Plomin, 2006), with estimations of close to 90% heritability rates (Tick, Bolton, Happé, Rutter, & Rijsdijk, 2016). However, studies looking for specific genetic markers have failed to provide consistent results. Many of the identified genes appear to only affect a small fraction of cases (e.g., less than 1%). For example, genetic conditions such as Tuberous Sclerosis and the Fragile X anomaly which exhibit clearer links with autism, account for only a small proportion of cases (Persico & Napolioni, 2013; Rutter, 2005). Furthermore, many so-called “autism genes” are also involved in other psychiatric conditions, including schizophrenia and intellectual disability

(Persico & Napolioni, 2013). As such, this has led to suggestions of the importance of a potentially large number of genetic variants, some of which are common within the condition and have a small effect, and some of which are rare but have a large effect (Lord, Elsabbagh, Baird, & Veenstra-Vanderweele, 2018).

In terms of neurobiology, current views do not subscribe to specific impairments, instead, most suggest problems in brain reorganisation and maturation which eventually predispose an individual to altered functional connectivity which gives rise to the autistic phenotype (Ecker, Bookheimer, & Murphy, 2015; Lord et al., 2018). Structural imaging studies have also emphasised brain overgrowth as represented in large brain volume (Hazlett et al., 2017).

Functional imaging studies, on the other hand, largely highlight problems in interconnectivity which may reflect atypical physical connectivity or causal interactions in patterns of activity in different brain regions (Lord et al., 2018; O'Reilly, Lewis, & Elsabbagh, 2017). Overall reduced connectivity and patterns of local hyper-connectivity have been documented in autism leading to many hypotheses of neural excitation-inhibition imbalance (Hadjikhani et al., 2018; O'Reilly et al., 2017; Rane et al., 2015).

Diagnosis. The diagnosis of autism is largely based on behavioural phenotypes. Assessment, therefore, focuses on comparing symptoms to descriptions of the condition, such as the ones provided in DSM-5. Additionally, a neurodevelopmental history of deficits is needed such that symptoms must be present in the early developmental period, particularly in the first two years of life (APA, 2013). As such, in addition to the DSM criteria, the diagnostic process of autism typically involves assessment of executive and social functioning in form of standardised clinical interviews and observations (e.g., Autism Diagnosis Observation Schedule, ADOS), with the input of family or caregivers where possible (Pruette, 2013; Rutter, Couteur, & Lord, 2003). For verbal and non-cognitively compromised individuals,

self-report questionnaires measuring autistic traits are also typically employed, for example, the Autism Spectrum Quotient (AQ-50) measuring social skills, communication, imagination, attention to detail, and attention switching (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001). Genetic testing is also recommended, particularly when there are risk factors for certain genetic conditions (e.g., Fragile X), or when certain prenatal risk factors are present, such as advanced age during pregnancy (Lord et al., 2018; Rutter, 2005).

While a neurodevelopmental history of deficits is essential for diagnosis, it is not uncommon for a formal diagnosis to happen much later in life (Huang, Arnold, Foley, & Trollor, 2020; Tromans, Chester, Kiani, Alexander, & Brugha, 2018). This may be related to the fact that while intellectual disability and language impairments are highly comorbid symptoms in autism (APA, 2013), some autistic individuals do not have appreciable cognitive impairments, particularly if they are classed as high functioning (Ben, 2014; Lincoln, Courchesne, & Kilman, 1988; G. Russell et al., 2019). While not being representative of the autistic population as a whole, these samples are particularly useful for investigating higher-order socio-cognitive processes which often require good language comprehension, and for testing socio-cognitive impairments in absence of intellectual disability (Harms, Martin, & Wallace, 2010).

1.1.1 Autism and socioemotional processes

Whilst autism is a complex and multifaceted condition with deficits across many cognitive areas, socio-cognitive deficits have captured the interest of many researchers since early descriptions of the condition. Socio-cognitive processes are relevant for various aspects of normal life with implications on the ability to form, develop and maintain relationships and to lead independent lives (Adolphs, 2001). Deficits in socio-cognitive processes in autism may therefore explain the increased prevalence of other mental disorders in autism, including

depression and anxiety (Lai et al., 2019), and social problems resulting from the economic burden associated with the condition, e.g. due to unemployment (Ganz, 2007).
Despite thousands of studies published to date exploring various aspects of socioemotional functioning in autism, there are still limited to no widely accepted and effective interventions (Grynszpan, Weiss, Perez-Diaz, & Gal, 2014). Part of the difficulty is that the nature of socioemotional deficits in autism is still poorly understood, and models of social cognition have not been able to provide an integrated account of the diverse profile of social skills in the condition (Gaigg, 2012). Furthermore, specific models of autism are often micro-theories or very narrow interpretations of models of social cognition, emotion, and social attention (Leekam, 2016). Furthermore, the mechanisms that support many typical socio-cognitive processes, such as face and person perception, as well as experiential aspects of emotion, have continued to elude psychologists (Adolphs, 2001, 2010).

Understanding the social symptoms of autism is therefore not only useful for developing interventions for autistic individuals, but may ultimately inform socio-cognitive models for normative behaviour (Leekam, 2016; Pelphrey, Adolphs, & Morris, 2004). For example, social cognition research for the past 40 years has largely been driven by the discovery of developmental effects on the representation of the mental states of others and false belief understanding (Premack & Woodruff, 1978). This discovery inspired many studies on the development of atypical social cognition in autism, where failures on false-belief tasks led to the hypothesis that autistic individuals lack a theory of mind (Baron-Cohen, Leslie, & Frith, 1986). Such simplistic theories have come under increasing scrutiny since their development, however, for example research suggests that many typical and autistic children fail false-belief for reasons unrelated to representing others' minds (Pellicano, 2012). Novel models, therefore, have emerged which attempt to account for both typical and atypical social cognition (Baker, Jara-Ettinger, Saxe, & Tenenbaum, 2017; Schaafsma, Pfaff, Spunt, &

Adolphs, 2015) including explaining the variability of how humans represent others minds (Bird & Viding, 2014; Conway et al., 2020; Conway, Catmur, & Bird, 2019; Tamir & Thornton, 2018).

This thesis, therefore, presents several experiments exploring mechanisms underlying the socioemotional functioning symptoms of autism. Specifically, mechanisms underlying problems in emotion processing, atypical social attention (eye gaze), as well as experiential components of emotion are investigated. Much of this work, however, has implications for understanding typical socioemotional processing. In the next section, I will briefly review the theoretical approaches that will guide the experimental work in this thesis.

1.1.2 Linking emotion, attention and physiology in autism

Most theorists agree that emotions reflect a core quality that defines the tone of the interaction between humans and their social world (Adolphs & Anderson, 2018; Gendron, 2010). This core quality can be manifested in terms of homeostatic processes and action tendencies (e.g., physiological reactions that drive approach or avoidance behaviours) (Norman, Necka, & Berntson, 2016).

In practice, emotion is often studied in terms of two components – the communicative component (facial expressions and other non-verbal behaviour), and an experiential component (physiological correlates of emotion and subjective experience). It is through the communicative and experiential components that emotions have consequences for interpersonal relationships (Adolphs, 2001; Parkinson, Fischer, & Manstead, 2005). A disruption in the ability to process or respond appropriately to emotional information has important consequences for how we interact with the world around us. Many studies have therefore suggested that autism is related to deficits in the communicative and experiential

components of emotion which may result in deficits in empathy and theory of mind (Adolphs, Sears, & Piven, 2001; Baron, 1995; Hobson, 1986).

Of note, however, is that emotional processes do not operate in isolation from other cognitive processes. It is clear that emotional processes interact with a wide range of cognitive processes, including attention, perception, and memory, and these interactions are the norm rather than the exception (Phelps, 2006). Furthermore, increased understanding of brain organisation and functioning highlights that the conceptual dichotomisation between emotion and cognition is not warranted, as purely "affective" and "cognitive" brain regions or networks have consistently failed to materialise (Pessoa, 2008).

The interaction between emotion and cognitive processes is relevant for understanding several accounts of socioemotional deficits in autism. For example, two related mechanisms have been suggested to underlie atypical emotion recognition and processing abilities. The first, which is explored in **Chapters 2** and **3**, involves a disruption of visual social attention processes, resulting in atypical modulation of gaze during face-to-face interactions, including reduced sensitivity to gaze direction and reduced eye contact. The second mechanism involves atypical neurophysiological modulation to socioemotional stimuli (e.g., eye contact). Atypical neurophysiological modulation has been a key feature of theories of autism and is thought to explain the common profile of hyper- and hypo-sensitivity which has frequently been described in autism, in both anecdotal and autobiographical accounts (Blackman & Attwood, 1999), as well as in empirical research (Klintwall et al., 2011; Robins, Bakeman, & Adamson, 2009). In addition to dynamically influencing and being influenced by processes such as visual social attention, atypical neurophysiological responses are also thought to have an impact on the experiential component of emotion via interoceptive processes (processes which allow the physiological state of one's body to be available to consciousness).

Therefore, many models of autism have argued that physiological or interoceptive processes may be atypical in autism.

Emotion processing, visual social attention, and physiological mechanisms are, therefore, invariably included in key theoretical frameworks of autism attempting to explain specific socioemotional deficits. I will briefly introduce the relevant theoretical frameworks in the next section. The various chapters and experiments throughout the thesis will also provide an overview of specific interpretations of these general frameworks to the specific questions explored in each chapter (see also **1.4. Guiding research questions**).

1.2 Theoretical frameworks

The working hypotheses guiding research on socioemotional processes in autism can be loosely subdivided into three main categories:

1- Physiological arousal and social attention-based models.
2- Emerging 'top-down' models, with a particular focus on the Bayesian accounts of autism.
3- Models of individual differences, with a particular focus on the alexithymia hypothesis, which attempts to account for the heterogeneity in the socioemotional profiles of autism.

1.2.1 Physiological arousal-models of socio-emotional processing in autism

One component that has been central to many theories of emotion is physiological responses. Darwin proposed early evolutionary accounts of emotions, wherein physiological signals were linked to behaviours (e.g., facial expressions, bodily changes) (Darwin, 1872). Evolutionary accounts inspired by Darwin proposed physiological responding as a mechanism to mobilise and regulate metabolic processes in the organism, and to optimise behavioural responses that are essential for survival or maintenance of the organism (e.g., approach or avoidance behaviours) (Ekman & Davidson, 1994; Kreibig, 2010; Stemmler, 2004). William James, one of the most influential theorists on emotion also conceived emotions as causal consequences of physiological changes in the body, going as far as to suggest that affective events without the accompanying physiological component cannot be considered "emotion proper" (James 1948).

The arousal-based models of autism at their core are based on this idea that physiological responses are central to emotional processes. These views highlight that hyper- and hypo-sensory responding is commonly reported in autism, such that aberrant resting and/or phasic physiological modulation may be a mechanism underlying atypical socioemotional processes (Cuve, Gao, & Fuse, 2018; Rogers & Ozonoff, 2005). Most of the empirical research comes from the measurement of autonomic (Cuve et al., 2018; Lydon et al., 2016; Top, Luke, Stephenson, & South, 2018) and brain responses during socioemotional tasks (Hadjikhani et al., 2017; Kliemann, Dziobek, Hatri, Baudewig, & Heekeren, 2012; Monk, 2010), although the nature of the tasks is varied and taps into different emotion components (e.g., emotion recognition vs emotional experience).

For example, the **hyperarousal hypothesis** is a specific (group of) arousal based model(s), which argue that emotion regulation difficulties in autism result in heightened sensitivity to socioemotional stimuli (Cuve et al., 2018; Hadjikhani et al., 2018; Tanaka & Sung, 2016). Problems recognising emotion from faces and atypical gaze (e.g., eye contact), and stereotyped behaviour are explained in terms of strategies reduce this physiological dysregulation (Lassalle et al., 2017; Tanaka & Sung, 2016). In social attention studies, this view is also often referred to as the eye-avoidance hypothesis.

A contrasting arousal-based model is the **hypoactivity or hypoarousal model,** which is closely linked to physiological models of orienting behaviour (Margaret M Bradley, 2009) and sociomotivational theories of autism (Chevallier, Kohls, Troiani, Brodkin, & Schultz, 2012). This hypothesis claims that physiological lability and disengagement in response to socioemotional stimuli in autism results in atypical learning of the intrinsic reward linked to socioemotional stimuli, for example the human face (Cuve et al., 2018; Rogers & Ozonoff, 2005). **Chapter 2** partly explores the application of this framework to the study of visual social attention, whereas **Chapter 4** explores in more detail the experiential aspects of emotion and physiological responses.

1.2.2 Top-down influence models: Bayesian accounts of autism

The Bayesian theory of autism has emerged in recent years as a result of the application of Bayesian models of perception to autism (Elizabeth Pellicano & Burr, 2012; Sinha et al., 2014). These models argue that atypical integration of sensory input and problems with learning statistical regularities about the environment (priors) underlie most atypical sensory and perceptual experiences in autism (Pellicano & Burr, 2012). Like general Bayesian models of perception, the key idea is that perceptual decisions and sensory experiences are influenced by both incoming sensory information and prior knowledge which exerts a top-down

influence on sensory processing (Helmholtz, 1867). As such, autistic deficits can be explained in terms of one or more differences in Bayesian parameters which describe how perception is modulated by priors and sensory information.

A few studies have explored Bayesian accounts of autism in the context of sensory abnormalities (Lawson, Mathys, & Rees, 2017) and in visual processing of abstract information (Król & Król, 2019; Maule, Stanworth, Pellicano, & Franklin, 2017), yet not many have explored applications to socioemotional processing (Pell et al., 2016). Nonetheless, much of the empirical work on visual processing speaks to the potential of Bayesian models for understanding social attention and emotion processing in autism. This includes previous work demonstrating top-down effects of priors on eye-movements (Friston, Adams, Perrinet, & Breakspear, 2012; Tatler, Hayhoe, Land, & Ballard, 2011), as well as the sensitivity of visual processing to stimulus-driven sensory information, including image statistics and biological motion (Johansson, 1973; Land, 1999; Thompson, Hansen, Hess, & Troje, 2007) which are also relevant for social stimuli like faces and emotional expressions (Furl et al., 2020; Jack & Schyns, 2015). This thesis, therefore, explores relevant aspects of the application of Bayesian accounts of autism to socioemotional processing, particularly to social attention, in experiments described in **Chapters 2 and 3.**

1.2.3 Models of individual differences: The alexithymia hypothesis

One common theme in reviews and meta-analyses of socioemotional deficits in autism is that findings are highly heterogeneous. This is true for emotion recognition deficits (Harms et al., 2010; Uljarevic & Hamilton, 2013), atypical visual social attention (Cuve et al., 2018; Nation & Penny, 2008; Papagiannopoulou, Chitty, Hermens, Hickie, & Lagopoulos, 2014), and atypical autonomic and neurophysiological responses to socioemotional stimuli (Ecker et al., 2015; Lydon et al., 2016). While part of the heterogeneity is certainly due to methodological

differences across studies, including task, stimuli, and analytical approaches, there is potentially as much heterogeneity in profiles of social functioning across autistic individuals.

This heterogeneity is well recognised within autism (Martinez-Murcia et al., 2017; Mottron & Bzdok, 2020), but socioemotional research has failed to explore its underlying factors. Recent accounts of autism suggest that heterogeneity concerning socioemotional functioning within the autistic population may be systematic, and explained by co-occurring conditions such as alexithymia (Bird & Cook, 2013). Alexithymia is a subclinical condition characterised by an impaired ability to describe and identify emotions and bodily signals (Nemiah, 1976; Sifneos, 1973). Alexithymia and autism display a high level of comorbidity, with estimations suggesting that close to 50% of people with autism also have alexithymia at clinically significant levels, in comparison to 10% of the general population (Berthoz & Hill, 2005; Kinnaird, Stewart, & Tchanturia, 2019). However, alexithymia is also highly prevalent in other somatic and mental disorders, suggesting that it may constitute a vulnerability marker for emotional problems across psychopathology (Lundh & Simonsson-Sarnecki, 2001; Papciak, Feuerstein, & Spiegel, 1985; Wingbermühle, Theunissen, Verhoeven, Kessels, & Egger, 2012).

In an interesting parallel, alexithymia has been implicated in many processes that have also been reported to be affected in autism, including difficulties with identifying emotions (Ola & Gullon-Scott, 2020), atypical visual processing of facial expressions (Cuve, et al., 2021) and atypical physiological and interoceptive responses that can also be described in terms of hypo and hyperarousal profiles (Panayiotou, Panteli, & Vlemincx, 2018; Shah, Hall, Catmur, & Bird, 2016).

Under the alexithymia hypothesis, alexithymia drives qualitatively distinct profiles of socioemotional functioning in autism (Bird & Cook, 2013). Nonetheless, autism research has

generally failed to control for alexithymia (and alexithymia research for autistic traits) making it difficult to test the specificity of emotion processing problems to either condition. Accounting for common and separate contributions of autism and alexithymia is particularly relevant to inform ongoing debates regarding the nature of emotional deficits in autism, as well as to inform clinical practice and theorising about typical and atypical socioemotional processes (Bird & Cook, 2013; Cuve, Murphy, et al., 2021).

Given the key focus of this thesis in providing a test for the alexithymia hypothesis, in the next section I briefly review the current views on the aetiology of alexithymia, its underlying mechanisms and assessment, and how well it can be differentiated from autism.

1.2.3.1 Alexithymia, aetiology and mechanisms

The **aetiology** of alexithymia remains unknown, and this area of research has received much less attention than research describing its associations with socioemotional processes. The atypicality in physiological and interoceptive responding typically associated with alexithymia led to proposals of disrupted transmission of interoceptive emotional information to the anterior cingulate cortex which is involved in the generation of emotional responses and dorsal areas implicated in the appraisal and expression of emotion (dorsal ACC, mPFC) (Etkin, Egner, & Kalisch, 2011; Goerlich & Aleman, 2018; Miyake et al., 2012). Atypical dorsomedial prefrontal cortex and amygdala function during both conscious and unconscious processing of emotions has also been reported in alexithymia (Goerlich, 2018; Katharina S. Goerlich & Aleman, 2018).

Even less research has focused on potential genetic underpinnings of alexithymia. However, recent genetic and endocrine studies of alexithymia highlighted a degree of heritability of alexithymia based on familial links in alexithymia scores between parents and offspring (Kano, Grabe, & Terock, 2018). Suggestive disruption in gene variants including oxytocin

receptors and serotonin reuptake genes has also been reported (Kano et al., 2018). Furthermore, because of the physiological profile of alexithymia which has been associated with impaired emotion regulation, sympathetic responding, and stress tolerance, some have suggested abnormalities in the hypothalamic–pituitary–adrenal axis which regulates neuroendocrinal responses to stress (Kano et al., 2018). However, most of this research is still very suggestive, with most samples being highly heterogeneous involving other conditions (e.g., PTSD and Depression), and most of the genetic and endocrinal mechanisms have been implicated in these other conditions as well (Kano et al., 2018).

In terms of **assessment**, objective markers of alexithymia are still an area of active research. Furthermore, alexithymia is currently not included as a formal diagnosis in the DSM, as such diagnosis is typically only possible via a combination of clinical interviews and self-reports (Sekely, Bagby, & Porcelli, 2018). The most popular and widely-validated measure of alexithymia is the Toronto Alexithymia Scale (TAS-20) (Bagby, Parker, & Taylor, 1994; Michael Bagby, Parker, & Taylor, 2020), which was designed to cover the original descriptions of the condition proposed in early work by Nemiah and Sifneous (Nemiah, 1977; Sifneos, 1973). It covers difficulties identifying feelings (DIF), difficulties describing feelings (DDF), and externally oriented thinking (EOT). The somatic problems included in the original descriptions are typically included within the feelings components (Bagby et al., 1994; Bagby et al., 2020), which may partly reflect the views of somatic symptoms in alexithymia as failures to process underlying emotional arousal (Maclean, 1949; Taylor, Bagby, & Parker, 1991). While there are no population norms for the TAS-20, a preliminary cut-off score of 61 has been suggested to indicate alexithymia, while scores below 51 indicate the absence of alexithymia (Bagby & Taylor, 1997). While scores for each subscale have been used in the literature, the authors have argued that the total score provides the most reliable measure of alexithymia (Parker, Keefer, & Taylor, 2008).

This thesis will explore the influence of alexithymia on several supposed autistic symptoms affecting social attention, emotion recognition and physiological responses. Additionally, in **Chapter 5**, I explore one issue that is crucial for the logical coherence of the alexithymia hypothesis and subtyping approaches in autism based on comorbidity – that is the degree to which autism and alexithymia reflect separate conditions, by investigating the degree of construct overlap between and their interactions.

1.3 Guiding research questions

In light of the theoretical frameworks introduced in the previous section, this thesis explores three groups of related research questions aiming to clarify the mechanisms underlying socioemotional symptoms in autism.

A central and overarching question in this thesis concerns the role of alexithymia and autism in socioemotional processes.

Are atypical socioemotional processes specific to autism or alexithymia?

Can alexithymia explain the heterogeneity in socioemotional processes in autism?

Across the studies and chapters, crucial tests of the alexithymia hypothesis are provided by evaluating the contributions of alexithymia to emotion processing, social attention, and physiological responses to emotion in autism.

Related to the alexithymia hypothesis, this thesis also addresses the question of autism and alexithymia overlap and comorbidity. In other words, **are autism and alexithymia distinct?** This question is central to the logical coherence of the alexithymia hypothesis and has implications for theories of autism as well as diagnosis, assessment, and intervention. A combination of experimental approaches and statistical modelling is used to disentangle the contributions of both autism and alexithymia across the relevant processes.

The second group of questions focuses on the nature of visual social attention deficits in autism.

Is social attention universally atypical in autism and/or alexithymia?

What mechanisms underlie atypicality and heterogeneity in visual social attention?

Specifically, in **Chapters 2 and 3**, I combine novel methodological approaches to examine social attention hypotheses of autism. I also explore whether Bayesian accounts of autism can account for atypical socioemotional processing and social attention in autism. Specifically, this research hopes to answer the following question:

Does atypical gaze in autism and/or alexithymia reflect atypical modulation of social attention by priors, atypical 'bottom-up' effects of socioemotional stimuli, or both?

Focusing particularly on social attention, novel methodologies were developed to quantify sensory information about social cues and novel experimental tasks were used to test top-down effects on visual behaviour during emotion processing tasks (discussed in Chapters 2 and 3).

The third group of questions relates to the experiential nature of emotion in autism and alexithymia.

Is physiological responding to emotion atypical in autism and/or alexithymia?

What is the specific profile of typical and atypical physiological responding to emotions?

The experimental work on these questions aimed to map the profile of physiological responses using multimodal recordings and multivariate analytical approaches that provide

more optimal tests to questions regarding the specificity of physiological and autonomic atypicalities, and how they relate to autism and alexithymia.

Overall, the empirical work addressing all three groups of questions is supported by both conceptual and methodological advances to study typical socioemotional processing. As such, each experiment and chapter are also relevant for informing the general field on typical socioemotional processes as well as approaches to subtyping heterogeneity in psychopathology. For example, the exploration of atypicality in social attention as well as Bayesian accounts also contribute knowledge about how social attention is modulated by stimuli-driven sensory information and top-down influences. The studies on experiential aspects of emotion are relevant for understanding the profile of typical physiological responses to emotion, and the contribution of physiological and interoceptive processes to attention processes. They also elucidate unknowns about the coherence of autonomic activation and physiological responding to emotion.

In **Figure 1.1** I provide a schematic descriptive heatmap with the density summarising how these various questions and models are reflected in the chapters and experiments included throughout the thesis.

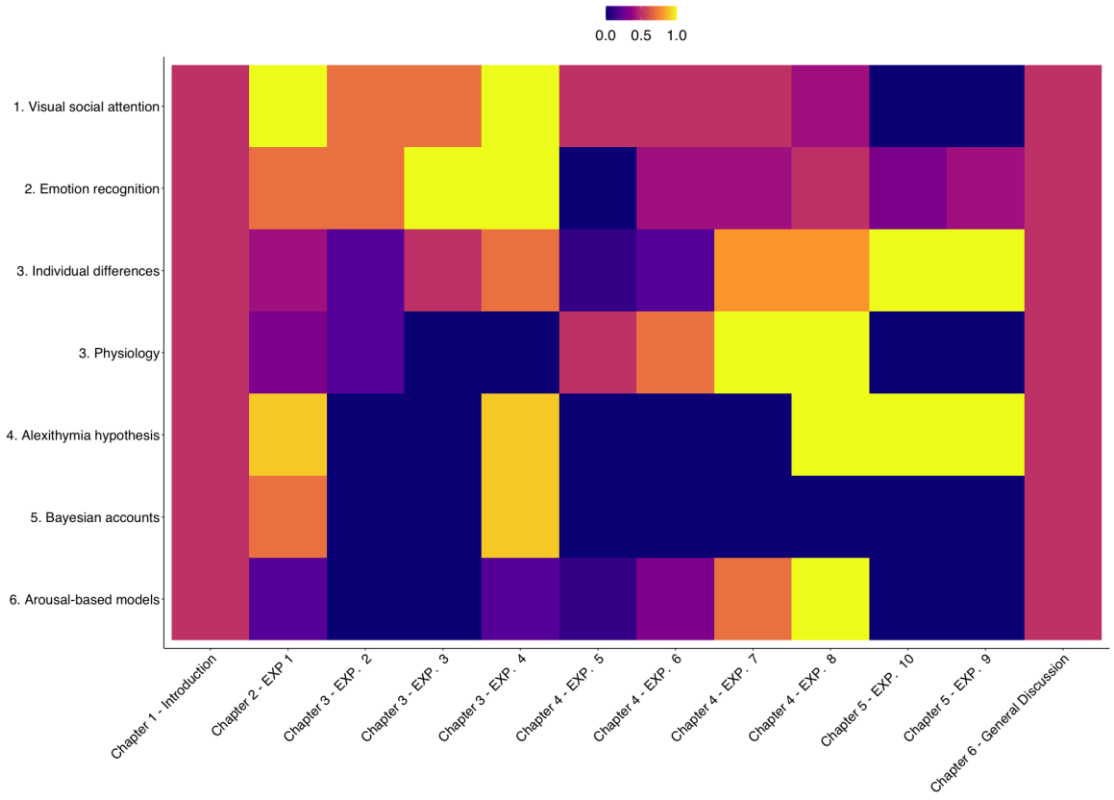

Figure 1.1 Heatmap of topics and theoretical models across the thesis.

High values (warmer colours) indicate an increased incidence (based on term frequency) of the topic and/or the model in the specific chapter and experiment (EXP).

1.4 What comes next?

Chapter 2 presents an eye-tracking paradigm to investigate whether autism or alexithymia are related to atypical gaze. A novel approach is used to study the dynamics of gaze allocation to facial expressions and to quantify gaze modulation effects by task- and emotion-relevant priors, to test the application of Bayesian accounts of autism to social attention.

2 CHAPTER 2. SPATIOTEMPORAL DYNAMICS OF GAZE ALLOCATION IN AUTISM AND ALEXITHYMIA

An edited version of this chapter has been published as the following article:

Cuve, H. C., Castiello, S., Shiferaw, B., Ichijo, E., Catmur, C., & Bird, G. (2021). Alexithymia explains atypical spatiotemporal dynamics of eye gaze in autism. *Cognition*, *212*, 104710.

Abstract

Recognition of emotional facial expressions is considered to be atypical in autism. This difficulty in emotion recognition is thought to be due to the way that facial expressions are visually explored. Evidence for atypical visual exploration of emotional faces in autism is, however, equivocal. In this chapter, I propose that, where observed, atypical visual exploration of emotional facial expressions is due to alexithymia, a distinct but frequently co-occurring condition. The eye-tracking study described in this chapter tested the alexithymia hypothesis using a number of recent methodological advances to study eye gaze during several emotion processing tasks (emotion recognition, intensity judgements, free gaze), in 25 adults with, and 45 without, autism. A multilevel polynomial modelling strategy was used to describe the spatiotemporal dynamics of eye gaze to emotional facial expressions. Converging evidence from traditional and novel analysis methods revealed that atypical gaze to the eyes is best predicted by alexithymia in both autistic and non-autistic individuals. Information theoretic analyses also revealed differential effects of task on gaze patterns as a function of alexithymia, but not autism. These findings highlight factors underlying atypical emotion processing in autistic individuals, with wide-ranging implications for emotion research.

Keywords: spatiotemporal; eye gaze; eye-tracking; autism; alexithymia; emotion recognition.

2.1 Experiment 1

2.1.1 Introduction

Autism is a condition defined by atypical social interaction and communication, and restricted patterns of thought and behaviour (APA, 2013). As discussed in chapter 1, the social diagnostic criteria specify a cluster of symptoms[1] that are likely related – a lack of socio-emotional reciprocity, impaired emotion recognition, and reduced eye gaze.

While the exact mechanisms linking these symptoms have not been determined, two hypotheses specify a role of arousal. Under one hypothesis, atypical emotion recognition is a result of active or reflexive avoidance of the eye region as a result of hyper-arousal induced by direct eye-contact (Kliemann et al., 2012; Hadjikhani et al., 2014). Under the other hypothesis, reduced eye gaze stems from hypo-arousal, resulting in diminished social motivation and orienting responses to social stimuli (Dalton et al., 2005). While there is evidence for both hypotheses (for a review see: Cuve, Gao & Fuse, 2018; Kliemmann et al., 2012, Senju et al., 2011), our understanding of how allocation of gaze to faces influences emotion recognition remains far from complete. For instance, previous work demonstrates that an individual's attention to the eye region of another's face is predictive of the degree to which the individual can recognise the other's emotions (Schurgin et al., 2014). Other studies, however, demonstrate that accurate emotion recognition can occur without overt visual attention to specific face features (Calvo et al., 2014; Peterson & Eckstein, 2012), and with varying patterns of gaze fixations (Yitzak et al., 2020).

It should also be acknowledged that most previous studies used static stimuli, but recent work highlights the importance of dynamic information as facial expressions evolve over time

[1] I use the term 'symptom' to mirror clinical practice but note that several autism advocates prefer the term 'traits' as it avoids equating autism with a disease.

(Jack et al., 2014). Thus, the predictive value of gaze with respect to emotion recognition may be dependent not only on the allocation of gaze to specific facial features, but also on the spatiotemporal relationships between gaze and the dynamic aspects of facial expressions. Furthermore, the redundancies in information conveyed through the spatiotemporal dynamics of facial expressions are likely to optimise recognition of emotions expressed in the face. For example, the kinematics of facial movements may provide additional cues as to the valence and intensity of emotion (Sowden et al., 2020). Such redundancies may explain why allocation of gaze to facial features is not always predictive of emotion recognition, particularly with dynamic stimuli. Importantly, such redundancy is the norm in real-life social interaction, where communication is often multimodal (e.g., involving speech, hand gestures, and gaze direction; Aviezer et al., 2011; Hessels et al., 2020) and benefits from context (Barret et al., 2011).

Considering the influential hypotheses concerning the role of atypical eye gaze in emotion recognition in autism (see Hadjikhani, Zurcher, et al., 2017; Kliemann, Dziobek, Hatri, Steimke, & Heekeren, 2010), and the fact that atypical eye gaze is widely regarded as a key diagnostic feature of autism (Senju & Johnson, 2009), it is significant that eye-tracking studies investigating how individuals with autism visually explore emotional faces have produced remarkably mixed results. Although several studies report atypical eye gaze in autism (Hadjikhani, Zurcher, et al., 2017; Kliemann, Dziobek, Hatri, Steimke, & Heekeren, 2010; Senju & Johnson, 2009), others do not (Black et al., 2017; Cuve, Gao, & Fuse, 2018; Guillon, Hadjikhani, Baduel, & Rogé, 2014; Kwon, Moore, Barnes, Cha, & Pierce, 2019). Even recent studies incorporating more naturalistic dyadic gaze interactions have produced conflicting results. Some studies found atypical eye gaze only when traditional screen-based measures were used (Grossman, Zane & Micthe, 2019), whereas other studies found a

reduction of eye-contact with elevated autistic traits in dyadic gaze interactions (Hessels, Holleman, Cornelissen, Hooge, Kemner, 2018).

There are at least two groups of factors that may explain these inconsistent results. The first relates to individual differences within the autistic population, and the second concerns methodological features relating to task format and how gaze behaviour is operationalised and measured. I discuss these factors below.

2.1.1.1 Individual differences

Although heterogeneity within the autistic population is well recognised, appeals to heterogeneity to explain inconsistent results are often vague, with no clear hypothesis about which individual differences contribute to variance in results. However, variability in the socioemotional domain in autism may be systematic, and related to alexithymia, as proposed by the alexithymia hypothesis introduced in Chapter 1.

Under the alexithymia hypothesis, inconsistent results when testing various socioemotional abilities in the autism literature are due to sampling variance with respect to alexithymia (Bird & Cook, 2013; Brewer, Happé, Cook, & Bird, 2015). Previous data support the alexithymia hypothesis, demonstrating that alexithymia, not autism, predicts reduced empathic brain activity and impaired emotion recognition (Bird et al., 2010; Desai et al., 2019; Heaton et al., 2012; Mul, Stagg, Herbelin, & Aspell, 2018; Trevisan, Bowering, & Birmingham, 2016). Preliminary evidence also points to reduced allocation of gaze to eyes being explained by alexithymia in both autistic (Bird, Press, & Richardson, 2011) and neurotypical individuals (Fujiwara, 2018). Thus, examining effects of autism independent of alexithymia (as is typical practice for co-occurring depression and anxiety; Cuve et al., 2018; Black et al., 2018), may be crucial in resolving inconsistencies in the literature on autism and eye gaze.

2.1.1.2 Methodological features

2.1.1.2.1 Spatiotemporal dynamics

In addition to a failure to consider co-occurring alexithymia, there are at least three potentially significant methodological limitations of previous studies. First is the fact that previous studies did not explore the temporal evolution of gaze, instead averaging, or otherwise aggregating, eye gaze across time. This practice does not adequately describe gaze behaviour, which is a dynamic process serving to optimise eye movements for sequential selection of relevant visual information (Land, 1999). By reducing such a dynamic process into aggregated measures, studies may fail to capture group differences with respect to the temporal patterns of gaze allocation.

While it is possible that differences between groups in the timecourse of gaze allocation to facial expressions are captured by aggregate metrics, some divergence between aggregate and timecourse metrics is to be expected. Non-linearities in the distribution of gaze allocation are likely in response to more naturalistic and moving stimuli (Tatler et al., 2011). This is particularly true given the spatiotemporal dynamics of facial expressions, as the diagnosticity of eye and mouth cues for emotion may vary over time, as expressions unfold, and different action units may activate and peak at different (or multiple) timepoints, in a synchronous or asynchronous manner (Jack, Garrod, & Schyns, 2014; Krumhuber & Scherer, 2011). This means that gaze optimised for dynamic emotional facial expressions will likely vary in a non-linear fashion to facial regions over time.

A number of approaches using non-linear modelling (e.g., growth curves) have been proposed to describe the timecourse of gaze allocation to stimuli in other contexts (e.g., word processing in visual world paradigms), under the assumption that the properties of the task, stimuli and/or relevant individual differences (e.g., clinical diagnosis) influence the

underlying probability distribution of gaze to regions of interest, and that these distributions provide an estimate of cognitive processing (Barr, 2008; Mirman, Dixon, & Magnuson, 2008; Seedorff, 2018). Importantly, these data (e.g., gaze data in visual world studies) have highlighted that the underlying distribution of gaze is not always well captured in the aggregate ("average") pattern of gaze data collapsed across time (Barr, 2008; Mirman et al., 2008, Seedorff, 2018).

2.1.1.2.2 Eye-movement differences and data quality

Eye movements are typically parsed into fixations (to gaze targets) and saccades. Parsing is based on the characteristics of gaze behaviour in neurotypical individuals - not those with autism. Problematically, data suggest that some aspects of eye movement kinematics differ between autistic and neurotypical individuals (Schmitt, Cook, Sweeney, & Mosconi, 2014; Takarae, Minshew, Luna, & Sweeney, 2004). It is also the case that eye movement data from clinical groups are typically noisier, resulting in low precision and robustness (e.g., more missing datapoints) than those from neurotypical individuals (van Renswoud et al., 2018; Hessels et al., 2017; Wass et al., 2013). Individual differences in oculomotor behaviour and the quality of eye-tracking data may contribute to inconsistent parsing of eye movements into fixations and saccades, which may impact the validity of any group-level comparison between a neurotypical and a clinical group.

Even when eye movements are classified correctly, the gaze target must be identified. To do so, areas of the visual scene are determined to contain 'Areas of Interest' (AOIs, e.g., the eye-region of a face). Typically, the experimenter's judgement determines the size and shape of AOIs. This process is subjective and has been shown to be problematic when comparing autistic and neurotypical groups. Hessels and colleagues (Hessels, Kemner, van den Boomen, & Hooge, 2016) showed experimentally that the variability introduced by different AOI

production methods can greatly alter the pattern of gaze metrics to face stimuli for atypical groups (e.g., those with autism), more so than for neurotypical individuals. Consequently, variability in gaze parsing and AOI discretisation procedures makes published studies in this area difficult to compare and may partly explain the mixed findings.

2.1.1.3 Task demands and perceptual priors

It is important to situate any theory of gaze allocation to emotional facial expressions within wider theories of the function of gaze and visual perception. Within wider theories, the important role of top-down factors, particularly perceptual predictions, in modulating gaze behaviour is consistently recognised (e.g., Parr & Friston, 2017; Brook et al., 2019). According to these theories, eye-movements are optimised to reduce sensory uncertainty in order to (dis)confirm perceptual predictions about incoming visual information (Friston, Adams, Perrinet & Breakspear, 2012). Furthermore, beyond salience of the visual scene (a bottom-up factor; Itti & Koch, 2000), the top-down effects of task on gaze behaviour are thought to offer a plausible account of fixation selection (see, e.g., Yarbus, 1967; Tatler et al., 2011). Task demands and goals are thought to determine (and therefore to differentiate) the predictions one makes about upcoming perceptual input, which would be expected to result in different gaze patterns.

Consistent with this idea, gaze behaviour to faces is task-dependent, with task structure and instructions influencing eye gaze more strongly than stimulus-driven factors (Del Bianco, Mazzoni, Bentenuto, & Venuti, 2018; Hessels, Holleman, Kingstone, Hooge, & Kemner, 2019). There is also evidence from neurocomputational models of emotional face processing that humans evaluate the spatiotemporal dynamics of facial expressions with respect to established norms (Furl et al., 2020). In an emotion processing context, these norms can be

formalised as perceptual expectations or priors (Jack & Schyns, 2015) which may drive the allocation of gaze and attention to faces.

It is, therefore, notable that previous mixed findings regarding atypical eye gaze in autism were derived from studies using a variety of tasks. Participants have variously been asked to freely explore (Hadjikhani, Åsberg Johnels, et al., 2017), recognise (Kliemann, Dziobek, Hatri, Baudewig, & Heekeren, 2012) and judge the intensity (Tsang, 2018) of emotional expressions. Given the demonstrated dependence of gaze patterns on the task to be performed, thought to be mediated by task-dependent perceptual predictions, it is perhaps unsurprising that different patterns of results have been found across these studies.

The effect of top-down predictions is further complicated in autism with theories suggesting that autistic perception is less influenced by predictions than neurotypical perception (Pellicano & Burr, 2012; Sinha et al., 2014). Under this hypothesis, autistic participants may be expected not to modulate their gaze behaviour based on task requirements or priors about the stimuli, or not to do so to the same degree that neurotypicals do (Król & Król, 2019).

The influence of task- and emotion-relevant priors on gaze can be estimated using information theory (Shannon, 1948) to quantify the degree of 'entropy' or predictability of gaze behaviour (Mirza, Adams, Mathys, & Friston, 2018; Shiferaw, Downey, & Crewther, 2019). If a task generates strong perceptual predictions (for example, if participants know that they will be asked to judge the intensity of a disgust expression (primarily signalled by the mouth), or a fear expression (primarily signalled by the eyes), one expects a less random gaze pattern than if participants do not know which of these two stimuli they will be asked to judge. However, if autism (or alexithymia) is characterised by a reduced reliance on priors, these effects of task on the predictability of gaze behaviour should be reduced.

2.1.1.4 The present study and experimental hypotheses

The overarching goal of this study was to examine whether atypical eye gaze to emotional facial expressions is a product of alexithymia or autism. The primary hypothesis, therefore, is that:

H.1. Any atypical eye gaze to emotional facial expressions will be better explained by alexithymia than autism.

This study also aimed to address previous methodological issues in several ways. Perhaps most important was the consideration of the temporal dynamics of eye gaze. The evolution of eye gaze was described by assessing how well gaze behaviour was described by linear and non-linear orthogonal polynomial terms within Generalised and Linear Mixed Models – GLMMs (Mirman, Yee, Blumstein, & Magnuson, 2011; Seedorff, 2018). With respect to the evolution of eye gaze over time, it was hypothesised that:

H.2. Gaze behaviour to the eye region of emotional facial expressions will be best described by non-linear terms, representing behaviour in which the non-eye regions of the face are explored but where gaze frequently returns to the eyes over the viewing window.

Hypotheses 1 and 2, and previous research on the effects of task on gaze behaviour (Tatler, Hayhoe, Land, & Ballard, 2011; Hessels et al., 2019; Król & Król, 2019), lead to hypothesis 3, that:

H.3. The evolution of eye gaze behaviour will be modulated as a function of task and alexithymia and/or autism.

To account for individual differences in data quality and eye movement profiles (which may vary as a function of autism), gaze metrics were derived on a per-participant and per-trial

basis using a data-driven adaptive gaze parsing algorithm that accounts for individual differences in eye movements, and variance in data quality (van Renswoude et al., 2018). Thus if, compared to neurotypical participants, individuals with autism exhibit atypical or idiosyncratic eye movements such that, for example, gaze data shows low robustness (increased data loss) or high levels of noise (low precision), gaze events should still be accurately and reliably classified. Furthermore, AOIs were derived objectively, on a frame-by-frame basis, by adopting a recently validated automatic AOI specification method (Baltrušaitis & Robinson, 2016; Hessels, Benjamins, Cornelissen, & Hooge, 2018).

To investigate the role of task- and emotion-related priors on gaze behaviour, each participant's gaze was recorded when they were, and were not, cued to the upcoming emotion, and when they were asked to complete a range of tasks (*free-gaze, emotion recognition, and intensity judgements*). It can therefore be determined whether any autism- or alexithymia-related atypicality is dependent upon task parameters, or instead a general feature of gaze behaviour. Two measures of entropy (stationary gaze entropy and gaze transition entropy, Shiferaw et al., 2019) were used to index the complexity of gaze behaviour, as these metrics are thought to reflect the degree of goal-directed visual search prompted by each task (e.g., one might expect more focused, structured, gaze behaviour when one is cued to the upcoming emotional expression than when not). In relation to the use of priors, it was hypothesised that:

H.4. Gaze will be more predictable (as demonstrated by reduced entropy) in the emotion recognition and intensity judgement tasks, and when participants are cued to the upcoming emotion, compared to the free-gaze condition. Similar to H.3., any task effect may be reduced in autistic or alexithymic participants.

2.1.2 Methods

2.1.2.1 Participants

A total of 75 participants were recruited from a database of research volunteers and compensated with either an honorarium or course credit. Five participants (3 autistic, 2 neurotypical) were subsequently removed from the study due to a failure to complete the tasks (most commonly because they fell asleep or failed to calibrate properly). Data from 45 neurotypical individuals and 25 individuals diagnosed with autism were therefore included in the final sample (for details see Table 8.1 and Table 8.2. in Supplementary Materials – SM - Appendices). Autistic participants were diagnosed by specialist clinicians, all of whom were independent from the research team. Each individual's diagnosis was confirmed using their official diagnosis letter. To the best of our knowledge, participants were diagnosed using DSM-5 (or DSM-IV) criteria. As part of their inclusion in the participant database used for recruitment in this study, participants were assessed using the original ADOS algorithm administered by a certified professional. As is typical for samples of autistic individuals with a high mean IQ (as in the current sample - see **Table 2.1 Demographic** data) and who lead independent lives, not all participants met the cut-off score on the ADOS. Specifically, 7 participants had a total score below 5 on the ADOS, however, all 7 of these participants reached the social cut-off of 2, and all scored above the screening cut-off of 26 on the AQ-50 (with 5 of the 7 scoring above the more stringent criterion on this measure, > 32).

2.1.2.2 Procedure

Participants completed the questionnaires (see Questionnaires section bellow) online before the lab visit. On the day of the visit, participants were tested individually in a soundproofed, dimly-lit room, and took short breaks between tasks. After the eye-tracking session participants completed an IQ assessment, after which they were debriefed. Research was

conducted in accordance with the revised 2013 Declaration of Helsinki and was approved by the local research ethics committee.

Table 2.1 Demographic data

Variable	NT		ASD				
	n = 45		*n = 25*				
	M	*SD*	*M*	*SD*	*t*	min	max
TAS20	41.16	14.06	60.44	13.52	5.63^{***}	11	88
AQ28	11.33	4.83	27.24	16.25	4.78^{***}	2	72
DASS21	19.06	13.80	52.08	29.77	5.24^{***}	0	108
ADOS	-		6.92	3.73	-	2	15
IQ	117.76	13.52	121.68	16.49	1.02^{ns}	89	150
Age (years)	26.13	6.66	37.68	11.86	4.49^{***}	18	62
Gender							
Male	16		13		$\chi^2 = 4^{ns}$	-	-
Female	29		11			-	-

Notes. All t-tests were Bonferroni corrected. * $p < .05$; ** $p < .01$; *** $p < .001$; *ns* = non-significant; NT = neurotypical; ASD = Autism Spectrum Disorder; *M* = Mean; *SD* = Standard deviation. Note that these analyses do not control for alexithymia.

2.1.2.2.1 Dynamic Emotional Face Paradigm

The dynamic emotional face paradigm consisted of four conditions in which participants were shown videos of naturalistic dynamic emotional facial expressions from a validated dataset (Yitzhak et al., 2017). Stimuli were displayed in full-screen mode such that the faces occupied approximately 23° (degrees of visual angle) vertically and 10° horizontally at a

distance of approximately 60 cm. Videos lasted between 8 and 10 seconds, starting with a neutral face that changed to an emotional expression and then returned to neutral in the last second. A total of 192 trials were presented, 48 in each condition (*free-gaze*, *emotion recognition*, *cued*, and *intensity judgement*), eight of each emotion (neutral, happy, sad, angry, fear, disgust) displayed by four male and four female actors.

In the *free-gaze* condition, participants were instructed to explore the videos freely as they were presented. This condition is of independent interest, and also serves as a baseline to which the other conditions can be compared. The *emotion recognition* and *intensity judgement* conditions involved the participant being informed that they were to recognise the emotion, or judge the intensity of the emotion, respectively. The *cued* condition was the same as the *free-gaze* condition, but participants were informed as to which emotion would be portrayed before the face appeared. Condition order was fixed (*free-gaze*, *emotion recognition*, *cued* and *intensity judgement*), but trial order was pseudo-randomized per condition for each participant. A delay of 1000 ms $\pm$ 250 ms was used as a jittered inter-trial interval which was converted to the closest delay appropriate for the screen refresh rate (60Hz). This was supplemented by a short system delay of random length at the end of each trial sequence to allow for time-critical logging and trial preparation. This provides for more accurate temporal synchronisation within a trial, with more variable delays between trials. These two processes resulted in a total delay ranging between 1476ms to 1720ms from the end of a trial to the presentation of the next stimulus video. For the *emotion recognition* and *intensity judgement* conditions, 5 practice trials were included. The experiment was programmed and presented in PyGaze (Dalmaijer, Mathôt, & Van der Stigchel, 2014).

2.1.2.3 Eye-tracking

A Tobii TX300 (Tobii, Sweden) screen-based remote eye-tracker was used with the original screen, sampling at 300Hz and tracking an area of 1920 x 1080 pixels. Participants were positioned approximately 60 cm from the screen. No chin-rest was used during the study, instead participants were instructed to avoid sudden body and head movements, although they could freely move their eyes while watching the videos. A 5-point calibration was administered at the start of each block and repeated until all points were successfully calibrated according to the eye-tracker's default criteria. Validation of the calibration was conducted using the PyGaze routine, and via inspection of the plot of the deviation of gaze to each of the calibrated points. Calibration was accepted if all 5 points had been successfully calibrated, with accuracy < 1°. To account for the deterioration of calibration quality expected over time (Holmqvist et al., 2012; Hessels et al, 2017), recalibration was performed after every 20 trials, and additionally when deemed necessary. Recalibration after every 20 trials was based on a pilot experiment showing that participants could complete approximately 20 trials at a time while maintaining an optimal position, with minimal movement, and without triggering drift correction. Overall, calibration accuracy error was below 1° and precision below 0.3°, and not statistically significantly different as a function of group (see Additional data quality checks in SM).

2.1.2.3.1 Eye-tracking data processing

Pre-processing and analysis of the eye-tracking data were completed with custom and adapted processing pipelines in R (R-Core Team, 2013; Dink & Ferguson, 2016). The first 150ms of each trial were excluded from analysis to account for gaze reorientation during screen transitions and fixations initiated before stimulus onset, and the viewing window was locked to 7800ms of full data after re-zeroing. Exclusion of the first 150ms was based on visualisation of the gaze data (both raw and parsed) which indicated consistent rapid

reorientation of gaze to facial features after, but not before, this period. This pattern could not have been due to screen change, nor was it due to a fixation cross, since a fixation cross was not presented to limit the central bias induced when viewing stimuli on screens (Hart et al., 2009; Foulsham, Walker & Kingstone, 2011; Tatler, 2007).

Trials and participants with more than 35% of data loss were not analysed. On average, participants contributed 90% of valid data after correction for data loss, and there were no statistically significant group differences in data loss. For fixation analyses, gaze was parsed using a data-driven velocity-based algorithm that accounts for individual differences and quality fluctuations in gaze data (van Renswoude et al., 2018).

2.1.2.3.2 Derivation of AOIs

Objective AOIs were derived using an adapted implementation of a recently proposed Limited-Radius Voronoi-tessellation method (LRVT) that builds on open-source facial recognition and landmarking software (Baltrušaitis & Robinson, 2016; Hessels et al., 2018). The method allows identification of the location of facial features in a video over time. AOIs are then created by segmenting the faces using the distances to predefined points indicating distinct features (eyes, mouth, nose). Gaze points were assigned to the AOI that had the smallest Euclidean distance between gaze coordinates and the centre of the AOI, with an AOI radius of approximately 4°; and average eye-to-eye distance of approximately 2°. This method is noise-robust for sparse stimuli like faces and recommended for group comparisons of gaze metrics (Hessels et al., 2018).

2.1.2.4 Questionnaire measures

2.1.2.4.1 Toronto Alexithymia Scale – TAS-20

The TAS-20 was used to measure alexithymia and was scored according to the original norms, with each item rated on a scale from 1 (completely disagree) to 5 (completely agree) which were then summed for a total score (Parker, Taylor, & Bagby, 2003).

2.1.2.4.2 Autism Spectrum Quotient – AQ28 and AQ50

To quantify autistic traits, participants completed the AQ28 (Hoekstra et al., 2011). One total score was computed by scoring the 4-point Likert-scale according to the original AQ scoring criteria. The AQ28 was used to quantify autistic traits as recent work suggests it is more consistent with clinical judgment than the AQ50 (Ashwood et al., 2016), and that the AQ50 in its entirely likely contains redundancies that do not necessarily provide improved precision of measurement (Cuve et al., 2021*;* Lundqvist & Lindner, 2017, see also discussed **Chapter 5**). AQ50 scores were also available for the autistic participants, and these were used to establish whether autistic participants' scores were above established clinical thresholds.

2.1.2.4.3 Depression, Anxiety and Stress Scale – DASS21

To control for any effect of depression and anxiety, these traits were assessed using the DASS21 (Lovibond & Lovibond, 1995). Each item was scored on a Likert-scale from 0 to 3, and a total score was computed by summing all item scores and scaling according to standard scoring methods. DASS scores were used in control analyses.

All questionnaires demonstrated good internal reliability as measured by Cronbach's alpha (.85, .84, and .93, respectively).

2.1.2.5 Analysis strategy

The analysis workflow is described in **Figure 2.1.** Gaze position data were split into 78 100ms time bins and the proportion of gaze points within each AOI in each bin was calculated. By using all available gaze points, this approach eliminates the robustness problems with velocity thresholds of parsing algorithms which can affect gaze metrics. To account for the bounded nature of proportional data, these were transformed to empirical logit (elog) and a small epsilon value (.05) added to avoid undefined logarithms (i.e. when the proportion is exactly zero or one).

Data were analysed using GLMMs implemented in lme4 package (Bates, Mächler, Bolker, & Walker, 2014) in R. GLMMs were used rather than other linear models for several reasons. First, GLMMs are appropriate for autocorrelated data such as the timecourse of gaze allocation (Barr, 2008; Mirman et al., 2011). Second, GLMMs are more robust to missing data and unbalanced designs (Bates et al., 2014; Jaegar, 2008), which is typical in eye-tracking experiments.

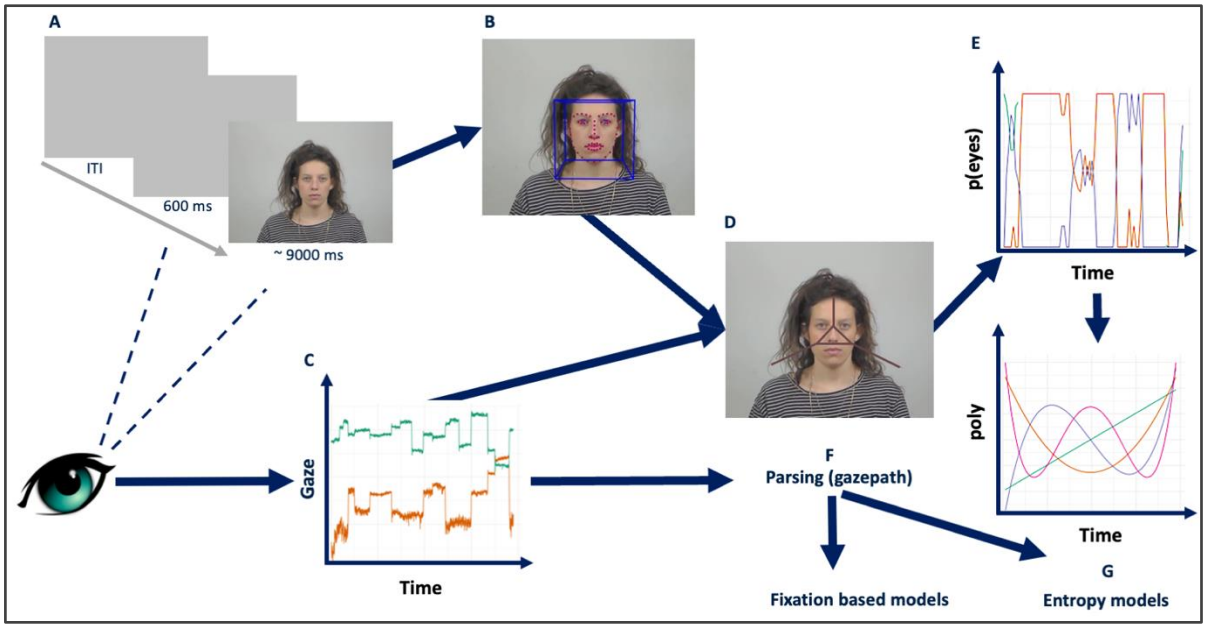

Figure 2.1. Study design and analysis workflow.

A. An example trial from the free-gaze condition. B. Sample stimuli with facial landmarks from OpenFace – facial landmarking software (Baltrusaitis et al et al., 2015). C. Sample time series of raw eye movements. D. Sample stimuli with superimposed AOI segmentation. E. Sample gaze data for a single trial for an example participant, showing the proportion (elog) of gaze points in the eye AOI vs. not eyes across time (top). Gaze points per AOI were then modelled using the orthogonal polynomials (bottom) in generalised linear mixed models (GLMMs). F. Additional standard analysis of fixation and saccades using data-driven parsing. G. Stationary and transition entropy calculation on the parsed gaze.

Third, participant and stimulus variance can be explicitly accounted for. For example, some participants may have a bias to look at the eyes more often than others, and some stimuli may attract eye gaze more strongly than others (Kanana, Bseiso, Ray, Hsiao, Cottrell, 2015). Finally, most of the assumptions about the distribution and nature of the residuals which affect the validity and sensitivity of general linear models are often violated in eye-tracking studies. GLMMs have the advantage of allowing for the non-normal distributions that are likely to be encountered with eye-tracking data (Jaegar, 2008; Seedorff, 2018).

2.1.2.5.1 Timecourse Analyses

For the timecourse analyses, a method to model the evolution of gaze allocation over time was required that could account for the expected non-linear timecourse of gaze allocation to eyes (Mirman et al., 2008; Seedorff, 2018). Accordingly, orthogonal polynomials, created as powers of the time variable, were used in the GLMM to model the evolution of gaze to AOIs over time (this method is also known as polynomial regression). As such an approach has not been used before to analyse autistic gaze behaviour, no firm predictions could be made about which polynomial terms should be included in the model. The simplest terms (linear and

quadratic) were therefore included to aid interpretability, and model selection (see below) was also used in order to determine the higher-order polynomial terms to be included before the increase in model complexity was not compensated for by increased predictive power (i.e., before evidence of overfitting was obtained).

Unlike traditional analyses which aggregate gaze behaviour across time, modelling gaze behaviour in this way allows four metrics to be derived. The first is the average behaviour across time (model intercept: for example the average proportion of gaze to the eye-region). The second is the way gaze behaviour changes across time (beta weights for each of the orthogonal polynomial terms: for example, if the beta weight for the linear term is significant, it suggests that gaze to eyes can be described as linearly increasing or decreasing over time; if beta weights for both the linear and quadratic terms are significant then it suggests that the change in gaze behaviour across time can be described as a combination of a linear and a U-shaped pattern. Higher order terms like cubic and quartic, can describe gaze distributions which oscillate more frequently (e.g., as a result of gaze shifts between AOIs), or asymmetries in the curvatures of lower-order shapes (e.g., the quadratic curve).

The third is average differences in gaze behaviour across time as a function of autism or alexithymia (beta weights for group main effects, autistic traits or alexithymia). The fourth is how predictors of interest, e.g., autism and alexithymia, alter the evolution of gaze behaviour across time (e.g., beta weights for interactions between group effects and the orthogonal polynomial terms).

2.1.2.5.2 Time clustering analysis

The polynomial timecourse analysis using GLMMs described above allows testing significant effects (e.g., significant effects of autism or alexithymia) on the global timecourse of eye

gaze but does not allow it to be determined precisely *when* those effects occur or their relative onset. For example, if a significant effect of autism is identified, one does not know whether this effect is largely found at the start of the stimulus video (potentially identifying an atypical orienting response), or whether the effect is sustained throughout the stimulus period. In order to identify when significant effects were observed, a non-parametric cluster-based bootstrapping analysis was performed (Maris & Oostenveld, 2007). Specifically, in step 1, the data in each time bin were analysed using GLMMs to identify significant effects at $p <$ .05. In step 2, when adjacent time bins were significant, their statistics were summed and they were considered a 'cluster', where a cluster reflects a continuous period of time in which significant effects (e.g., of autism or alexithymia) on gaze behaviour were observed. In step 3, the data were then bootstrapped (2000 iterations), such that predictor values (for example diagnostic status) are randomly reassigned and steps 1 and 2 repeated at each iteration, such that a probability distribution of summed cluster statistics can be created and used to determine the significance of clusters identified with the real data (Maris & Oostenveld, 2007). Such an analysis is appropriate as unlike traditional multiple comparison corrections which are unreasonably conservative for most time-series data, non-parametric clustering has been shown to provide a reasonable control for Type I errors while preserving power (Maris & Oostenveld, 2007), and it conserves the temporal effects inherent in the data. Note, however, that this analysis does not obviate the need for the polynomial timecourse GLMM analysis, as that does, and the time clustering analysis does not, allow the shape of gaze evolution to be determined.

2.1.2.5.3 Entropy

In addition to standard aggregated analyses across time, the timecourse and time clustering analyses outlined above are essential to address hypotheses 1-3, addressing whether alexithymia or autism is a better predictor of eye gaze (H1), whether the evolution of gaze to the eyes is non-linear (H2), and whether the evolution of eye gaze is affected by task and/or autism or alexithymia (H3). Hypothesis 4 is that gaze will be more predictable (as demonstrated by reduced entropy) in the emotion recognition and intensity judgement tasks, and when participants are cued to the upcoming emotion, compared to the free-gaze condition. In addition, we wanted to investigate whether any task effect may be reduced in autistic or alexithymic participants.

Entropy can be used as a measure of predictability as it reflects the amount of information necessary to describe patterns of gaze behaviour. The more random the pattern, the more information is needed to describe that pattern (i.e., entropy is increased). More predictable, structured, patterns can be described with less information (reduced entropy) and, therefore, more structured and predictable patterns of gaze behaviour in response to the emotion recognition and intensity judgement tasks, and when participants are cued to the upcoming emotion, should be reflected in reduced entropy. Specifically, two measures of entropy were computed: stationary gaze entropy (SGE) and gaze transition entropy (GTE). SGE quantifies the overall spatial dispersion of gaze fixations across the visual scene and, therefore, addresses the spatial predictability of gaze behaviour. GTE provides an estimate of the complexity of gaze transition patterns and, therefore, addresses the spatiotemporal predictability of gaze behaviour and is thought to reflect efficiency in visual scanning behaviour (Shiferaw et al., 2019). SGE was calculated using the formal application of the Shannon equation (Shannon, 1948) as follows:

$$H(x) = -\sum_{i=1}^{n} (p_i) log_2(p_i)$$

Where H is the entropy value of a fixation sequence *x* (e.g., trial), *i* indexes the individual AOI, *n* is the number of AOIs fixated in a sequence, and p_i is the proportion of fixations in each AOI.

To reflect the fact that longer fixations likely imply deeper processing of visual information, the duration of each fixation was used to compute the SGE (Hessels et al., 2019).

GTE was computed as follows:

$$H(x) = -\sum_{i=1}^{n} p_i \sum_{j=1}^{n} p(i|j) log_2 p(i|j)$$

Where *H* is the uncertainty of *x* when its prior state is known. Thus, the current fixation is best predicted by considering the previous fixation location; p_i is the stationary distribution and *p(i|j)* is the probability of transitioning from *i* to *j* AOI (Krejtz, Szmidt, Duchowski, & Krejtz, 2014; Shiferaw et al., 2019). SGE and GTE were normalised by the maximum entropy for all AOIs to improve interpretability.

2.1.2.5.4 Fitted models

The aggregated gaze, timecourse of gaze data, and gaze entropy were modelled using GLMMs. Several models were built in order to address the study hypotheses and model comparison used to evaluate these models. For example, if one wants to determine whether autism predicts eye gaze one can compare a model of eye gaze which includes all other factors but not autism, with a model including all factors plus autism. If the model including autism fits the data better than the model without (i.e., the addition of autism improves the

model fit and does not lead to overfitting), then one can conclude that autism contributes to the prediction of eye gaze and the model parameters can be interpreted. Similarly, if one wants to determine whether autism or alexithymia is a better predictor of eye gaze behaviour then one can compare the fit of a model including autism but not alexithymia, with the fit of a model including alexithymia but not autism (and further compare these models to those containing both autism and alexithymia, and those containing neither autism nor alexithymia).

Fitted models therefore ranged from simpler (e.g., models accounting for random effects only, predicting eye gaze using the orthogonal polynomials only, or orthogonal polynomials plus the effect of the experimental conditions) to more complex models (which included these predictors plus autism and/or alexithymia and interactions between predictors). These models could then be directly contrasted to test whether adding alexithymia or autism improved model fit over simpler models.

To summarise, three groups of statistical models for both the gaze timecourse and entropy were tested:

Group A. Separate models for Autism and Alexithymia:

A.1. Autism Diagnosis Models: Simple models (orthogonal polynomials: linear, quadratic, cubic and quartic for timecourse analyses; condition: free gaze, emotion recognition, intensity judgement, cued) compared to more complex models including autism diagnosis, condition, polynomials (for timecourse analyses) and their interactions.

A.2. Autistic Trait Models: As A.1 but where continuous scores from the AQ-28 - indexing autistic traits - replace the binary autism diagnosis coding.

A.3. Alexithymia models: As A.2 but where alexithymia scores from the TAS-20 replace scores on the AQ-28.

Group B. Joint Autism and Alexithymia Models:

Models in Group B included the crucial predictors of interest as in Group A, but alexithymia was included as a covariate in autism models (i.e., all models included autism and alexithymia). Including autism (diagnosis or traits) and alexithymia in the same model is of use as the validity of direct comparisons of models with different fixed effects (as fitted in Group A) is debated. While some fit indices such as AIC and BIC can be used to compare non-nested models fitted with Maximum likelihood (Akaike, 1985), model comparison techniques such as the Likelihood Ratio Test are not valid for non-nested models (Boker et al., 2009; Matuschek et al., 2017).

Group C. Control Models.

This group of models included important control variables that were not the focus of this study (e.g., depression and anxiety, IQ, age, gender) that may still influence gaze and emotion processing, as well a series of sensitivity analyses (e.g., separate analyses by group).

For all models, quasi-maximal random structures were initially fitted. For polynomial models, all models included participant and stimuli random effects on all time terms (see Participants in SM). Convergence issues were solved by simplifying the random structure. This allowed us to fit principled quasi-maximal models while avoiding the incremental computational and power cost of maximal models (Barr, Levy, Scheepers, & Tily, 2013; Bates, Kliegl, Vasishth, & Baayen, 2015). Poisson and binomial distributions were used to fit non-Gaussian count and binary data (e.g., fixation count), or data were log-transformed to meet normality assumptions and correct for exponential distributions (e.g., fixation duration)

and additional zero-truncated Poisson models were used to fit zero peaked (inflated) distributions (e.g., entropy).

2.1.2.5.5 Significance testing and model comparison

Model selection was performed by comparing the fitted model of interest, e.g., including alexithymia or autism, to nested models containing all terms, minus these fixed effect(s) of interest via the *anova* function in R. Alexithymia and autism (trait and diagnosis) were also assessed in the same model and the standardised estimates were used to compare which factor (e.g., alexithymia or autism) had the larger influence on gaze. An additional validation used the *step* function from the lmerTest package to fit a backwards elimination regression and identify eliminated terms. P-values for fitted models were derived using Satterthwaite's approximation (Kuznetsova, Brockhoff, & Christensen, 2017). As part of the model assessment, information criteria (AIC and BIC) were also used to contrast different models (e.g., alexithymia vs. autism models). The full set of comparisons are listed in Table 8.3 Model building and comparison in SM.

2.1.2.5.6 Control analyses

Control analyses ensured that data quality or effects of other covariates (e.g., autistic traits, age, IQ, anxiety, ADOS and DASS scores) did not change the results of the main analyses (due to space limitations these are presented in the SM – see Control and additional analyses).

2.1.3 Results

2.1.3.1 Aggregated fixation analyses

To enable comparison with previous literature, I present first the standard fixation measures that aggregate over the stimulus viewing window. Two metrics are reported, the number of fixations, and the total duration of fixations.

Gaze distribution to face regions followed expected patterns (see **Figure 2.2** A & B). Specifically, participants spent more time looking at the eyes than the mouth (Estimate = -0.86, SE = 0.18, $p < .001$), and nose (Estimate = -0.61, SE = 0.14, $p < .001$). Similarly, participants fixated the eye regions more times than both the mouth ($Z = -4.78$, $p < .001$) and nose ($Z = -3.53$, $p < .001$). These results are line with other research on face scanning and supported the focus on eye gaze in subsequent analyses. There was also a slight preference for the nose over the mouth, and interactions with condition; however, to simplify interpretation of results in relation to the research hypotheses, separate models for each region of interest were fitted (see below).

2.1.3.1.1 Fixation count to the eyes

Free-Gaze condition: effects of alexithymia and autism

Alexithymia outperformed the (null) random effects model ($\chi^2 = 5.98$, $\Delta AIC = 4$, $p < .01$) whereas the autism diagnosis ($\chi^2 = .64$, $\Delta AIC = 2$, $p = .42$), and autistic traits ($\chi^2 = .18$, $\Delta AIC = 2$, $p = .67$) models did not. Additionally, alexithymia emerged as the most predictive term in the joint models ($Z = -2.63$, $p < .01$), in comparison to autism diagnosis ($Z = -0.49$, ns), and autistic traits ($Z = 1.56$, ns). Increasing alexithymia was related to a reduced number of eye fixations during free-gaze (Estimate = -.17, SE = .07, $p = .01$).

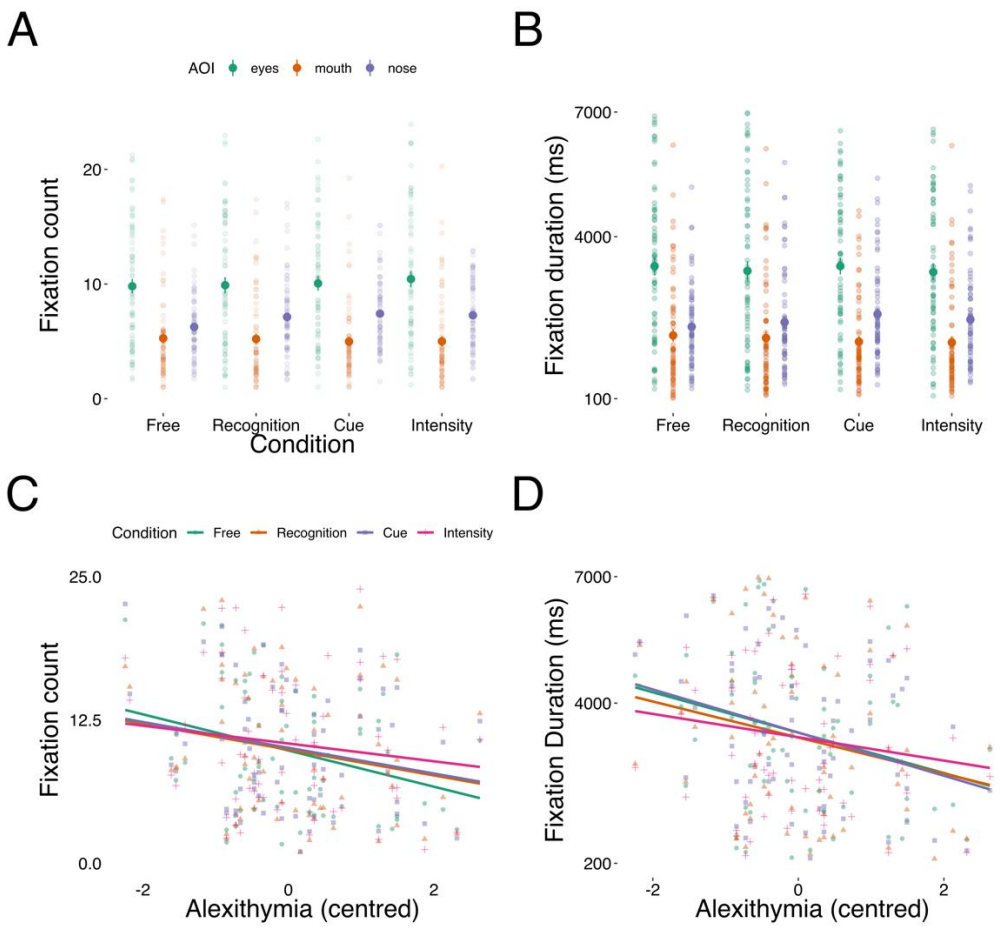

Figure 2.2. Aggregated gaze plots

Aggregated fixation count (A) and duration (B) for eyes, mouth and nose. Participants showed preferential looking to the eyes. Fixation count (C) and duration (D) as a function of alexithymia (condition indicated by line colour). Alexithymia predicted both reduced fixation count and reduced duration of eye gaze.

Condition effects relative to the free-gaze baseline: effects of alexithymia and autism

For the analysis including all conditions (**Figure 2.2**), the alexithymia model outperformed the condition model (χ^2 = 13.23, ΔAIC = 123, ΔBIC= 93, p < .001). The autism diagnosis model also provided a better fit to eye fixation data relative to the condition model (χ^2 = 125.48, ΔAIC = 112, p < .001) as did the autistic traits model (χ^2 =41.68, ΔAIC = 33, p <

.001). Once again, however, the fit indices and the jointly-estimated model indicated a larger influence of alexithymia (Z = -2.69, p < .01) compared to autism diagnosis (Z = -.29, ns) and autistic traits (Z = .054, ns). Inspection of the alexithymia model revealed a main effect of increasing alexithymia predicting a reduced number of eye fixations (Estimate = -.17, SE = .06, p = .009).

There was also a significant interaction between alexithymia and condition, characterized by a relative increase in the number of fixations to eyes in the *emotion recognition* (Estimate = .06, SE = .01, p< .001), *cued* (Estimate = .05, SE = .01, p< .001) and *intensity judgement* conditions (Estimate = .11, SE = .009, p < .001; see **Figure 2.2**) in relation to the *free-gaze* condition, as alexithymia increased.

Subsequent analyses showed that despite individuals higher in alexithymia demonstrating a reduced number of eye fixations in the free-gaze condition, they do not show a significant reduction in attention to the eyes in the *recognition* (Estimate = -.14, SE = .08, p = .06) *cued* (Estimate = .11, SE = .06, p = .07) and *intensity judgement* conditions (Estimate = -.08, p = .28). Thus, data from aggregate measures suggest that individuals with alexithymia adapt their gaze behaviour according to the task to be performed.

Overall, traditional analyses of fixation count support H1; that alexithymia is a better predictor of reduced eye gaze than autism.

2.1.3.1.2 Fixation duration

Free-gaze condition: effects of alexithymia and autism

Model selection analyses of fixation duration to the eyes in the *free-gaze* condition indicated no significant improvements in model fit for the alexithymia or autism models (χ^2 = .09, ΔAIC = -2, p = .8). Therefore, none of the models were interpreted further.

Condition effects relative to the free-gaze baseline: effects of alexithymia and autism.

When analysing all conditions, the alexithymia model outperformed the condition model (χ^2 = 78.433, ΔAIC = 70, p < .001). The autism diagnosis model also outperformed the condition model (χ^2 = 11.871, ΔAIC = 4, p < .02). Importantly the stepwise backwards elimination validation dropped all autism and autistic traits terms but not alexithymia, suggesting a stronger predictive effect of alexithymia and condition on fixation duration to eyes over the other predictors.

The alexithymia model indicated that increased alexithymia is associated with reduced fixation duration to eyes in the *cued* (Estimate = -.10, SE = .01, p < .001) and *intensity judgement* conditions (Estimate = -.09, SE = .01, p < .001), with a non-significant trend for reduced fixation duration to eyes in the *emotion recognition* condition (Estimate = -.3, SE = .01, p = .06). Similar results were obtained when analysing each condition separately.

Alexithymia models also performed better than autism diagnosis and traits models in predicting aggregated fixation metrics to the mouth and nose, yet models accounting for alexithymia and autism were typically penalised for complexity. This suggests that simpler models including experimental conditions only are sufficient to explain the mouth fixation data (see **'Aggregated fixation analyses – mouth AOI'** in SM for details).

Overall, analyses of fixation duration data, like fixation count data, support H1 – that alexithymia is a better predictor of eye gaze than autism.

2.1.3.2 Timecourse models for eye gaze

The timecourse models of eye gaze data use polynomial regressors locked to the stimulus viewing window to describe the changes in eye gaze proportion across time and how these patterns are impacted by alexithymia and autism (whether measured as the presence or

absence of an autism diagnosis or the degree of autistic traits) and conditions. Initially, an analysis of the free-gaze condition alone is presented, which is followed by an analysis of how the pattern of eye gaze changes when comparing each condition to the free-gaze condition. This latter analysis also describes how alexithymia and autism impact the changes in eye gaze patterns as a function of condition. Only critical terms are described in the text below.

Free-gaze condition: Effects of alexithymia and autism

Model comparison indicated that the alexithymia model outperformed the polynomials only (null) model (χ^2 = 77.38, ΔAIC = 67, $p < .001$), as did the autism (diagnosis) model (χ^2 = 14.947, ΔAIC = 4, $p = .01$) whereas the autistic traits model did not provide an improved fit over the null model (χ^2 = 10.157, ΔAIC = 0, $p = .07$). When comparing standardised estimates in a joint model, alexithymia consistently emerged as the most influential predictor (Std. Estimate = -.23, Std. SE = .07, p < .001) compared to autism diagnosis (Std. Estimate = .09, Std. SE = .08, ns) and autistic traits (Std. Estimate = .11, Std. SE = .07, ns). Similarly, the backwards stepwise validation analysis based on AIC eliminated both autism diagnosis and autistic traits and their interactions with polynomial terms, but not alexithymia and interactions with higher order polynomials.

Estimates of the alexithymia model suggested a significant intercept (Estimate = 2.72, SE = .15, $p < .001$) indicating that overall, participants were more likely to look at the eyes than not. There was a significant main effect of alexithymia (Estimate = -. 50, SE = .14, $p < .001$), indicating that alexithymia is associated with decreased gaze allocation to the eye region. Alexithymia interacted with higher order polynomials suggesting that temporal evolution of gaze behaviour differs as a function of alexithymia, with highly alexithymic individuals reaching near asymptote after reduced attention to the eyes early in the trial (see **Figure 2.3**).

Full details and mouth gaze models are presented in **8.1.3 Control and additional** analyses in **SM**.

Condition effects relative to the free-gaze baseline: Effects of alexithymia and autism

Timecourse data and model predictions are illustrated in **Figure 2.3**. According to all model fit criteria, the alexithymia model significantly outperformed the condition models (χ^2 = 2083.3, ΔAIC = 2044, $p < .001$). The autism model (diagnosis) also provided a better fit than condition models (χ^2 = 1146.7, ΔAIC = 1107, $p < .001$) as did the autistic traits model (χ^2 = 412.38, ΔAIC = 373, $p < .001$). However, when directly contrasted within the same model, standardised estimates for alexithymia effects were significant and consistently larger (Std. Estimate = -.28, Std. SE = .06, p < .001) than those for autistic traits (Std Estimate = .07, Std. SE = .07, ns) or autism diagnosis (Std. Estimate = .08, Std. SE = .08, ns).

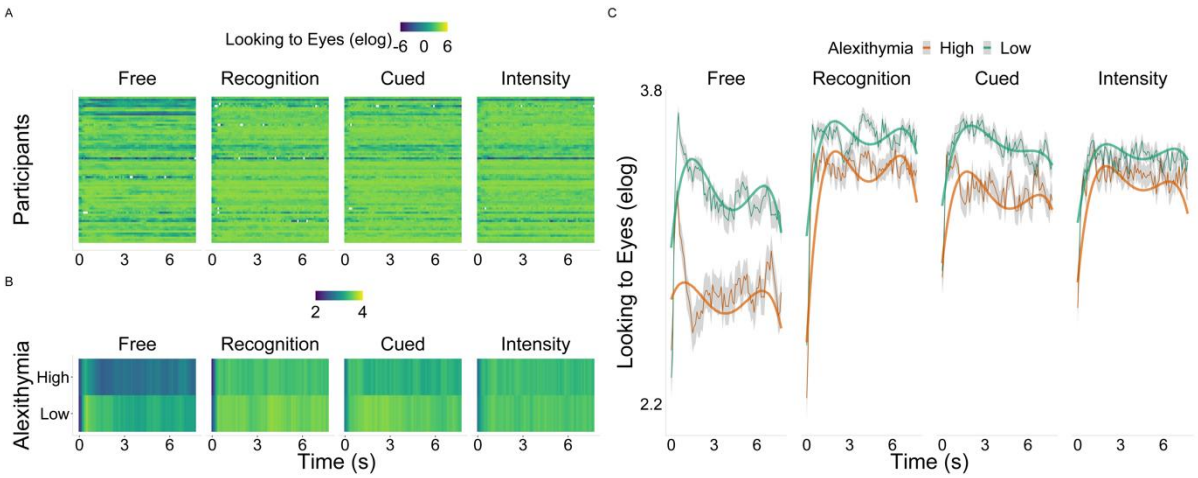

Figure 2.3 Timecourse analysis.

A – B. Raster plots showing reduced gaze to the eye with increasing alexithymia (A) and separated into groups with high and low alexithymic traits (for visualisation only; B); C. Timecourse model for gaze to the eye region. Shaded ribbons represent 95% Confidence Intervals. Solid lines represent predictions obtained from the multilevel polynomial models.

The alexithymia model outperforms both the autism diagnosis and autistic traits models in predicting non-linear eye gaze behaviour. The analyses above excluded the first 150 ms of gaze data; the results remained consistent when excluding the first 500 ms or 1000ms. Note that the dichotomisation between high and low alexithymia is for visualisation purposes only, as alexithymia was treated as a continuous predictor.

Similarly, the backwards stepwise selection validation analysis removed the majority of the autism terms and none of the alexithymia terms. These results suggest that a model including alexithymia is a better predictor of eye gaze than autism diagnosis and trait models.

Inspection of the best fitting model (the alexithymia model) revealed a significant interaction between alexithymia and condition for all higher-order time polynomials but not for the linear term, indicating that alexithymia is related to the condition-specific non-linear trajectory of gaze to the eyes across conditions. This effect suggests that alexithymia is associated with atypical dynamic gaze exploration of face regions, in particular the eye region. Notably, alexithymia was also related to reduced overall eye gaze across conditions (Estimate = -.48, SE = .10, $p < .001$).

A potential concern that can be visualised in **Figure 2.3** relates to the rapid increase in gaze to the eyes seen at the start of the viewing window. This increase, although an accurate description of participants' eye gaze, likely relates to the onset of the video stimulus and as such may be less representative of real-world viewing behaviour. In terms of analysis, this increase may unnecessarily require that data be fitted by higher order polynomials. Therefore, to guard against the possibility that high-order polynomial terms were improving fit artefactually, additional analyses were conducted excluding the first 500ms and the first

1000ms of gaze data. When the first 500ms of data were excluded it was still the case that models with higher order terms (up to the quartic order) provided significantly better fit to the probability distribution of eye gaze over time compared to just the linear term (χ^2 =974.12, ΔAIC = 954, $p <$ .001) or even the quadratic term (χ^2 = 387, ΔAIC = 375, $p <$ .001), and including alexithymia outperformed models with no clinical predictors (χ^2 = 1746005, AIC = 1746005, p< .001) and with autistic traits (ΔAIC = 1743948) and diagnosis (ΔAIC = 904). Thus, the reported effects remained consistent with those described above, such that alexithymia had significant and large effects on eye gaze over time compared to smaller and/or non-significant effects of autistic traits or diagnosis (see

Figure 8.3 Time course model of eye gaze without the first 500ms in SM). Excluding the first 1000ms resulted in similar results, with the only notable difference being that the best fitting model was achieved with up to the cubic polynomial, without the quartic term being required.

As a result, the analyses excluded only the first 150ms. Including as much early data as possible was thought to be important as hypotheses of atypical eye gaze in autism make predictions about gaze orientation and rapid eye avoidance, meaning that early visual attention is theoretically relevant to analysis of eye gaze in autism. While all individuals may show rapid orientation to the eyes, groups of individuals may differ at the rate at which they do so, or at the rate at which they move away from the eyes. A number of published studies in autism use early gaze data, including first fixation (Bours & Bakker, 2018; Kleberg et al., 2017; Kliemann et al., 2012; Madipakkam, Rothkirch, Dziobek, & Sterzer, 2017; Schauder et al., 2019), precisely to test atypical orientation and or eye avoidance. Here, the agreement between the three analyses excluding different amounts of early data provides a sensitivity test of the main findings and suggests that they are robust.

Thus, with respect to the study hypotheses, the timecourse analysis supported H1 in showing that alexithymia explained atypical eye gaze to emotional facial expressions better than autism (either the presence of a diagnosis or autistic traits). H2 was also supported, as the evolution of eye gaze over the stimulus viewing window was non-linear. H3 was also supported for alexithymia: the (non-linear) trends of eye gaze behaviour over time were modulated as a function of task, and this task modulation was moderated by alexithymia.

2.1.3.3 Non-parametric cluster-based analysis

Whereas the previous analyses allow the overall trajectory of gaze to the eyes to be described across time, they do not allow identification of the particular time period(s) over the course of a trial when effects of alexithymia or autism can be seen. To that end, nonparametric time-based cluster analyses were performed on every condition while regressing eye gaze proportions on each bin onto autism, alexithymia or autistic traits separately, in order to uncover any time-specific effects of each factor (see **Figure 2.4**).

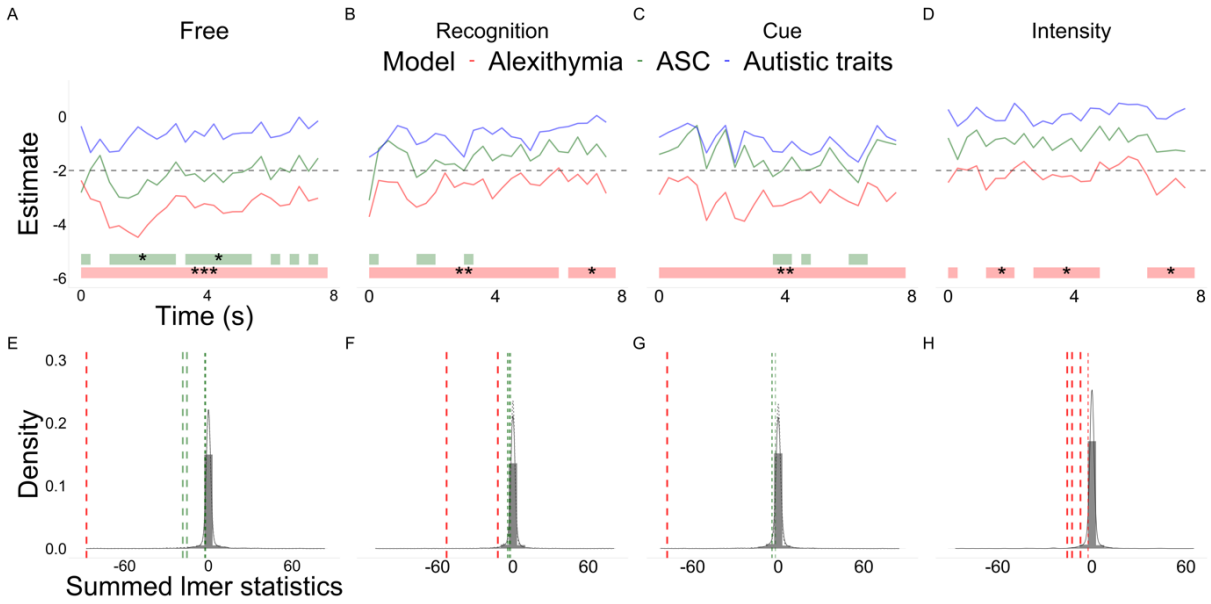

Figure 2.4. Cluster analysis of timecourse effects.

A-D. Shaded areas indicate clusters. If significant, clusters are marked: *** p < .001, ** p < .01, * p < .05. Y axis represent the size of the effect of the standardized predictors on eye gaze. Dashed y intercepts represent lower critical significance limits for estimates from Linear Mixed Models. Alexithymia consistently predicted significant reduced eye-looking behaviour across time and conditions (red shades); E-H. Density plots from the nonparametric cluster bootstrapping. X axis indicate summed estimates for all the time bins in clusters and x intercepts indicate individual clusters coloured by alexithymia (red) and autism diagnosis (green), autistic traits (blue).

In the *free-gaze* condition, there was one cluster spanning the entire stimulus viewing window which was explained by alexithymia. Bootstrapping indicated that this cluster was significant ($Cluster_{0\text{-}7.8s}$ Sum Statistic = -87.38, $p < .001$). In contrast, the autism diagnosis model suggested six smaller clusters, of which only two were significant ($Cluster_{0.9\text{-}3s}$ Sum Statistic = -18.39, $p = .03$, $Cluster_{3.3\text{-}5.4s}$ Sum statistic = -15.42, $p = .03$). There were no significant clusters for the autistic traits model.

For the *emotion recognition* condition, the alexithymia model accounted for two significant clusters occupying nearly all of the viewing window ($Cluster_{0\text{-}6s}$ Sum Statistic = -52.82, $p = .001$; $Cluster_{6.3\text{-}7.8s}$ Sum Statistic = -12.03, $p = .03$), demonstrating that alexithymia predicts reduced eye gaze across the duration of the trial. Three small clusters in the first half of the timecourse were attributed to the autism diagnosis model, none of which reached significance when assessed with the bootstrapping analysis ($Cluster_{0\text{-}0.3s}$ Sum Statistic = -3.11, $p = .12$; $Cluster_{1.5\text{-}2.1s}$ Sum Statistic = -4.28, $p = .10$; $Cluster_{3\text{-}3.3s}$ Sum Statistic = -2.00, $p = .23$). No significant time clusters were observed for the autistic traits model.

In the *cued* condition, alexithymia also predicted reduced eye gaze across the timecourse (Cluster $_{1\text{-}7.8s}$ Sum Statistic = -77.85, p = .002). For the autism diagnosis model, three clusters suggested reduced eye gaze in autism, yet none were significant when assessed with the bootstrapping analysis (Cluster$_{3.6\text{-}4.2s}$ Sum Statistic = -4.2, p = .11; Cluster$_{4.5\text{-}4.8s}$ Sum Statistic = -1.997, p = .22; Cluster$_{6\text{-}6.6s}$ Sum Statistic = -4.5, p = .09). There were no significant clusters for the autistic traits model.

For the *intensity judgement* condition, there were four clusters for the alexithymia model, of which the three largest were significant (Cluster$_{1.2\text{-}2.1s}$ Sum Statistic = -7.31, p = .046; Cluster$_{2.7\text{-}4.8s}$ Sum Statistic = -15.75, p = .02; Cluster$_{6.2\text{-}7.8s}$ Sum Statistic = -12.66, p = .03), with a non-significant early cluster (Cluster$_{0\text{-}0.3s}$ Sum Statistic = -2.45, p = .11). There were no significant clusters for the autism diagnosis or autistic traits models.

Overall, these analyses highlight that alexithymia is related to consistently reduced gaze to the eyes across conditions and over time (see **Figure 2.4**), and therefore support H1; that alexithymia explains reduced eye gaze better than autism. Timecourse models predicting gaze to the mouth also favoured alexithymia models over autism diagnosis and autistic traits models. However, simpler models including only the effects of condition and the polynomials were favoured in model selection for the mouth (see **8.1.3.1 Timecourse for the mouth AOI** in SM).

2.1.3.4 Stationary (SGE) and transition (GTE) gaze entropy

Hypothesis 4 stated that gaze will be more predictable (as demonstrated by reduced entropy) in the emotion recognition and intensity judgement tasks, and when participants are cued to the upcoming emotion, compared to the free-gaze condition. Furthermore, in line with the Bayesian hypothesis of autism (Pellicano & Burr, 2012), any task effect may be reduced in autistic (or alexithymic) participants. Accordingly, entropy analyses were used to examine

the degree of structure/randomness in the pattern of fixations (SGE) and gaze transitions (GTE), and how the degree of structure was affected by autism, alexithymia, and the experimental conditions. Manipulation checks and additional details for the entropy analyses are provided in the **Control and additional** analyses section of the SM.

2.1.3.5 SGE

Free-gaze condition: effects of alexithymia and autism

There were no significant effects of any regressor on SGE in the *free-gaze condition*, and no improvement in model fit by the inclusion of alexithymia or autism. None of the models were therefore interpreted further.

Condition Effects Relative to the Free-gaze Baseline: Effects of Alexithymia and Autism

When analysing condition effects, the alexithymia model outperformed the condition model (χ^2 = 33.75, DAIC = 25.7, p < .001) whereas the autistic traits model (χ^2 = 5.3211, DAIC = 2.7, ns), and the autism diagnosis model (χ^2 = 4.66, DAIC = 3.34, ns) did not. When estimated in a joint model, the backwards stepwise selection validation analysis did not suggest any eliminations, yet this maximal model did not provide any significant improvement in explained variance compared to the alexithymia model, and the fit indices suggested overfitting. The alexithymia model indicated a significant effect of condition. Participants showed less dispersed fixation patterns during the *emotion recognition* (Estimate = -.007, SE = .003, p < .05), *cued* (Estimate = -.01, SE = .003, p < .001), and *intensity judgment* conditions (Estimate = -.04, SE = .003, p < .05). These effects are consistent with top-down priors acting to guide gaze fixations in order to complete the task or confirm the cued emotion. There was also a significant interaction between alexithymia and condition: higher levels of alexithymia predicted increased fixation dispersion during the *cued condition* (Estimate = .01, SE = .003, p < .001; see **Figure 2.5**), suggesting weaker modulation of gaze

by top-down priors relating to cued emotional expressions in alexithymia. No other alexithymia by condition interactions were significant.

The SGE analysis – providing an index of the predictability of fixations to dynamic face stimuli – supports Hypothesis 4 concerning alexithymia. Gaze was more predictable in the emotion recognition and intensity judgement tasks, and when participants are cued to the upcoming emotion, compared to the free-gaze condition. Furthermore, the effect of task was reduced in alexithymic participants.

2.1.3.6 GTE

Free-gaze condition: effects of alexithymia and autism

The GTE analysis provides an index of the predictability of gaze transitions (as opposed to the spatial predictability of gaze fixations measured by the SGE analysis). As with the SGE analysis, however, condition, alexithymia and autism models did not improve model fit in predicting the complexity of gaze transitions in the *free-gaze* condition. Therefore, these models were not further interpreted.

Condition effects relative to the free-gaze baseline: effects of alexithymia and autism

When analysing condition effects, the alexithymia model showed a significant improvement in fit compared to the condition model (χ^2 = 39.0, DAIC = 31, p < .001). The autism diagnosis model also outperformed the condition model (χ^2 = 29.43, DAIC = 21.5, p < .001), whereas the autistic traits model did not (χ^2 = 2.6, DAIC =5.3, p = .6). A joint estimation of autism diagnosis, autistic traits, and alexithymia effects on eye gaze, did not provide any significant improvement over the alexithymia model and tended to overfit the data according to the BIC (χ^2 = 23.23, DBIC = 51, p < .001), showing that the alexithymia model was sufficient to predict the complexity of fixation transitions (see **Figure 2.5**).

The alexithymia model revealed a significant reduction in GTE in the *emotion recognition* condition (Estimate = -.03, SE = .005, p<.001), and a significant effect of alexithymia in the *emotion recognition* (Estimate = .01, SE = .005, p = .01) *cued* (Estimate = .03, SE = .005, p < .001) and *intensity judgment* conditions (Estimate = .01, SE = .005, p = .03); whereby increasing alexithymia was related to reduced predictability (increased entropy) of gaze transitions in these conditions.

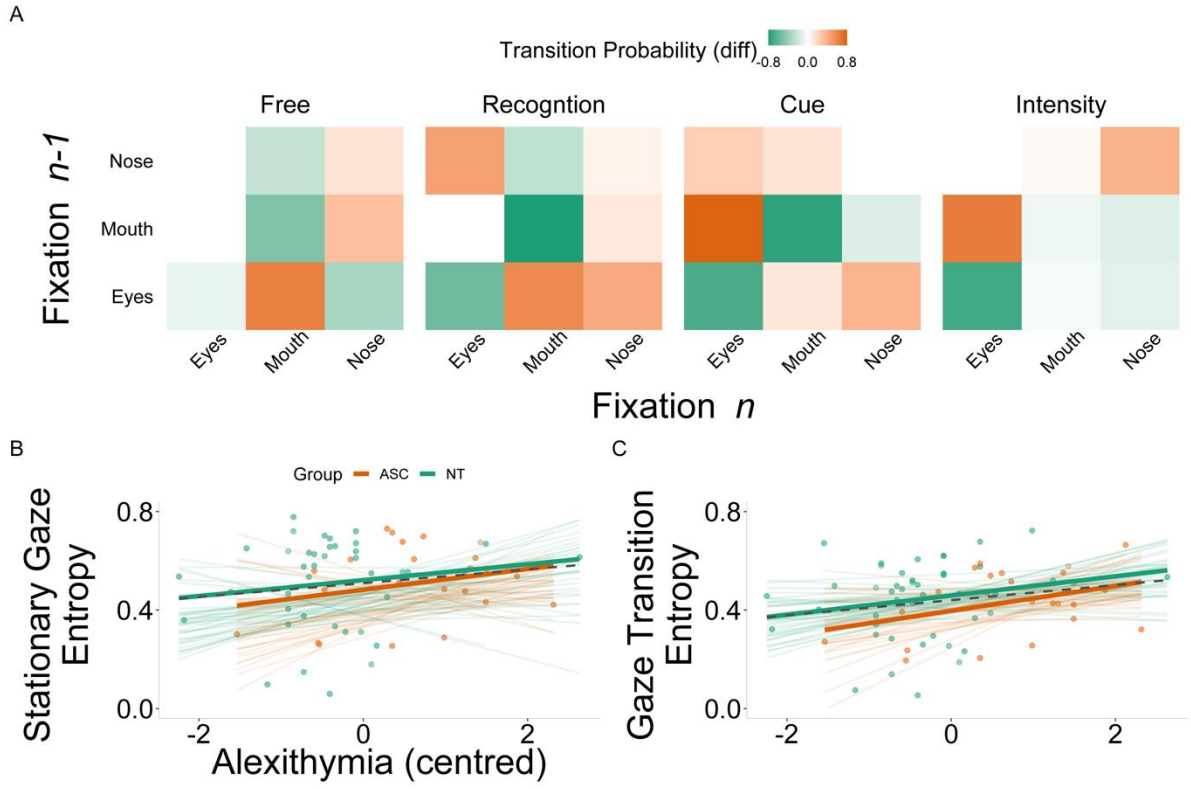

Figure 2.5 Entropy effects.

A. Transition matrices showing the difference in conditional transition probabilities between AOIs (eyes, mouth, nose) as a function of alexithymia (contrasted as high vs. low; median split for visualisation). Positive values indicate increased transition probability for highly alexithymic individuals whereas negative values represent decreased probability of transitions for highly alexithymic individuals. B-C. Scatter plots for linear mixed models showing that increasing alexithymia is associated with increased entropy in both autistic and

non-autistic individuals. Dashed line represents global trend line and faded lines illustrate random variability across trials.

Thus, consistent with the entropy analysis of the dispersion of gaze fixations (SGE), the predictability of gaze transitions as a consequence of condition reveals less of an influence of top-down priors relating to task, or knowledge of the upcoming emotional face stimulus, in alexithymic individuals. Thus, both the GTE and SGE data support Hypothesis 4.

2.1.3.7 Emotion recognition and gaze

Both autism diagnosis and alexithymia predicted emotion recognition accuracy. Specifically, neurotypical individuals were more accurate than autistic individuals ($Z = -3.79$, $p < .001$), and increasing alexithymic traits predicted poor emotion recognition ($Z = -2.69$, $p = .007$); a similar effect was observed for autistic traits ($Z = -3.24$, $p = .001$), however, in model comparison all of these modes had comparable fit and more complex models tended to overfit.

There was however no effect of looking time to eyes on emotion recognition accuracy ($Z = .80$, $p = .4$) nor interactions between looking time and clinical predictors on this measure (all $p > .05$), which was consistent when replacing eyes with nose and mouth looking measures. Similarly, correlations between gaze measures, accuracy and intensity ratings were all below .1 and non-significant (see Table 8.2 Zero-order correlations for aggregated data in SM). There were also no effects of SGE ($Z = .12$, $p = .9$) or GTE on accuracy ($Z = 1.3$, $p = .19$) nor interactions with clinical predictors (all $p > .05$).

2.1.4 Discussion

This study investigated whether autism or alexithymia was the better predictor of eye gaze while individuals with and without autism and alexithymia completed four tasks with dynamic emotional facial expressions. Previous studies addressing eye gaze in autism have typically aggregated gaze behaviour across time, analysing the number of fixations on, and the total duration of attention to, the eyes (Grynszpan & Nadel, 2014; Hadjikhani, Åsberg Johnels, et al., 2017; Kliemann et al., 2012). Using these standard analyses, the current data indicated that alexithymia was a better predictor than autism of reduced eye gaze to dynamic emotional facial expressions. Furthermore, alexithymia was a better predictor of how these aggregated metrics of overt attention to the eyes changed as a function of condition.

These results are consistent with the only previous study of the impact of alexithymia on attention to the eyes in autism (Bird, Press, & Richardson, 2011) and recent similar observations in non-clinical participants (Fujiwara, 2018; Wiebe, Kersting, & Suslow, 2017) and autism (Clin, Maes, Stercq, & Kissine, 2020). These results go significantly beyond these earlier studies however, as previous studies sometimes consisted of small sample sizes (Bird et al., 2011), used hand-drawn AOIs to define gaze to the eyes or used static stimuli, and used standard algorithms developed for neurotypical individuals to parse eye gaze data from autistic individuals into fixations and saccades. These methodological features are limiting for the reasons outlined in the Introduction.

Moving beyond traditional gaze metrics, separate analyses incorporated the temporal domain by modelling eye gaze behaviour over time. Three hypotheses were tested using these analyses. H1 stated that alexithymia would be a better predictor of eye gaze than

autism. H2 predicted that gaze behaviour to the eye region of emotional facial expressions would evolve in a non-linear fashion over the stimulus viewing window. H3 predicted that the evolution of eye gaze behaviour will be modulated as a function of task and alexithymia and/or autism.

Results supported the "alexithymia hypothesis" - demonstrating that alexithymia provides the best explanation of gaze behaviour to the eyes over time, influencing the non-linear changes in eye gaze in both the neurotypical and autistic group, and modulating the effect of task on the evolution of gaze behaviour. The temporal cluster-based analyses were consistent with this, indicating significant effects of alexithymia that persisted across the viewing window across conditions, with very few significant periods in which effects of autism were observed. The persistence of the alexithymia effects mean they are unlikely to reflect atypical reflexive responses or orientation tendencies, and instead likely reflect atypicality in the goal-directed visual exploration of emotional faces in alexithymia.

The present study manipulated the task required of participants when presented with the emotional faces. Participants were either free to gaze at the stimuli as they wished when they were unaware of the upcoming emotion, or when they were cued as to the emotion they would see; or they were asked to recognise the emotion or judge the intensity of the emotion. Such manipulations were motivated by previous findings of task-dependant gaze behaviour (Del Bianco et al., 2018; Hessels et al., 2019; Ricciardelli, Carcagno, Vallar, & Bricolo, 2013) and theories suggesting that autistic perception is less affected by priors than neurotypical perception (Pellicano & Burr, 2012). One might therefore have expected individuals with autism to modulate their gaze patterns less when cued to the upcoming emotion, or when required to complete a task, compared to a free-gaze situation. In fact,

alexithymia was a better predictor than autism of modulation of attention to the eyes by task.

The effect of perceptual priors on gaze was addressed by the fourth hypothesis which stated that gaze will be more predictable (as demonstrated by reduced entropy) in the emotion recognition and intensity judgement tasks, and when participants are cued to the upcoming emotion, compared to the free-gaze condition. Hypothesis 4 also stated that any condition effect may be reduced in autistic or alexithymic participants. Results of the entropy analyses supported Hypothesis 4 in showing reduced entropy (reduced randomness/increased predictability) in the spatial dispersion of gaze fixations and in gaze transitions in all conditions compared to the free-gaze condition. Furthermore, the effect of the task on entropy was reduced in alexithymia such that individuals with alexithymia showed an increase in entropy (more dispersion and randomness in gaze) during cued conditions. This latter finding is not necessarily inconsistent with other studies showing increased randomness in face scanning in autistic children (Shic, Chawarska, & Bradshaw, 2008; Wang et al., 2020), increased exploratory gaze by autistic individuals (Vettori et al., 2020), and a reduced effect of priors on recurrence of fixations (Król & Król, 2019), as these studies did not account for alexithymia or used non-emotional static visual stimuli.

It is worth considering why alexithymia would lead to a reduced effect of condition on gaze entropy, given that the primary prediction relating to condition was an effect of autism, due to theories suggesting that autistic perception is less influenced by predictions than neurotypical perception (Pellicano & Burr, 2012; Sinha et al., 2014). One explanation for the effect of alexithymia on the use of priors is related to imprecise emotion concepts in alexithymia. Previous studies have shown high-level confusion between emotion categories in alexithymia (Brewer, Cook, & Bird, 2016; Cook, Brewer, Shah, & Bird, 2013). Such

confusion may mean that alexithymic individuals struggle to individuate emotion categories, which in the context of visual exploration of emotional facial expressions may be reflected in a reduced ability to modulate gaze in response to task demands or to show confirmatory gaze behaviour reflecting predictions about upcoming emotional stimuli.

Interestingly, the effect of alexithymia on eye gaze was specific; effects of alexithymia and/or autism on gaze to other face regions were not robust or non-significant. As such, these results reinforce earlier claims of the importance of reduced eye gaze in the autistic population (Kliemann et al., 2010; Stephenson, Luke, & South, 2019), but suggest that the reduced eye gaze to emotional stimuli may be due to co-occurring alexithymia (Bird et al., 2011; Brewer, Cook, Cardi, Treasure, & Bird, 2015). These results might also be indicative of distinct factors underlying modulation of attention to eyes and mouth. Whereas higher-order emotion processing affects eye gaze, it may be the case that attention to mouth regions is largely dependent on functional requirements of the task, e.g., decoding speech.

The current results can inform future research in four ways. First, because the heterogeneity in emotion processing and social attention in autism may be driven by alexithymia, assessment of alexithymia in studies of socioemotional processing and social attention in autism is essential. Second, models of social attention in autism need to incorporate potential causal influences of alexithymia on socioemotional behaviour. Third, looking time measures are likely to underestimate group variability, and for dynamically changing behaviour such as gaze to dynamic expressions, aggregated metrics may not reflect the underlying distribution of visual exploration over time depending on the task (Barr, 2008; Mirman et al., 2008). Describing the temporal dynamics of gaze also offers practical and conceptual advantages over traditional eye-tracking research in autism. For example, data can be analysed without being unreasonably penalised for the multiple comparisons

incurred in the use of multiple eye-tracking metrics (entry times, fixation duration, dwell), the interpretation of which is often not clear when related to eye gaze hypotheses in autism (Cuve et al., 2018; Hessels, 2020). Fourth, the current results demonstrate that participants modulate their attention to the eyes as a function of the task they were performing. Free-gaze conditions may therefore be more likely to reveal reduced eye attention in the autistic population, as a result of co-occurring alexithymia, than active tasks such as emotion recognition. Conversely, experimental conditions manipulating the effect of priors on gaze may be more sensitive to illustrate atypical modulation of eye gaze which may not be apparent in looking time measures (e.g., when using entropy to measure the complexity of gaze patterns).

A limitation of the current study is that it was not optimised to measure the relationship between eye gaze and the accuracy of emotion recognition. Overall, autism and alexithymia were reasonably comparable in how well they predicted emotion recognition accuracy, yet when controlling for alexithymia neither autism diagnosis nor autistic traits had a significant effect on accuracy. Nonetheless, the emotion recognition task was only one of four conditions in the experiment, and only a limited amount of data could be recorded per person in this condition. It is certainly the case that not enough data were recorded to examine how the relationship between attention to the eyes and emotion recognition accuracy might vary depending upon the emotion portrayed. There is, however, consistent evidence from previous studies using carefully matched samples and sensitive emotion recognition tasks that alexithymia is the driver of atypical emotion recognition in both autistic (Bird et al., 2011; Cook et al., 2013; Ola & Gullon-Scott, 2020) and non-clinical volunteers, including correlations with atypical eye gaze (Fujiwara, 2018) and diminished sensitivity to emotion intensity (Starita, Borhani, Bertini, & Scarpazza, 2018). Subsequent studies should therefore

explore how autism and alexithymia may impact the relationship between spatiotemporal dynamics of eye gaze and emotion recognition abilities.

Finally, it also needs to be acknowledged that the group of autistic individuals who volunteered their participation in this study were not representative of the autistic population as a whole. They were a group of autistic adults with a very high mean IQ, and relatively low levels of overt symptoms (at least when assessed with the ADOS). This experiment therefore needs replicating with a more representative sample of autistic adults and children.

2.1.5 Conclusion

Across different analytical approaches, this study demonstrated that spatiotemporal differences in eye gaze patterns to emotional expressions are best explained by alexithymia rather than autism diagnosis or autistic traits. The findings highlight potential underlying visual processing mechanisms of atypical emotion processing in alexithymia and autism. They echo growing calls for the need to account for the effects of alexithymia when studying emotion processing. They may also go some way towards explaining previous inconsistencies in the literature and may offer a useful model for studying the gaze patterns of atypical groups.

Supplemental materials: see 8.1 Chapter 2. Supplementary Materials

Data availability: Code and data available at https://osf.io/nxdkj/.

2.2 What comes next?

Chapter 2 provided the first test of the alexithymia hypothesis and Bayesian accounts of autism on gaze modulation to facial expressions, using novel methodological approaches to characterise the dynamics of gaze allocation to faces. In Chapter 3, I expand on this work to provide further tests of the alexithymia hypothesis and Bayesian account of social attention and extend the current approach to the study of spatiotemporal dynamics of facial expressions and their effects on social attention.

3 CHAPTER 3. CONTRIBUTION OF FACE DYNAMICS AND PRIORS TO SOCIAL ATTENTION AND EMOTION PROCESSING

3.1 REPRESENTATION OF EMOTION IN LINEAR AND NON-LINEAR DYNAMICS

Abstract

Faces are one of the main stimuli used to investigate emotion recognition and social attention. Although research has traditionally utilised static photographs of emotional expressions, there has recently been a shift towards the use of dynamic stimuli. The use of dynamic stimuli introduces complexities in stimulus control, however, relating to the fact that face stimuli can vary greatly in their dynamic properties (speed, displacement patterns). Although frequently not analysed, the impact of this dynamic variability on emotion processing is the focus of this paper. In Experiment 2, stimuli analyses show differences in emotion-related spatiotemporal information extracted from posed facial expressions typically used in emotion research, as well as the influence of morphing on face dynamics when used to generate stimuli. Experiment 3 reveals that the varying linear and non-linear dynamics in face stimuli influence judgements of how intense and natural those stimuli are, while having less impact on valence judgements. Analyses of recognition accuracy suggest that judgements of facial expressions are optimised according to the expected spatiotemporal profile of the emotion. These results highlight the need for greater consideration of the impact of the dynamics of face stimuli on emotion processing, including the implications of animating faces with techniques that affect emotion-specific non-linear dynamics. This study also provides a pipeline that can be used to 1) analyse face stimulus dynamics and select stimuli in a reproducible and objective manner, and 2) derive statistics describing dynamics that can be used for further analysis.

Keywords: face dynamics, emotion, faces, prototypicality, morphing, quality.

3.1.1 Introduction

Face stimuli are commonly used within emotion research for investigating the ways in which humans signal and decode affective information during face-to-face communication (Jack & Schyns, 2015). Notably, early theories hypothesising the existence of universal configurations of emotional facial expressions (Paul Ekman & Friesen, 1986) have largely dictated the choice of face stimuli. Accordingly, most research has used static photographs depicting prototypical facial expression configurations as specified by the Facial Affect Coding System (FACS), based on Ekman and Friesen's work (Paul Ekman & Friesen, 1986).

It has recently been argued, however, that in order to study emotion perception as it occurs in real-life social interactions, dynamic stimuli depicting facial expressions of emotion should be used instead of static photographs. It is argued that the motion in facial expressions provides an additional source of information useful for emotion recognition, leading to a 'dynamic advantage', where emotion recognition is improved for dynamic compared to static stimuli (Arsalidou, Morris, & Taylor, 2011; Liu, Chen, Ward, & Takahashi, 2016; O'Toole et al., 2011). However, the degree of dynamic advantage observed is modulated by characteristics of the particular stimuli employed and how they vary in prototypicality, intensity, and naturalness (Dobs, Bülthoff, & Schultz, 2018; Fiorentini & Viviani, 2011), and whether the expression was performed spontaneously, or as a deliberate communicative act (Barret et al., 2019; Motley & Camden, 1988).

Findings that the size of the dynamic advantage varies as a function of stimulus characteristics highlight that dynamic stimuli are highly variable (Krumhuber, Skora, Küster, & Fou, 2017). Perhaps most well-known are the motion and morphological differences observed between posed and spontaneous facial expressions (Namba, Makihara, Kabir, Miyatani, & Nakao, 2017). But even within posed expressions, the most common expression

type employed in psychological studies, the intended communicative goal of the expresser as well as the type of expression elicitation method (e.g., uninstructed vs. FACS template) affects the spatiotemporal dynamics of the expression. This in turn changes how prototypical and intense expressions appear (Krumhuber, Kappas, & Manstead, 2013; Yitzhak, Gilaie-Dotan, & Aviezer, 2018), and how well they are classified by both human and machine classifiers (Yitzhak et al., 2018; Krumhuber et al., 2020).

How these dynamic properties of facial expressions affect inferences about emotion has only been explored directly in a few studies. For example, Cosker et al. (2010) and Kolkorova et al. (2018) showed that observers preferred, and were better at recognising, non-linear expressions over animations depicting artificial linear expressions, and rated them as more natural. Dobs et al. (2014) extended this finding by demonstrating a complex relationship between the different types of linear and non-linear approximations of natural face motion and similarity judgements, which varied as a function of the emotion category. In addition, Jack et al. (2014) identified random patterns of face action synthesised from a few spatiotemporal parameters (including latency, speed, and shape of action unit activation) that reliably predicted observers' representations of emotional expressions. Finally, Sowden et al. (2020) showed causal links between the speed of facial motion and the emotion attributed to facial expressions. This research suggests that expression dynamics are important determinants of emotion perception and that observers are likely to learn some aspects of these dynamics. This is consistent with proposals of a motion-based face-space, in which the dynamic properties of facial expressions are represented and evaluated in a norm-based fashion and through which face-related computations (e.g., emotion recognition, conversational identity recognition) are implemented (Dobs, Bülthoff, & Schultz, 2016; Furl et al., 2020).

An implication of these findings is that facial dynamics need to be accounted for when selecting and/or generating experimental stimuli. Their impact on perceptual processes must also be accounted for, otherwise group or individual differences that are found in the processing of facial expressions may be related to the specific dynamics of a particular stimulus set, and not generalise to other stimuli. Accounting for variability in dynamics is not common, but expressions show marked variability in real life (e.g., Brewer et al., 2016; Zane et al., 2019), and in stimulus sets used for research (Dobs et al., 2018; Krumhuber et al., 2017). Changes in stimuli - with accompanying changes in the spatiotemporal dynamics of emotional expressions - may produce sizable variance in how emotional expressions are perceived and processed.

The present study therefore has two aims. First, to investigate the extent to which variability in emotional expression stimuli produces variance in spatiotemporal dynamics for different emotions. This was achieved by quantifying the degree to which the dynamics in stimuli from high and low prototypicality stimulus sets differed from one another, as well as how these original stimuli compared with their simple linear (morphed) approximations. Second, to determine how changes in expression dynamics affect observers' judgements (including valence, intensity, naturalness, and recognition accuracy).

It was hypothesised that emotion-related dynamics would differ depending on stimulus set (high vs. low prototypicality) and type (non-linear vs. linear). As a consequence, given previous findings (Dobs et al., 2014), it was predicted that judgments of the non-linear and linear expressions would differ depending on emotion and stimulus set. In light of research showing dissociable effects of dynamics on emotion categorisation and intensity judgments, with spatial dynamics influencing categorisation and temporal aspects influencing intensity

(Chen et al., 2021), it was also predicted that intensity and naturality judgements will be more strongly impacted by morphing than valence judgements and recognition accuracy.

EXPERIMENT 2: Spatial-kinematic analyses of facial expressions

3.1.2 Methods

3.1.2.1 Stimuli

Two sets of dynamic emotional facial expressions were chosen to represent the stimuli typically used in psychological research, displaying both prototypical and intense emotional expressions – Amsterdam Dynamic Facial Expressions Set – ADFES; (van der Schalk, Hawk, Fischer, & Doosje, 2011) and subtler, non-stereotypical expressions – Jerusalem Emotional Facial Expressions – JeFEE (Yitzhak et al., 2017). Hereafter, the ADFES stimulus set will be referred to as the high prototypicality and the JeFFE stimulus set will be referred to as the low prototypicality stimulus set (**Figure 3.1**). Note that, as is typically the case in the literature, intensity and prototypicality are confounded, with highly prototypical expressions also rated as more intense and vice versa. Both stimulus sets include videos of young male and female actors (20 – 30 years old) and have been previously validated in terms of valence, intensity, and recognition accuracy (van der Schalk et al., 2011, Yitzhak et al., 2017). The final set contained 8 identities from each stimulus set, four female, all expressing anger, happiness, sadness, fear, surprise, and disgust.

3.1.3 Stimulus processing

Stimuli were equalized across stimulus sets as much as possible on low and mid-level features (face size, lighting). First, all videos were trimmed to the length of six seconds, with actors displaying a neutral expression for approximately one second at the start of each video. A linear expression was then created for each of the original videos by selecting a neutral and a peak frame from each video and morphing them. The number of frames for the morphed

expression was matched to the original videos by making the number of morphed transitions between the neutral and peak frames the same as in the original video. All morphed videos were screened for naturalness and distortions. Distortions were corrected in individual frames using photo editing software.

3.1.4 Data analyses

3.1.4.1 Data pre-processing and reduction

All data analysis pre-processing was performed using custom R-markdown scripts (R-studio 1.2.5033, running R version 3.6.3). O*penFace*, an opensource toolbox implementing face recognition and tracking (Baltrusaitis, Zadeh, & Lim, 2018) was used to extract coordinates for 67 face landmarks for each video frame (see **Figure 3.1**).

These coordinates were then used to compute nine face distances that have been previously identified to reliably capture face actions involved in emotional expressions (e.g., mouth opening, eye-brow widening; see (Ekman et al., 1987; Furl et al., 2020; Sowden, Schuster, Keating, Fraser, & Cook, 2021; Zane et al., 2019).

The face actions were computed as Euclidean distances of the 2D coordinate time series (x and y coordinates) of the face landmarks (**Figure 3.1-C**). This calculation was carried out for all frames, creating a time series of distances associated with each face action for each video. To simplify analyses, a covariance matrix was then computed for all the face action distances to identify the actions least correlated with the other actions and with D1 (overall face action), resulting in 4 distances with correlations below .5. This resulted in two eye distances representing eye opening (D3 and D4), one eyebrow widening (D2), one nose length (D7), and one representing mouth width (D8). The identified distances explained the highest amount of unique variance in face actions, ensuring that no face action was overrepresented. The distances were then averaged at each frame, resulting in one average

face distance for each frame that represents the average amount of action in that frame of the expression.

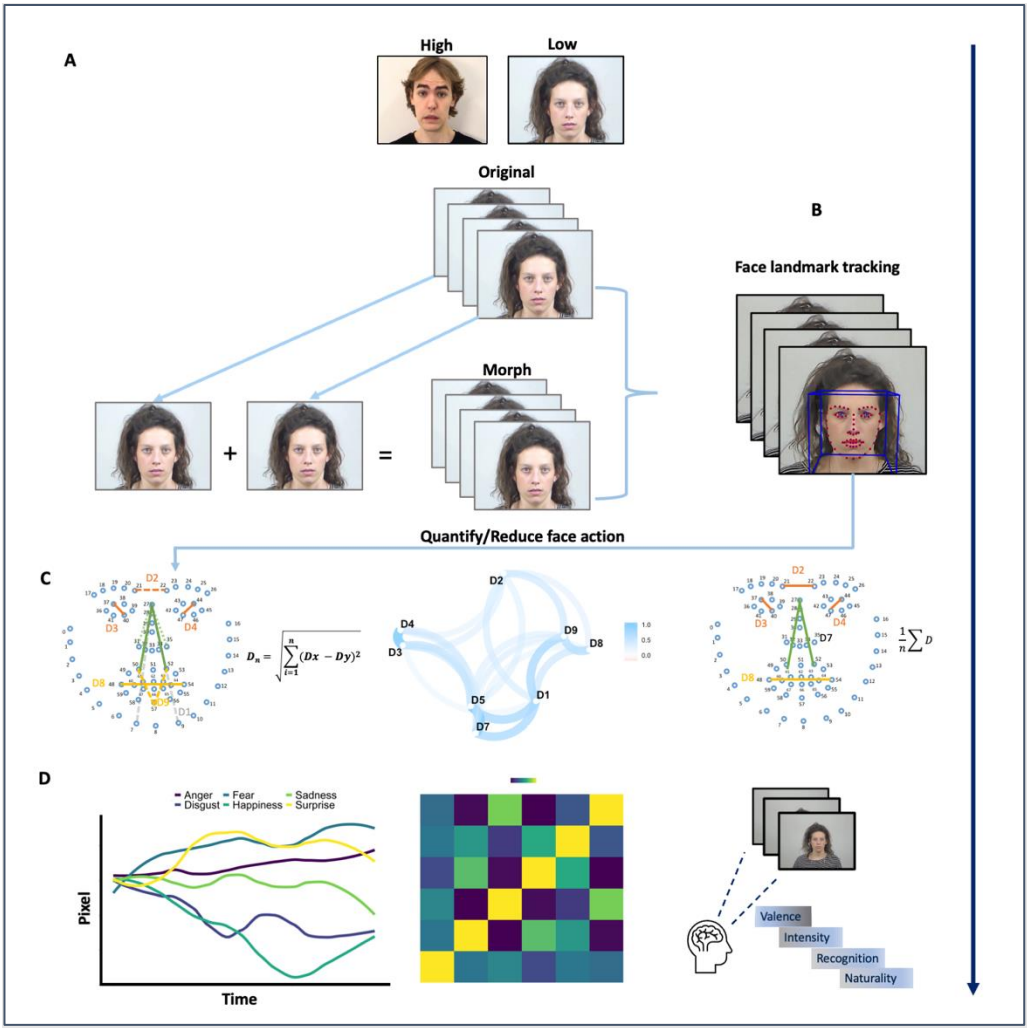

Figure 3.1. Experiment 2 –Analysis of stimuli

A. Sample stimuli from the high and low prototypicality stimulus sets, also used to create the morphed stimuli. B. Face movements were tracked over time using OpenFace. C. Distances representing face actions were computed for each frame. D. These distances were used to create a time-series of average face action for use in subsequent analyses.

3.1.4.2 Multilevel polynomial modelling

Using multilevel models, the timeseries of the average face action at each frame was regressed onto stimulus set (high vs. low prototypicality), stimulus type (morphed/linear vs. original/non-linear) and emotion (anger, happiness, sadness, fear, surprise, and disgust) at the stimulus level. To capture how the dynamic patterns of face actions vary over time, orthogonal polynomial powers of the time term (linear, quadratic, and cubic) were computed and fitted as interactions with the fixed effects. These polynomials were chosen based on a smoothed visualisation of the timecourse of face actions, which indicated these trends. This approach is also known as growth curve modelling and has been described in greater detail in Mirmam (2014) and was also discussed in Chapter 2. After simplifying the maximal model structure to eliminate convergence errors, the model included random effects for stimulus set, stimulus id and the fitted polynomial terms:

*face distance ~ stimulus set * stimulus type * emotion * (ot1 + ot2 + ot3) + (1 + ot1 + ot2 + ot3 | stimulus id) + (0+stimulus set | stimulus id),* with *ot* representing the polynomial terms.

This analysis permits modelling both the effect of stimulus set and stimulus type on average face action changes, and the inclusion of the polynomials allows assessment of the linear and non-linear patterns of face actions over time (e.g., differences in the rate and shape of the face signals in addition to differences in the average face action).

3.1.4.3 Nonparametric Time Clustering Analysis of Face Dynamics

In addition to comparing the two original stimulus sets, morphed was used to create simple linear approximations of the original stimuli. To investigate the effect of this manipulation of the stimulus dynamics, time clusters that distinguished morphed and original stimuli on the basis of face action changes were identified. By contrasting morphed and original time series, the most salient differences in dynamics can be identified, with temporal clusters of

differences representing either periods of significant loss of dynamic information in morphed expressions, or artefactual changes in dynamics relative to the original expressions.

Accordingly, the time series of average face action changes was divided into time bins (n = 40, 4 frames per bin) and a non-parametric clustering technique described in (Maris & Oostenveld, 2007) – also described in Chapter 2, was implemented to identify contrast effects on spatiotemporal auto-correlated timeseries data. As a recap, this method accomplishes three objectives: 1) it avoids detecting spurious differences, for example, at a frame level, as any meaningful differences in dynamics are expected to cluster temporally; 2) unlike traditional multiple comparison corrections which are unreasonably conservative for most time series data, nonparametric clustering controls for Type I error while preserving power, regardless of the threshold used for selecting significant time bins; and 3) this method preserves the temporal dynamics that may be sacrificed by analyses that aggregate dynamics across time.

This approach consisted of 4 steps:

1. A mixed model contrasting stimulus type (*morphed* vs. *original*) was fitted to predict face action change at each bin for each stimulus set.
2. Significant adjacent time bins (at $p < .05$) were clustered by summing their statistics.
3. A null distribution was created by randomly shuffling predictor labels (*morphed* vs. *original* expressions).
4. Repeating steps 1 to 3, 1000 times and comparing the number of times the summed statistic was larger than the random summed statistics.

Finally, the covariance in dynamics across stimuli was assessed by correlating their timecourses across emotions for each data set and stimulus type, producing a similarity matrix for face dynamics.

3.1.5 Results

3.1.5.1 Multilevel polynomial modelling

The average face action for each emotion, stimulus type and stimulus set are displayed in **Figure 3.2**, along with the emotion timecourses for each stimulus type and stimulus set.

As expected, the high prototypicality stimulus set showed larger face actions on average than the low prototypicality stimulus set (Estimate = 2.93, SE = 0.86, $p < .001$; High > Low prototypicality). There was also an effect of morphing on the average face action (Estimate = 2.09, SE = 0.86, $p = .02$; Original > Morph) and a main effect of emotion on the amount of action, with significant pairwise differences for all pairs (p corrected < .05) except fear vs. surprise, disgust vs. happiness, and anger vs. sadness.

There was a significant interaction between emotion and stimulus set ($F_{(5,192.01)} = 36.71$, MSE =1938, $p < .001$): post hoc analyses showed that there were 50% fewer significant pairwise differences between emotions for the low prototypicality stimulus set (see **Figure 3.2**) compared to the high prototypicality stimulus set. Similarly, there was an interaction between stimulus type and emotion, showing that morphed stimuli resulted in fewer distinct average face action patterns for emotions, compared to original expressions.

For the timecourse of face actions, there was an interaction of stimulus set and higher order polynomial terms (quadratic and cubic), suggesting that in addition to differences in average face action, stimulus sets showed systematic differences in the temporal patterns of face action change over time. In particular, prototypical (original) expressions showed marked non-linear timecourses, compared to less prototypical expressions (see **Figure 3.2**-B).

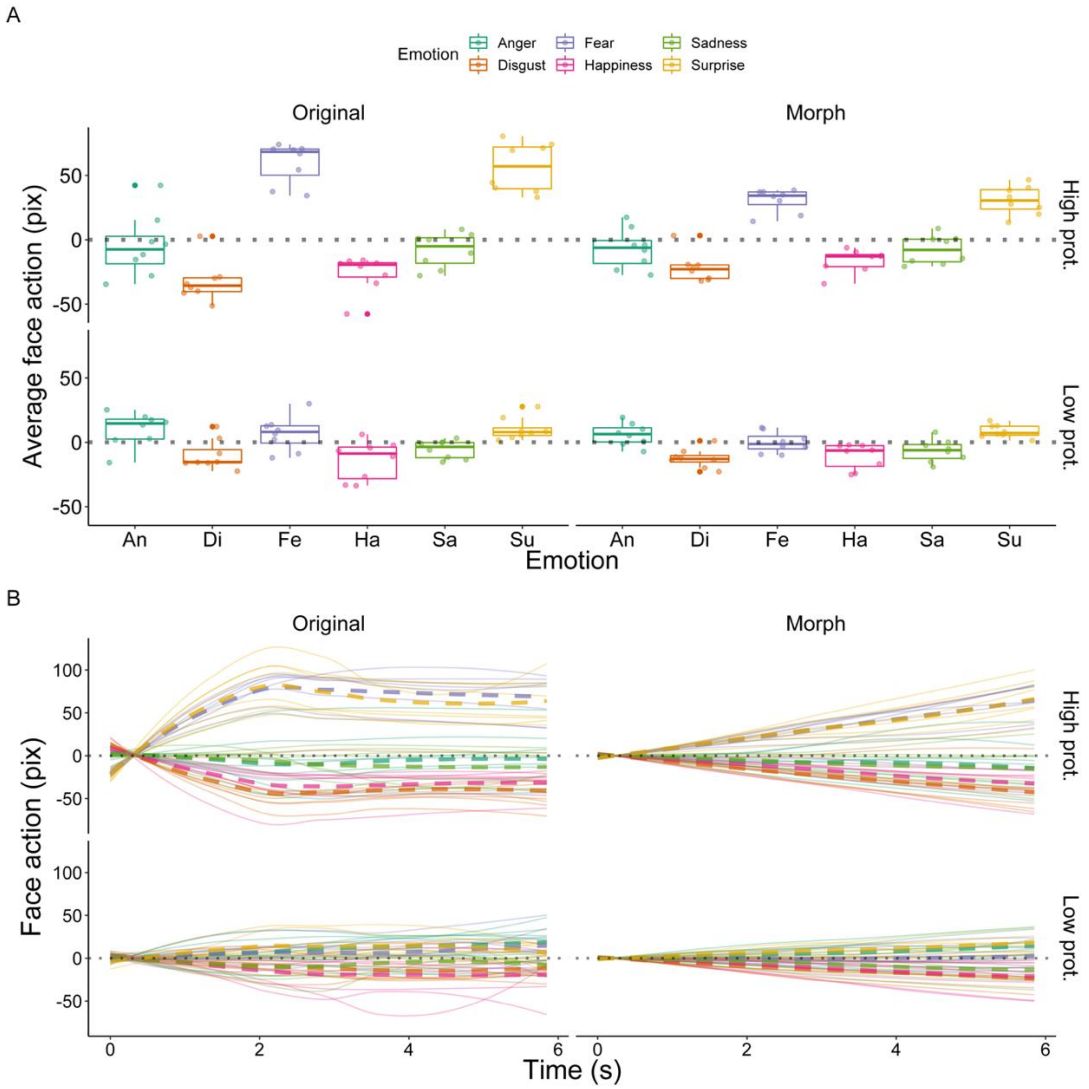

Figure 3.2 Aggregate and timecourse of face-actions

A. Average face action (baseline corrected) by emotion, stimulus type and stimulus set. Separate points indicate individual stimuli with colour representing emotion. Dashed y intercept represents baseline. B. Expression timecourses varied, with prototypical posed expressions showing non-linear timecourses which were not captured well in morphed expressions. Highly prototypical expressions had more distinct timecourses compared to less prototypical expressions. An = Anger. Di = Disgust, Fe = Fear. Ha = Happiness. Sa = Sadness. Su = Surprise. High prot = High prototypicality. Low prot = Low prototypicality. *** $p < .001$.

There was also an interaction between all the polynomials and emotion (see Table 8.5 Full Model Details – Experiment 2 in Appendices). These effects show that differences between emotions are captured by different functional shapes that characterise how these expressions evolve over time (see Figure 3.2). There was a significant three-way interaction between stimulus type, stimulus set and the polynomial terms. Exploratory visualisations demonstrated that this interaction was driven by non-linear patterns that approximated sigmoidal trends in face action in high prototypicality expressions, and subtler non-linearities in low prototypicality stimuli, compared to morphed stimuli which were best fitted by linear trends (see Figure 3.2).

3.1.5.2 Nonparametric time clustering of face dynamics

The timecourse analysis sought to identify time clusters that distinguished morphed and original facial expressions. Overall, morphed and original stimuli differed by emotion, with some emotions showing particularly marked differences in the average face action signals between stimulus types in both early and later time clusters ($anger_{morph>original}$, $sadness_{morph<original}$), throughout the timecourse ($fear_{morph<original}$, $disgust_{morph>original}$) or only in the middle cluster ($surprise_{morph<original}$) – see **Figure 3.3** for each individual emotion.

Both when analysing at the stimulus set level and when collapsing across stimulus set and emotion, morphed vs. original differences in face action clustered mostly at the earlier stages of expression, especially for the low prototypicality expressions.

These results indicate that moment-to-moment differences in face action changes are apparent in early time clusters, suggesting that early dynamics in posed facial expressions may carry temporal non-linearities (related to the activation pattern of face actions) that are not well-modelled by simple morphed animations of expressions (assuming a linear change of expression).

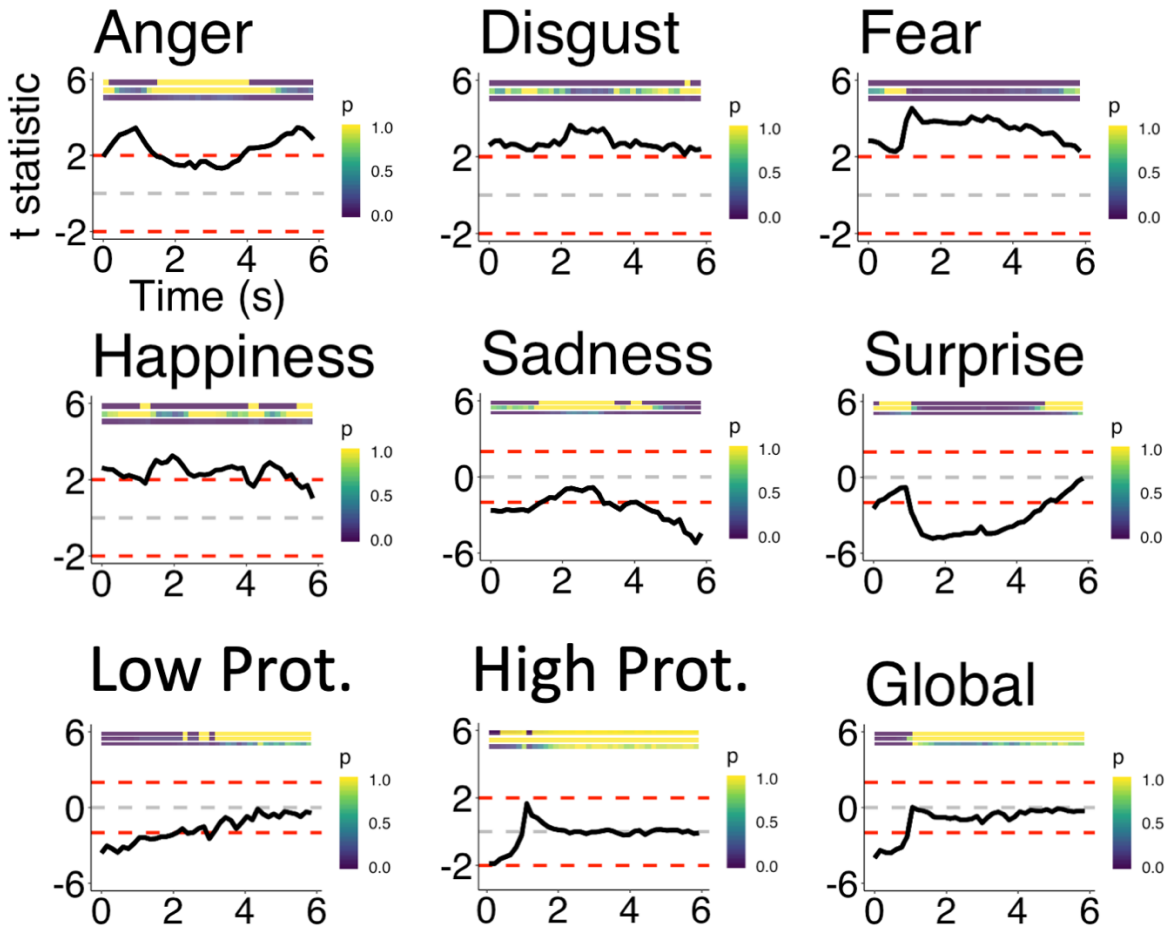

Figure 3.3 Nonparametric time clustering of face actions.

Contrasting morphed vs. original stimulus types. Horizontal colour bars represent p values: uncorrected (lower), Bonferroni corrected (middle) p-values, and significant vs. non-significant clusters based on non-parametric bootstrapping (top). Darker colours indicate lower p-values. Positive test statistics indicate greater face action change for morphed, and negative test statistics indicate greater face change for original, expressions. Dashed red lines indicate test statistic cut-offs.

In turn, this indicates that initial face movements exhibit high variability and non-linear patterns (e.g., rapid acceleration), consistent with the quadratic effects in the polynomial models reflecting differences between stimulus type, emotion and stimulus set.

Finally, as an exploratory analysis, a set of correlations was computed between timecourses of face action changes across emotions, allowing covariance of the dynamic face patterns to be assessed statistically (rather than absolute differences as in the analyses above). The covariance matrices indicate more distinct patterns for the highly prototypical expressions (more divergent heatmap – see **Figure 3.4**) resulting in higher covariance patterns (R^2 = .49, p < .01, off-diagonal = .28, p < .05) compared to less prototypical expressions (R^2 = .31, p < .05, off-diagonal = .01, *ns*).

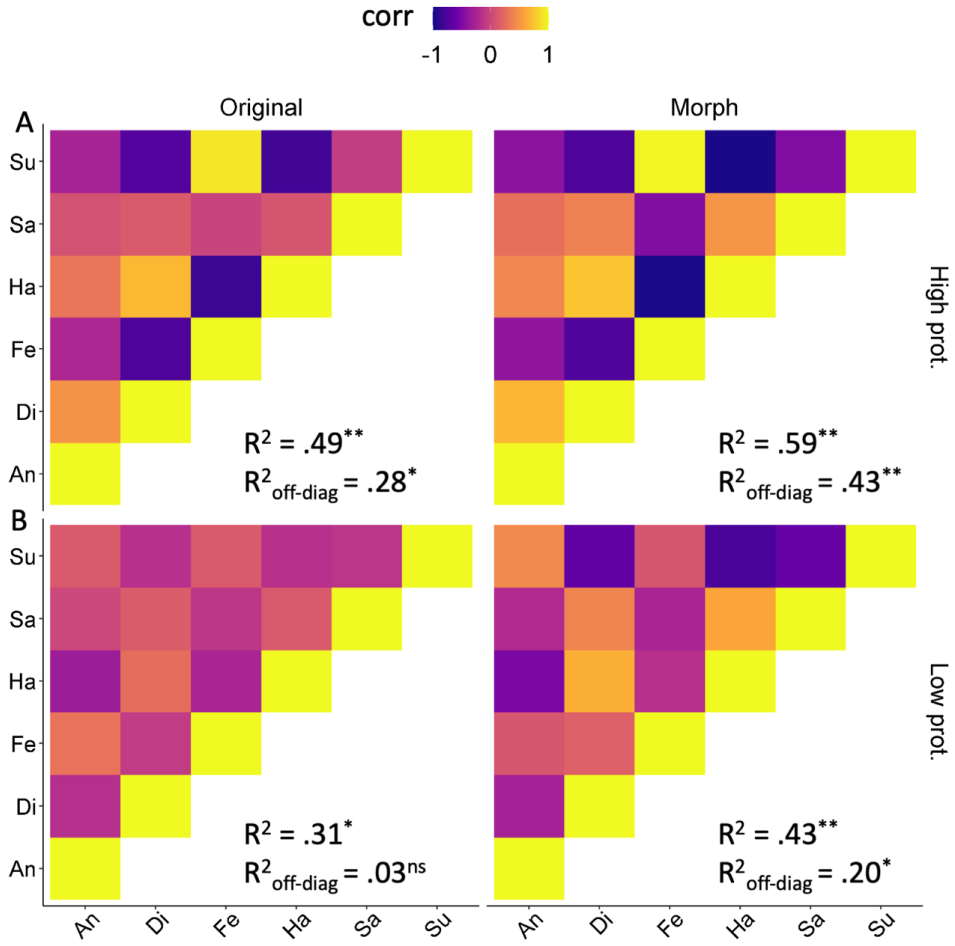

Figure 3.4 Similarity matrices for spatiotemporal dynamics.

Shows the correlation of spatiotemporal patterns of face action across emotions by stimulus set and stimulus type. Corr = Correlation (Pearson). An = Anger. Di = Disgust. Fe = Fear. Ha = Happiness. Sa = Sadness. Su = Surprise. ** p < .01; * p < .05, ns = non-significant.

In all cases morphing increased the covariance of face action patterns almost two-fold while also resulting in more divergent heatmaps. This suggests that morphing artificially exacerbated differences between expressions in the spatial pattern of facial action while blurring differences in their temporal dynamics.

3.1.6 Discussion

Analysis of two stimulus sets using timecourse analyses provides evidence that expressions varying in levels of prototypicality differ in their dynamic statistics. These results confirm the variability within posed expressions, showing that prototypical expressions have clearer and more distinct spatiotemporal patterns for different emotions, but also show stronger temporal covariance, compared to less prototypical expressions. Clustering analyses highlighted that differences in dynamics between stimuli are mostly evident in early time clusters that represent initial face movements. Importantly, linear approximations of original expressions (morphs) affected the emotion-specific non-linear dynamics that characterised prototypical original expressions to varying degrees. Similarly, morphs had a loss of specific temporal dynamics while maximising the spatial appearance linearly over time.

The current results support the need to investigate the effects of the dynamic variability of facial expression stimuli on observers processing, which may have implications for the generalisability of results obtained from studies using a single stimulus set or a limited number of emotions.

EXPERIMENT 3. Effect of expression dynamics on emotion processing

Experiment 3 aimed to investigate how the variability in expression dynamics revealed in Experiment 2 influences observers' emotion categorisation and ratings of valence, intensity, and naturalness.

3.1.7 Methods

3.1.7.1 Participants

A total of 106 participants (61 female) after exclusions (N = 5 because they failed attention checks), with no current or recent (in the previous 6 months) mental health conditions took part (M_{age} = 32.9, SD_{age} = 8.79 [18 – 50]). Sample size was determined a priori through simulation based on pilot data, indicating that approximately 60 to 70 participants would provide 80% power to detect a small effect size of interest. Participants were recruited via *Prolific.ac* and compensated for their time. All participants reported English as their primary language and resided in the UK at the time of the study.

3.1.7.2 Dynamic emotion task

Participants were shown original and morphed versions of the facial expression stimuli used in Experiment 2 and asked to rate them for valence, intensity, and their degree of naturalness ('naturality'). Participants were also asked to categorise the emotion (see **Figure 3.1**, panel B) by choosing from 7 labels (anger, disgust, fear, happiness, sadness, surprise, and neutral). The task was designed and deployed via *Gorilla.sc*, an online platform for behavioural experiments (Anwyl-Irvine, Massonnié, Flitton, Kirkham, & Evershed, 2020). Because there were more stimuli than could be practically completed by a single participant in an online setting, each participant was randomly assigned a portion of the task comprising of 96 trials. Random attention checks were included which displayed unrelated words (e.g., air, fuss, cup) for less than 200ms in less than 5% of trials. Participants were asked to choose the word they

had seen and those who made more than one mistake in reporting the words were excluded from further analyses (N = 3). 2 additional participants were excluded for showing implausible completion times.

3.1.7.3 Data analysis

3.1.7.3.1 Generalised and linear mixed models

Generalised and Linear Mixed Models (GLMMs) were fitted in lme4 (Bates et al., 2014) to predict participants' scalar ratings (valence, intensity, and naturality) and a logistic binomial model was used for modelling trial-by-trial recognition accuracy (binary accuracy). The starting model always had the form:

DV ~ *stimulus type* * *emotion* * *stimulus set* + (1+ *stimulus type** *stimulus set* | *participant id*) + (1 | *stimulus id*).

Where stimulus type (morphed vs. original), emotion (anger, happiness, sadness, fear, surprise, and disgust) and stimulus set (high vs. low prototypicality) were estimated as fixed effects, and participant and stimulus id were estimated as random effects. Slopes were dropped where appropriate due to convergence errors, near-zero variance and or almost perfect correlation with intercepts. To avoid overfitting, models were simplified in three steps: first, by using the *step* function from the lmerTest package v. 3.1.0 (A Kuznetsova, Brockhoff, & Christensen, 2015) to iteratively select and remove terms; second, by refitting models dropping eliminated terms in step 1 and computed an R-squared equivalent measure for mixed models proposed in (Barton & Barton, 2015); and third, ANOVA Type III tables were computed to test the significance of each term. It was settled on a model when changes in explanatory power remained relatively constant with addition of further terms. This method was compared to the Likelihood Ratio Test using an *anova* method in R to contrast nested models, with the model with the lowest AIC and BIC (delta > 4) preferred, which

agreed with the methods described above. The package *emmeans* (Lenth, Singmann, Love, Buerkner, & Herve, 2018) was used to probe planned and post hoc contrasts and interactions.

3.1.7.4 Similarity analyses

In addition to looking at differences in the ratings (absolute values), it was assessed whether the rating pattern within each judgement (valence, intensity, naturality, recognition, and confusion) was consistent within and across participants, for morphed and original stimuli. This analysis is useful as, for example, despite potential differences in valence ratings (e.g., original > morph), participants may still show consistent ratings such that the covariance of ratings across emotions remains consistent (e.g., anger expressions are still judged as having stronger negative valence than sad expressions). To do this, vectors were created containing each rating (valence, intensity, naturality, and recognition), for morphed and original expressions, independently for each stimulus set. These were paired by stimulus id, and a correlation coefficient computed between all pairs for each participant. A correlation of zero would indicate no relationship between a participant's ratings of expressions across morphed and original stimuli, whereas a positive correlation indicates that morphed and original expressions were rated consistently by a given participant. These correlations were then transformed using the Fisher r-to-z method and submitted to a single sample t-test. A significant test at $p < .05$ can be taken as an indication that this correlation pattern is significantly different from zero across individuals and the magnitude and direction of the relationship can be compared between stimulus sets (Kuhn, Wydell, Lavan, McGettigan, & Garrido, 2017). In other words, stronger positive values indicate increased similarity in the pattern of ratings which would suggest limited effects of spatiotemporal differences induced by morphing, whereas smaller or negative correlations would indicate that morphing affected rating patterns.

For recognition accuracy scores, it was also possible to compute confusion matrices, which were used to assess the overall pattern of responses across stimuli for morphed and original expressions. Like the similarity analyses, confusion matrices were computed for each participant, stimulus set, and stimulus type, and then correlations between confusion matrices calculated for morphed and original stimuli. A single sample t-test was performed on the Fisher r-to-z transformed correlation scores for statistical inference at the group level. Finally, the same analysis was also repeated after excluding diagonal elements (correlations for the same emotion category), given their strong correlations.

3.1.8 Results

3.1.8.1 Predicting emotion ratings

Mixed models were fitted to predict the influence of stimulus type (morphed vs. original), stimulus set (high vs. low prototypicality) and emotion (anger, disgust, fear, happiness, sadness and surprise), on valence, intensity, naturality and recognition accuracy.

3.1.8.1.1 Valence

The final model had 67.79% of explanatory power of which 62.30% was explained by the fixed effects. Within this model, there was no significant effect of stimulus type (Estimate = -0.87, SE = 1.94, $t_{(191)}$ = -0.45, p = .800). There was a small effect of stimulus set (Estimate = 10.09, SE = 1.94, $t_{(191)}$ = 5.20, p < .001; low > high) and a significant effect of emotion ($F_{(5.\ 194.51)}$ = 693.96, p < .001; see **Figure 3.5**). There was also an emotion by stimulus type interaction ($F_{(5,\ 195.09)}$ = 4.07, MSE = 616, p = .001), indicating that valence ratings for different emotions varied systematically depending on whether participants rated morphed or original expression stimuli. Specifically, post-hoc tests indicated significant differences for anger (Estimate = 2.72, SE = 1.37, z-ratio = 4.71, p = .047; morph > original) and happiness

(Estimate = -4.83, SE = 1.37, z-ratio = -8.52, $p < .001$; morph < original). There were no significant effects of morphing on other emotions.

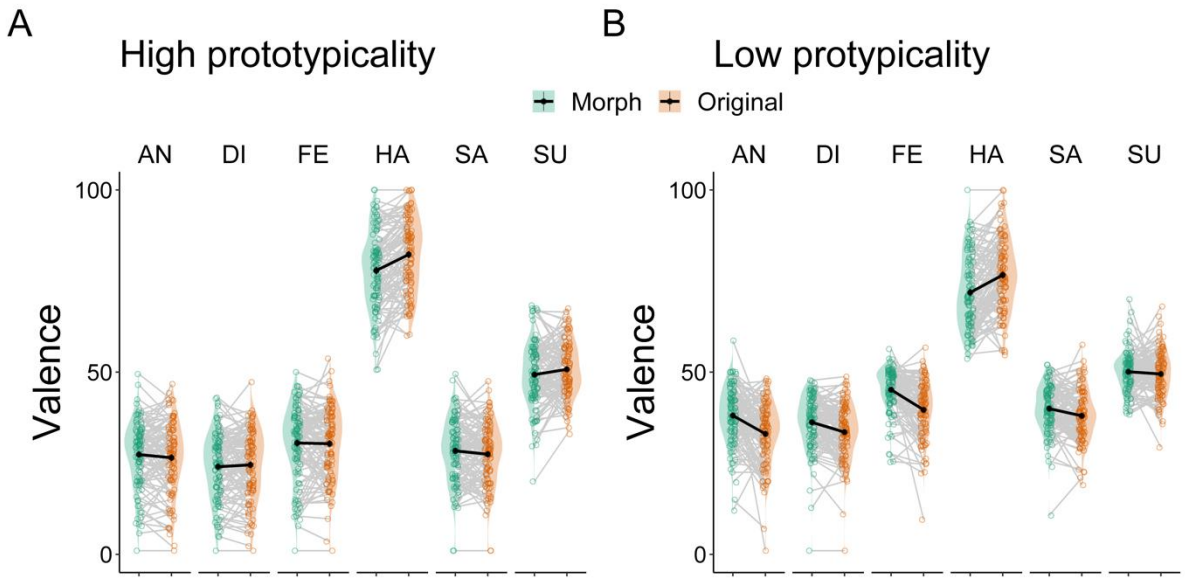

Figure 3.5 Valence ratings.

Low prototypicality stimulus sets were rated as more positive, and valence ratings for emotion varied as a function of stimulus type (morph vs. original) and stimulus set. AN = Anger. DI = Disgust. FE = Fear. HA = Happiness. SA = Sadness. SU = Surprise.

The interaction between stimulus type and stimulus set was not significant ($F_{(1, 190.61)} = 2.03$, MSE = 308, $p = 0.15$), suggesting that morphing impacted valence ratings of both stimulus sets equally. However, there was an interaction effect for stimulus set by emotion ($F_{(5, 194.51)}$= 27.63, MSE = 4179, $p < 0.001$), suggesting that the valence rating for emotions differed depending on the stimulus set.

3.1.8.1.2 Intensity

The final model selected for intensity ratings had a total explanatory power of 54.66%, of which the fixed effects explained 37.17% of the variance (stimulus type and emotion). Within this model, there was a small significant effect of stimulus type (Estimate = 4.31, SE = 1.11, $t_{(190)} = 3.88$, $p < .001$; original > morphed). There was also a significant effect of stimulus set (Estimate = -27.41, SE = 1.11, $t_{(190)} = -24.72$, $p < .001$; high > low prototypicality). Finally, the effect of emotion was significant, suggesting that intensity ratings varied significantly by emotion ($F_{(5, 200.01)} = 25.5$, MSE = 7221, $p < .001$; see **Figure 3.6**).

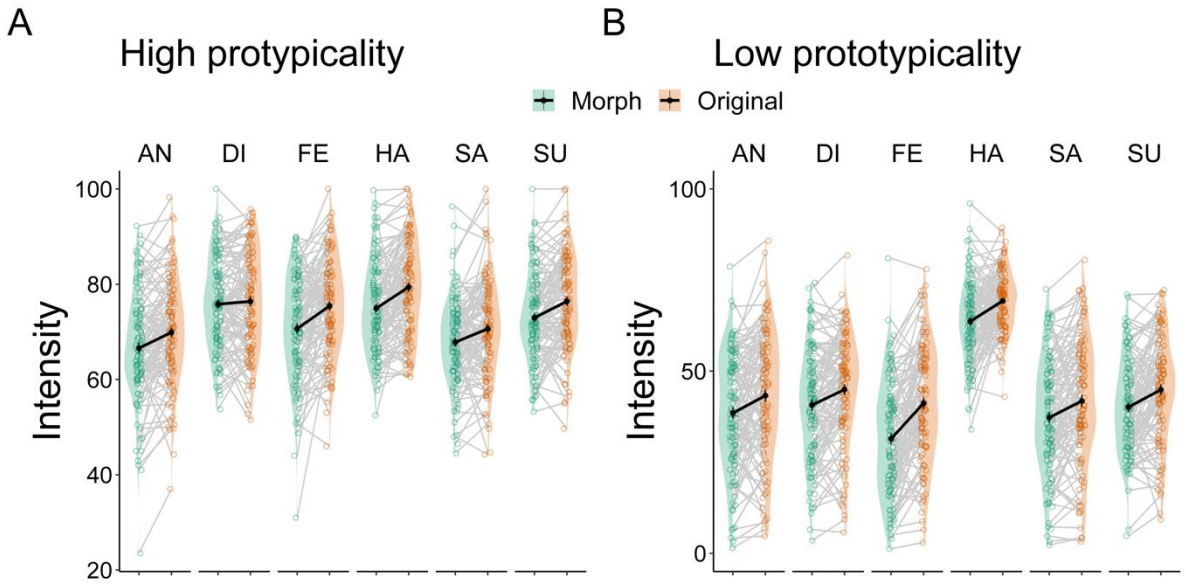

Figure 3.6 Intensity ratings.

Morphed expressions are rated as slightly less intense than original expressions, and high prototypicality stimuli as more intense than low prototypicality. AN = Anger. DI = Disgust. FE = Fear. HA = Happiness. SA = Sadness. SU = Surprise.

3.1.8.1.3 Naturality

The final model predicting naturality had a total explanatory power of 28.05%, of which the fixed effects explained 11.37% of the variance (stimulus type, stimulus set and emotion, and their two-way interactions). Within this model, the effect of stimulus type was small, but significant (Estimate = -6.44, SE = 2.02, $t_{(190)}$ = -3.18, $p < .01$; morph < original). There was also a small, but significant effect of stimulus set (Estimate = 5.97, SE = 2.02, $t_{(190)}$ = 2.95, $p < .01$, low > high). The main effect of emotion was significant ($F_{(5, 193)}$ = 39.05, MSE = 16545) suggesting that emotions differed in their perceived naturalness, driven primarily by happy expressions being perceived as more natural than all other expressions.

There was a significant two-way interaction between stimulus type and stimulus set ($F_{(5,193.00)}$ = 5.30, MSE = 2247, $p < .001$). Interestingly, while original expressions were perceived as more natural for low prototypicality stimuli (Estimate = -4.80, SE = 1.08, z-ratio = .434, $p < .001$), the opposite was true for high prototypicality stimuli, with morphed stimuli perceived as more natural (Estimate = 3.50, SE = 1.08, z-ratio = 3.23, $p = .006$). The stimulus type by emotion interaction was also significant ($F_{(5, 193.00)}$ = 5.3051, MSE = 2247, $p < .001$). Post-hoc analyses indicated a significant difference between morphed and original stimuli for happy expressions (Estimate = -9.25, SE = 1.87, z-ratio = -4.94, p < .001, morphed < original). There was also a significant stimulus set by emotion interaction ($F_{(5,193.00)}$ = 6.20, MSE = 2629, $p < .001$), suggesting that naturality ratings varied by stimulus set; see **Figure 3.7**.

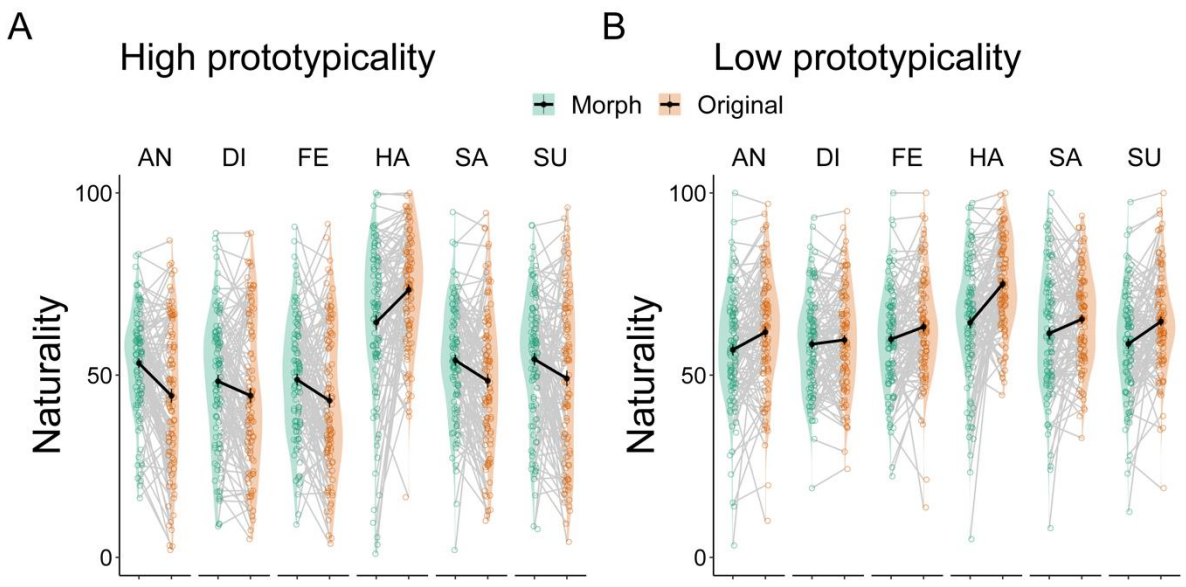

Figure 3.7 Naturality ratings

Morphed expressions are perceived as less natural for low prototypical expressions and more natural for high prototypical expression. Low prototypical expressions are perceived as more natural overall. AN = anger, DI = disgust, FE = fear, HA = happiness, SA = sadness, SU = surprise.

3.1.8.2 Accuracy

The final model had 59% explanatory power, of which 44% was explained by the fixed effects (stimulus type, emotion, stimulus set and their interactions). There was a significant effect of stimulus type (Estimate = -0.567, SE = 0.19, z-ratio = 2.935, $p = 0.003$, morph < original; see **Figure 3.8**).

There was a significant effect of stimulus set, with high prototypicality expressions being recognised more accurately than low prototypicality expressions (Estimate = 2.57, SE = 0.18, z-ratio = 13.97, $p < .001$).

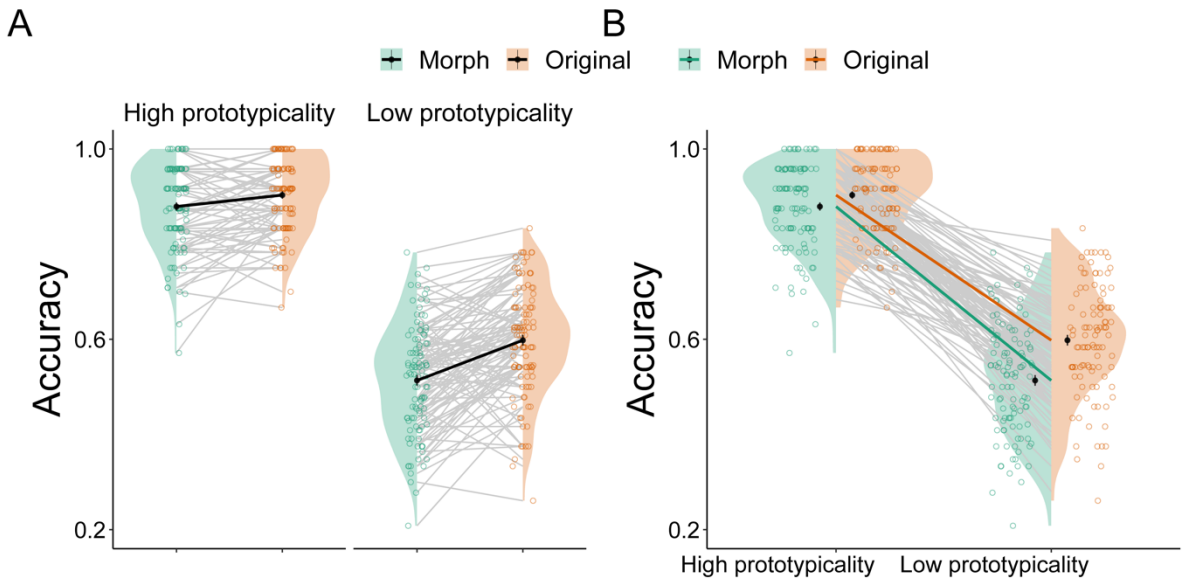

Figure 3.8 Emotion Recognition Accuracy.

High prototypicality stimuli were recognised more accurately compared to low prototypical stimuli. Original expressions were recognised better than morphs for low but not high prototypicality stimuli. A & B represent the same data but illustrating different within effects, stimulus set and stimulus type respectively.

Interestingly, the difference between morphed and original expressions was significant for low prototypicality stimuli only (Estimate = 0.71, SE = 0.25, z-ratio = 2.88, $p_{adjusted}$ = .008). There was also a main effect of emotion ($F_{(5\ 184.24)}$ = 36.85, MSE = 36.85, $p < .001$), with happiness recognised more accurately than all other emotions.

In summary, there were effects of stimulus type, stimulus set, and emotion observed for valence, intensity, and naturality ratings, and for the accuracy of emotion recognition. Valence ratings varied significantly depending on stimulus set, emotion but not stimulus type. Interestingly, morphing increased the naturality of high prototypicality expressions whereas it decreased naturality judgments for low prototypicality expressions. Finally, recognition

accuracy was worse for morphed low prototypicality expressions compared to their respective original expressions, whereas recognition for highly prototypical expressions was equivalent for morph and originals. This suggests that spatiotemporal information is more relevant to judgements of subtler and less stereotypical expressions.

3.1.8.3 Similarity analyses

Figure 3.9 shows the results of the similarity analyses for original and morphed expressions for ratings of valence, intensity, naturality, and emotion recognition accuracy.

For valence, there was strong similarity between ratings of morphed and original stimuli ($t_{(105)}$ = 27.02, p < .001, $Estimate_{r\text{-}z}$ = 0.98[95%CI = 0.91, 1.05]; Mean-r = .70, SD = .20), with 56% of shared variance. There was, however, a significant difference between stimulus sets ($t_{(104)}$ = 243.98, p < .001). The high prototypicality stimulus set showed a higher similarity in valence ratings (Z = 1.42, r = 0.89, SE= 0.05; Mean-r = .83, SD = .19) than the low prototypicality expressions (Z = 0.77, r = 0.65, SE = 0.05; Mean-r = .60, SD = .25).

The test for the similarity of intensity ratings between morphed and original stimuli was also significant ($t_{(105)}$ = 11.44, p < .001, $Estimate_{r\text{-}z}$ = 0.31[95%CI: 0.25, 0.36], Mean-r = .26, SD = .24). However, unlike valence ratings, the amount of shared variance between morphed and original intensity ratings was much smaller (7%) and no significant differences between stimulus sets was observed ($t_{(105)}$ = 1.96, p = .17). Thus, although intensity ratings for morphed and original stimuli were significantly correlated, the size of the correlation suggests that morphing impacts perceived expression intensity to some degree.

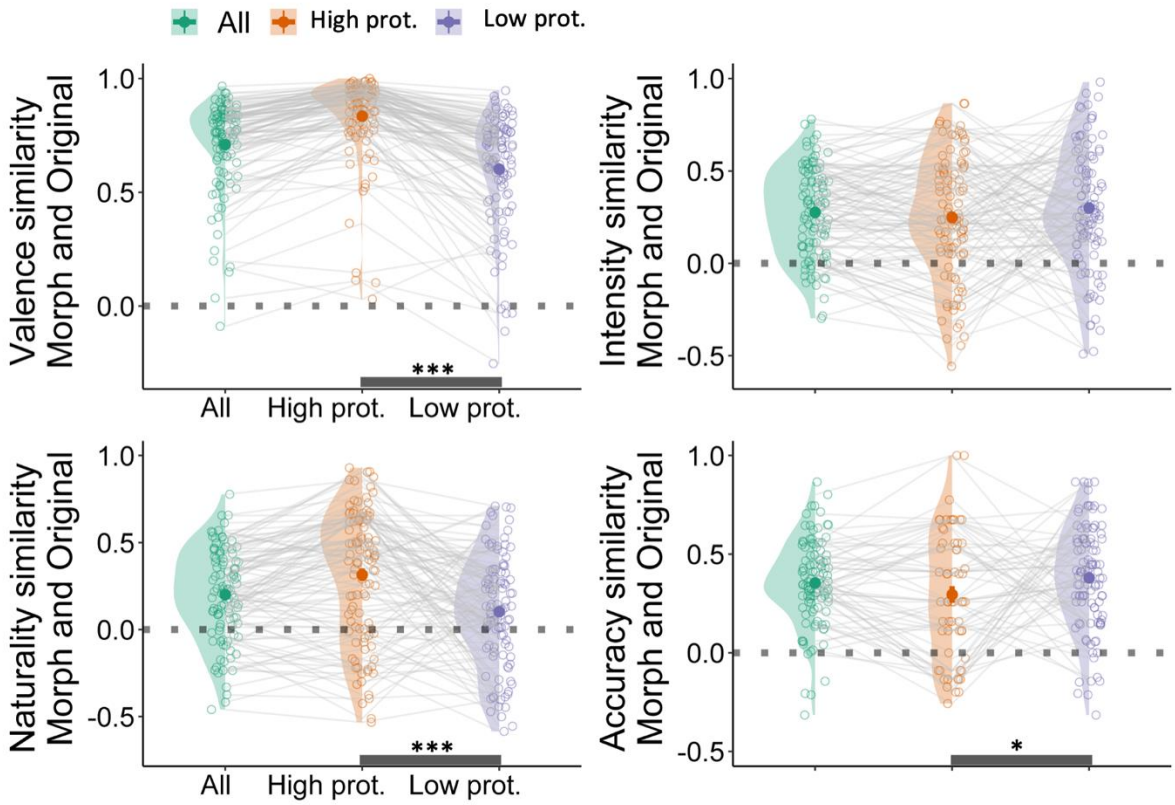

Figure 3.9 Similarity of ratings and emotion recognition patterns.

Across morphed and original expressions. X-axis represents stimulus set (High vs. Low prototypicality, All = combined) and the y-axis indicates the correlation between responses to original and morphed stimuli. Individual dots and lines represent individual participants. *** p < .001; * p < .05.

There was also a significant relationship between naturality ratings for morphed and original stimuli ($t_{(105)}$ = 7.76, *p* = .001, $Estimate_{zr}$ = 0.22[95%CI: 0.16, 0.27], Mean-r = .20; SD = .26). However, there was less than 5% shared variance, indicating reduced similarity of ratings across stimulus types. There was a significant effect of stimulus set ($F_{(1, 105)}$ = 26.93, GES = .099, *p* < .001), such that the high prototypicality stimuli showed higher consistency (z = 0.39, SE = 0.04, Mean-r = .31, SD = .37) than the low prototypicality stimuli (z = .11, SE =

0.04, Mean-r = .10, SD = .32). These data suggest that morphing impacts the perceived naturality of expressions.

Finally, for accuracy, there was a significant relationship between morphed and original expressions ($t_{(105)}$ = 15.02, p < .001, $Estimate_{zr}$ = 0.39[95%CI: 0.45, 0.34], Mean r = .35, SD = .22). However, recognition accuracy for original expressions only accounted for approximately 12% of the variance in accuracy for morphed expressions. There was also a significant difference between stimulus sets ($t_{(1, 56)}$ = 4.06, p < .04), with high prototypicality stimuli showing less similar accuracy profiles between morphed and original expressions (z = 0.311, SE = 0.05, Mean-r = .29, SD = .32), than low prototypicality stimuli (z = 0.38, SD = .26, Mean-r = .36, SD = .267).

3.1.8.4 Confusion patterns

Unlike the accuracy analyses above, analyses of confusion matrices collapse across stimuli and allow it to be established how similar the overall recognition pattern (including both correct and confused responses) is across stimulus type (morphed and original) and stimulus sets. The participant-level correlations for morphed and original confusion patterns were significantly different from zero ($t_{(105)}$ = 62.57, df = 105, p < 0.001, $Estimate_{rz}$ = 1.29, [95%CI: 1.25, 1.33], Mean-r = .86; SD =.25; see **Figure 3.10**). The positive estimate indicates that overall, confusion matrices were consistent across individuals with the two matrices sharing 78% of the variance. There was a significant effect of stimulus set ($F_{(1, 105)}$ = 7.47, MSE = 22.91, GES = .098, p < .001), such that the confusion matrices between morphed and original stimuli were more similar for highly prototypical stimuli (Z = 2.74, SE = .26, Mean-r = .99, SD = .25) than the low prototypicality expressions (Z = 0.95, SE = 0.26; Mean-r = .74, SE = .25).

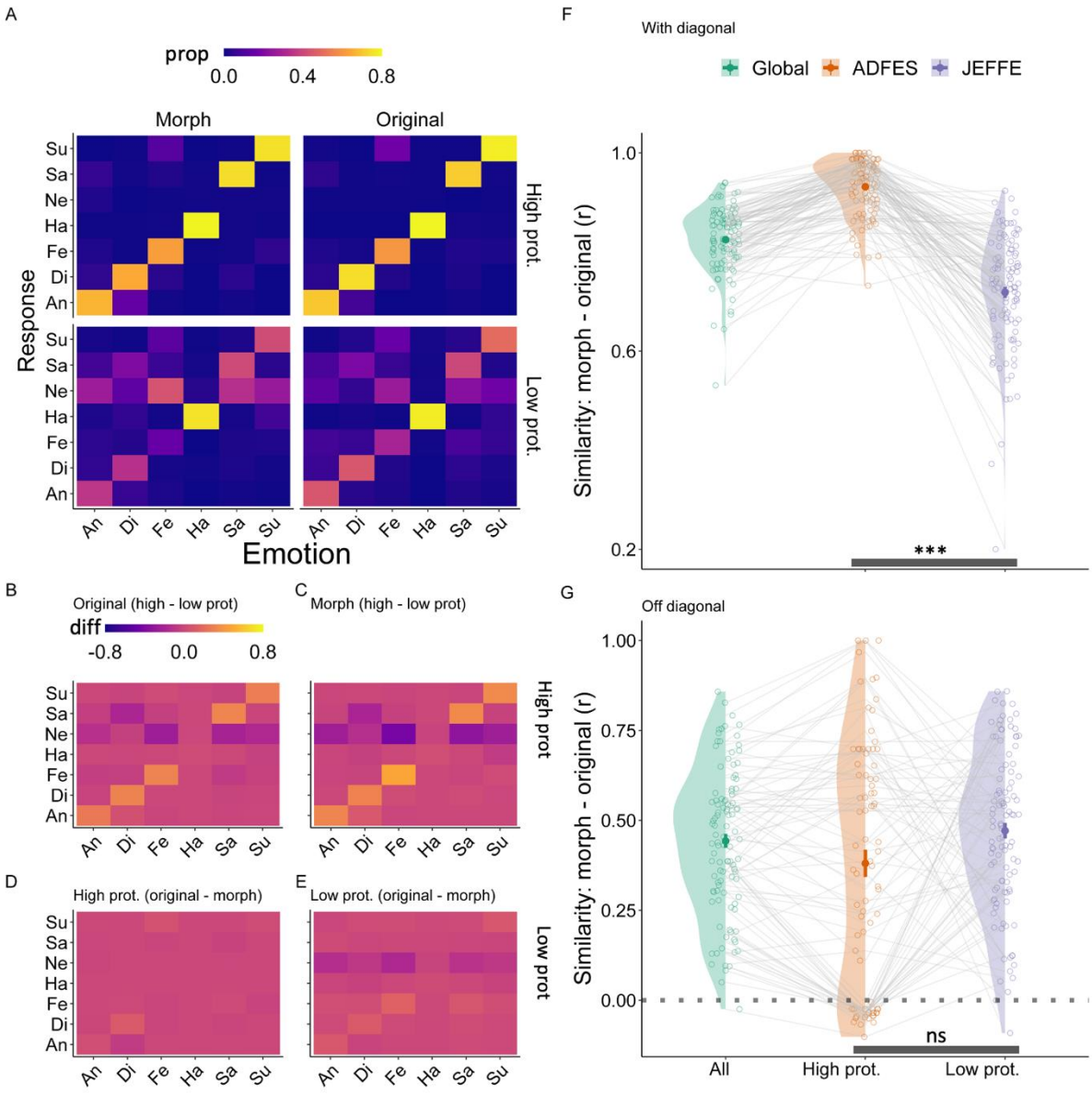

Figure 3.10 Confusion matrices and similarity analyses.

A. Confusion matrices for morphed and original expressions. B – E: Difference matrices for recognition patterns. F – G: Similarity plots for confusion matrices with and without diagonal elements. An = anger, Di = disgust, Fe = fear, Ha = happiness, Sa = sadness, Su = surprise; *** $p < .001$, ns = non-significant. Prop = proportion; diff = difference. Prot = prototypicality.

The same analysis was also significant on the off-diagonal elements (i.e., pattern of inaccurate responses), $t_{(105)} = 22.9$, $p < 0.001$, $Estimate_{rz} = 0.54$, 95% CI: 0.49, 0.59], Mean-r = .49, SD = .27). Note, however, that the shared variance was reduced to 24%. Additionally, the difference between stimulus sets was no longer significant in the off-diagonal analysis ($F_{(1,76)} = 1.66$, MSE = 0.18, $p = .201$). This suggests that the increased consistency of emotion recognition between morphed and original stimuli in highly prototypical expressions is driven by trials in which participants make accurate responses (see **Figure 3.10**) while errors vary by stimulus type.

In summary, the similarity analyses show that, in addition to differences in predicting judgements demonstrated in the generalised and linear mixed model analyses, there is a high degree of variability in the covariance pattern of ratings, particularly for intensity and naturality judgements and recognition accuracy scores. This suggests that differences in dynamics due to stimulus set and disruption of temporal dynamics due to morphing lead to increased inconsistence in judgements of emotional facial expressions, within individuals and at a group level.

Lastly, an exploratory analysis tested whether the covariance in stimulus dynamics computed in Experiment 2 (Figure 3.4) could predict confusion matrices observed in Experiment 3 (Figure 3.10), as it could be expected that stimuli with a higher degree of similarity in their dynamics may be more easily confused, and that this may in turn explain previously reported confusions in the literature such as anger and disgust, or fear and surprise (Calvo, Avero, & Fernández, 2016). This analysis indeed suggested that different dynamics in stimulus types and stimulus sets impact the patterns of errors observers make when trying to recognise or label emotions. Specifically, face action dynamic similarity was positively related to average confusion between emotion pairs for original high ($r = .40$, $p < .05$) and low prototypical

expressions (r = 37, $p < .05$) and high (r = .44, $p < .05$) but not low prototypical morphs (r = .07, ns; see **Figure 3.11**).

3.1.9 Discussion

Experiment 3 shows that observers are sensitive to the dynamics of emotion stimuli. Specifically, valence, intensity, and naturality ratings, as well as the accuracy of emotion recognition, were all influenced by morphing and effects often differed depending on the stimulus set.

3.1.10 General Discussion

The vast majority of faces we encounter are moving, rather than static, which is why many researchers recognise the need to adopt more ecologically-valid dynamic face stimuli when studying social perception (Dobs et al., 2018; Krumhuber et al., 2017). Adopting such stimuli introduces a further complexity, however, given that dynamic information conveyed by the face varies with stimulus type (here simple linear morphs vs. original video stimuli with non-linear dynamics), and by stimulus set. How the variance in dynamic information impacts processing of facial information is currently not well understood, but if effects are large, it may mean that results which are found using one type of stimulus or stimulus set do not generalise well to other stimuli. Importantly, high levels of individual variability in the effects of these dynamics could preclude normative studies on emotion perception from faces (e.g., answering which emotions are likely to be confused, which expressions are processed in a more featural manner, or studies of group or individual differences). The question of how differing dynamics of emotional expressions impact the judgements of emotion from facial stimuli was the question addressed here.

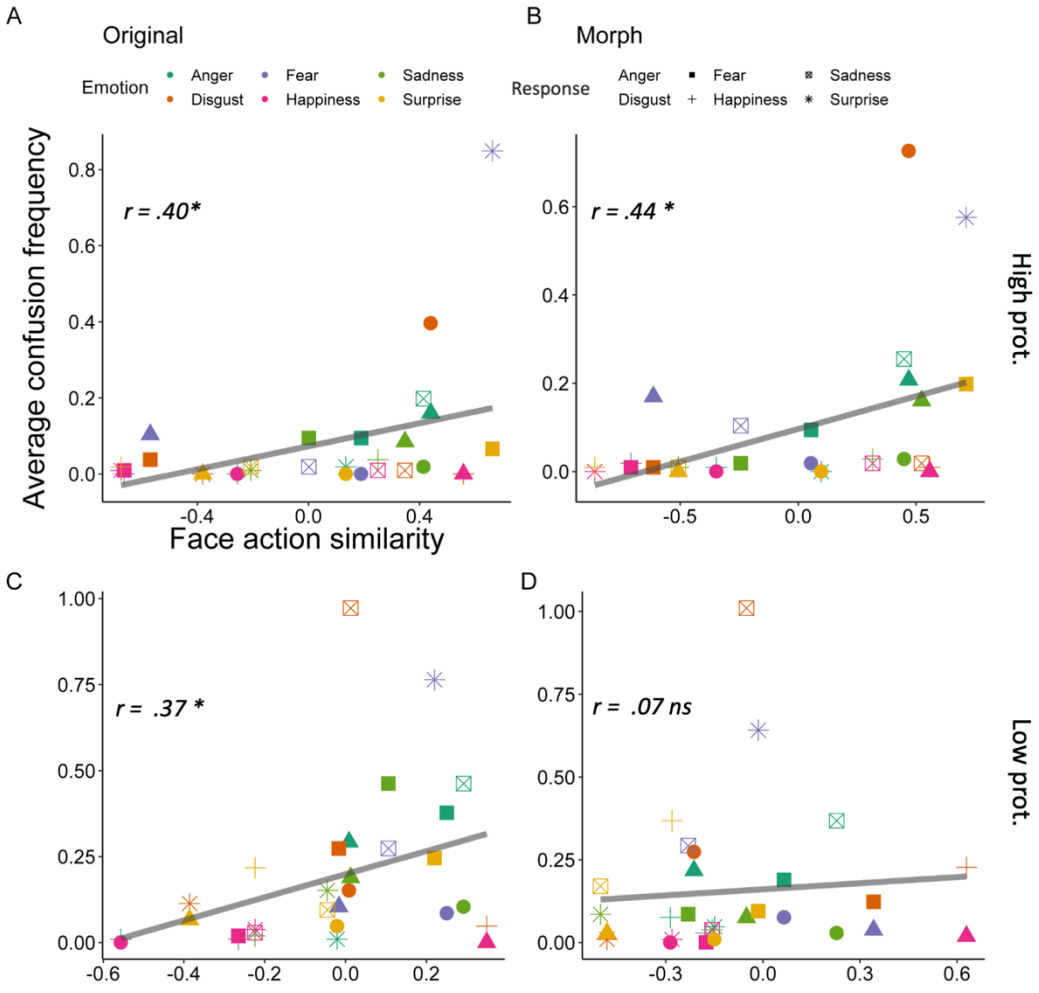

Figure 3.11 Face action similarity correlates with confusion between emotions.

While judgements of valence were highly consistent across morphed and original stimuli, judgements of intensity and naturality, as well as recognition accuracy, were less consistent.

Findings from Experiment 2 add to the growing body of work suggesting that different emotions have different movement profiles and evolve over different timescales (Cook et al, 2020, Jack et al., 2014). Although only two stimulus sets were included (high and low prototypicality), it is likely that the differences found in the dynamics of the expressions between stimulus sets would extend to other stimulus sets. This is likely given that stimulus sets differ according to whether they contain spontaneous or posed expressions; and, when

posed, whether expressions are FACS-instructed or natural; and these differences will be reflected in artificial dynamic facial expressions when based on human motion (Dobs et al., 2014; Krumhuber et al., 2017). Furthermore, analyses show that simply adding linear motion (through morphing) to create the appearance of a dynamic facial expression results in a loss of important non-linear dynamics which may individuate specific emotions.

Experiment 3 demonstrated the importance of non-linear dynamics for emotion perception judgements when comparing morphed stimuli with their video equivalents. For example, while overall accuracy of emotion recognition was broadly similar when morphed and original stimuli were compared (though markedly less so for the low prototypicality stimulus set), and valence ratings were broadly consistent, intensity and naturality ratings were not consistent across morphed and original stimuli. Furthermore, the pattern of confusions resulting from errors observers made in recognising emotions varied as a function of stimulus set and stimulus type. The distinction between the effects of morphing (and thus of altering the temporal characteristics of expressions) on valence and intensity ratings is consistent with claims that intensity judgements rely more on temporal information than valence judgements, which rely more on spatial information (largely preserved by morphing; Chen et al., 2020).

The current findings go beyond previous observations of reduced recognition accuracy for morphed expressions (Korolkova, 2018), and preference for non-linear over linear motion profiles (Cosker, Krumhuber, & Hilton, 2015). The similarity analyses highlight high levels of individual variability in intensity and naturality judgements, and confusion similarity patterns, across stimulus sets and emotions (see Figure 3.9-B-D, and Figure 3.10-D). These findings raise important questions regarding the generalisability of findings from studies of emotion processing from faces, when the majority of studies are limited in how well they capture the spectrum of dynamics that make up emotional facial expressions.

Low consistency of judgements across morphed and original stimuli suggests that emotion processing is optimised for the expected dynamics of moving expressions, as disruption of dynamics results in judgement inconsistency (particularly for intensity, naturality, and recognition accuracy). This result is compatible with the idea of representation of motion within face space (Furl et al., 2020), and with observers' preference for reconstructed linear and spline approximations of timecourse dynamics varying systematically by emotion (Dobs et al., 2014). Results also show that highly prototypical expressions are judged as less natural than their morphed counterparts, highlighting those preferred dynamics do not only vary by emotion, but also vary by the type of expression (high or low prototypicality). This results also suggest that highly prototypical stimuli do not benefit from a 'dynamic advantage' when processed, given that morphing was expected to impoverish naturalness due to removal of important temporal dynamics of the stimuli.

These findings have potentially important implications. In addition to affecting behavioural ratings, variability in the dynamics of facial expressions is likely to impact cognitive and neural mechanisms implicated in processing of emotion and identity from faces. For instance, the enhanced neural activity linked to dynamic facial expressions (Sato, Kochiyama, Yoshikawa, Naito, & Matsumura, 2004; Zinchenko, Yaple, & Arsalidou, 2018) may well be modulated by factors such as the rate with which facial actions evolve, or with the predictability of face movements (Yoshikawa & Sato, 2008), all of which vary by stimulus and stimulus set.

In the same vein, it is worth noting that while Experiment 2 demonstrated systematic variance in face action dynamics that evolve over time, Experiment 3 utilised single judgments recorded after the entirety of the expression had been viewed. Studies investigating perception of transitions of dynamic expressions have shown that dynamics impact ongoing

behavioural ratings (Falagiarda & Collignon, 2019; Korolkova, 2018). Future studies could therefore benefit from including dynamic measures of emotion processing. One could investigate how varying face dynamics impact continuous visual attention, eye-movements, pupil fluctuations or physiological arousal, perhaps in combination with the use of reverse correlation and mutual information techniques to reveal how information about the dynamic quality of face movements is integrated into perceptual judgements of emotion (Chen, Messinger, Duan, & Ince, 2020; Jack & Schyns, 2017).

The current study offers an approach to selecting dynamic face stimuli for research and reveals the potential pitfalls of not accounting for the role of dynamics on behavioural or neurophysiological responses to emotional expressions. Perhaps above all, it highlights that it would be useful for the field to decide what is required for an expression to be 'dynamic', while not assuming that all dynamic stimuli are equally ecologically valid. For example, it could be argued that repurposing static stimulus sets through morphing is not enough for them to be considered 'dynamic' nor does it confer any "dynamic advantage", as the use of morphed stimuli does not provide information about the dynamic quality inherent to various emotional expressions. This does not imply that morphed stimuli lack utility – they are useful for parametric manipulations of stimulus features (e.g., Cook et al., 2009; Gray et al., 2020) – but such stimuli might be less likely to engage the neural and perceptual processes that support dynamic face processing in face-to-face communication.

One way of addressing the impact of the variability in stimuli is to sample more widely across stimulus sets varying in dynamics in order to increase the generalisability of findings. Another suggestion is to use data-driven approaches to generate stimuli, where spatiotemporal parameters are sampled from the whole spatiotemporal space (e.g., Yu, Garrod, Schyns, 2012). Similarly, photorealistic rendering or retargeting of face movements

based on machine learning approaches trained on naturalistic face movements may help overcome these issues, although they are likely to require increased inter-disciplinary collaboration (Krumhuber et al., 2018). However, it is important that these methods are able to incorporate the individual differences in expressivity and face movements which are marked in real life (Cohn, Schmidt, & Gross, 2002; Friedman & Riggio, 1981; Whittaker, Rogers, & Petrovskaya, 2021), and are used by observers to, for example, perceive identity in conversational expressions (Dobs et al., 2016). These considerations are also likely to be crucial to transfer knowledge of emotion expression production and processing to equip digital agents and social robots (Whittaker et al. 2021).

Another more readily available solution is for researchers to quantify dynamics in facial expressions stimuli used for research. This would enable researchers to report summary information regarding these dynamics, from low level motion (e.g., motion energy), to kinematics (e.g., speed) and semantic dynamics describing aggregated and timecourse metrics of action units (or face actions, as used here). It should also be emphasized that more research is needed on how these different levels of dynamics affect perceptual processes more broadly, and whether any emotion/expression or face–to-face communication effects can be dissociated from general effects of low- and high-level dynamics. Thanks to the proliferation of face and motion tracking algorithms (e.g., OpenFace, Baltrusaitis et al., 2018; blenderFace, Zinkernagel et al., 2018) this is achievable. Even if the accuracy for action unit detection (e.g., based on FACS) is still typically below the capabilities of commercial algorithms or human observers (Dupré, Krumhuber, Küster, & McKeown, 2020), they are likely sufficient.

Making it routine to analyse and report the spatiotemporal dynamics of stimuli would also allow to select stimuli in a more informed manner. Perhaps the biggest ensuing benefit is that

this information can be regressed onto other variables akin to the practice of regressing natural image statistics (e.g., color, intensity, higher order: edge, contours) onto perceptual processes and neural representations (Simoncelli & Olshausen, 2001).

Open Practices Statement: Materials and data will be made available with publication at https://osf.io/gk3xf/.

Supplementary Materials: **8.2.1 Experiment** 2

3.2 EXPERIMENT 4. Alexithymia differentially modulates the effect of spatial-kinematics and task on visual processing of emotional expressions in Autism

Abstract

One of the key tasks when observing or participating in face-to-face interactions is to decode transient non-verbal communicative signals such as facial expressions and gaze direction. Recent research has demonstrated the importance of the spatial-kinematics of observed facial actions in shaping observers' inferences about facial actions. Additionally, research on gaze control has highlighted that visual exploration is strongly modulated by task or stimuli-related priors. If these priors influence processing of the spatial-kinematics of facial actions, it may explain why individuals with autism – who are thought to be less influenced by priors – show atypical social attention and a deficit in the recognition of emotional facial expressions. However, alternative hypotheses argue that atypical social attention and emotion recognition in autism is driven by alexithymia, a frequently co-occurring condition in autism which is rarely accounted for. This study (N = 100) used a novel gaze location estimation paradigm to quantify the extent to which the spatiotemporal characteristics of facial expressions influence passive and active gaze behaviour of autistic and non-autistic participants, across various emotion processing tasks (passive-view, emotion-recognition, cued-gaze, and intensity ratings). Results provide evidence that gaze allocation to facial expressions is determined by both the spatial-kinematics of stimuli and task-related priors. Furthermore, this study showed that the influence of spatial-kinematics and priors on gaze behaviour can vary according to clinical predictors. Specifically, in line with the alexithymia hypothesis, alexithymic traits modulated the relationship between spatial-kinematics, task, and gaze during emotion

processing. These results suggest that reduced influence of priors, when applied to social attention in autism, may actually reflect alexithymia and not autism.

Keywords: emotion, face, dynamics, autism, alexithymia, priors.

3.2.1 Introduction

One of the key tasks for observers or participants in face-to-face interactions is to attend to, and to decode, transient communicative signals including non-verbal information like facial expressions and gaze direction. These non-verbal cues have been shown to shape social perception at very early stages of processing (Willis & Todorov, 2006), and as such these processes are thought to be key to engage and maintain successful social interactions (Jack & Schyns, 2015; Riggio, 1992). In support of this proposition is that non-verbal communication deficits have been implicated in atypical social functioning in autism. For example, as discussed in previous chapters, it is thought that the ability to process facial expressions is impaired in autism (Uljarevic & Hamilton, 2013), and this has traditionally been explained in terms of atypical social attention, such as atypical gaze to social cues during face-to-face communication (Dratsch et al., 2013; Guillon, Hadjikhani, Baduel, & Rogé, 2014; Senju & Johnson, 2009). Therefore, understanding the mechanisms driving atypical gaze is potentially relevant to understand and devise interventions for social difficulties in autism.

Research on both gaze allocation to faces and emotion recognition in autism has, however, produced mixed results. Many studies have reported some emotion recognition deficits and/or atypical gaze behaviour (Grynszpan & Nadel, 2014; Kirchner, Hatri, Heekeren, & Dziobek, 2011; Kliemann, Dziobek, Hatri, Steimke, & Heekeren, 2010), while others find typical emotion recognition and visual social attention (Rutherford & Towns, 2008; Tang, Chen, Falkmer, Bölte, & Girdler, 2019). Despite emotion recognition and social attention being two of the most studied aspects of autism, many reviews and metanalyses highlight the

inconsistency of results (Cuve et al., 2018; Harms et al., 2010; Lozier, Vanmeter, & Marsh, 2014).

A possible explanation for the inconsistency of findings across studies are the many methodological differences across those studies, including task demands (e.g., emotion recognition vs passive view), and stimulus characteristics (e.g., dynamic vs static; posed vs naturalistic; for a review see Cuve et al., 2018, Harms et al., 2020, Lozier et al., 2014). Yet, the extent to which the mixed evidence can be attributed solely to methodological factors remains unclear. Some argue that the mixed evidence underscores important individual variability relevant to emotion processes within the autistic spectrum. In particular, individual differences in alexithymia, a condition that frequently co-occurs with autism (Kinnaird et al., 2019) and affects the ability to describe and interpret one's own feelings and bodily states (Sifneos, 1973), have been recently proposed to explain atypical emotional processing (Bird & Cook, 2013; Cook, Brewer, Shah, & Bird, 2013) and gaze behaviour in autism (Geoffrey Bird, Press, & Richardson, 2011; Cuve, Castiello, et al., 2021) – see also Chapter 2. Nonetheless, autism research has generally failed to control for alexithymia (and alexithymia research for autistic traits), making it difficult to test the specificity of emotion processing problems to either condition.

Recent models of autism have also argued that atypical integration of sensory input and task-relevant priors may underlie atypical perceptual experiences (Pellicano & Burr, 2012). This hypothesis builds on a specific interpretation of Bayesian models of perception discussed in Chapters 1 and 2. At their core, such models argue that perceptual decisions are influenced by both incoming sensory information and prior knowledge which exert a top-down influence on sensory processing. As described by Helmholtz, retinal images are inherently ambiguous, and therefore perception is an unconscious inferential process that necessitates both the

immediate sensory experience as well as prior knowledge from past percept's (Helmholtz, 1867). In the context of autism, the Bayesian hypothesis proposes that deficits in the integration of sensory evidence and priors may cause atypical perception and sensory experiences. While some versions prescribe hypo-priors as the exact mechanism (Elizabeth Pellicano & Burr, 2012), which results in an overreliance of sensory information and reduced reliance on prior knowledge, deficits in the integration of sensory information and priors have been also proposed (e.g., precision weighted integration) (Coll, Whelan, Catmur, & Bird, 2020; Friston, Lawson, & Frith, 2013).

The Bayesian hypothesis provides an account of general sensory abnormalities in autism (Lawson et al., 2017), and while it has also been applied to visual processing (Król & Król, 2019; Maule et al., 2017), only a few studies have considered it in the context of social attention and visual processing of non-verbal cues like gaze direction. These studies have provided mixed results in terms of supporting the Bayesian hypothesis or not (Pantelis & Kennedy, 2017; Pell et al., 2016). Nevertheless, there is compelling evidence for the importance of both 'bottom-up' stimulus driven effects on emotion recognition and gaze towards facial expressions, and of the effect of 'top-down' priors on the same processes. Thus, considering these processes in autism within a Bayesian framework is sensible. With respect to stimulus-driven effects, socioemotional non-verbal cues like dynamic facial expressions are rich in sensory information, particularly in terms of their spatial-kinematics (Jack & Schyns, 2015). As shown in Experiments 2 and 3 in this chapter, these spatial-kinematics influence perceptual judgements of dynamic facial expressions across dimensions such as valence, naturality, intensity and emotional categorisation. Manipulation of spatial-kinematics via increase or decreases in speed, has also been shown to causally impact observers' attributions of anger and sadness to facial movements (Sowden et al., 2021).

With respect to the existence and importance of priors, given the rich and diverse experience that humans have with faces it's plausible that observers have priors that are organised as perceptual representations that account for how faces move across time to convey various social messages (Furl et al., 2020; Jack, Caldara, & Schyns, 2012). These prior representations may encompass associations between spatial-kinematics (e.g. speed) and valence (e.g., rapid face movements - danger) or facial actions and emotion categories (e.g. expression prototypes), or the informative value of various facial actions to various social processing decisions (e.g. that eyes can be used to differentiate disgust and anger) (Wiese, Wykowska, & Müller, 2014).

Top-down effects on visual processing are well-recognised as discussed in Chapter 2, for example, early work showed that an observer's gaze to a complex social scene is dependent on task instructions (Yarbus, 1967). Computational work has shown that many patterns of gaze behaviour can only be accounted for by models which integrate task modulation effects on gaze control (Tatler et al., 2011). Top-down effects have also been shown specifically in the processing of faces (Rolls, 2008), including findings that visual exploration of faces is modulated primarily by task instructions rather than stimulus factors (Hessels, Holleman, Kingstone, Hooge, & Kemner, 2019). Furthermore, the ratio of eye to mouth gaze (thought to be a crucial variable in social attention) has been shown to vary depending on whether observers need to judge the intensity of an emotion, or recognise which emotion is portrayed (Tsang, 2018).

The effect of stimulus-driven sensory information and priors (stimulus and task-related) can be estimated from how they modulate visual processing of emotional faces. Interestingly, results of a few studies already suggest that gaze allocation in autism and alexithymia can be explained in terms of atypical modulation by task and stimulus-related priors (Cuve,

Castiello, et al., 2021; Del Bianco, Mazzoni, Bentenuto, & Venuti, 2018; Loth, Gómez, & Happé, 2010) – see also Chapter 2. Nonetheless, the quantification of the stimulus-driven sensory information and task-related priors in these studies was limited. For example, faces were either static with no dynamic information, or their dynamic range was limited (as was the case in Chapter 2). Nonetheless, as shown in the previous two experiments in this Chapter, facial cues of emotion vary drastically in their dynamic properties. Furthermore, while posed expressions provide clear communicative signals with clear patterns of spatial-kinematics , spontaneous expressions tend to exhibit more complex dynamics (Cohn & Schmidt, 2004; E. G. Krumhuber, Küster, Namba, Shah, & Calvo, 2021; Namba et al., 2017)**.** Nonetheless**,** successful social communication relies on the ability to process a wide range of dynamic signals communicated using facial expressions, from spontaneous to purely communicative expressions which may, in fact, be posed (e.g., when people try to portray an emotion for communicative purposes). Ensuring that the whole range of dynamic properties of facial expressions are captured is therefore important to estimate its influence on visual processing. This is also important to ensure a more valid and generalisable measure of emotion recognition (Barrett, Adolphs, Marsella, Martinez, & Pollak, 2019), especially in autism where different types of stimuli appear to show different effects on different subgroups of autistic participants (Harms et al., 2010).

The current study therefore investigated whether atypical gaze and emotion recognition of facial cues in autism and alexithymia reflect differential modulation by sensory evidence and priors in line with Bayesian accounts. Sensory evidence here is characterised by the spatial-kinematics representing dynamic information of how the various facial cues evolve over time, derived using the approach developed in Experiment 2. Priors here are characterised by the experimental conditions in which facial cues are presented, namely passive view, emotion recognition, and cued gaze (as in Chapter 2). Thus, priors are given by the task goal and the

presence or not of an emotion cue (emotion label) to the facial cue. If participants have task and emotion-related priors, then gaze in these conditions should differ compared to passive viewing. If stimulus-related priors are utilised, then knowing the upcoming emotion should change gaze to the expression in a confirmatory fashion (see Chapter **2** and Cuve et al., 2021). Contrasting both autism and alexithymia will allow testing of the specificity of effects attributable to these individual difference factors.

3.2.2 Methods

3.2.2.1 Participants

A final sample of 100 participants were included in this study after exclusions for data quality issues (see below). 72 (56 female) participants were neurotypical participants recruited from prolific and 28 (16 Male) were autistic participants from a volunteer database. All autistic participants had an official diagnosis confirmed via an official letter and an ADOS assessment with a licensed practitioner in the research team. All autistic participants also scored above the AQ-28 cut off 70 (Hoekstra et al., 2011) – see also below. Groups were age-matched (NT: M = 29.8[18 – 60] ASD M = 30[19-56]). All participants reported normal or corrected-to-normal vision, were resident in the United Kingdom and were fluent in English. All participants were financially compensated for their time. All research procedures were approved by the Medical Sciences Ethics Committee (IDREC) – reference: R60697/RE004 and were in accordance with the revised 2013 Declaration of Helsinki.

3.2.2.2 Procedure

Participants completed the experiment entirely online via Gorilla (Anwyl-Irvine et al., 2020). The experiment lasted approximately half an hour, with none of the accepted participants exceeding an hour. Once the participant had completed a rudimentary bot check they were

then asked if they would consent to use eye-tracking software which utilised the participant's webcam. Participants who consented to eye-tracking via webcam, first calibrated the software and then both groups completed the facial expression viewing tasks (online camera eye-tracking data is reported elsewhere – see also Gaze Estimation section). Following this, participants completed a series of questionnaire measures, namely the TAS-20, AQ-28 and DASS21 as well as a short version of the WASI IQ test (these measures were detailed in Chapter 2). The order of the individual questionnaires and the WASI test were randomised according to a Latin square design.

3.2.2.2.1 Task and stimuli

In a series of trials, participants were presented with emotional dynamic face stimuli from various datasets, extensively validated in a recent project by both human observers as well as machine learning algorithms (Krumhuber et al, 2020). The use of videos from different datasets aimed to achieve a set of stimuli that displayed a broad heterogeneity across relevant stimulus parameters (e.g., posed/spontaneous, spatiotemporal dynamics) and to mirror naturalistic face processing that generalises across stimuli types (Barrett et al., 2019). Due to the established similarities in expression between disgust and anger, as well as fear and surprise (Jack et al, 2014), only the expressions of happiness, sadness, disgust, and fear were used.

3.2.2.2.2 Gaze estimation task

A new gaze estimation method adapted from Rudoy and colleagues (Rudoy et al, 2012) was used to estimate gaze allocation during the online task[2]. This method can be leveraged with tasks where gaze follows stereotypical patterns, and stimuli are sparse and contain easy to

[2] This alternative method was used because traditional lab-based eye-tracking could not be used due to Covid-19 related restrictions. Piloting using web-based eye-tracking solutions (relying on participants' webcam) available at the time also showed large heterogeneity in data quality and reliability.

segment features (e.g., faces). It relies on flashing grids of digit/letter pairs at certain times during the stimuli and relying on participants' self-reports of the pair they were fixating to estimate the attended locations. In the current implementation, face landmarks retrieved from a face-tracking open-source software - OpenFace (Baltrušaitis et al, 2018) were used to overlay a grid of digits, centred around the middle of the face (approximately the nose region; see **Figure 3.12**). The time of presentation for the grid was systematically varied so that whilst the exact presentation time was random, a third of the stimuli had grids presented early, a third in the middle portion of the stimuli, and a third in the late portion of the stimuli. Pilot studies were conducted with multiple repetitions of the grid per stimuli, and showed that presenting only one grid per stimulus, at varying times, had the best trade-off in terms of not affecting performance of the main task and reliability of gaze location estimation. By randomising the grid onset time across stimuli and participants, the task captures gaze across the whole spectrum of motion on average.

In all trials, participants were shown a fixation cross followed by a video stimulus such as that shown in the figure below (Task). Participants were then asked to identify which portion of the face they were looking at when the grid was presented by remembering the digit/letter pair they saw, as shown in the figure below (orthogonal gaze estimation task).

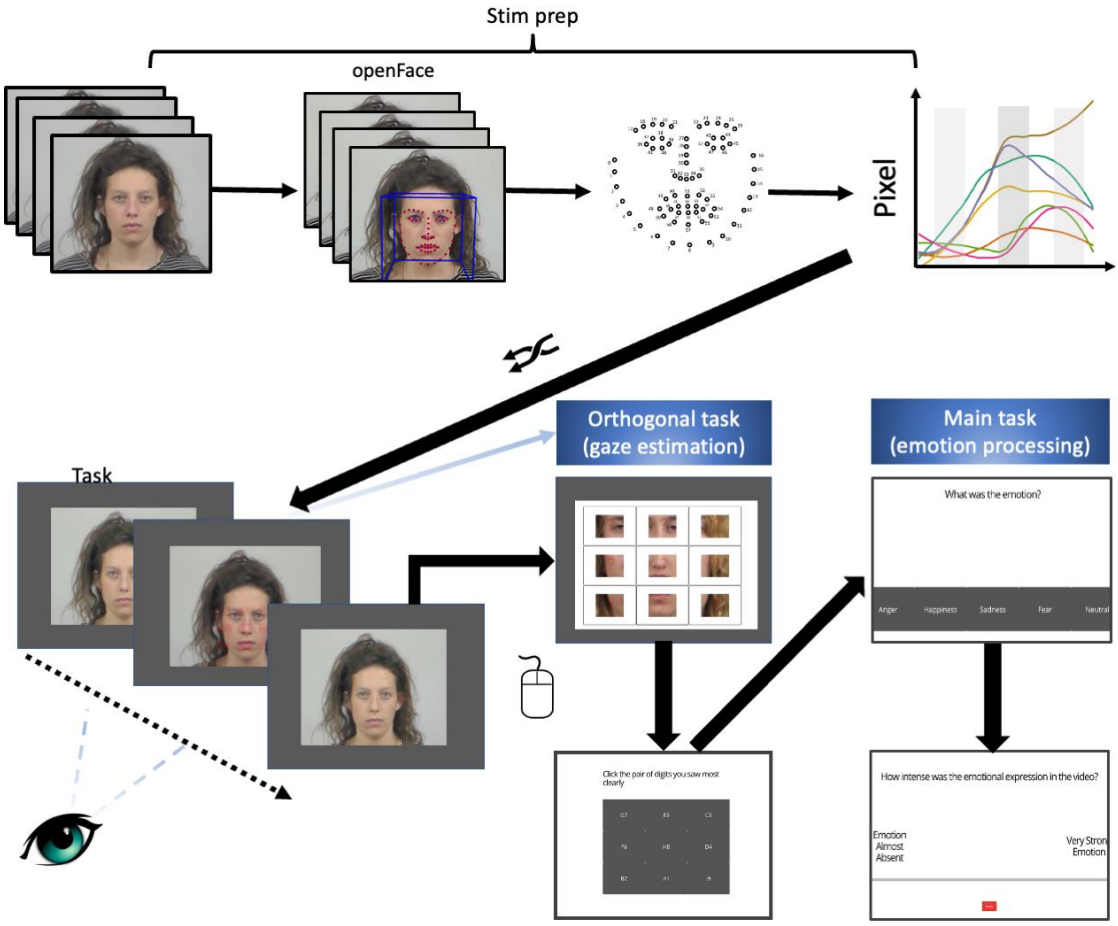

Figure 3.12 Task and gaze estimation approach.

Participants were shown the same letter/digit pairs (A1, B2, C3…) in a randomised order for each trial for 750ms at some point during the expression video. The text was translucent and red (based on piloting tests of readability). Once the video had finished participants were presented with a screen of the face split into 9 different areas. The image used for this was the first frame of the video, chosen to prompt the participants without giving any emotional information. The participant then had to choose the location of the letter/digit pair they saw and which pair in the randomised grid of digits shown. These responses were then used to score whether the chosen pair matched the location based of the grid position presented during the video. This information was used to compute a metric of gaze direction in terms of distances to the various facial features (eyes, nose, and mouth).

This was achieved by showing the participant the face split into 9 different areas. Once the participant clicked the location of the digit they saw most clearly, they were then asked to click the digit pair they saw most clearly. The location of the digits in the grid was used to judge whether the combined selection of digit response and location was correct when compared to the original position of grids and letters during the video. For example, if A1 was presented in the region the participants said they were looking in, the participant must then select A1 as the digit-pair they saw in order for the selection to be recorded as correct. A continuous vector was calculated to reflect the Euclidean distance between the digit the participant clicked and the coordinates of the eyes, nose, or mouth. This measure was used as a proxy for gaze allocation in the analyses (see also **3.2.2.4.3**).

Trials were split into three condition blocks (passive view, emotion recognition, cued), each with 16 trials (4 per emotion) as well as 3 practice trials. First, participants took part in a passive view condition, where they were simply to pay attention to the faces and report where they were looking when the grid was presented. This served as a baseline to which the other conditions can be compared. In the second condition (emotion recognition), after the necessary gaze location estimation questions, participants were asked to report the intensity of the expression they saw (on a scale of 1-100) as well as the emotion on display (choice between anger, happiness, sadness, fear and neutral). In the third and final block (cued), participants were cued with the emotion they were about to see and at the end were asked the intensity of the expression they saw. The condition order was fixed, but the order of stimuli was pseudo-randomized and each block had an approximately even distribution of relevant features (gender, intensity, emotion, grid presentation time).

3.2.2.3 Questionnaire measures

Participants also completed the Toronto Alexithymia Scale (TAS-20), Autism Spectrum Quotient - AQ28, Depression Anxiety and Stress scale - DASS21, and Weschler Abbreviated Scale Intelligence Test – WASI, already described in Chapter 2.

The neurotypical and control groups were matched on the range of alexithymia scores (TAS-20), despite a lower average score for the control group (which included more participants with low alexithymia scores). Participants were also matched for age and IQ. Additionally, participants were given a questionnaire specifically focusing on current COVID-19 stressors to filter out participants who may have completed studies with significant mental health problems during the pandemic.

3.2.2.4 Analytical approach

3.2.2.4.1 Quantifying spatial-kinematics of facial expression stimuli

Facial expressions can be described by spatiotemporal properties of corresponding face actions, namely displacement (amplitude of facial actions) and kinematics (e.g. speed of face actions). Similar metrics have been used in several studies (Dobs et al., 2014; Furl et al., 2020; Jack, Garrod, & Schyns, 2014; Sowden et al., 2021), and also in Experiment 2 in this chapter. I will refer to these dynamics as spatial-kinematics.

The first stage in determining these metrics was calculating static distances between different regions of the face. Utilising OpenFace (Baltrušaitis & Robinson, 2016a), the coordinates of key areas of the face were identified – see **Figure 3.13.** The Euclidean distance between key areas was then determined. For the purposes of this study, eight distances were computed, one for the whole face (D1), three for the eyes (D2, D3, D4), one for the nose (D5), and three for the mouth (D7, D8, D9) (labelled for consistency with Experiment 2). In line with the focus on gaze to face regions, however, the selected face distances represented specific face

actions for eyes nose, and mouth (D2, D3, D4 for eyes and eyebrows, D5 for nose, D8, D9 for mouth (excluding D1 and D7) – see **Table 3.1.** As in Experiment 2, displacement was then computed based on inter-frame distances normalised to the first few frames (mean of the first 5 frames). For temporal kinematics, the distances from each frame were taken to calculate the derivative to determine the speed of motion in the face. These measures capture how fast or slow face actions change over time. Displacement and speed kinematics for the entire video as well as for the letter/digit grid periods were used to estimate gaze location distances. Spatial-kinematic measures were normalised within each stimulus such that they reflect a change relative to the dynamic range of each actor and to remove inter-individual differences.

Table 3.1 Facial distances

Action code	Descriptive action
D1	Overall face action
D2	Eyebrow widening
D3/D4	Eye open/closing
D5	Nose lengthen/widen
D7	Upper lip/nose raise
D8	Mouth widening
D9	Mouth opening/closing

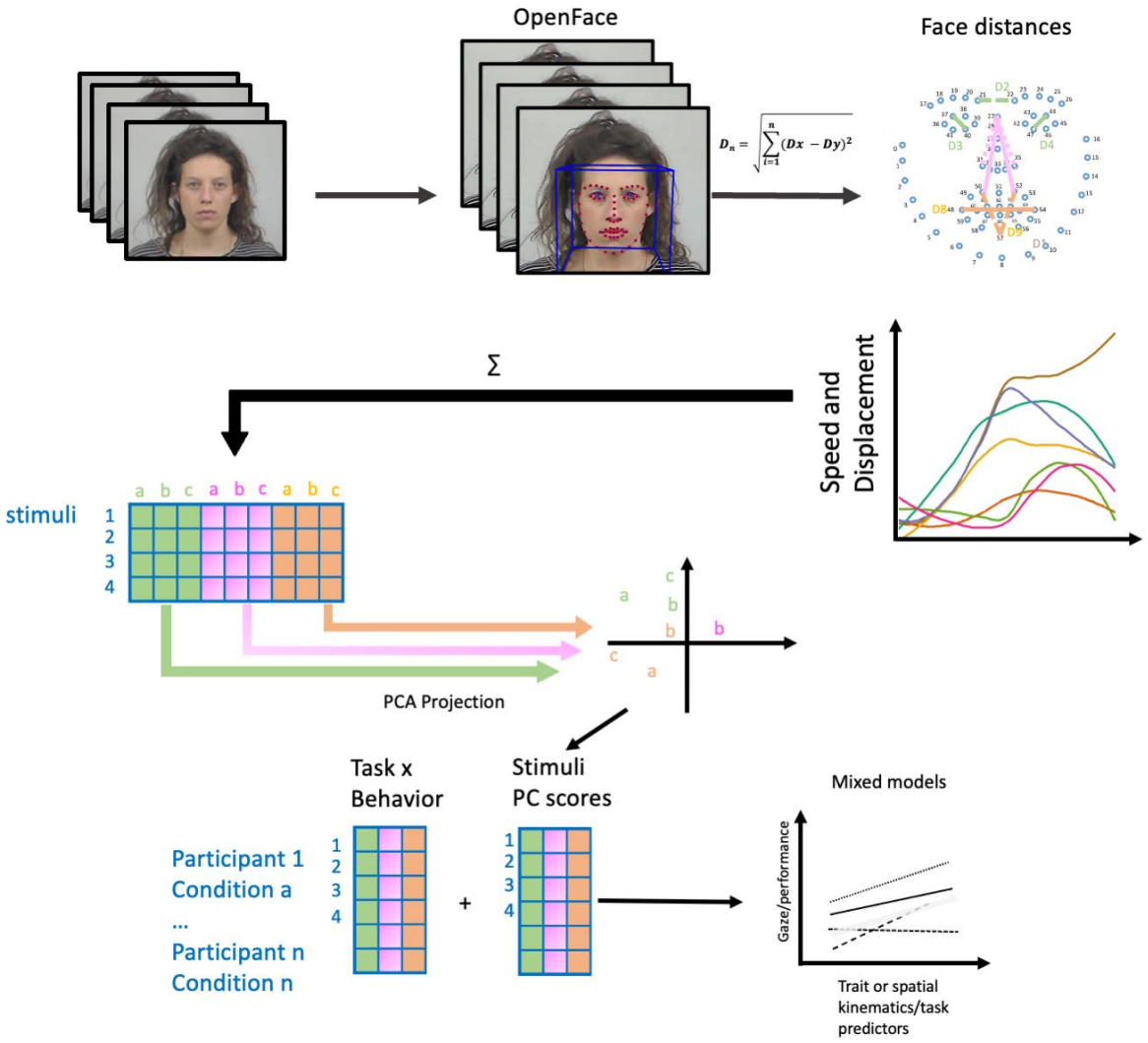

Figure 3.13 Illustration of the analytical approach.

Face landmarks were used to compute distances representing semantic activations of face actions (e.g., mouth opening, eye closing) which were used to derive spatial-kinematic parameters (displacement and speed). Using PCA, important spatial-kinematic variables were identified, and component scores were assigned to each stimulus and used for regression analyses along with participant and task-relevant predictors.

3.2.2.4.2 PCA on spatial-kinematics of facial expressions

The spatial-kinematics of face regions are not completely independent, as various facial expressions can be described in terms of temporal co-activation of various facial actions (Krumhuber, Manstead, & Kappas, 2006). Nonetheless, not all face actions are likely to contribute equally or to be equally informative to the covarying patterns of facial activity. Therefore, an inferential PCA was used to determine which spatial-kinematics variables are important such that they significantly contribute variance to the observed patterns of coactivation of emotional expressions. This also had the practical advantage of allowing a reduction of potential multivariate patterns of coactivation into a lower-dimensional space in a data-driven fashion, without assumptions about which facial actions are relevant or how they cluster with other face actions. Furthermore, scores for each stimulus on each projected component can be used in regression analysis, which is preferable to having to regress all individual spatial-kinematic variables, which would make interpretation unnecessarily complex, in addition to introducing multiple comparison problems in the absence of specific predictions for all individual spatial-kinematic variables.

The analyses were conducted using the inferential PCA battery of tests implemented in the TinPosition package in R (D Beaton, Rieck, & Abdi, 2013). In these tests, the significance of variable contributions to components is determined via bootstrapping and permutation tests. The PCA solution was used to describe the pattern of spatial-kinematics of facial expressions and as the basis for testing the effect of spatial-kinematics on gaze allocation and emotion processing performance, along with clinical trait predictors and task conditions.

3.2.2.4.3 Regression analyses of gaze behaviour and emotion processing

Because of the non-normality of residuals observed when attempting to predict gaze distance metrics (a proxy for gaze allocation), robust mixed models were fitted in R using the robustlmm package (Bates, 2010; Koller, 2016). Specifically, mixed models were fitted to predict gaze location (at the grid presentation moment) to eyes, nose, and mouth, separately, from spatial-kinematics, clinical traits, and task conditions. All models were fitted with a random intercept for participants and stimuli using the maxima of parsimonious modelling (Bates, Mächler, Bolker & Walker, 2014). For example, the model predicting gaze to the eye region had the form:

*Distance from gaze location to eyes ~ 1 + task * (spatial-kinematics components) * (Alexithymia + Autism traits) + (1 | Stimuli) + (1 | Participant).*

Where 1 is the overall intercept, task (passive, emotion recognition, cued), spatial-kinematics (stimuli scores from the PCA) and clinical predictors (main effects and interactions) are fixed effects, and stimuli and participant IDs were included as random intercepts.

The main effects represent the direct effect of task, spatial-kinematics, or clinical predictors on gaze location. Interactions between task and spatial-kinematic components can be interpreted as differential modulation of the effect of sensory information (stimulus dynamics) on gaze by top-down priors (task). Likewise, interactions between clinical predictors and spatial-kinematics or task, indicate that alexithymia (or autism) is related to different modulation of gaze based on sensory information or atypical task- and emotion-relevant priors. Similar models were fitted to predict emotion recognition performance and intensity ratings.

3.2.3 Results

3.2.3.1 Preliminary validation of gaze location estimation

The data from the gaze location estimation task suggests that the grid method is capturing expected patterns of visual exploration to faces. Specifically, participants looked more at the eyes than mouth during passive viewing (Estimate = 0.178, SE = 0.047, df = 140, t= 3.756, p < .001). They also looked more to the eyes than the nose (Estimate = 0.153, SE = 0.048, df = 140, t = 3.224 p = 0.005), whereas no differences were observed between nose and mouth regions (Estimate = 0.025, SE = 0.0475, df = 140, t= 0.531, p = 0.8562). In contrast only for the emotion recognition task, the probability of looking to the nose region was higher than the mouth region (Estimate = 0.123, SE = 0.047 , df =140, t = 2.580, p = 0.029). No differences in gaze allocation were observed between the cued and emotion recognition conditions - see **Figure 3.14 Grid task results.**). These results resemble patterns observed in gaze behaviour obtained using standard eye-tracking (see also **Figure 2.2. Aggregated gaze plots in Chapter 2**) also reported in (Cuve et al., 2021, Peterson & Eckstein, 2012; Schurgin et al., 2014).

Crucially, the matching accuracy for the grid task (assigning the correct location to the correct letter/digit pair) was above chance levels. This indicates that participants were not simply choosing digits and locations at random (see **Figure 3.14**). Additionally, the grid task did not interfere with performance on the main face tasks (at least it did not interfere to too great a degree) as illustrated by above-chance performance in the face tasks (Figure 3.14-D). The internal reliability of intensity ratings was also excellent, ranging from alpha = .86 to alpha = .90 which suggests that the grid task did not disrupt the assessment of stimulus intensity. As such, these results indicate the current task is a valid indicator of gaze allocation to faces.

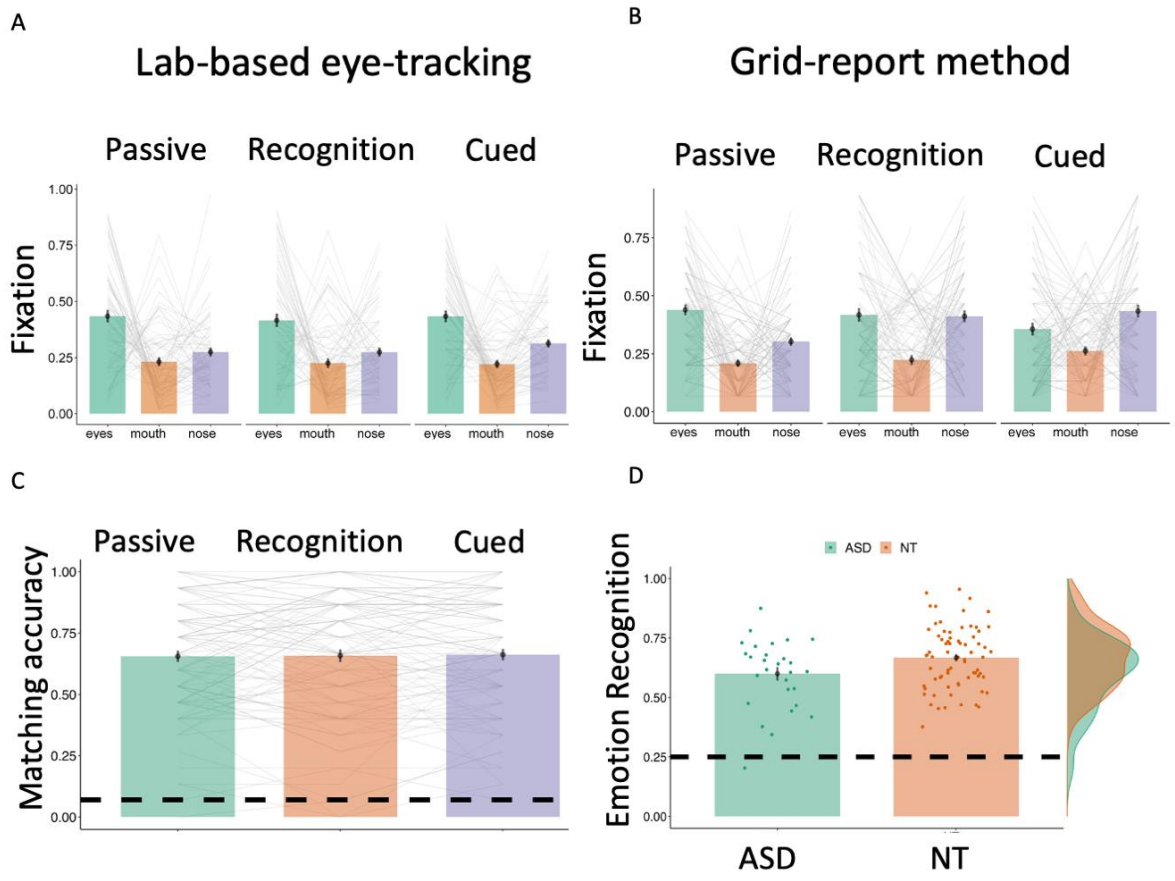

Figure 3.14 Grid task results.

A and B compare gaze patterns obtained from a similar paradigm in the lab with a standard eyetracker (TX300) from Chapter 2 and the grid method for the current study, respectively. C and D show accuracy rates for the gaze estimation task (that did participants remember the location of the digit/letter pair they saw most clearly) and emotion recognition task, respectively. Dashed lines indicate chance levels.

3.2.3.2 PCA on spatial-kinematics

Inferential PCA was used to identify the facial actions and spatial-kinematics variables that significantly contributed to the observed covariance patterns of facial expressions. The PCA solution represents a lower-dimensional space that describes the spatial-kinematics of the facial expressions.

After 1000 permutations, inferential results suggested two significant components, explaining a total of 53.16% of the variance – see **Figure 3.15**. Component 1 (R2 = 32.7%, p = 001) was dominated by negative loadings for all speed variables, and a positive loading for eye and nose displacements. As such this component appears to be largely capturing temporal properties of facial expressions (e.g., speed of change). The fact that all speed metrics loaded on the same component with the same direction suggests that the temporal kinematics of different facial actions tends to covary (see **Table 3.2**). High scores on this component, therefore, indicate slow temporal changes (speed) of the various face actions (because of the negative loadings).

Component 2 (R2 = 20.39%, p = 001) was characterised primarily by facial action displacements. Specifically, eye and mouth displacements loaded negatively. Thus, a high score on this component indicates inward displacements tendencies (e.g., constriction of mouth) whereas low scores indicate outward displacements (e.g., mouth widening, eye-widening). Of note, is that eye displacements (e.g., blinking) and speed for eyebrow activity and mouth opening contributed to both components 1 and 2 - see **Figure 3.15.**

Table 3.2 Loading Matrix for spatial-kinematics

Variable	Descriptive action	PC1	PC2
Speed D2	Eyebrow widening	-0.236405	-0.394881
Speed D3/D4	Eye open/closing	**-0.415505**	-0.273391
Speed D5	Nose lengthen/widen	**-0.439383**	-0.321608
Speed D8	Mouth widening	**-0.383723**	-0.050985
Speed D9	Mouth opening/closing	**-0.407579**	0.2198708
Displacement D2	Eyebrow widening	-0.201942	-0.12053
Displacement D3/D4	Eye open/closing	0.3690079	**-0.372327**
Displacement D5	Nose lengthen/widen	0.2771521	**-0.382609**
Displacement D8	Mouth widening	0.0982993	**-0.543753**
Displacement D9	Mouth opening/closing	0.0403036	-0.1405

Showing 1 to 10 of 10 entries, 10 total columns. Insignificant components were omitted (3 to 10). Variables were computed as peak summaries (maximum activation).

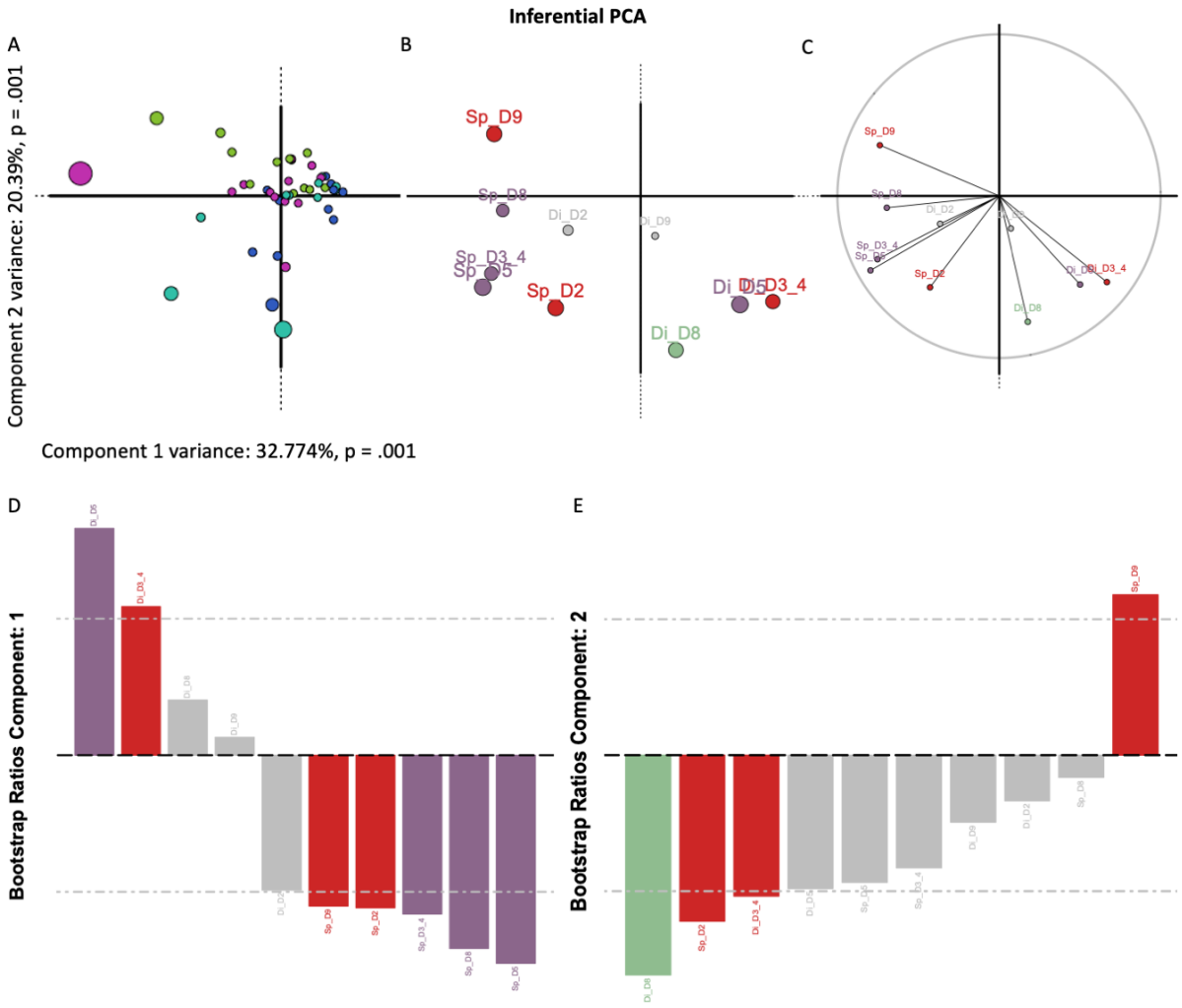

Figure 3.15 PCA results of Spatial-kinematic analyses of facial expressions.

A. Factor map with row scores (stimuli), colour represents different emotions. B. factor map for columns (variables: facial action speed and displacements). The size of the dot represents variance contributed by the variable for the respective dimension. Plum represents variables that load on component 1 (temporal/speed), green load on component 2 (displacement) whereas red contributes to both components. C. Circle plot: variables closer to the circle line contribute more strongly to their respective components. D and E illustrate bootstrap ratio tests for each variable. Gray indicates non-significant variables. The dashed line is the ratio test threshold (similar to a z score).

The PCA scores for each stimulus on each significant component were used as the basis to represent spatial-kinematics (sensory evidence) predictors in regression analyses of gaze and emotion processing (emotion recognition and intensity ratings). To aid the understanding of this PCA solution, it is useful to visualise the component scores for the stimuli used, which were intentionally selected to vary on spatial-kinematics, due to varying underlying dynamics resulting from emotion expression categories and emotion elicitation methods, e.g., posed prototypical vs. spontaneous expressions as discussed in Experiment 2. **Figure 3.16** provides visualisations of the component scores by stimulus types and chosen emotional expressions.

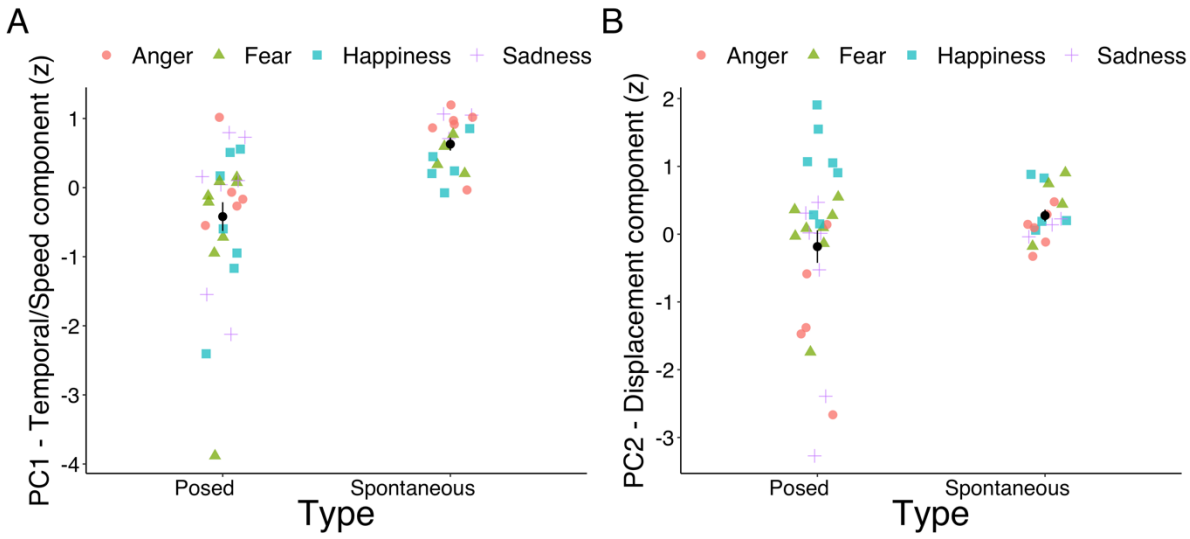

Figure 3.16 Illustration of principal component scores for temporal/speed (PC1) and displacement (PC2) components by stimulus categories.

This visualisation highlights some differentiation of expression and stimuli type. For example, Component 1 shows that spontaneous stimuli are largely characterised by slower changes (positive scores are related to negative speed loadings) and smaller displacements when compared to posed expressions. Similarly, there is a trend for better differentiation of

different emotions in posed expressions compared to spontaneous expressions. As such, the PCA scores can be taken as capturing meaningful variance in facial dynamics.

Note, however, that fine-grained analysis of temporal courses of facial actions over time may offer more sensitive differentiation of stimuli, but this is beyond the scope of the current study. Furthermore, Experiment 2 and other studies have tested for temporal discrimination of facial expressions based on spatial-kinematics (Jack et al., 2014; Krumhuber, Küster, Namba, & Skora, 2021). For the purposes of this study, it suffices to show that the principal component scores capture meaningful covariation in spatial-kinematics of various face actions, such that they can be used as a metric for the dynamic information conveyed by expressions in analyses of how they drive gaze and emotion processing.

3.2.3.3 Effects of spatial-kinematics and task on gaze allocation to facial expressions

3.2.3.3.1 Gaze towards the eye region

When predicting gaze towards the eyes, no main effect was observed for Component 1 which primarily defines temporal coactivation of face actions (Estimate = -8.27, SE = 9.35, t= -0.88, p = .377). However, there was a significant main effect of Component 2 (displacement component: primarily of eye and mouth). Specifically, for high scores on PC2, participants looked closer to the eye region (Estimate = -20.86, SE = 9.35, t = -2.23,p = 0.026). In other words, during rapid face movements and smaller mouth and eye displacements (constriction actions), participants were more likely to attend to the eye region.

There was also a significant effect of condition, indicating that gaze to eyes was significantly modulated by task. Specifically, there were significant pairwise differences between emotion recognition and cued rating conditions (Estimate = -4.55, t = -2.553, p = 0.029, Cued > Recognition). This indicates that gaze distance to eyes was greater in the cued condition

when they knew which emotion they would see, while participants looked closer to eyes in the emotion recognition condition when trying to recognise the emotion. No differences were found for passive vs. emotion recognition (Estimate = 2.49, SE= 1.78, t = 1.402 p = 0.34) or passive vs. cued condition (estimate = -2.05, SE = 1.78, t = -1.157, p = 0.47). There were also no significant main effects of alexithymia (Estimate = -0.38, SE = 2.96, t = -0.13, p = .9) or autistic traits (Estimate = 2.11, SE = 2.96, t = 0.71, p = .47).

Looking at the two-way interactions, there were only two significant interactions between clinical predictors and task, and clinical predictors and spatial-kinematic scores. Specifically, a significant interaction was found between alexithymia and PC1 (temporal/speed component) (Estimate = 1.93, SE = 0.899, t = 2.139, p = 0.03). Slope analysis showed that high alexithymic participants had lower modulation of gaze to eyes by expression kinematics, whereas low alexithymic participants looked closer to the eyes on average with increasing speed of facial actions. No significant interaction was observed between autistic traits and PC1 (Estimate = 0.31, SE = 0.89, t = 0.350, p = .7), which suggests that atypical gaze modulation by spatial-kinematics (sensory information) is primarily linked to alexithymia and not autism (see **Figure 3.17**).

There was also a significant interaction between alexithymia scores and condition (Estimate = 2.490, SE = 1.256, t = 1.983, p = .04). Specifically slope analysis and visualisations indicated significant differences in gaze allocation towards the eye region, in the emotion recognition condition, with alexithymia scores being related to increased gaze allocation away from the eye region, whereas low alexithymic participants looked primarily at the eyes when engaged in emotion recognition (see **Figure 3.17**). No other effects were significant.

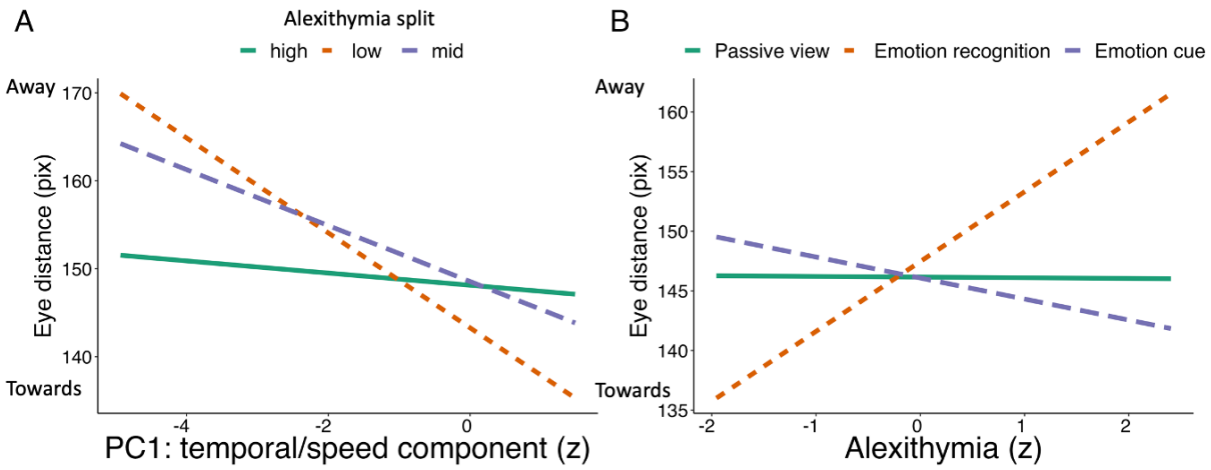

Figure 3.17 Summary results for gaze towards the eye region.

A. Low alexithymia split (n = 36), midsplit (n = 43), high split (n = 21) for visualisation purposes only. Lower values on PC1 indicate higher temporal change (speed). Lower values on the y axis (eye distance) indicate gaze towards the eyes with higher values indicating gaze away from the eyes. B. Alexithymia by condition interaction.

3.2.3.3.2 Gaze towards the mouth region

There were no significant main effects of spatial-kinematics or clinical predictors (autism and alexithymia) on gaze allocation to the mouth region. However, there was a significant main effect of task, suggesting that gaze allocation to the mouth region (like for the eye region) is modulated by task- and emotion-relevant priors. Specifically, participants were more likely to be looking towards the mouth in the cued compared to the emotion recognition (Estimate = 7.07, SE= 2.2, z = 3.21 p = 0.003) and passive viewing conditions (Estimate = 14.39, SE = 2.19, z = 6.56, p <.001) and more during emotion recognition compared to passive viewing (Estimate = 7.48, SE = 2.20, z = 3.33, p = 0.003).

For the two-way interactions, there was a significant interaction between autistic traits and displacement scores (PC2 – mouth and eye displacements) (Estimate = -1.39, SE = 0.11 , p =

0.039). Slope analysis showed that high autistic traits were related to reduced gaze modulation towards the mouth region (see **Figure 3.18**).

Similar to eye region results, alexithymia interacted significantly with condition (Estimate = -3.32, SE =1.53, t = 1.994 p = .03), with higher levels of alexithymia showing less task differentiation effects on gaze to the mouth region (see slope visualisations in **Figure 3.18**).

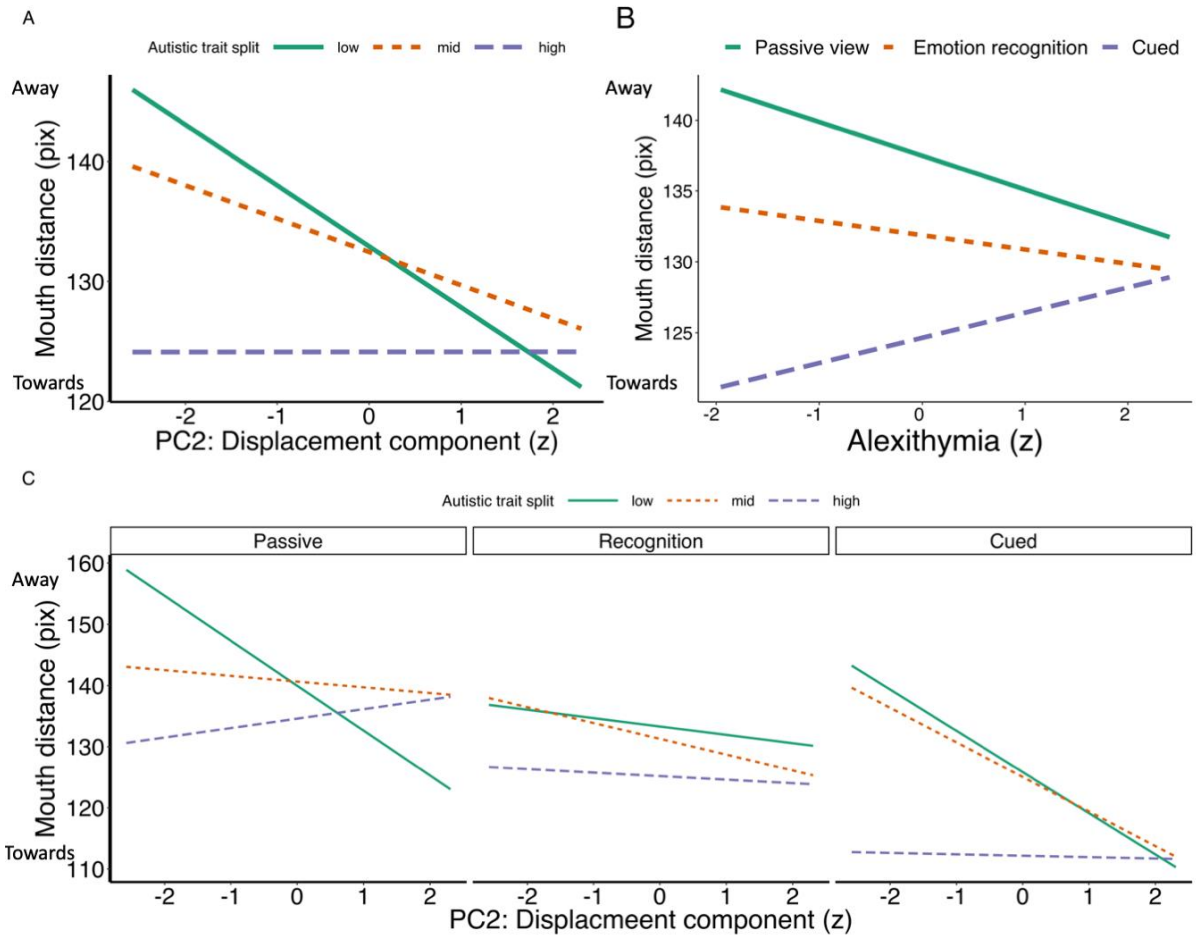

Figure 3.18 A visual summary of results for gaze towards the mouth region.

A. Low displacement scores reflect larger outward movements (mouth widen, eye open), whereas higher values reflect constriction movements. Small face movements predicted attention to mouth in low and mid but not high autistic traits. B. High alexithymia shows less

differentiation of mouth gaze by task. C. Three-way interaction between displacement scores, autistic traits and task.

Lastly, there was also a significant three-way interaction between autistic traits, displacements (PC2) and condition (Estimate = 3.34, SE = 1.56, t = 2.14, p < .05). Slope analyses indicated significant differences in the influence of displacements in gaze allocation to the mouth region for high autistic traits in the passive compared to emotion recognition conditions (see **Figure 3.18**).

These results suggest influences of both autistic and alexithymic traits in attention to the mouth, with alexithymia influencing task modulation effects and autistic traits influencing spatial-kinematic modulation of gaze towards the mouth region.

3.2.3.3.3 Gaze towards the nose region

Gaze distance to the nose region was predicted by the temporal (speed) component - PC1 (Estimate = -5.44, SE = 2.31, p < .019). Patterns of increasing speed of face actions was related to increasing gaze allocation to the nose region. There was no main effect of PC2 (displacement component) on gaze allocation to the nose region (Estimate = -3.28, SE = 2.30, t = -1.42, p =.15). Similarly, no main effects were found for alexithymia (Estimate = 2.46, SE = 3.21, t = .766, p = 0.423) or autism (Estimate = -2.97, SE =3.2, t = -.09 , p = 0.355).

Similar to results for eye and mouth region, there was a main effect of task, with two out of three pairwise comparisons indicating significantly more attention towards the nose region in the recognition condition compared to passive viewing (Estimate = 13.31, SE = 1.9, z = 7.013, p.adjusted < .001), and cued compared to passive viewing (Estimate = 16.02, z = 8.415, p.adjusted <.001). However, there was no difference in gaze towards the nose for emotion recognition vs. cued condition (Estimate = 2.71, z = 1.42, p = .33).

For the two-way interactions, there was a significant effect of alexithymia and temporal component – PC1 (Estimate = 2.51, SE = 0.961, t = 2.61, p = .009). Slope analyses and visualisations indicated a reduced effect of temporal kinematics influences on gaze at higher levels of alexithymia (see **Figure 3.19**). There was also a significant interaction between component 2 (displacement) and condition, showing the reduced influence of face action displacements on gaze allocation to the nose region for passive viewing compared to cued condition (Estimate = 2.38, SE = 1.1128, t = 2.142, p = .032).

Also, similar to results for gaze towards the eye and mouth regions, alexithymia also modulated gaze to the nose region differently depending on the task- and emotion-related priors. Slope analyses and visualisations indicated that alexithymia was related to increasing gaze towards the nose region on the passive condition (Estimate = -2.97, SE =1.32 , t = -2.24, p =.025) whereas low alexithymic had increasing gaze towards the nose on recognition and cued conditions (Estimate =2.66, SE =1.340, t = 1.98, p = .047). No other effects were significant.

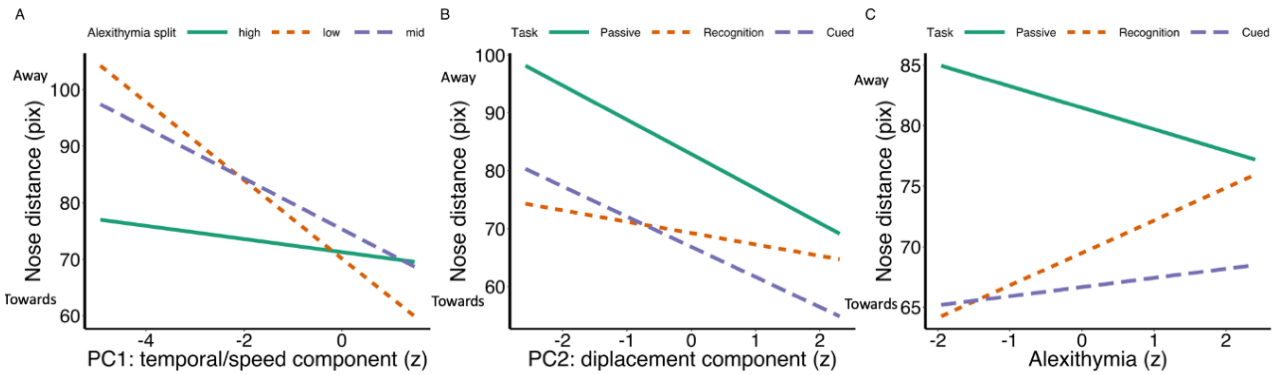

Figure 3.19 Visual summary of results for gaze towards the nose region

A. Alexithymia by PC1 interaction – split for visualisation purposes only. B. Displacement by task interaction. C. Alexithymia by task interaction.

3.2.3.4 Effects of spatial-kinematics on intensity ratings and emotion recognition.

3.2.3.4.1 Intensity ratings

There was a significant effect of PC1 (temporal/speed component) on intensity ratings. Specifically, increased speed of coactivated face actions (negative values in PC1) resulted in stronger intensity ratings (Estimate = -0.20, SE = .09, t = -2.18, p = .030). This might reflect the fact that spontaneous expressions – normally rated as less intense (Kumhuber et al., 2021) show reduced speed of facial actions. No main effects were observed for displacement scores (PC2), autistic or alexithymic traits.

For the two-way interactions, there was a significant effect of alexithymia and PC1 (temporal/speed component) (Estimate = 0.034, SE = 0.016, t = 2.178, p = .029). This effect indicates that, at fast (but not slow) facial actions, low alexithymia participants rated faces as more intense compared to highly alexithymic participants (see **Figure 3.20**). Similarly, there was a significant interaction between temporal component and task, with low scores on PC1 (higher speed of facial actions) leading to stronger intensity ratings in cued condition compared to the emotion recognition condition (estimate = 0.79, SE = .3177, t = 2.50, p = .012).

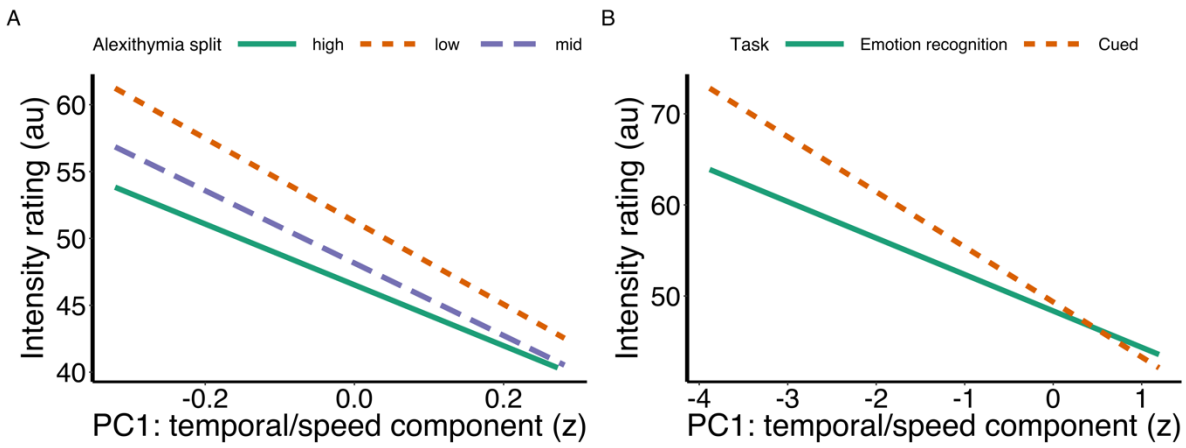

Figure 3.20 Visual summary of results for intensity ratings

Spatial-kinematics affect ratings differently as a function of alexithymia and task. A – split for visualisation purposes only. High values in PC1 represent reduced speed in facial actions. B. temporal component by task interaction.

3.2.3.4.2 Emotion recognition

Only one of the three conditions included emotion recognition ratings by design. Therefore, only the effects of spatial-kinematics on emotion recognition were assessed. There was a significant effect of PC1 (temporal coactivation), with higher temporal coactivation leading to reduced recognition rates (Estimate = -0.94, SE = .34 , z =-2.760, p = .006).

When looking at the clinical predictors, there was a significant effect of autistic traits (Estimate = -0.23, SE = .11, z = -2.129, p = .04) indicating that increasing autistic traits are associated with reduced recognition rates and no significant effect of alexithymia despite the predicted direction (Estimate = -0.139, SE = 0.110, t = -1.262, p = .21). However, a further inspection of this pattern suggests that this effect was primarily driven by high autistic traits and alexithymia in autistic participants. In fact, when breaking down the analysis by group there was no significant effect of autistic traits on emotion recognition for either group,

whereas the expected trend was observed for alexithymia (predicting reduced recognition) (see **Figure 3.21**). No other effects were observed.

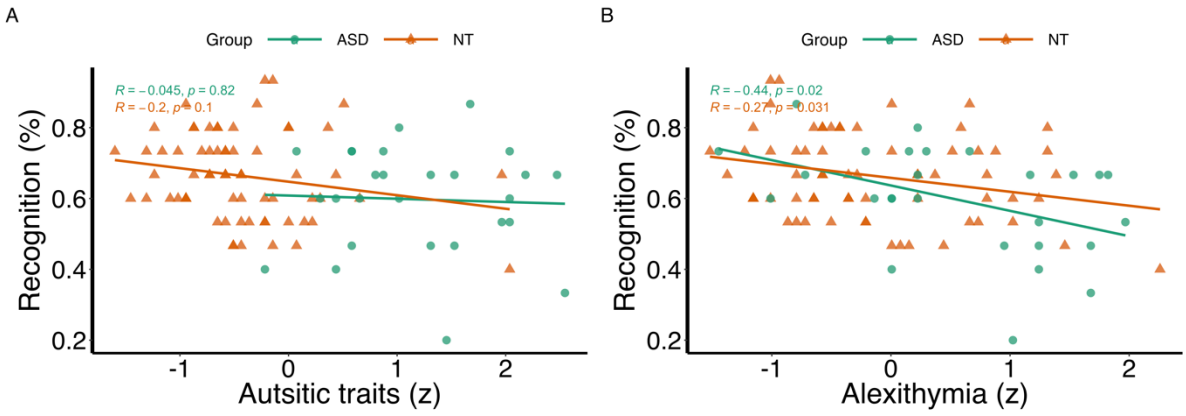

Figure 3.21 Visual summary of results for emotion recognition accuracy.

When matched, aggregated performance (for illustration purposes) was not significantly predicted by autism, whereas alexithymia predicted impaired emotion recognition.

3.2.3.5 Summary of results

Multivariate analysis of spatial-kinematics of facial actions identified two significant components that explained patterns of coactivation of various face actions involved in emotional expressions. Component 1 is related to temporal aspects of face movements (speed), and Component 2 is related to displacements (e.g., mouth and eye constriction or widening movements). The analyses of behavioural responses showed that spatial-kinematic information in facial expressions modulated gaze along with task- and emotion-relevant priors. Furthermore, gaze allocation analyses showed primarily contributions of alexithymia, which differentially affected the degree to which gaze behaviour was modulated by spatial-kinematics and task- and emotion-relevant priors. There was also some indication that both autism and alexithymia may contribute to modulate gaze allocation to the mouth region via different mechanisms, with autism modulating the impact of spatial-kinematics and

alexithymia modulating the impact of task and stimuli related priors on gaze towards the mouth region. These results were also consistent with behavioural ratings with effects of spatial-kinematics and conditions significantly influencing intensity ratings and emotion recognition and atypicalities in emotion processing being explained primarily by alexithymia rather than autism predictors.

3.2.4 Discussion

The current study investigated the contribution of spatial-kinematics and task and emotion-related priors on gaze behaviour and emotion processing. In particular, inspired by the Bayesian accounts of autistic perception (Elizabeth Pellicano & Burr, 2012) and results in Chapter 2 (Cuve et al., 2021) and Experiments 2 and 3 in the current chapter, this work aimed to explore whether atypical gaze and emotion processing can be explained in terms of differential modulation by both stimulus dynamics (bottom-up stimulus factors) and task- and emotion-relevant priors in autism. Furthermore, this study aimed to test the specificity of autistic influences on social attention and emotion processing when compared to alexithymia, in light of the alexithymia hypothesis discussed in the Introduction.

Perhaps unsurprisingly, the current results highlight that visual attention during emotional face processing is in part determined by spatial-kinematics of facial expressions. This is, however, the first quantitative demonstration of the effects of the spatiotemporal dynamics of nonverbal facial cues on gaze behaviour. In particular, spatial-kinematics representing temporal changes of facial actions associated with emotional expressions predicted the degree of attention to eyes, nose, and mouth. Rather than supporting general ratios of gaze allocation to different face regions during the processing of facial expressions, these results suggest that the amount of information expressed in terms of coactivation of facial actions has an

important role in dynamically modulating gaze allocation to faces. This result is important for research focusing on the processing of emotional faces where often the role of expression dynamics is ignored (Dobs et al., 2018; Krumhuber et al., 2013; Krumhuber et al., 2006; Namba, Matsui, & Zloteanu, 2021), and their influence on gaze allocation is rarely assessed (Cuve et al., 2021). As such, previous patterns of generalised eye preference, or reduced eye preference for some emotions and groups, may not generalise across stimuli varying in spatial-kinematics, which may explain the mixed findings of the effect of autism on social attention (Cuve et al., 2018; Guillon et al., 2014; Papagiannopoulou et al., 2014).

The results also highlight a simple but important observation, that gaze allocation to faces is task-dependent. Even with the relatively coarse gaze location estimation method used here (when compared to high resolution gaze sampling in standard eye-tracking as in Experiment 1), task effects were clear, with participants showing increased eye preference for passive viewing and emotion recognition conditions, but not cued conditions. Furthermore, in line with top-down effects of priors on gaze behaviour, reduced eye gaze in the cued condition likely reflects the effect of confirmatory visual behaviour in accordance with participants' representations (priors) of temporal dynamics of facial expressions. If participants have models for how emotional expressions evolve on average, then those models likely differentiate the usefulness of eye-related cues over time for various emotions, causing their gaze to be directed to the most informative parts of the face, which is not necessarily the eye region for all emotions or for the entire expression (see Jack et al., 2014).

These results are consistent with the eye-tracking and gaze entropy results reported in **Experiment 1** in Chapter 2, as well as to task modulation of gaze documented across various social and non-social visual processes (Tatler et al., 2011; Yarbus, 1967), including in social attention to faces (Hessels et al., 2019; Hessels, 2020). However, studies looking at

modulation of gaze to faces by top-down factors have typically failed to account for the dynamic nature of visual information in facial expressions, relying on more rudimentary characterisation of stimuli (e.g., the occurrence or not of a certain actions such as blinking rather than a proper quantification of spatial-kinematics). I suggest the latter approach may be more sensitive in capturing both stimulus effects on visual processing and interactions between stimulus and top-down factors.

One of the main goals of this study was to explore whether stimulus and task factors could explain the atypical gaze allocation reported in autism and alexithymia. While there was no direct effect of autism and alexithymia on gaze (in contrast to Experiment 1), likely due to limited sampling of gaze behaviour, atypical gaze modulation by task- and emotion-relevant priors and stimulus dynamics was observed for alexithymia for gaze to the eyes nose, and mouth. Alexithymia also interacted with task, replicating results from Experiment 1 in Chapter 2, suggesting that alexithymic impairments in the visual processing of nonverbal emotional cues are related to suboptimal priors to guide visual exploration of facial expressions. However, a novel finding compared to Chapter 2, is that alexithymia also interacted with spatial-kinematic components suggesting that alexithymia may be related to altered sensitivity to spatiotemporal dynamics of facial expressions. However, whether the latter reflects atypical low-or-high level processing of dynamics is unclear. One possibility is that alexithymia is related to ‘low-level’ deficits in biological motion processing. This has been suggested by earlier studies showing that alexithymia scores predicted performance with point-light displays (Lorey et al., 2012) and differences in early ERP components during motion processing (Borhani, Borgomaneri, Làdavas, & Bertini, 2016). However, it’s also possible that these effects reflect atypical representation of ‘higher-level’ spatiotemporal dynamics cues like facial expressions.

Furthermore, if these results are to inform Bayesian accounts of autism applied to social attention, the evidence here suggests that atypical sensitivity to sensory cues and atypical modulation by priors to non-verbal cues might characterise alexithymia rather than autism. This result is consistent with findings in Chapter 2 using a similar task (although with a restricted range of stimulus dynamics), where alexithymia was found to moderate the effects of task on the complexity of gaze patterns during emotion processing (Cuve et al., 2021).

One recent study provided evidence that was somewhat inconsistent with these results however - autism effects on emotion processing were shown to remain even after accounting for alexithymia when processing biological motion related to facial cues of anger (Keating, Sowden, Fraser, & Cook, 2020). This discrepancy may be related to the fact that the current study sampled a wide range of expressions, naturalistic and posed, and used actual faces rather than point-light displays which prioritise biological motion. Deficits in the processing of biological motion might be a point of confluence for alexithymia and autism influences on emotion processing. Recent metanalyses also suggest a higher likelihood of deficits in processing biological motion in autism (Han, Tijus, Le Barillier, & Nadel, 2015; Todorova, Hatton, & Pollick, 2019). While studies often attempt to dissociate biological motion conditions from emotion, facial expressions are non-verbal cues that inherently contain biological motion. As such it's possible that biological motion processing deficits in autism and alexithymia have different causes but similar consequences. Future studies should therefore aim to explore other types of non-emotion-related biological motion to investigate the specificity of any effects of alexithymia and autism effects on motion dynamics.

In summary, these results show that emotion processing can be studied in terms of sensory-driven stimulus influences as well as task- and emotion-relevant priors relevant to Bayesian accounts of social attention. The current results support the idea that both stimulus dynamics

and task- and emotion-relevant priors influence emotion processing, and that atypical integration of sensory information related to stimulus dynamics and top-down effects related to task- and emotion-relevant priors might be primarily a product of alexithymia, not autism. Rather than requiring a specific Bayesian theory of alexithymia, however, these results likely suggest that alexithymia may disrupt perceptual learning of emotional information during development, giving rise to atypical or suboptimal representations/priors that guide visual processing of socioemotional stimuli.

3.2.4.1 Limitations and future directions

Several limitations of this study should be discussed to inform future research. First, while the novel gaze location estimation method has some advantages in that it can be deployed outside the lab allowing for more naturalistic viewing behaviour and contexts (Rudoy, Goldman, & Shechtman, 2012), and shows remarkable consistency with patterns of gaze from previous studies using standard eye-tracking, this method has undoubtedly reduced the temporal sampling capacity and cannot describe spatiotemporal properties of eye movements (as done in Chapter 2). As such, some direct effects of autism and alexithymia (e.g., on reduced gaze) might have been harder to detect. Therefore, replication of this work with similar approaches employed in Chapter 2 and standard eye-tracking is needed.

Second, the autistic sample used in this study is defined as high functioning and with high IQ, which does not necessarily represent the autistic population as a whole. Although this is a selection bias that is not specific to this study (Russell et al., 2019), it carries important implications for the generalisability of autism-related research to diverse populations (e.g., those with intellectual disabilities).

Third, the current study relied on quantifying naturally-occurring dynamics and manipulating conditions to study the influence of sensory information and priors on emotion processing.

However, it is important to highlight that Bayesian models of vision provide not only a theory but also an empirical and methodological framework to test the computational mechanisms that implement the principles of Bayesian perception (e.g. active inference) (Friston et al., 2013; Lawson, Rees, & Friston, 2014). For example, while this study used priors in a general sense to refer to knowledge or expectations about facial expressions and then assumed this knowledge is used to accomplish the different tasks (emotion recognition, intensity judgements), or can be activated to produced confirmatory gaze (via emotion cueing), the current study does not assess several elements that are crucial for Bayesian models of vision and perception. A key idea of Bayesian accounts of perception is that higher brain networks which code for statistical probabilities associated with the environment generate top-down predictions that meet bottom-up stimulus-related signals from lower sensory areas and generate a prediction error, which reflects the discrepancy between actual sensory input and top-down predictions (Friston et al., 2013; Lawson et al., 2014). However, the current study did not make any direct measurement of prediction errors, priors, or their precision nor the precision of sensory information. Rather, their influence is inferred based on the modulation of gaze behaviour and emotion ratings as a function of the experimental conditions and stimulus properties. As such, future work exploring the application of Bayesian accounts of autism to social attention and emotion processing should devise tasks where these key elements of the Bayesian theory are tested, including manipulating (and measuring) the precision of sensory information and priors about communicative facial cues. This research should also aim to generate experimental conditions where prediction errors about upcoming sensory information (relating to nonverbal cues) can be tested (e.g., through a presentation of incongruent facial actions to expected emotional cues).

3.3 What comes next?

In Chapter 3, I extended findings from Chapter 2 by showing that both stimuli dynamics and task-related factors modulate gaze allocation to faces and emotion processing, and that atypical modulation of gaze by these factors reflects primarily influence of alexithymia rather than autism, in line with the alexithymia hypothesis. Chapter 4 will turn the focus to the experiential components of emotion thought to be atypical in autism as discussed in Chapter 1. First, Experiment 4 and 5 presents a validation study of a new eye-tracking and psychophysiology setup used in several tasks in Chapter 4. Then, Experiments 6 and 7 will investigate physiological and interoceptive processes implicated in emotion and how they are affected by alexithymia and autism.

4 CHAPTER 4. PHYSIOLOGICAL MECHANISMS OF SOCIOEMOTIONAL PROCESSING

This chapter starts by presenting a validation of a new setup, used in the psychophysiology and eye-tracking experiments. This validation supported the inclusion of some psychophysiology measures which could reduce participant burden associated with setup (e.g. attaching many sensors), particularly relevant when studying clinical participants. This also supported the use of the new low-cost eye-tracker in the following experiments, given that it was not routinely used for research applications. This experiment will also introduce the main pre-processing pipeline for analysing the physiological signals that will be used in subsequent experiments.

4.1 Validation of Gazepoint low-cost eye-tracking and psychophysiology bundle

This section is based on the published version of related work below edited specifically for the thesis:

Cuve, H. C., Stojanov, J., Roberts-Gaal, X., Catmur, C., & Bird, G. (2021). Validation of Gazepoint low-cost eye-tracking and psychophysiology bundle. *Behavior Research Methods*, 1-23.

Abstract

Eye-tracking and recording of physiological signals are increasingly used in research within cognitive science and human-computer interaction. Gaze position and measures of autonomic arousal, including pupil diameter, skin conductance (SC) and heart rate (HR), provide an indicator of cognitive and physiological processes. The growing popularity of these techniques is partially driven by the emergence of low-cost recording equipment and the proliferation of open-source software for data collection and analysis of such signals. However, the use of new technology requires investigation of its reliability, and validation with respect to real-world usage and against established technologies. Accordingly, in two experiments (total N = 69) we assessed the Gazepoint GP3-HD eye-tracker and Gazepoint Biometrics (GPB) system from Gazepoint. Results show that the accuracy, precision, and robustness of the eye-tracker are comparable to competing systems. While fixation and saccade events can be reliably extracted, the study of saccade kinematics is affected by the low sampling rate. The GP3-HD is also able to capture psychological effects on pupil dilation in addition to the well-defined pupillary light reflex. Finally, moderate-to-strong correlations between physiological recordings and derived metrics of SC and HR between the GPB and the well-established BIOPAC MP160 support its validity. However, low amplitude of the SC

signal obtained from the GPB may reduce sensitivity when separating phasic and tonic components. Similarly, data loss in pulse monitoring may pose difficulties for certain HR variability analyses.

Keywords: eye-tracking; psychophysiology; gaze position; pupillometry; skin conductance; heart rate; validation; Gazepoint GP3-HD.

4.1.1 Introduction

Eye-tracking and psychophysiological recordings[3] have gained popularity in recent years as a way to gain insight into cognitive processes, particularly the timecourse of those processes (Cacioppo, Tassinary, & Berntson, 2016; Holmqvist, Nyström, Andersson, & Dewhurst, 2011). The use of these techniques for research is not new; attempts to track human gaze and link physiological signals (e.g., heart rate, skin conductance and pupillary change) to cognition, although highly expensive and invasive, can be found even in the nineteenth century (see, Buswell, 1935; Dodge & Cline, 1901, and Cacioppo, Tassinary & Berntson, 2016, for a review).

While of interest to researchers for decades, this technology was until recently, mostly limited to research groups that could afford their high cost along with the proprietary software necessary to analyse the data (Funke et al., 2016). However, the proliferation of technology companies working on virtual reality, human-computer interaction, and marketing, has diversified research into, and applications of, eye-tracking and psychophysiology technologies.

Manufacturers are now starting to offer low-cost eye-trackers (e.g., GP3 and GP3-HD from Gazepoint; Tobii Eye Tracker 4C and Tobii Eye Tracker 5 from Tobii or the now

[3] Note that although eye-tracking is also considered to be a psychophysiology technique, the terms are used separately simply to aid exposition of the validation of the eye-tracking device and the biometrics bundle (for skin conductance and heart rate).

discontinued EyeTribe), and there is increased availability of open-source data acquisition and analysis software (e.g., OpenSesame - Mathôt et al., 2012; PsychoPy - Peirce et al., 2019; PyGaze - Dalmaijer et al., 2014; GazeR – Geller, Winn, Mahr, & Mirman, 2020). Similarly, there are several psychophysiology devices for skin conductance and heart rate measurement targeted at both consumers – e.g., Fitbit bracelets, Apple Watch, and smartphone apps (Mühlen et al., 2021), and researchers (e.g., Shimmer by Tobii, the E4 wristband by Empatica), alongside the more conventional (and more expensive) devices traditionally used for scientific research. A number of established open-source tools for analyses of signals like SCR (Ledalab, Benedek & Kaernbach, 2010; PSPM - Bach & Staib, 2015; EDAExplorer – Taylor et al, 2015) and heart rate and variability (ArtiFact, Kaufmann et al., 2011; Kubios – Tarvainen et al., 2014; RapidHRV; Kirk, Garfinkel and Robinson, 2021) have made it easier to automate often cumbersome pre-processing procedures.

These inexpensive eye-tracking solutions represent a very attractive option, not only for researchers operating on a limited budget, but also for those interested in more portable and less cumbersome eye-tracking devices that can be easily moved and retrofitted according to specific study purposes and environments. There are also several potential advantages provided by the newer and simpler devices to measure SC and HR. For instance, traditional psychophysiological recording takes time to setup and can be invasive (e.g., attaching specialised ECG sensors to the participant's chest and torso, often requiring the removal of clothing) which can add burden particularly to special participant populations (e.g. clinical groups).

While the diversification of eye-tracking and psychophysiology solutions provides considerable opportunities, it can also represent a risk to research validity and reproducibility if researchers are not adequately informed about the limitations of the low-cost devices available on the market (Orquin & Holmqvist, 2018; Society for Psychophysiological

Research Ad, 2012). For eye-tracking applications, manufacturers commonly specify spatial accuracy - the average distance between a known target in space and the gaze position estimated by the eye-tracker; and spatial precision - the average distance between consecutive gaze position data points where gaze is assumed to have remained relatively stationary (Holmqvist, Nyström, & Mulvey, 2012). However, manufacturers' performance evaluations are usually conducted under optimal conditions with trained participants using chinrests, or even using artificial eyes (Hessels, Cornelissen, Kemner, & Hooge, 2015). As a result, relying solely on performance estimates provided by the manufacturers can yield unjustified optimism when evaluating the suitability of low-cost eye-tracking devices to answer certain research questions. Aware of the need for performance evaluations in more realistic experimental conditions, eye-tracking researchers have conducted extensive validation and comparison studies for some of the most frequently used eye-tracking devices (see, Funke et al., 2016; Hessels, Andersson, Hooge, Nyström, & Kemner, 2015; Janthanasub & Meesad, 2015; Leube, Rifai, & Wahl, 2017; Mannaru, Balasingam, Pattipati, Sibley, & Coyne, 2017; Niehorster, Cornelissen, Holmqvist, Hooge, & Hessels, 2018). A common observation across these studies is that, even under ideal conditions, there is still a great deal of variability in how well different eye-tracking systems perform.

In addition to gaze position eye-movement researchers often study saccades. However, systematic analysis of saccades in validation studies is often overlooked. While system accuracy and precision can inform saccadometry research, there aren't many established baselines for saccade metrics and most research has relied on direct comparison of different eye-trackers (e.g., Dalmajer, 2014; Nyström, Niehorster, Andersson & Hooge, 2020). Nonetheless, it is possible to assess saccade parameters descriptively, for example, by looking at known regular relationships between saccade parameters (e.g. duration, amplitude, velocity) known as the saccadic 'main sequence' (Bahill et al., 1975; Gibaldi & Sabatini,

2021). The shape of the main sequence is well-known – for small to medium saccades (between 10 to 20 degrees of visual angle in size), one should expect the relationship between these saccade metrics to be approximately linear (Gibaldi & Sabatini, 2021). Similarly, for a given task where the size of the expected saccade is known, researchers could use the actual observed saccades of typical participants to assess undershooting or overshooting, as well the degree of saccade curvature (Leeuwen & Belopolsky, 2018).

This study aimed to assess the performance of a new relatively low-cost eye-tracker, the GP3-HD (Gazepoint), with a sampling rate of 150 Hz and incorporating a high-definition machine-vision powered camera. The GP3-HD replaces the previous model, the GP3, which recorded at 60 Hz and for which independent validations exist (Brand, Diamond, Thomas, & Gilbert-Diamond, 2020; Mannaru, Balasingam, Pattipati, & Sibley, 2017). This setup will be used for the other experiments in this chapter.

In addition to the GP3-HD, Gazepoint recently launched a Biometrics system (GPB) for the measurement of autonomic responses, specifically, skin conductance (SC) and heart rate (HR). SC and HR provide an indication of the degree of an individual's physiological arousal, and the physio-anatomical mechanisms underlying changes in SC and HR are relatively well understood. As is the case with the GP3-HD, however, the reliability and validity of the GPB is currently unknown. A comparison of raw and derived SC and HR metrics obtained from the GPB and from a well-established device would provide a useful insight into the potential of the GPB to provide valid measurements. Therefore, this study also aimed to validate the GPB in order to include it as an option that can be used with certain special populations.

In Experiment 5, common data quality indicators (calibration quality, data loss, accuracy, precision) were obtained for the GP3-HD eye-tracker. This study also provides information on sampling rate variability and fixation and saccade metrics (Holmqvist, Nyström, Andersson, & Dewhurst, 2011). In addition to gaze position and saccade analyses, we provide pupillometry analyses tracking the pupillary light reflex (PLR), a physiological process in which the pupil constricts in response to increased light intensity and dilates in response to reduced light intensity (Mathôt, 2018). In Experiment 6, a second validation of the GP3-HD system is provided that enabled to study its performance under the conditions encountered in a typical psychological experiment, as well as to further test measurement of pupillary responses. Additionally, in Experiment 6, data were collected simultaneously from a well-established psychophysiological recording system (BIOPAC-MP160) and from the GPB system to assess the validity of SC and HR data, and derived metrics, recorded from the GPB system. Experiment 6 is particularly relevant as it represents the main experimental task used for studying psychophysiological responses to emotion in this thesis. Finally, recommendations for researchers planning to use this technology are provided.

4.1.2 EXPERIMENT 5

4.1.2.1 Method

4.1.2.1.1 Participants

A total of 13 university students (7 women, 13 right-handed) took part in Experiment 1 after exclusion of one participant due to a failure to calibrate, and one for excessive data loss and difficulties tracking). They ranged in age from 19 to 28 years (M = 22.08, SD = 2.40). All participants had normal or corrected-to-normal vision and reported being able to complete the study without relying on vision correction. Hence, no participants wore glasses or contact lenses throughout the experiment. Finally, no participants wore make-up during the experiment.

4.1.2.1.2 Apparatus and task environment

The experiment was run on a Dell computer (Intel Core i7-3610QM @ 2.30 GHz, 16 GB RAM, Windows 10) and the task stimuli were presented on a monitor (53 x 30 cm, 60 Hz refresh rate, 1920 x 1080 pixels, 45.99 x 27.01 degrees of visual angle). The experiment was completed in a dimly lit, sound-proof testing room.

4.1.2.1.3 Eye-tracking

The remote GP3-HD eye-tracker, recording at 150 Hz, was used in this study. The eye-tracker was controlled through a custom script in PsychoPy (Peirce et al., 2019). The eye-tracker was placed at a 45° angle and 60-65 cm from the participants' eyes ($M_{DISTANCE}$ = 62.45 cm), in line with the instructions provided in the Gazepoint manual. The Gazepoint control and monitoring window, and physical measurement (before and between tasks) were used to aid setup and find an optimal position. Eye-tracker specifications provided by the manufacturer are summarized in Table 4.1.

Table 4.1 GP3-HD eye-tracker specifications offered by the manufacturer (Gazepoint)

GP3-HD specifications	Values
Accuracy	0.5 - 1º
Headbox	35 x 22 cm
Precision	NS
Sampling rate	60 or 150 Hz
Tracking distance	50-100 cm (65 cm recommended)

Notes. NS = Not specified by the manufacturer.

Prior to starting the main tasks, calibration was performed using a nine-point grid followed by a validation sequence. Satisfactory calibration criteria for continuing with the task were determined *a priori*: (a) all nine calibration points had to be deemed valid according to the Gazepoint Control software; (b) average calibration error had to be below or equal to 40

pixels (approximately 1 degree of visual angle); and finally, (c) using a real-time gaze relay nine-point grid, where participants' gaze was shown as moving green dots on the screen, participants were asked to report how good they thought the eye-tracker was at approximating where they were actually looking using a 0-10 scale after explicitly attending to each of the gaze targets. Only answers equal to, or above, 8 were accepted.

4.1.2.1.4 Tasks

Fixation-Saccade task

Participants completed two main tasks. The Fixation-Saccade task was designed to provide data for the calculation of fixation and saccade metrics, and for accuracy and precision analyses. Participants were presented with nine black dots on the screen (size: 40 pixels, approximately 1 degree of visual angle; with an average distance of 11 degrees between target dots; see **Figure 4.1- A**). A target (blue dot) started on the central dot and then transitioned from the centre to each peripheral dot at random throughout the task. All target positions were sampled before any were repeated. The duration of time for which the central dot was blue was varied between 2 and 5 seconds on each transition to prevent participants from trying to predict when and where the dot will move next. The task finished when each peripheral dot had turned blue twice.

Pupillary Light Reflex task

The second task was designed to evoke the pupillary light reflex (PLR). Participants were presented with a grey dot in the middle of the screen (size: 50 pixels, approximately 1.2 degrees of visual angle) and instructed to fixate on it while black and white backgrounds[4]

[4] Please note that the screen was not exactly white as the change from a completely black to a completely white screen would have been straining for participants and would have caused excessive blinks and data loss during this transition period. The exact colour of the screen was [0, 0, 0] in the RGB colour space.

interchanged every 5 seconds. Each screen was presented 12 times (24 changes of colour, see **Figure 4.1- B**).

4.1.2.1.5 Procedure

Participants completed the setup and the calibration procedure, followed by the tasks detailed above. Each task was completed twice, once with the participants' heads placed on the chinrest to limit their head movements, and once without. In both conditions participants were asked to avoid head and body movements. The order of tasks and conditions (chinrest vs. no-chinrest) was counterbalanced. The calibration procedure was performed prior to each task. After the experiment participants were debriefed. All experimental procedures were conducted in accordance with the revised 2013 Declaration of Helsinki and were approved by the local research ethics committee.

4.1.2.1.6 Pre-processing

Eye-tracking data were pre-processed using custom code in R (version: 3.6.1). Gaze samples falling outside screen coordinates were eliminated, as well as the samples labelled as invalid by the eye-tracker (in total, 2.72% of the samples were excluded, out of which 0.74% fell outside the screen boundaries, and 1.98% were labelled as invalid by the eye-tracker). A simple implementation of the adaptive velocity-based algorithm proposed by Engbert and Kliegl (2003) was used to detect fixations and saccades in the 'Fixation-Saccade' task. Saccades were defined as periods of at least 20 ms (the duration of three adjacent gaze samples) where velocity exceeded an adaptive threshold set for each participant and condition (chinrest and no-chinrest) based on the level of noise in the data. The velocity threshold was defined as 5 median absolute deviations above the median velocity for each participant and condition. Finally, to prevent artificial improvements in accuracy and precision, no smoothing, filtering, or interpolation was applied to gaze position coordinates.

For the PLR task, pupil data were first cleaned by removing pupil sizes outside the range of 2-10 mm. Pupil data were then pre-processed using functions from the R package GazeR (Geller, Winn, Mahr, & Mirman, 2020). Data loss (e.g., blinks) up to 150ms in duration were imputed using linear interpolation. Finally, a subtractive baseline correction was applied in line with the recommendations in Mathôt et al. (2018). Median pupil size during the last 20 samples of the preceding trial and the first 20 samples of the current trial (approximately 240 ms, incorporating an equal duration of light and dark screens) was taken as baseline pupil size, from which all individual pupil sizes were subtracted on each trial.

4.1.2.1.7 Metrics and analyses

Calibration quality. To assess the calibration quality, two metrics were used based on the manufacturer's calibration procedure: 1) the number of calibration attempts it took until the experimenter accepted the calibration, and 2) the average error of the accepted calibration. All calibrations needed to have nine valid calibration points to be accepted so the number of valid calibration points was not considered in further analyses.

Sampling rate variability. As the GP3-HD eye-tracker has a sampling frequency of 150 Hz, the expected average inter-sample time is approximately 0.0067 seconds (6.7 ms). Sampling rate variability was assessed by calculating the mean and the standard deviation of inter-sample time as well as their robust equivalents (median and median absolute deviation), for both the chinrest and no-chinrest condition. Sampling rate variability was assessed across both the Fixation-Saccade and the PLR task.

Data loss. Data loss occurs when the eye-tracker cannot detect the position of the eyes, and individual samples where this happens are labelled as invalid by the device. Proportion of lost gaze was computed for each trial and participant and compared between conditions (chinrest and no-chinrest).

Accuracy. Prior to computing accuracy and precision, the first and last 250 ms of each trial were removed to give participants time to fixate on the new target dot and to limit the extent to which participants' anticipatory saccades influenced these metrics. This interval was chosen after calculation and visual inspection of saccade latency. Accuracy was computed as the error between the estimated gaze location and the location of a known target (Holmqvist et al., 2012). Horizontal and vertical accuracy were calculated for each gaze sample in the Fixation-Saccade task by subtracting estimated x and y gaze coordinates from the pre-defined x and y coordinates of each target dot location. Sample-level global accuracies were calculated as Euclidian distances between estimated x and y gaze coordinates and pre-defined x and y coordinates of target dot locations (see **Figure 4.1**- C and 1 - D).

Having calculated all three types of sample-level accuracies, outlier samples were removed if they were greater than 4 median absolute deviations from the median respective accuracy for each participant, condition, and trial (2.1% of the samples were excluded for vertical accuracy, 3% for horizontal accuracy, and 2.9% for global accuracy – note that these values are not independent). These outliers corresponded mostly to saccades, with the size of the error matching the expected saccade sizes during the task (note that analyses with outliers yielded consistent results). This was performed to avoid biasing the accuracy calculation by including gaze samples where the participant was likely to have clearly moved their eyes away from the target dot. Finally, mean vertical, horizontal, and global accuracy were calculated for each participant, condition, and trial.

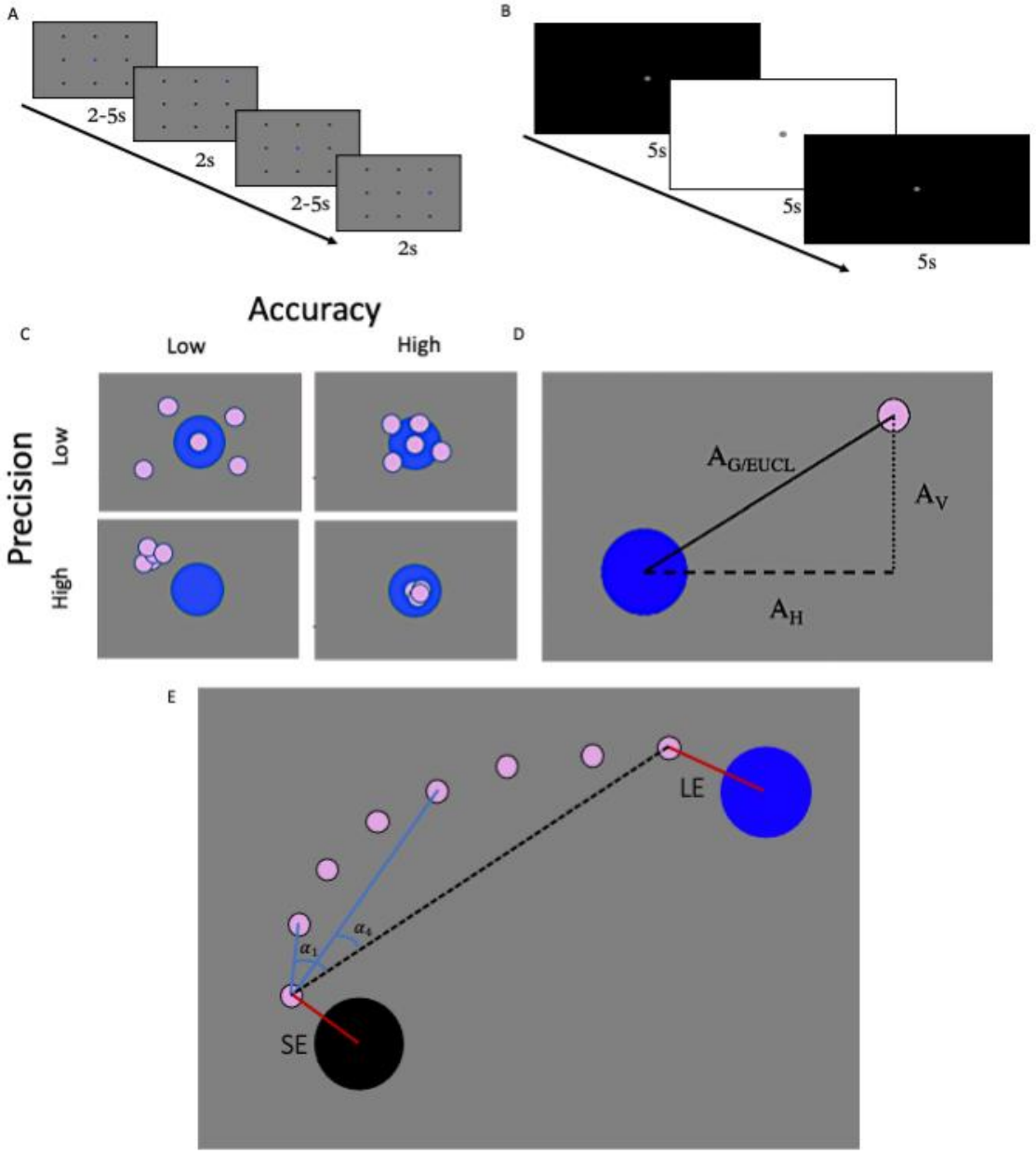

Figure 4.1 Schematics of task and eye-tracking measures.

(A) Schematic of the Fixation-Saccade task. A blue target appeared and switched from the central dot to each of the peripheral dots. (B) Schematic of the Pupillary Light Reflex task. Each screen appeared 12 times. Both tasks were repeated twice, once with, and once without, a chinrest. (C) Illustration of the concepts of accuracy and precision for eye-tracking data. The blue centre dot represents the known gaze target, and the smaller pink circles

represent gaze locations estimated by the eye-tracker. (D) Three types of eye-tracker accuracy calculations. AH = Horizontal accuracy for a particular gaze sample. AV = Vertical accuracy for a particular gaze sample. AG/EUCL = Global/Euclidian accuracy for a particular gaze sample. (E) Illustration of saccade metrics. SE = Starting error. LE = Landing error. Curvature = Median of all individual αi angles between each sample point and the straight line connecting the start and end point of the saccade.

Following the calculation of descriptive statistics, linear mixed models in lme4 (Bates, Mächler, Bolker, & Walker, 2014) were fitted to test whether global accuracy differed between conditions (chinrest and no-chinrest) and target dot locations (central and peripheral) while accounting for participant and trial random effects. Maximal models were always fitted first (Barr et al., 2013), and convergence and singularity warnings were resolved by simplifying the random structure using Principal Component Analysis to determine the most relevant random components (Bates et al., 2015).

Finally, it was decided to compare accuracy on the central dot location against accuracy on all the peripheral dot locations grouped together for two reasons: (a) in psychological research, stimuli are commonly presented at the centre of the screen, and it might be useful for researchers to know whether accuracy at this location is superior to accuracy at any peripheral location; (b) since trials with different target dot locations varied in frequency and duration (central target was presented more frequently and for a longer period of time in comparison to peripheral targets), grouping all peripheral locations allowed us to increase statistical power. Additionally, only sample-level accuracies within the first 2 seconds of the central trials were used in this comparison in order to match their duration with the duration

of peripheral trials. For analyses, accuracy was log-transformed to correct the violation of the assumption that the residuals of the model are normally distributed.

Precision. Precision is a measure of the spatial variance in accuracy when the eye is assumed to be relatively stationary (Holmqvist et al., 2012). Therefore, gaze samples were first parsed into fixations and saccades based on the adaptive velocity threshold described above. Only fixations longer than 80 ms were used for calculating precision to avoid including small saccades which may be inaccurately labelled as fixations. Horizontal and vertical precision were calculated on a trial-by-trial basis for each participant by computing the root mean square from successive gaze samples for each fixation to the target dot. Global precision was calculated by first estimating the Euclidian distances between pairs of adjacent gaze samples and then computing the root mean square over these distances. After calculating descriptive statistics, linear mixed models were fitted to test whether the three types of precision (horizontal, vertical, global) varied between conditions (chinrest and no-chinrest) and target locations (central and peripheral) while controlling for participant and trial variability. Comparison between central and peripheral target locations as well as the choice of model's random structure followed the same logic as the analysis of accuracy. Precision data was also log-transformed for analysis due to non-normality of residuals.

PLR. Following pupil pre-processing and baseline correction, the degree of PLR elicited by the changing stimulus was estimated (i.e., pupil constriction in response to light and pupil dilation in response to darkness). More specifically, linear mixed models were fitted for each condition (chinrest and no-chinrest) to compare pupil size changes in response to the two stimuli (black vs. white).

Saccade metrics. Saccade starting and landing error, amplitude, gain, curvature, latency, mean, and peak velocity were calculated (see, **Figure 4.1**). These metrics are provided to allow researchers to judge how well saccade parameters are reflected in gaze data from the

GP3-HD, as there are no standard norms to which these values can be judged against, other than direct comparisons with other systems.

Saccade starting error was calculated as the Euclidian distance between the gaze sample labelled as the saccade onset and the centre of the starting target, while saccade landing error was calculated as the Euclidian distance between the gaze sample labelled as the saccade end and the target centre (Dalmaijer, 2014). After calculating saccade amplitude (size of the saccade in degrees of visual angle), we proceeded to compute gain, the ratio between the observed saccade amplitude and the expected saccade amplitude, actual distance between the two consecutive target locations (Noto & Robinson, 2001). Saccades with gains less than 1 were too small (*hypometric*), while saccades with gains higher than 1 were too large (*hypermetric*). Gain provides an approximation of over- or under-estimation of the expected saccade amplitudes.

In order to capture saccade trajectories, curvature was calculated as the median angle between each gaze point in a saccade and an imaginary straight line connecting the start and the end of the saccade, following the strategy of van Leeuwen and Belopolsky (2018). Saccade latency is the time from target onset until the initiation of the saccade to that target.

Finally, velocity was calculated for each gaze sample making up a saccade by dividing inter-sample distance (in degrees of visual angle) by inter-sample time (in seconds), after which mean and peak velocity were computed for each saccade as a whole.

4.1.2.2 Results

4.1.2.2.1 Calibration quality

Calibration metrics included the number of attempts it took to achieve an acceptable calibration, and the average error of the accepted calibration. Due to the violation of

normality (Shapiro-Wilk test: $W(12) = 0.72$, $p < .001$), a Wilcoxon signed-rank test was performed to examine differences between the chinrest and no-chinrest conditions on both calibration quality metrics. No differences were detected in the number of calibration attempts ($M_{CHINREST} = 1.81$, $M_{NOCHINREST} = 1.58$, $W(12) = 30$, $p = .402$) nor in the average error of the accepted calibration ($M_{CHINREST} = 0.93$[5], $M_{NOCHINREST} = 0.99$, $W(12) = 47$, $p = .946$) (see **Figure 4.2**).

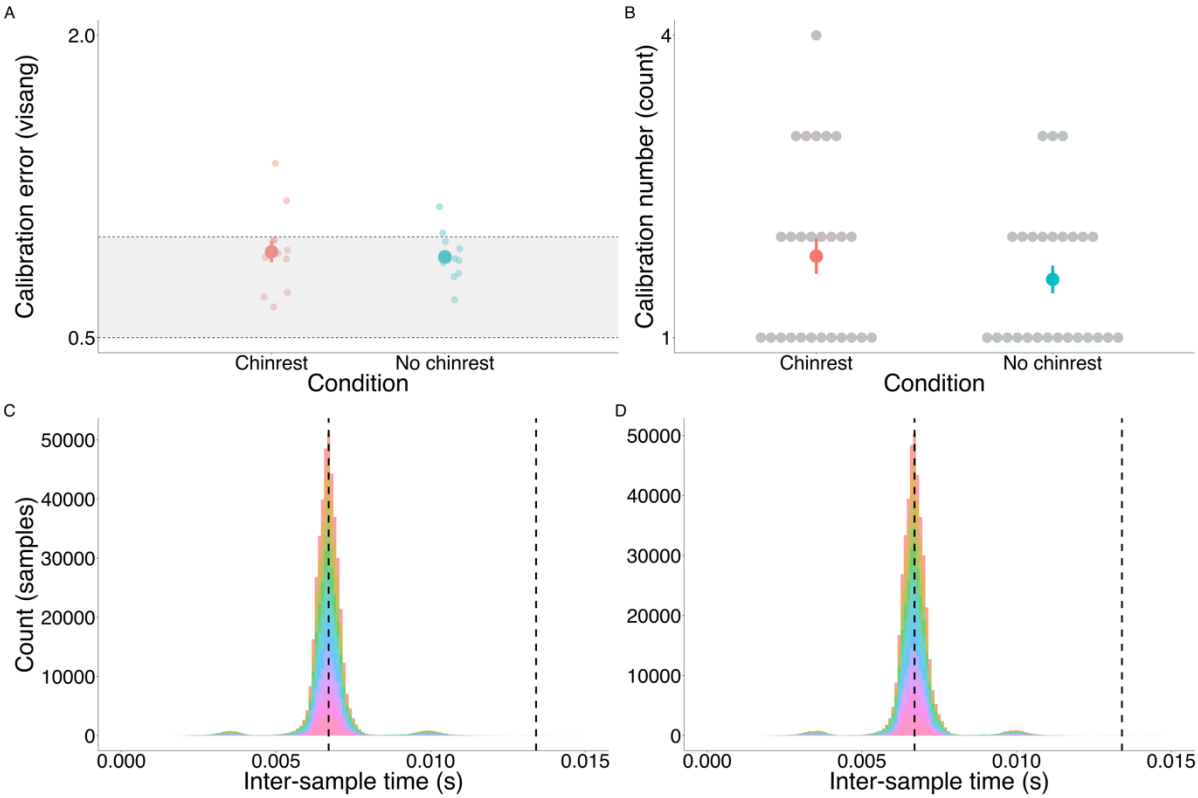

Figure 4.2 Calibration and sampling error.

(A) Average error of the accepted calibration in degrees of visual angle. The shaded part indicates average accuracy stated by the manufacturer (0.5 – 1 degrees of visual angle). (B) The number of calibration attempts before the calibration was accepted. Each participant went through the calibration twice in each condition. (C) Distribution of the inter-sample

[5] Average error of the accepted calibration is measured in degrees of visual angle.

times grouped in 0.0001 s (0.1 ms) bins in the chinrest condition, (D) and the no-chinrest condition. Different colours represent different participants (N = 13). Left dashed line indicates the expected inter-sample time based on the eye-tracker's sampling rate (150 Hz), and the right dashed line indicates the duration of two consecutive samples.

4.1.2.2.2 Sampling rate variability

As expected, the observed average inter-sample time was 6.7 ms and the standard deviation of the inter-sample time was 0.79 ms (robust descriptives: Mdn = 6.68 ms; MAD = 35.29 ms). Only 0.0003% of the inter-sample times were greater than the duration of two consecutive samples (13.4 ms). Distribution of inter-sample time is shown in **Figure 4.2.**

4.1.2.2.3 Data loss

A Wilcoxon signed-rank test was performed to compare whether the chinrest and no-chinrest condition differed in the proportion of lost gaze across tasks, and no differences were observed ($M_{CHINREST}$ = .031, $M_{NOCHINREST}$ = .029, $W(12) = 43$, $p = .583$). Additionally, the quality of the accepted calibration prior to each combination of condition and task (Fixation-Saccade task – chinrest condition; Fixation-Saccade task – no chinrest condition; pupil task – chinrest condition; pupil task – no chinrest condition) did not correlate with the proportion of lost gaze (Kendall's tau: $t_{FIX\_SACC\_CHINREST}$ = -.24, p = .228; $t_{FIX\_SACC\_NO\_CHINREST}$ = .01, p = 1; $t_{PUPIL\ \_CHINREST}$ = .01, p = 1; $t_{PUPIL\_NO\_CHINREST}$ = -.21, p = .331).

4.1.2.2.4 Accuracy

A visualisation of participants' gaze positions superimposed on target positions for the Fixation-Saccade task is provided in **Figure 4.3**. No differences in global accuracy, defined as the Euclidian distance of the actual gaze sample from the expected gaze position, were found between conditions with and without the chinrest (Estimate < 0.01, SE = 0.01, t = -

0.37, p = .710), while peripheral target locations had slightly better global accuracy in comparison to the central position (Estimate = -0.09, SE = 0.02, t = -4.29, p < .01). However, different accuracy profiles were observed when vertical and horizontal accuracies were analysed separately. In the case of vertical accuracy, no differences were found between conditions (Estimate = 0.02, SE = 0.01, t = 1.64, p = .102), while vertical error at the peripheral target locations was lower than at the central target location (Estimate = -0.19, SE = 0.03, t = -6.79, p = < .001). In the case of horizontal accuracy, better accuracy was achieved without the chinrest (Estimate = -0.04, SE = 0.01, t = -3.16, p < .01), and peripheral target locations had worse horizontal accuracy in comparison to the central target location (Estimate = 0.16, SE = 0.03, t = 4.95, p < .001). Descriptive statistics for vertical, horizontal, and global accuracy for each condition and target location can be seen in Figure 4.3.

These results suggest that globally, the accuracy of the GP3-HD is closer to the upper bound of the expected ~ 0.5-1º values, although horizontal accuracy is consistently closer to 0.5º. Nonetheless, the range of accuracy values is similar to what has been reported in previous evaluations of commercial eye-trackers (Funke et al., 2016; Holmqvist, 2017). Overall, the GP3-HD shows precision below 0.5º, which is also in line with what is reported using similarly priced eye-trackers and even some high-end systems (Funke et al., 2016; Holmqvist, 2017).

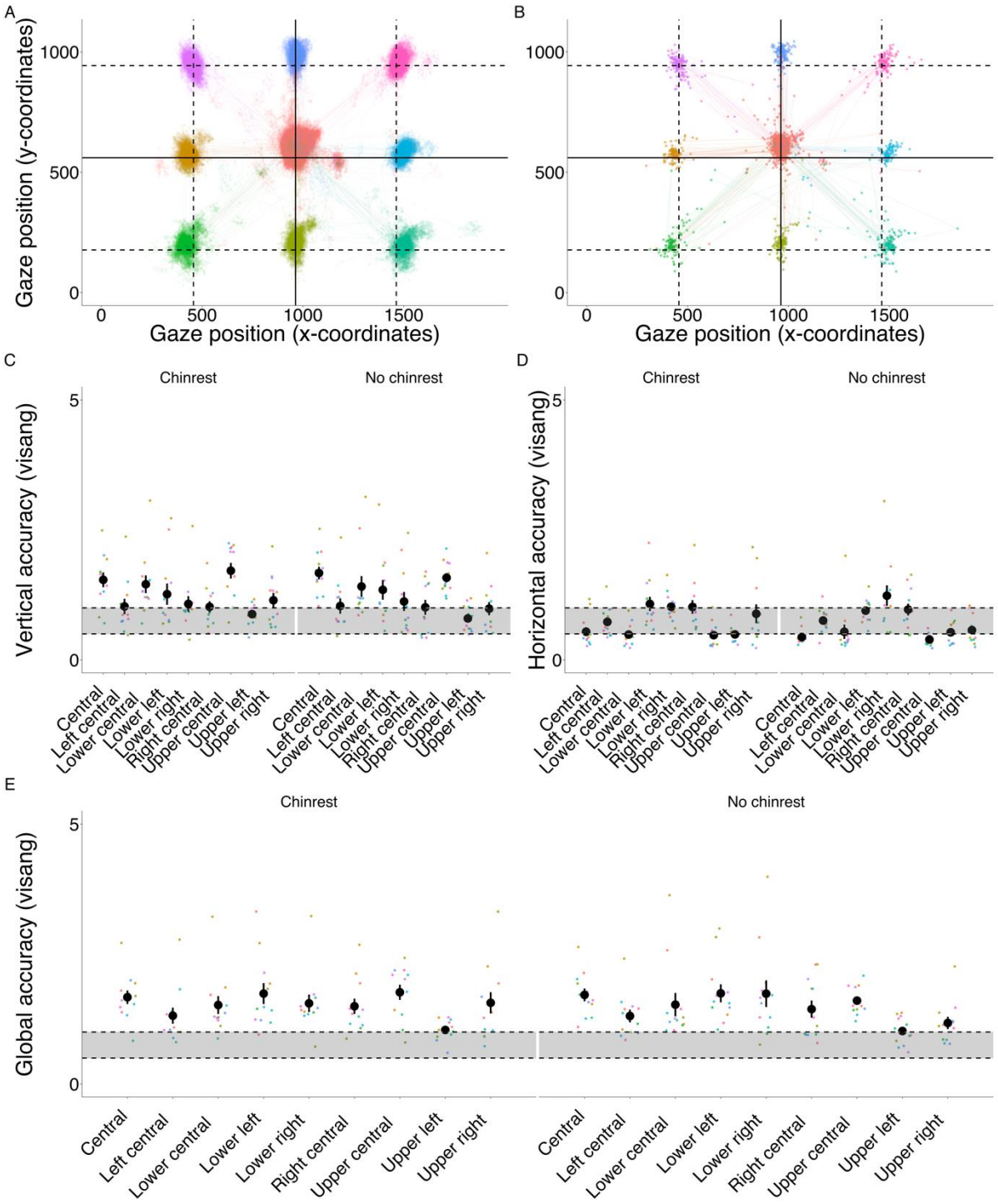

Figure 4.3 Eye-tracker accuracy metrics.

(A) Estimated gaze locations at each target location (individual gaze samples plotted). (B) Estimated gaze locations at each target dot location (mean fixation positions plotted). (C) Vertical, (D) horizontal and (E) global accuracy in degrees of visual angle for each condition (chinrest, no-chinrest) and target dot location in the Fixation-Saccade task. The shaded part

indicates average accuracy indicated by the manufacturer (0.5 – 1 degrees of visual angle). Coloured dots represent participants' accuracy in each iteration of target location, while the black dot and error bars represent means and 95% CIs.

4.1.2.2.5 Precision

No differences were detected between central and peripheral target locations in any of the precision metrics (Horizontal: Estimate = 0.01, SE < 0.01, t = 1.86, p = .069; Vertical: Estimate < 0.01, SE < 0.01, t = -1.07, p = .288; Global: Estimate < 0.01, SE = 0.01, t = 0.52, p = .607), whereas the differences between chinrest and no-chinrest conditions showed a less consistent pattern. The no-chinrest condition yielded increased vertical precision (Estimate = -0.01, SE < 0.01, t = -2.66, p < .01), whereas no differences were observed in horizontal (Estimate < 0.01, SE < 0.01, t = 1.53, p = .126) and global precision (Estimate < 0.01, SE < 0.01, t = -0.80, p = .426).

4.1.2.2.6 Pupillary Light Reflex

The PLR was reliably detected in both the chinrest (Estimate = -19.55, SE = 0.26, t = -74.50, p < .001) and no-chinrest conditions (Estimate = -20.75, SE = 0.30, t = -69.79, p < .001) - see **Figure 4.4-A** and **Figure 4.4-B**). As expected, the PLR effect is very large, accounting for 95% of the variance in both conditions.

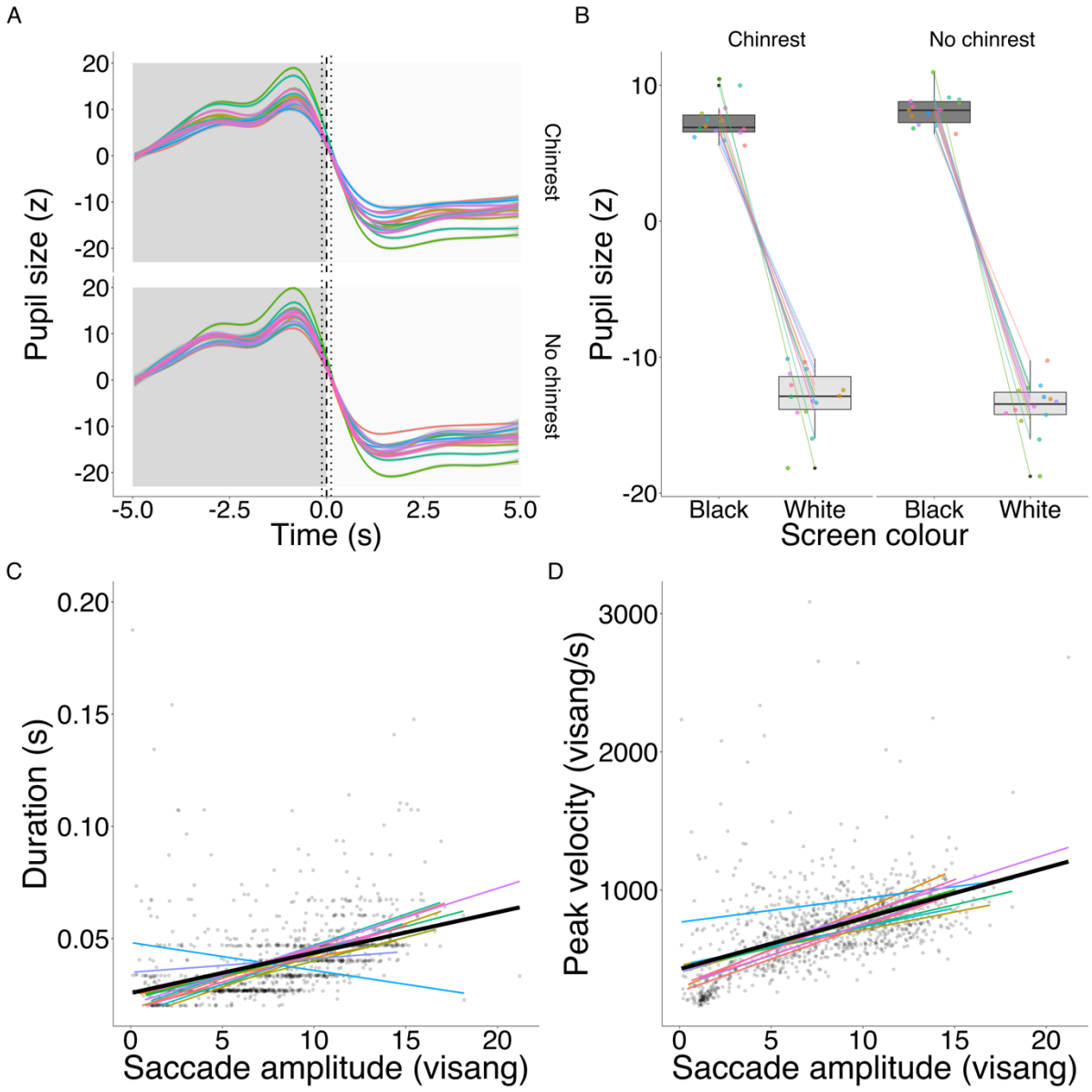

Figure 4.4 Pupillary Light Reflex and saccade parameters.

A-B: Pupillary light reflex in both chinrest and no-chinrest conditions. Dark-coloured time section (from -5 to 0 s) and dark boxplot indicate pupil dilation during the black screen, while light-coloured time section (from 0 to 5 s) and light boxplot indicate pupil constriction during the white screen. Dashed line on graph A indicates the switching point from black to white screen, while the dotted lines mark the baseline period used for calculating baseline pupil size. C-D: Main sequence of saccadic eye movements ($r = .288$, $p < .001$; $r = .420$, $p < .001$). Each coloured line represents a slope for each individual participant, while the black

line represents the slope across participants. Jittered points presented in the background represent individual saccades.

4.1.2.2.7 Saccadometry

Descriptive statistics for different saccade metrics are provided in **Table 4.2**. The relationships between saccade amplitude, duration and peak velocity are visualised in **Figure 4.4**-C and D.

Table 4.2 Saccade metrics calculated using GP3-HD data

Saccade index	GP3-HD M(SD)	Unit
Amplitude	8.87 (3.97)	(Degrees of visual angle)
Curvature	30.74 (12.94)	(Degrees)
Duration	48.80 (25.03)	(Milliseconds)
Gain	0.73 (0.21)	-
Latency	163.38 (369.75)	(Milliseconds)
Mean velocity	315.83 (95.73)	Degrees per second)
Peak velocity	756 (322.48)	-
Starting error	3.56 (1.96)	(Degrees of visual angle)
Landing error	2.16 (2.28)	-

As expected, since the majority of the saccades detected in the Fixation-Saccade task would be classified as small using the guidelines of Gibaldi & Sabatini (2021), the relationship between these metrics was approximately linear.

Finally, participant's saccade trajectories from the central target to each peripheral target were examined (see **Figure 4.5**). Individual saccade trajectories plotted in Figure 4.5 are not smooth, but appear broken and edgy, which is a sign of under-sampling (Dalmaijer, 2014). While clear identification of saccade events is possible, this suggests that the GP3-HD eye-tracker is less suitable for saccadometry research.

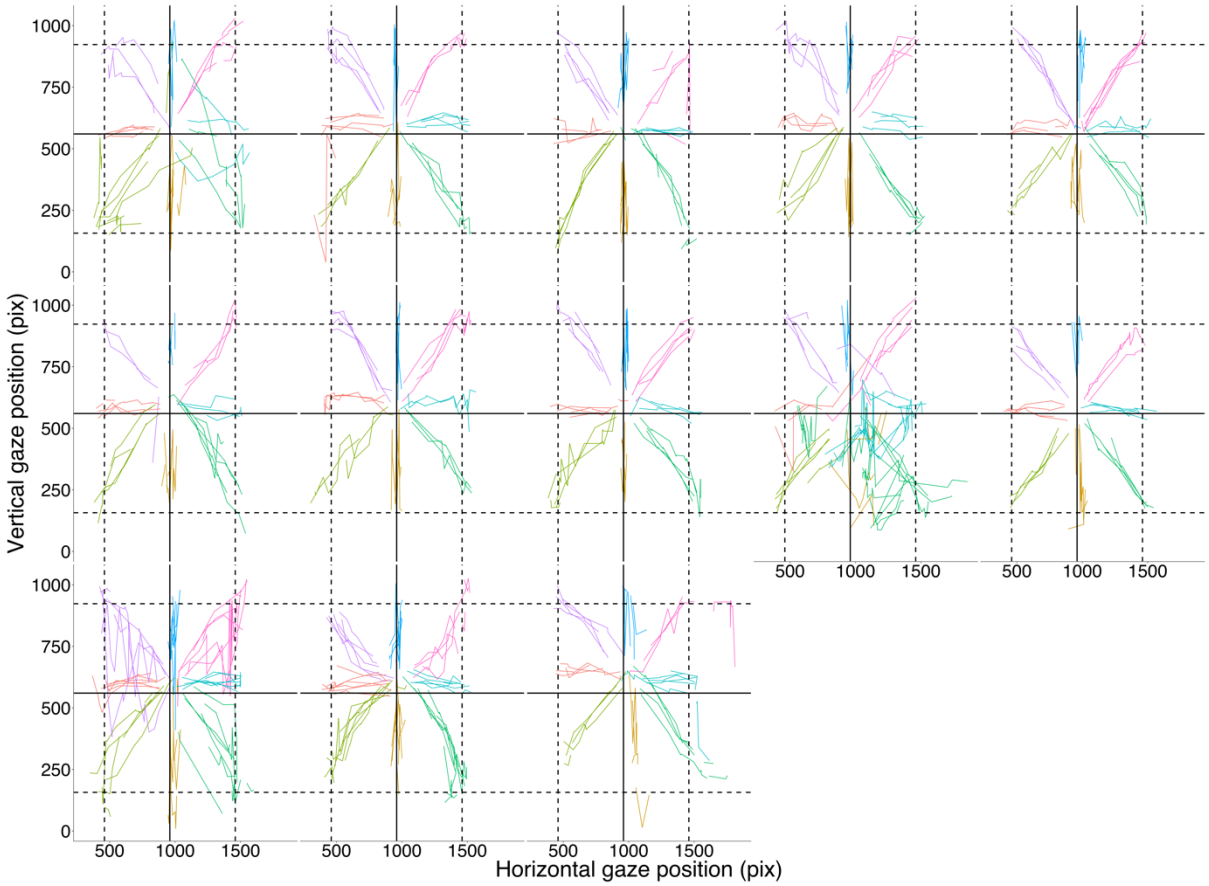

Figure 4.5 Saccade trajectories from the central target to each peripheral target.

Each graph shows saccade trajectories of a different participant (N=13; pix = pixel).

In summary, accuracy measures are comparable to the range reported in the existing eye-tracking literature for similar grade devices. In both ideal (chinrest) and non-ideal (no-chinrest) conditions, overall accuracy was at the upper limit or even higher than the values stated by the manufacturer (1 degree of visual angle). While this degree of accuracy is perfectly capable of capturing gaze behaviour reliably across most experimental tasks, tracking targets smaller than this error could be problematic. While clear separation of saccades and fixations is possible using the GP3-HD, study of the properties of saccade kinematics is compromised by the low sampling rate.

4.1.2.3 Discussion

Experiment 5 provided a standard validation of the GP3-HD eye-tracker. Overall, calibration and data loss were acceptable, with calibration achieved successfully for most participants after one or only a few attempts, and with a low rate of data loss over the experiment, comparable to more expensive eye-trackers based on previous reports (e.g., (Holmqvist, Nyström, Andersson, & Dewhurst, 2011). Accuracy and precision of gaze tracking was also acceptable, but closer to upper desired limits (~1 degree of visual angle). This means that during stimulus design and presentation, researchers should accommodate this tracking error by ensuring that target locations are separated by 3 degrees of visual angle or more, so that if a participant was looking exactly at the middle of two targets, the estimated gaze locations (even accounting for error) would not fall onto either of the targets. It is worth noting, however, that better accuracy values were achieved (0.5 degrees of visual angle) particularly for the horizontal dimension. This is useful to know, as it is possible to calibrate how gaze is assigned to areas of interest given the tracking error associated with a particular participant at a particular point in time (Hessels & Hooge, 2019; Orquin & Holmqvist, 2018). Similarly, individualised accuracy and precision profiles can be used to calibrate gaze parsing filters (see, Feit et al., 2017).

With regard to saccade metrics, while the identification of saccades appears good, the calculation of kinematic parameters appears to be affected by the low sampling rate. As a result, the study of the properties of saccades using the GP3-HD may lead to inaccuracies depending on the specific parameters being studied. For example, it appears that the amplitude of saccades is underestimated, as the gain value was < 1, indicating hypometric saccades, that is, saccades where the amplitude was smaller than expected. Looking at the plots in Figure 4.5, it is apparent that saccade patterns show breaks as a result of the sampling

rate. This is consistent with Nyquist's theorem which specifies that a signal must be sampled at more than twice the highest frequency component of the signal. Note, however, that saccade detection as well as peak velocity approximations are reliable with even 60 Hz sampling, and there are saccade reconstruction techniques that can improve the approximation of the “true” parameters of saccades (Wierts, Janssen, & Kingma, 2008).

The lack of consistent significant differences between chinrest and no-chinrest conditions suggests that the algorithm for gaze estimation used by GP3-HD is relatively robust to small head movements. However, increased infra-red ‘bounce’ was observed during the sessions with a chinrest, where small and temporary reflections from the chinrest would cause it to be mistakenly detected as a part of the eye. Improvements in tracking accuracy expected from use of the chinrest may have been lost as a result. While this problem is solvable by eliminating infra-red reflections, a possibility for development would be for Gazepoint to offer a user intervention enabling the operator to manually correct the misidentified reflections during calibration, such that the Gazepoint algorithm would subsequently underweight those regions when estimating gaze location. Similarly, providing the stream of estimated distances from the screen, in addition to the gaze coordinates and pupil samples, would be useful for researchers to accommodate changes in distance from the screen in non-chinrest conditions.

4.1.3 EXPERIMENT 6

Experiment 5 provided a standard validation of the GP3-HD eye-tracker in terms of its accuracy and precision, degree of data loss and the benefits of head stabilisation. While this provides a useful benchmark assessment of the GP3-HD, such eye-tracking validation studies do not reflect typical experiments in which task demands are usually greater and there are

more limited checks on the eye-tracker's performance imposed by study design (Niehorster et al., 2018). Therefore, Experiment 6 provides data from a real-world typical psychological experiment to assess GP3-HD performance. Importantly, as typical behavioural experiments may vary from a few minutes to hours, we also investigated how data quality parameters changed over time, across a one-hour-long experiment.

Experiment 5 also demonstrated that GP3-HD is able to capture pupil changes such as the PLR. In most cognitive and behavioural research, however, researchers are typically interested in how pupil changes are modulated by cognitive and affective factors rather than low-level visual properties. This can range from quantifying cognitive effort (Papesh & Goldinger, 2012; Piquado, Isaacowitz, & Wingfield, 2010) to emotional arousal (Bradley, Miccoli, Escrig, & Lang, 2008). Considering that cognitive and emotional effects on pupil diameter are much smaller in magnitude than luminance effects (Mathôt, 2018), it is unclear how well the GP3-HD is able to capture such effects. Experiment 6 used data from a paradigm that allowed for the exploration of how pupil size is modulated by emotional factors.

Specifically, the effect of viewing emotionally arousing images on pupil diameter was investigated. A positive correlation between self-rated arousal and pupil size was predicted. Since this task makes use of naturalistic visual stimuli varying in low-level properties, the luminance of the stimuli can also be regressed onto pupil size to model modulation of pupil size due to the PLR. Stimulus brightness should have a negative correlation with pupil size, such that brighter stimuli should lead to decreased pupil size (pupil constriction) whereas darker stimuli should predict increased pupil size (pupil dilation).

Additionally, in Experiment 6, SC and HR data was collected from participants simultaneously using both the GPB and a well-validated physiological recording system

(BIOPAC-MP160). Strong correlations between devices would indicate the capability of the GPB to capture physiological signals. Crucially, this experiment introduces the main paradigm which will be used to study typical and atypical physiological responses to emotions in Experiments 7 and 8.

4.1.3.1 Method

4.1.3.1.1 Participants

A total of 46 healthy participants with no recent history of mental health problems (28 female, 18 male, M = 23, SD = 5.54, 18 to 65 years old), with normal or corrected to normal vision and without makeup, took part in this study. Additionally, 10 participants with an independent diagnosis of autism were recruited at a later stage and their data used for a subset of the heart rate analyses (4 female, 6 male, M = 38, SD = 15.73, 18-65 years old). Participants took part in a psychological experiment where they had to view and rate emotional pictures, while their gaze and physiological signals were monitored. Participants were reimbursed £10 per hour or 3 course credits for their time.

4.1.3.1.2 Stimuli and task

Picture stimuli were 50 images from the International Affective Picture System (IAPS, Lang et al., 2008) designed to elicit emotional responses in observers (e.g., open lung surgery, naked bodies, etc). The stimuli were chosen to cover a wide range of valence (M = 5.07, SD = 1.92, Range = 1.46 – 8.19 – on a 1 to 9 scale) and arousal (M = 4.66, SD = 1.19, Range = 2.63 – 7.21) scores based on population norms. Participants viewed each stimulus and provided ratings for valence using a slider (‘How did you feel watching the image’) from ‘-10 (Extremely negative)’ to ‘+10 (Extremely positive)’; and arousal (‘How intense was your emotional response’) from ‘-10 (Extremely calm and relaxed)’ to ‘+10 (Extremely intense,

agitated)'. Each trial started with a fixation cross of variable duration (ranging from 7 to 15s), followed by presentation of the image for 6 seconds, after which participants provided their valence and arousal ratings – See **Figure 4.6**.

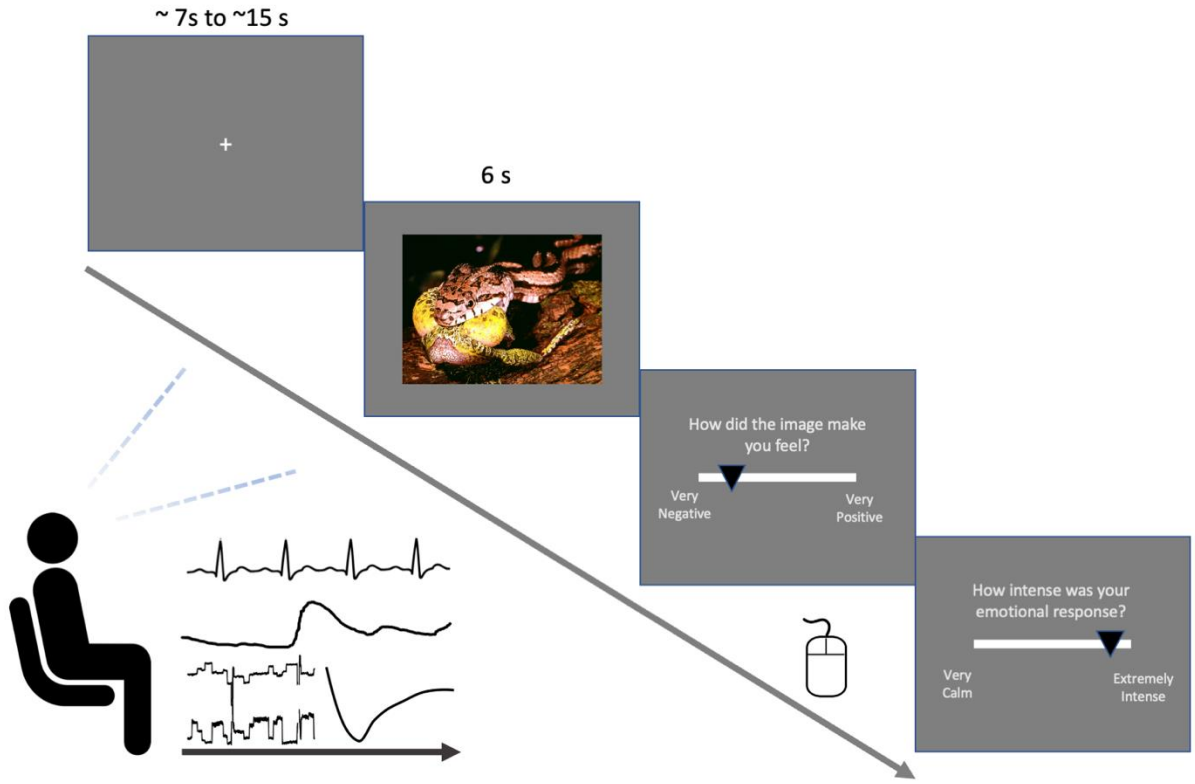

Figure 4.6 Schematics of Experiment 6.

4.1.3.1.3 Apparatus and task environment

The experiment was displayed on a computer monitor measuring 1920 x 1200 pixels with a fixed refresh rate of 60 Hz running on an Intel Core i9 Windows 10 computer (32GB RAM).

Eye tracking

The same GP3-HD eye-tracker from Experiment 5 was used to track eye-movements and pupil size with a sampling rate of 150 Hz. Setup and calibration were similar to Experiment 1. Recalibration was repeated after 25 trials (approximately 20 minutes). Participants were

asked to keep their head and body still and a chinrest was used. However, they could move their eyes freely to explore the images.

Accuracy and precision

Accuracy and precision were computed in a similar manner as in Experiment 5, however here computations were performed during the fixation screen that preceded each trial. The data were trimmed to 6 seconds and the first 250 ms after the start of fixation cross were removed. Outlier precision and accuracy values were removed as in Experiment 5.

Physiological recordings

Physiological recordings of SC, pulse and heartbeat data were obtained from two systems: the GPB and the BIOPAC M160 with EDA100C and ECG100C modules (details below).

Skin conductance – GPB. The GPB measures both heart rate and skin conductance via a sensor attached to two fingers (index and middle finger), without the need for extensive skin preparation or specialised electrodes. To record SC, the GPB uses an exosomatic recording method which applies direct current with a constant voltage source. The applied voltage is 5V through high impedance voltage division. The conditioning method uses an analog low pass filtered at 10 Hz with a sampling frequency of 150 Hz. The sensors use gold-plated steel electrodes strapped to distal phalanges of the fingers.

Heart rate – GPB. The SCR system described above allows detection of heartbeats at the middle or index finger using a photoplethysmography (PPG) method where a light beam is transmitted to the tissue and heartbeats are detected via intensity modulation of the reflected light. Note that the standard GPB system reports only heart rate data, however, through the provided API it is possible to store the raw pulse data using a custom script. This data was only available for a third of participants. Heart rate is computed using a moving average with

a window of 3 beats. For all measures, live monitoring was achieved via the Gazepoint control application.

Skin conductance – BIOPAC MP160. To validate the GPB, physiological data were also collected using a BIOPAC MP160 system sampling at 2000 Hz. SC data were recorded using the EDA100C module and TSR203 transducers. The skin where the GSR electrodes were placed was cleaned with water and dried with a cotton tissue. The EDA100C uses a constant voltage (0.5 V) technique to measure skin conductance. SC was collected via two electrodes that were placed on the inside of the left foot to measure GSR (see Section **8.3.1** in SM). Foot (rather than hand) placement was used to avoid creating interference between the two devices. The foot and palm fingers have been shown to be the best locations to measure SC and provide largely similar results (van Dooren, de Vries, & Janssen, 2012).

ECG - BIOPAC MP160. The ECG signal was recorded via the ECG100C electrocardiogram amplifier which records electrical activity generated by the heart. Two electrodes were placed on participants (see Section **8.3.1** in SM). One electrode was placed on the right collarbone and one on the lower left torso to measure HR. All sensors were well secured with surgical tape to prevent loss or disruption of signal. The skin where the ECG electrodes were placed was cleaned with Signagel Electrode Gel before attaching sensors.

4.1.3.1.4 Procedure

Following informed consent and the opportunity to ask questions, physiological recordings were prepared. A two-minute rest period was allowed for recordings to stabilise before preparations continued. The task started with a calibration, followed by another two-minute rest period, during which participants focused on a fixation cross in the centre of the screen, and a single practice trial. After 25 trials participants took a short break for recalibration. After the task they were debriefed and compensated. All research was conducted in

accordance with the revised 2013 Declaration of Helsinki and was approved by the local Research Ethics Committee.

4.1.3.1.5 Data pre-processing

Eye-tracking

Eye-tracking data were pre-processed following the same steps as in Experiment 5. For pupil analysis, pupil response was baseline corrected using an interval corresponding to one second before and one second after the stimulus, using the same method as in Experiment 5.

Skin conductance and heart rate

SC data from both the GPB and BIOPAC systems were analysed using a Continuous Decomposition Analysis (CDA) algorithm implemented in the open-source software Ledalab (Kaernbach, 2005). For analysis, data were first downsampled to 10 Hz and inspected for artifacts. Adaptive smoothing was used prior to analysis. All optimisations used the default values in Ledalab recommended for SC measurement and analysis (Boucsein et al., 2012). The global mean of the SC signal as well as the event-related phasic signal was computed.

Heart rate data

Established HR data-processing pipelines were used to process the heartbeat and ECG signal from the GPB and BIOPAC, respectively. Processing was accomplished via the ArtiiFact toolbox (Kaufmann, Sütterlin, Schulz, & Vögele, 2011). Artifact detection was achieved using the Berntson, Quigley, Jang, and Boysen (1990) algorithm based on individual thresholds derived from inter-beat-interval (IBI, also known as RR interval) distributions and their estimated real (not contaminated) distribution and interpolated using the cubic spline method. The peak of the R wave (heartbeat, see **Figure 4.7**) was detected using the global

threshold method (or local threshold method when drift was present), after low pass filtering at between 10 and 20 Hz.

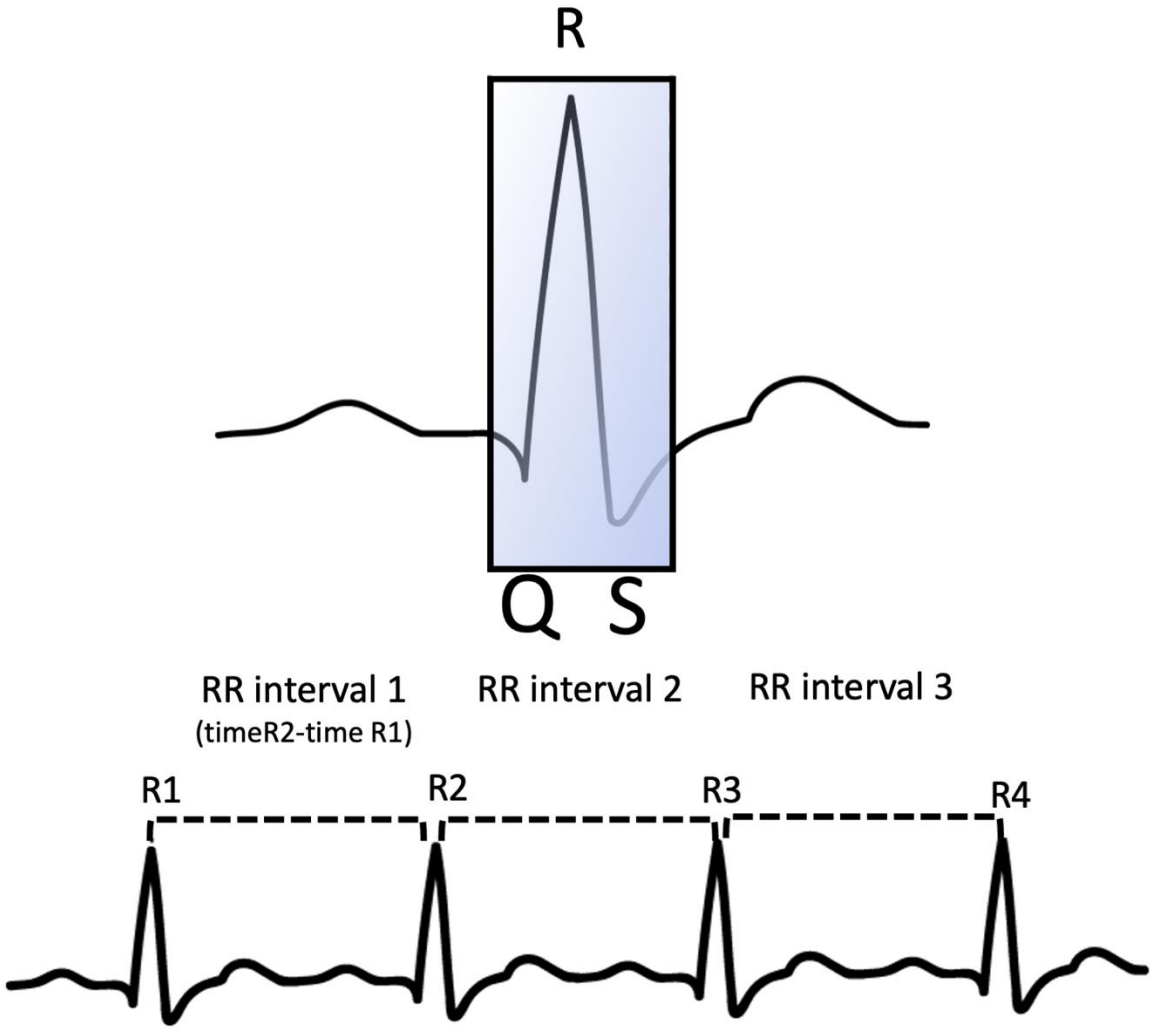

Figure 4.7 Heartbeat parameters.

Illustration of the elements of a heartbeat used to compute heart rate and heart rate variability. RR-interval (or inter-beat interval - IBI) is the time difference between successive heartbeats. NN-interval is a normalised RR-interval in which artefacts have been removed.

In addition to HR which was computed for the entire task, both time and frequency domain metrics of heart rate variability (HRV) were derived from the resting period. Here we report the standard deviation of NN intervals (SDNN), root mean square of successive RR interval differences (RMSDD), and percentage of successive RR intervals that differ by more than 50 ms (NN50) for the time domain; and absolute power of the high-frequency band (0.15–0.4 Hz) – HF, and low-frequency band (0.04–0.15 Hz) – LF for the frequency domain (Shaffer & Ginsberg, 2017).

4.1.3.1.6 Statistical analyses

Eye-tracking

In addition to descriptive metrics for all gaze position and calibration measures, variance in data loss, accuracy and precision during the task is also reported. To this end, linear mixed models implemented in R package lme4 (Bates et al., 2014), were fitted to assess the rate of change over time. For example, the rate of data loss over the course of the experiment was investigated by modelling each of the 25 trials immediately following successful calibration for a total of 25 trials. Linear and quadratic functions were used to approximate the rate of change over time as these could easily describe linear or accelerated/delayed changes in the metrics**.** This approach is known as polynomial modelling or growth curve analysis (see, Mirman, 2014).

Fitted models had a quasi-maximal structure:

(e.g., loss ~ (linear + quadratic) * block + (1 + linear + quadratic | participant id),

with linear and quadratic representing orthogonal polynomial terms created as powers of the trial number, and trial id and participant id were estimated as a random intercept. Block was dropped as a random slope due to near zero variance and convergence errors.

Pupil size

Baseline-corrected pupil responses to each picture were regressed onto self-reported ratings of arousal and mean brightness values for each image, while controlling for random effects of participant and stimulus id using linear mixed models. Random slopes were dropped due to near zero variance or convergence errors, and the final models had the form:

pupil ~ arousal rating + mean brightness + (1 | stimulus id) + (1 | participant id).

All continuous predictors were centred and scaled.

Skin conductance and heart rate

The average SC signal during each trial, event-related phasic SC responses and HR metrics were correlated between the GPB and BIOPAC systems. HRV measures were only computed for the subsample of participants for whom the raw timeseries of heart data was available for both the GPB and BIOPAC. This subsample had 20 participants with raw pulse measurements (10 neurotypical individuals included in the other analyses, and an additional 10 autistic participants). For this subsample all correlations are Kendal rank correlations due to small sample sizes.

4.1.3.2 Results

4.1.3.2.1 Eye-tracking

Calibration

Calibration quality and other descriptive statistics are provided in **Table 4.3**. The average calibration error across all three accepted calibrations during the one-hour session was within the expected range, with a mean of .98º (SD = .45º), and it took an average of 1.66 attempts to achieve an acceptable calibration.

Table 4.3 Descriptive measures for eye-tracking data.

Variable	Mean	SD	Range
Calibration count	1.66	0.98	1-5
Calibration error	0.98	0.45	0.49-4.92
Gaze loss	8.3%	15%	0-100
Accuracy			
Global	0.77	0.70	0.23 –13.53
Vertical	1.33	0.73	0.27 -9.50
Horizontal	0.54	0.50	0.30-6.34
Precision			
Global	0.27	0.11	0.15-2.14
Vertical	0.30	0.13	0.11-.95
Horizontal	0.24	0.09	0.09-.60

Notes. Calibration error, accuracy and precision data is reported in degrees of visual angle (range is reported at the trial level).

Data loss

On average, less than 10% of gaze data were lost, which is consistent with the typical rate of loss reported in the literature and even lower than some more expensive systems (e.g., Tobi TX300 based on (Holmqvist, 2017). During the task less than 1% of the data fell outside screen bounds.

Data loss increased at a linear rate (Estimate = .004, SE - .002, t = 2.05, p = .04) across the experiment. There was also a main effect of block, with trials after the recalibration showing reduced data loss (Estimate = -.12, SE = .02, t = -5.08, p < .001). The only significant interaction was between the quadratic term and block (Estimate = .05, SE = .02, t = 2.43, p = .02): post-hoc tests indicated that the rate of loss in the first block additionally followed an ‘inverted-U’ shape, that is, it was characterised by rapid increase in data loss in the first few trials until the rate of loss stabilised and reduced in the last few trials (Estimate = .01, SE = .002, t = 5.07, p < .001).

Accuracy

Accuracy values were in the expected range, averaging between 0.5º to 1.33º, with better accuracy on the horizontal dimension with values consistently ~ 0.5º (see **Figure 4.8**). Overall, the global accuracy changed at a linear rate such that for every trial, tracking accuracy deteriorated by 0.04º (Estimate = .04, SE = .01, t = 3.21, p = .001). There were also block differences such that the second block showed better accuracy, with re-calibration improving accuracy by .06º compared to the previous trial (Estimate = .06, SE = .02, t = 2.17, p = .01). Results were consistent for vertical and horizontal accuracy.

With respect to horizontal accuracy, for every trial after calibration tracking accuracy deteriorated by .03º (Estimate = .03, SE = .009, t = 3.21, p = .001) and recalibration after every 25 trials improved accuracy by on average .05º (Estimate = .05, SE = .02, t = 2.49, p = .01). For vertical accuracy there was an interaction between block and the linear parameter (Estimate = .05, SE = .02, t = 2.79, p < .001), such that in the first block accuracy declined more linearly than in the second block.

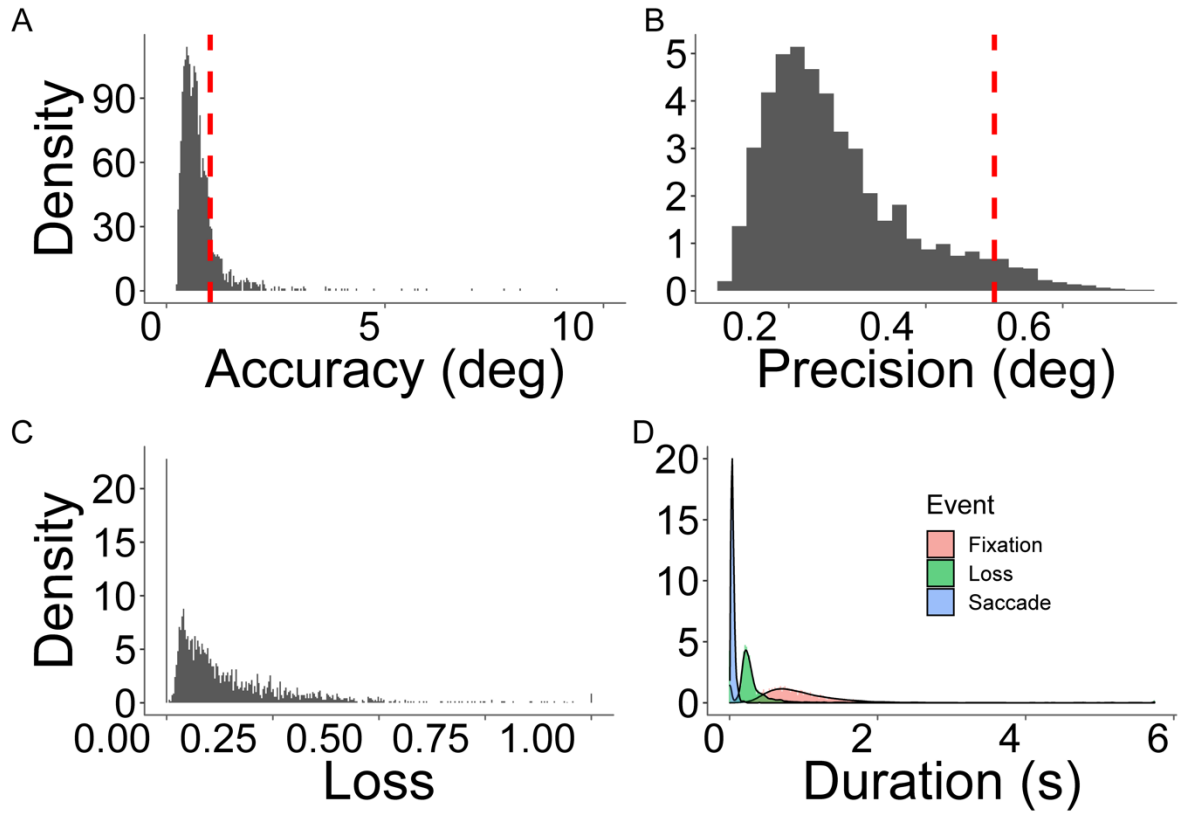

Figure 4.8 Density plots for accuracy.

(A), precision (B), and data loss (C). Frequency of fixations, saccades and data loss for each trial and participant (D). Data represents the density of every single observation for every participant for each trial.

Precision

All precision values were within an acceptable range < 0.5º, with the horizontal dimensions showing better tracking precision. There was no effect of trial on precision nor interactions with block. There was only a main effect of block, with the second block showing better precision (Estimate = .007, SE = .002, t = 2.93, p = .002). Both vertical and horizontal precision showed the same effect.

Pupil size

Experiment 6 aimed to explore whether the modulation of pupil size by emotional arousal is detectable with the GP3-HD, considering that such effects are orders of magnitude smaller than the PLR detected in Experiment 5. A main effect was observed for self-reported arousal in the predicted direction (Estimate = .24, SE = .05, t = 4.35, p < .001) which remained significant after statistically controlling for brightness. This shows that self-perceived arousal is positively related to pupil change. The predicted effect of brightness on pupil size was also significant, and larger (Estimate = -1.02, SE = .12, t = -8.59, p < .001), demonstrating again that brightness is negatively related to pupil change. As predicted, the effect of arousal on pupil size was less than 1% of the size of the brightness effect and showed more individual variability in comparison to the PLR (see **Figure 4.9**). The model explained 54% of the variance in pupil size, of which 23% was explained by the fixed effects (discounting the random effects of participants and trial). Thus, Experiment 6 provides support for the use of the GP3-HD eye-tracking system for the study of gaze position and pupil size in typical psychological experiments. Tracking capability is better horizontally than vertically, but the vertical accuracy also remained acceptable range (~ 1°).

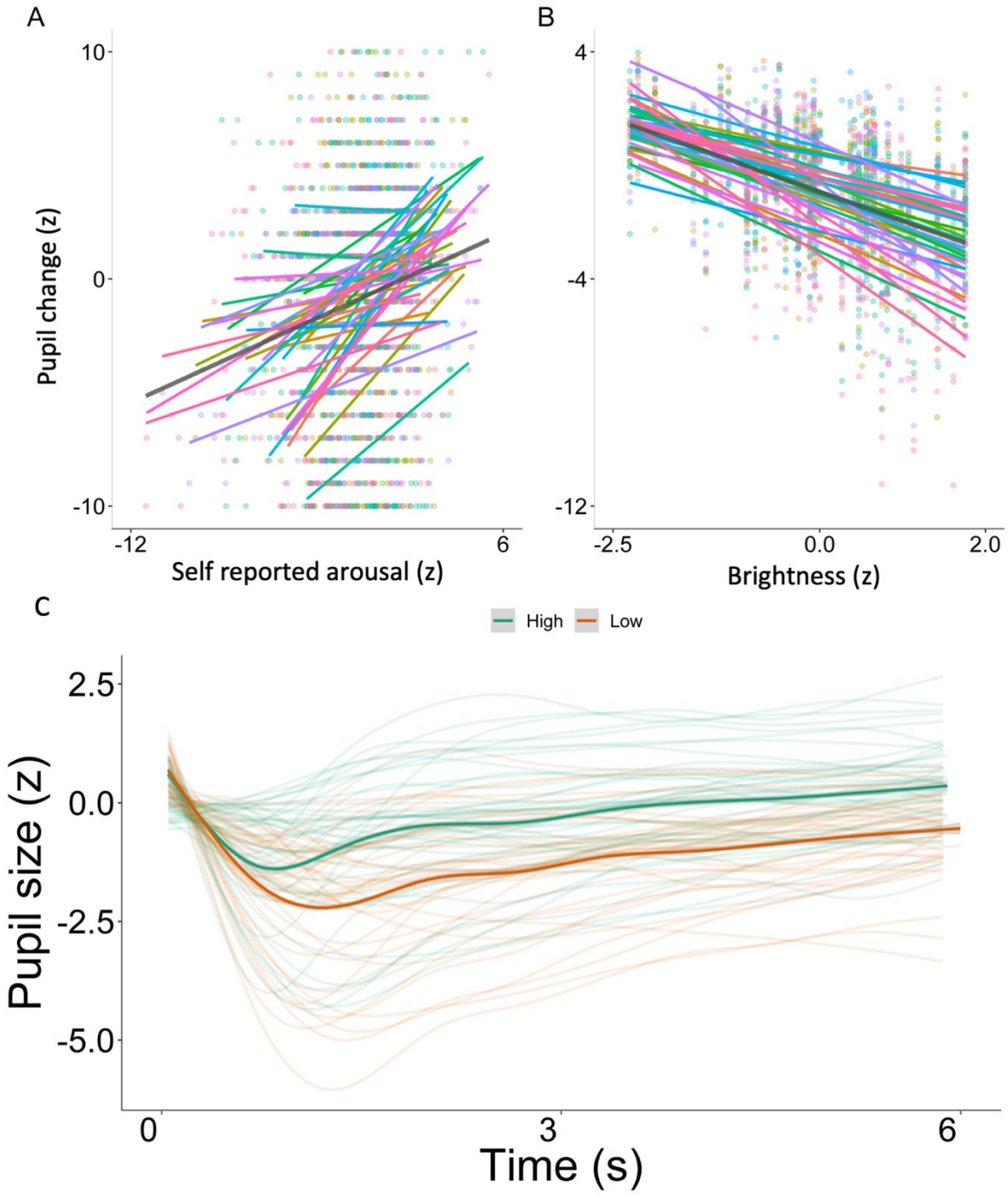

Figure 4.9 Pupil and arousal.

Self-perceived arousal in response to emotional stimuli correlated with pupil size (A) as did stimulus luminance (B). C. The timecourse of pupil response by high and low arousal stimuli (split for visualisation only). Individual lines represent individual participants average pupil trace.

A second goal of this study was to provide a validation of the GPB system, by comparing SC and HR obtained using the GPB with data collected from the well-validated BIOPAC MP160 (see **Figure 4.10**).

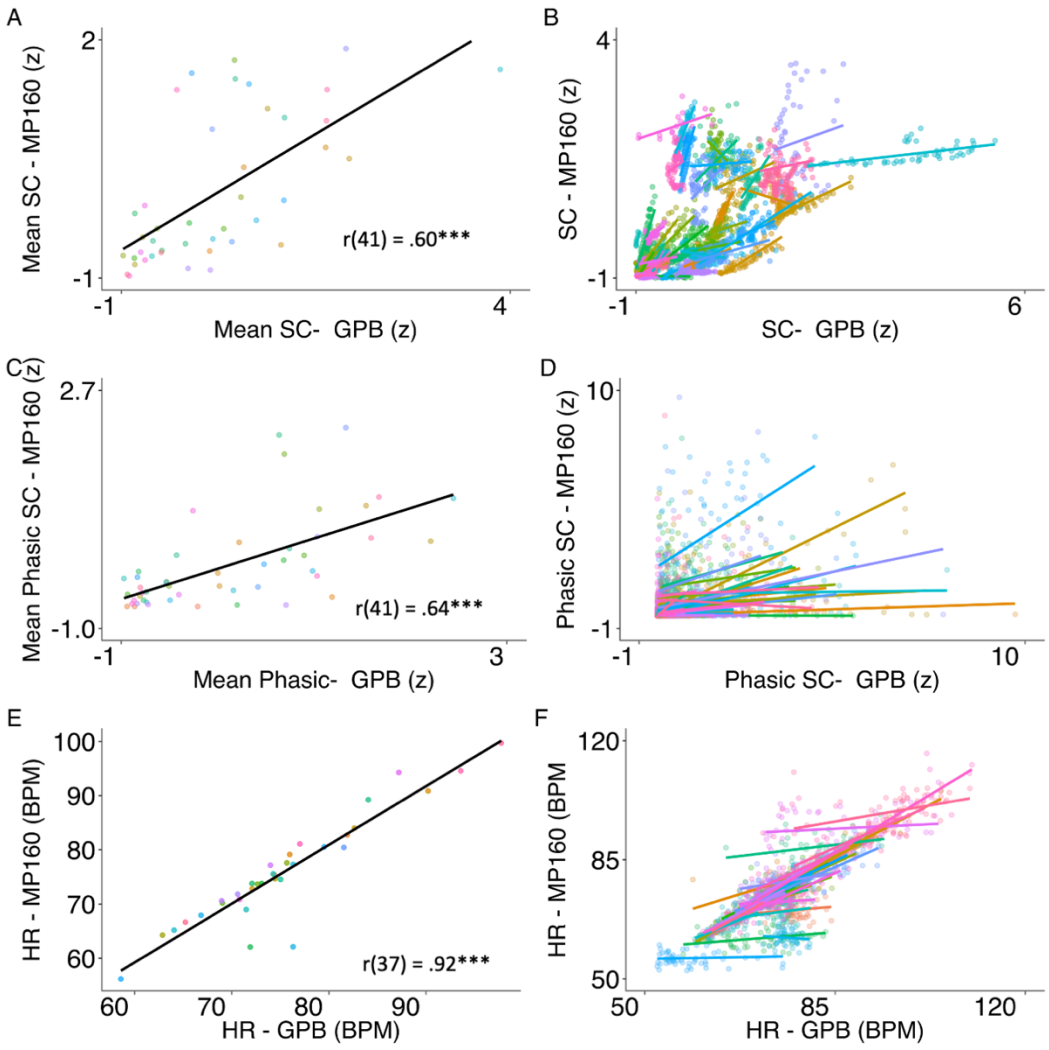

Figure 4.10 Skin conductance and heart-rate.

A-D: Scatter plots for raw averages of the skin conductance signal across the Gazepoint biometrics (GPB) and BIOPAC MP160 systems. E-F: Scatter plots for correlations of computed heart rate from Gazepoint biometrics system and BIOPAC MP160 ECG. Left plots show data aggregated by participant, and right plots show data for all trials and participants. Each colour and line represent a single participant, each dot represents a single trial. *** $p < .001$.

4.1.3.2.2 Psychophysiological responses

Skin conductance

The SC measurement capacity of the GPB showed excellent robustness, with less than .06% of loss compared to no loss of signal in the BIOPAC-MP160. The average SC signal from the GPB and BIOPAC showed a strong correlation ($r_{(37)} = .60$, $p < .001$). This was also consistent when looking at specific derived measures of skin conductance from Ledalab, such as a decomposed phasic signal ($r_{(41)} = .64$, $p < .001$), with measurements across both devices sharing 40% of the variance. However, as illustrated in **Figure 4.10** there is a significant degree of difference in how well these signals correlate at an individual level. This may be related to differences in how similar the physiological properties are in the measured locations for each participant (foot vs. palm), as well as the fact that the SC signal from the GPB is smaller in range compared to the BIOPAC signal.

A sample of the SC signal from an example participant is shown in SM - **Figure 8.6** for both the GPB and BIOPAC systems. One obvious difference is that the signal from the BIOPAC is much larger in magnitude compared to the GPB signal. This results in more signal being preserved after removal of tonic data in BIOPAC compared to GPB data.

Heart rate

Quality checks indicated that the GPB lost on average 19% (SD = .37) of data (impossible HR values or loss of signal) compared to no obvious data loss for the BIOPAC system (i.e., peaks were still present even in periods of relative noise). Importantly, however, participants were not more likely to lose data as the task progressed (Estimate = .004. SE = .005, $t = .69$, $p = .45$). This was a concern as the strap of the sensors may be thought to limit the blood flow to the finger, systematically affecting heartbeat tracking over time. HR recorded from the GPB and BIOPAC were, however, very strongly correlated ($r_{(37)} = .92$, $p < .001$, Figure

4.10), demonstrating that the GPB provides valid measurement of heart rate, corresponding to systems which are much more expensive. Results were similar whether using interpolated or non-interpolated GPB heart rate data. Notably, these correlations are much larger than the correlations between the systems for SC signals, however the loss of HR data is much greater than the loss of SC signals for the GPB system.

Another ECG metric of interest for many researchers is heart rate variability (HRV). Notably, HRV measures require more sensitive and less noisy recordings than HR, which is relatively stable.

Heart rate variability

Both time and frequency domain measures of HRV were considered. Overall, there were strong correlations between GPB and BIOPAC MP160 recordings and derived HRV metrics. All correlations were > .6 (see Table 4.4).

Table 4.4 Heart rate and variability correlations between the GBP and BIOPAC systems.

Variable	Correlation
HR	.92***
Time domain	
SDNN	.73***
PNN50	.63***
RMSSD	.61***
NN50	.65***
Frequency domain	
HF	.75***
LF	.76***

Notes. N = 20. Analyses are reported excluding outliers, but results did not differ significantly with the inclusion of outliers (there was only one outlier per analysis). *** $p < .001$. All correlations are Kendal rank correlations. *HR:* Heart rate; *SDNN:* Standard deviation of NN intervals; *RMSSD:* Root mean square of successive RR interval differences;

NN50: Percentage of successive RR intervals that differ by more than 50 ms; *HF:* Absolute power of the high-frequency band (0.15–0.4 Hz); *LF:* Low-frequency band (0.04–0.15 Hz).

Overall, the GPB system shows strong concordance with the well-established BIOPAC MP160 system. Notably, the HR measurements were much more consistent than SC, despite the increased data loss (see **Figure 4.11** Comparison plots of heartbeat data from the Gazepoint biometrics (GBP) and BIOPAC MP160 systems.).

4.1.3.3 Discussion

Experiment 2 provided further validation of the accuracy, precision, and robustness of the GP3-HD, with accuracy generally within 1º of visual angle and precision < 0.5º. In addition, correspondence was observed between self-reported arousal and pupil size, showing that the small changes in pupil size due to emotional arousal can be measured using the GP3-HD. Furthermore, the PLR was measured in response to luminance changes which were less marked than those in Experiment 1. The changes over time observed for data loss, accuracy and precision are consistent with observations that eye-tracking data quality deteriorates over time (Holmqvist, Nyström & Mulvey 2012; Hessels et al., 2017). Researchers should accommodate this in study design, for example by including frequent recalibration or breaks to reduce participants' fatigue.

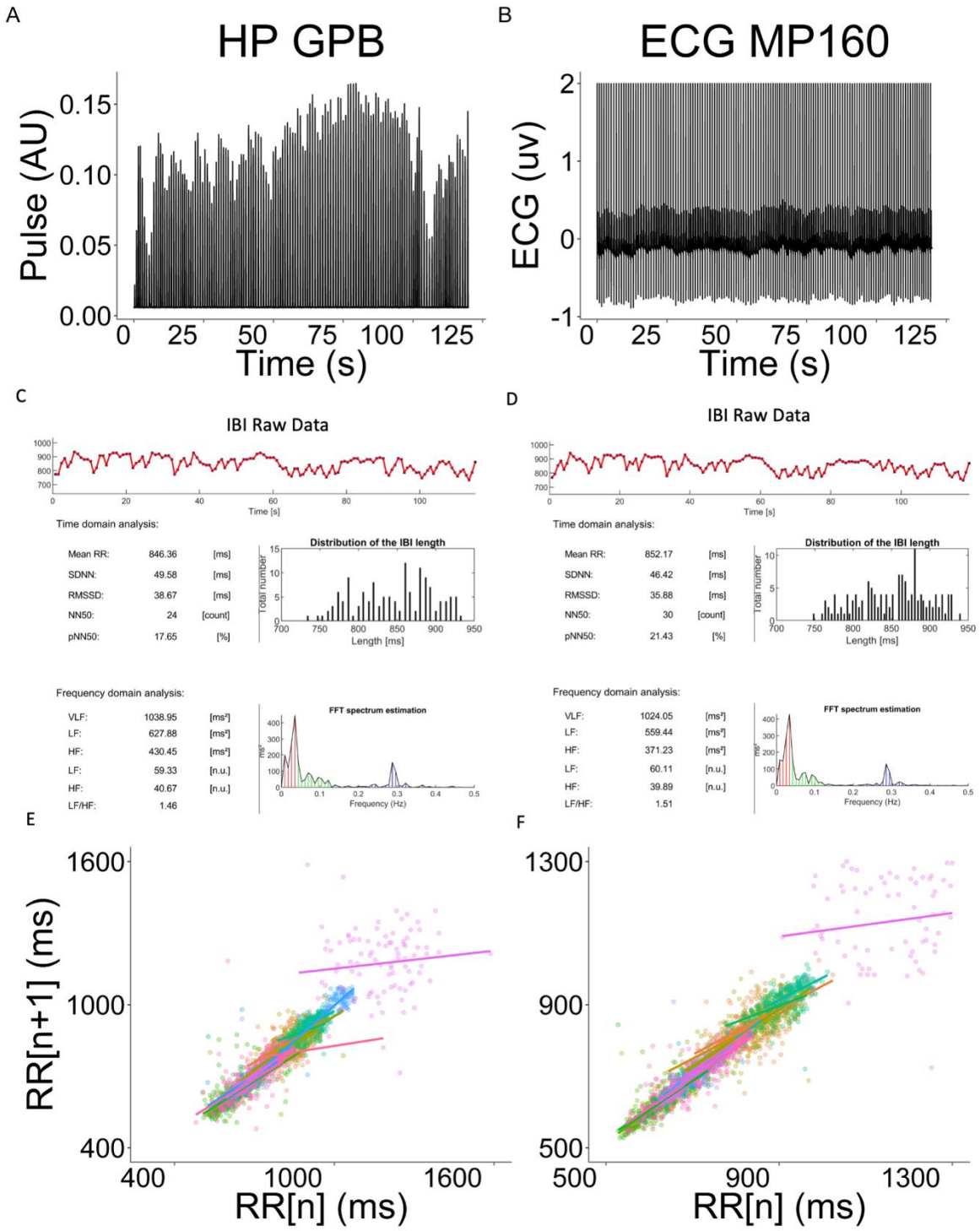

Figure 4.11 Comparison plots of heartbeat data from the Gazepoint biometrics (GBP) and BIOPAC MP160 systems.

A and B shows a representative participant's raw recording of pulse (GPB; A) and electrocardiogram (BIOPAC MP160; B) data. C and D shows derived heart rate variability

metrics for the same participant across the two devices (C = GPB, D = BIOPAC). E and F shows point-care plots for the inter-beat-interval (IBI) for all participants. Each colour denotes a single participant, each dot denotes a single RR (peak-to-peak) duration.

Finally, the comparison between SCR and HR raw and derived metrics suggests moderate to very strong agreement between the GPB and the well-established BIOPAC MP160 system, with correlations ranging from .6 to .95. However, the low amplitude of the SC signal from the GPB system means it is likely to make the separation of tonic and phasic components more difficult (Edelberg, 1993; Society for Psychophysiological Research Ad, 2012), than the SC signal from the BIOPAC system. Nonetheless, estimation of resting and stimulus-evoked SC responses is possible, as indicated by a relatively strong correlation of SC measurements between devices. For both heart rate and heart rate variability metrics (in both the frequency and time domain) the recordings from the GPB showed remarkable consistency with those from the BIOPAC system. However, measurement of the pulse by the GPB system was more prone to data loss than the ECG system used by the BIOPAC. This data loss may cause problems for heart rate variability analyses.

4.1.4 General Discussion

Experiments 5 and 6 aimed to assess the validity of the GP3-HD eye-tracking system. While the manufacturer's stated levels of accuracy are possible to achieve (0.5 – 1º), in two studies the average accuracy of the system was closer to 1º, with the horizontal accuracy and precision matching the stated ~ 0.5 and < 0.5, respectively. This is similar to what has been reported for similar grade (e.g., EyeTribe, Tobii EyeX) as well as higher grade devices (e.g., Tobii TX 300) in large scale eye-tracking comparison studies, where the measured accuracy averaged around

1º (Funke et al., 2016; Holmqvist, 2017; Niehorster et al., 2017). Data loss is also a good indicator of the capabilities of a system and in this regard, the GP3-HD also performs quite well, with discarded data after cleaning making up on average less than 10% of the collected data, compared to reported data loss in different systems which ranges from less than 2% to up to 20% (Holmqvist, 2017).

It is important to note that in behavioural studies, reduced accuracy can also result from participant behaviour rather than hardware limitations, as the computation of accuracy and precision is reliant on participants attending to the targets. Nonetheless, this type of validation represents the most likely scenario under which most eye-tracking systems will be used with human participants. The lack of any major effects on the quality of gaze estimation between chinrest and no-chinrest conditions in Experiment 5 suggests that the gaze estimation algorithm for the GP3-HD is robust. It is worth noting, however, that in both conditions participants were instructed to avoid body and head movements. Other studies have shown that body and head position can severely affect the quality of data obtained from remote eye-trackers (Niehorster et al., 2018), and that infants and participants with neuropsychiatric conditions are more likely to show poor data quality, in terms of calibration, accuracy, precision and data loss (Dalrymple, Manner, Harmelink, Teska, & Elison, 2018; Hessels & Hooge, 2019; Holmqvist et al., 2012). Therefore, it is recommended that experimenters systematically assess data quality parameters when using GP3-HD for experimental research.

One issue to consider when using the GP3-HD is that tracking participants' gaze when using chinrests or with additional objects near their head (e.g., headphones, glasses) may cause infrared bounce. This was observed during some recordings, where transient reflections from headphones (which was required for a separate task) or the chinrest would cause gaze estimation failures. While these data samples are typically flagged as invalid by the GP3-HD

algorithm, it can increase data loss, in some cases sufficiently to invalidate a full trial if no correction is made. Covering areas that are likely to be reflective with non-reflective tape - are usually enough to solve this issue.

Analyses of gaze position metrics show that detection of fixations and saccades is reliable, yet measurement of the kinematics of saccades are negatively impacted by the low sampling rate. Similarly, analyses of velocity profiles show that known relationships between saccade parameters, e.g., saccade velocity and amplitude, or saccade duration and amplitude, recorded from the GP3-HD only approximate the expected relationships. Nonetheless, any inaccuracy in the estimation of fixation and saccade metrics is likely systematic, such that comparisons between different conditions, individuals or groups should be possible, if all other factors are taken into account (e.g., differences in accuracy or precision).

Finally, in terms of the software for collection and analysis of data, the GP3-HD software is unlikely to meet the demands of most experimental research. However, using the Gazepoint API, a number of popular experimental software libraries now support Gazepoint eye-trackers, such as PsychoPy (Peirce et al., 2019), PyGaze (Dalmaijer, Mathôt, & Van der Stigchel, 2014), OpenSesame (Mathôt, Schreij, & Theeuwes, 2012), and psychtoolbox (Kleiner, Brainard, & Pelli, 2007). Similarly, for analyses, the output generated by Gazepoint can be imported into third party open-source software like Python and R (or proprietary software such as MATLAB) for further processing.

Gazepoint biometrics system

Overall, the GBP system showed a high degree of consistency with the well-established (and considerably more expensive) BIOPAC system, which is often considered to be the ‘gold-standard’ for physiological recording. However, specialised pre-processing of pulse data obtained from the GBP system is necessary for calculation of HRV metrics. Similarly, like

other PPG measures the study of properties of the pulse (or heartbeat) signal - such the QRS complex - is likely to be challenging (although see Chiu, Shuai, & Chao, 2020). While the SC signal is more robust to data loss, the low amplitude of the signal is likely to make the separation of phasic and tonic measures of SC more difficult (Edelberg, 1993; Society for Psychophysiological Research Ad, 2012).

Another problem relates to motion and respiration artifacts. Irregular respiration and deep breaths cause fluctuations in the SCR signal which may lead to inaccurate SCR detection (Posada-Quintero & Chon, 2020). In systems like the BIOPAC MP160, it is possible to also collect respiration (with additional modules) and using this information to remove artefactual SCRs caused by respiration. Such automation is impossible with the GPB. This also means that tasks involving physical activity cannot be used when measuring SC and HR with the GPB. The GPB design is also optimised for use in the right hand, while it works on the left hand the positioning is less ideal, which may be a problem in reaction-time tasks where the use of a dominant right-hand is needed.

In terms of software integration, at the time of writing, raw recordings of SC, HR and pulse are not accessible by default in the implementation of Gazepoint systems in PsychoPy, OpenSesame or psychtoolbox. However, it is possible to access these data through the provided Gazepoint API. Similarly, the current iteration of Gazepoint's collection and analysis software does not include the raw pulse rate, although it is likely to be included in future releases. As with the eye-tracking data, however, output from the GPB can be exported to be used in open-source toolboxes for analyses of SC such as Ledalab (Kaernbach, 2005) or PSPM (Bach & Staib, 2015), or heart rate variability such as Artifact (Kaufmann et al., 2011) or Kubios (Tarvainen et al., 2014). Based on our tests, simple k-means clustering on the raw

timeseries of pulse data from the GPB provided acceptable classification of heart beats, which means that basic processing pipelines can be used relatively easily.

4.1.5 Conclusion

Two experiments assess the validity of a new low-cost eye-tracking and psychophysiology system from Gazepoint. Results show that the GP3-HD eye-tracker shows acceptable accuracy and precision, with only the study of saccade kinematics likely to be problematic. The GP3-HD was also shown to reliably capture the PLR and arousal effects on pupil size. Measurement of SC, HR and HRV from the GPB show a high degree of consistency with the well-established BIOPAC MP160 system. However, the low amplitude of SC signal may make it difficult to parse small phasic responses, and the relatively high degree of pulse rate loss in some participants may render pulse data unsuitable for HRV analyses without extensive pre-processing.

Supplementary materials: 8.3 Chapter 4. Supplementary Materials.

Extended paper supplement https://osf.io/8qxk4/

Materials: Code and data are available at https://osf.io/8qxk4/.

4.2 EXPERIMENT 7: Limited autonomic space coherence for emotion: Comparing pupil, skin conductance and heart rate

Abstract

Physiological indices of autonomic state such as changes in pupil dilation, skin conductance (SCR) and heart rate (HR)**,** are frequently used interchangeably as markers of emotional experience. The degree of concordance between these indices is currently unclear and the potential of multivariate profiles of autonomic activation to emotions has been rarely explored. The current study, therefore, investigated fluctuations in the relationship between pupil dilation, SCR, HR, and subjective emotional experience (valence and arousal) during an emotion-inducing task. Multivariate profiles of physiological responding were explored using data-driven clustering. Results suggest asymmetries in the relationship between subjective arousal and pupil dilation, SCR, and HR based on self-reported valence, reflecting individual differences in autonomic reactivity, subjective emotional experience and their relationship. There was also limited concordance across the various physiological indices of autonomic state, suggesting they cannot be used interchangeably as markers of emotional experience. Despite this lack of concordance, data-driven clustering revealed subgroupings of multivariate profiles of physiological responses that loosely mapped onto self-reported valence and arousal. Future work should explore the robustness and replicability of data-driven multivariate patterning of pupil dilation, SCR and HR as a function of emotion.

Keywords: pupil, skin conductance, heart rate, emotion, autonomic state-space.

4.2.1 Introduction

Identifying the physiological markers of subjective emotional experience has long been a major goal of those interested in understanding emotion. Despite this, whether there are specific bodily and/or brain signatures for all emotional states has been the subject of great disagreement between emotion theorists; while some have claimed the existence of highly specific physiological correlates of at least basic emotions such as fear, disgust and anger (Ekman, 1992; Stemmler, 2004), others have argued that the physiological correlates of emotional states are undifferentiated (Barrett, 2006; Kreibig, 2010). Between these two positions, a general consensus has emerged that emotion involves some form of homeostatic signalling processes which result in measurable physiological modulation, and that this physiological modulation maps onto general dimensions of arousal and valence (Cacioppo, Berntson, & Larsen, 2000; Kreibig, 2010; although see Siegel et al., 2018).

In order to identify the physiological markers of subjective emotional experience, autonomic signals are often recorded; with three of the most common being pupil changes (dilation/constriction), skin conductance and heart rate (Cacioppo et al., 2000; Kreibig, 2010). With respect to the current study, of interest is that these changes are increasingly being used interchangeably (particularly across studies), with each of the above being used, for example, as a marker of the physiological arousal associated with emotional experience, as highlighted in several reviews and meta-analyses (Kreibig, 2010; Norman et al., 2016; Siegel et al., 2018). Whether such interchangeable use is appropriate is not clear: while the physiological signals share some similarities in their dynamics (e.g., relatively slow responses) (Bradley, Miccoli, Escrig, & Lang, 2008; Mathôt, 2018), they are partly controlled by different subcortical and cortical structures (Dawson, Schell, & Filion, 2016; Tranel & Damasio, 1994). Similarly, within the autonomic nervous system (ANS), different neuronal populations

are more or less engaged in arousal modulation of different autonomic outputs (Folkow, 2000; Jänig & Häbler, 2000).

Furthermore the ANS is affected by both afferent and efferent interactions with the central nervous system (CNS), which means that regulation of various autonomic signals may vary by the degree of afferent and efferent traffic specific to each autonomic modality (Hamm & Weike, 2005; Kreibig, 2010; Leuchs, Schneider, & Spoormaker, 2019; Quigley & Barrett, 2014). Perhaps as a result, the various physiological signals vary in their phasic properties, habituation, rate of change, and recovery profiles (Bacigalupo & Luck, 2018; Bradley, Lang, & Cuthbert, 1993; Leuchs et al., 2019; Snowden et al., 2016).

Therefore, while the use of SCR, pupil and heart rate measurement remains ubiquitous in emotion research and is increasingly used interchangeably to index subjective emotional experience (as well as other cognitive processes, e.g., fear conditioning), it is unclear how findings obtained from one modality translate to another, particularly given the prevalence of single modality recordings. For example, while some studies have shown that pupil changes and SCRs track arousal regardless of valence (Bradley et al., 2008), others have shown distinct or asymmetric response profiles for positive and negative emotional experiences (Babiker, Faye, & Malik, 2013; Vasquez-Rosati, Brunetti, Cordero, & Maldonado, 2017). Similarly, while some studies found similar patterns of relationships with subjective emotional experience (arousal or valence) across physiological signals (Bradley et al., 2008), others have not (Wang et al., 2018).

Of note is the fact that the correlation between different psychophysiological responses is at best modest, and mostly significant only at a group level (Bradley et al., 2008; Cacioppo et al., 2000; Kuzinas, Noiret, & Bianchi, 2016; Wang et al., 2018). Such heterogeneity of results across studies coupled with poor correlations between physiological signals has led many to

conclude that the link between physiological responses and subjective emotion states is tentative at best, and highly heterogeneous across individuals (Barrett, 2006; Cacciopo et al., 2000; Kreibig, 2010; Siegel et al., 2018).

Given the common use of pupil, SCR and heart rate to qualify subjective emotional experiences, more research is needed to characterise the profile that best accounts for the relationship between emotional experience and each signal in combination (Kreibig, 2010; Leuchs et al., 2019). It may be the case that emotional reactions are best characterised as a multivariate pattern of responses in a physiological state-space instead of set patterns across physiological modalities (Berntson et al., 1991; Stemmler, 2004). Examining the relationship between groups of physiological signals and subjective emotional experience allows one to identify whether multivariate combinations of physiological signals may be better than any one of those signals in isolation in providing a biological marker of emotional experience (Berntson, Cacioppo, & Quigley, 1991; Friedman, Stephens, & Thayer, 2014; Stemmler, 2004). One useful but seldom-employed approach to test the utility of combinations of physiological signals to predict emotional experiences is data-driven clustering. An advantage of multivariate approaches is that they can describe multidimensional response profiles with no assumptions about how many and which categories of subjective emotional experience exist, and whether or how boundaries separate emotional experiences - all of which are topics of fierce debate among emotion theorists and thought to drive at least some of the mixed findings regarding the relationship between physiological responses and subjective emotional experience (Kreibig, 2010; Norman, Necka, & Berntson, 2016; Siegel et al., 2018).

The current study, therefore, had two aims. First, to investigate trial-by-trial fluctuations in the relationship between pupil size, HR and SCR, and subjective emotional experience in

terms of valence and arousal. A particular focus was on how the relationship between subjective emotional experience and the physiological signals varies across modalities, as well as the degree of concordance between the different physiological modalities. Based on previous research, it was predicted that pupil size and SCR would show a positive relationship with subjective arousal regardless of valence. As discussed previously, the relationship between HR and subjective arousal has been observed to be both positive and negative across studies, thus, no directional predictions were made. Similarly, given the mixed findings regarding the relationship between the different modalities (Kreibig, 2010; Norman et al., 2016), no firm predictions could be made but any observed relationship between modalities was expected to be small in terms of effect size.

The second goal was to explore how physiological responses to emotion-inducing stimuli clustered across the three modalities (pupil, SCR and HR), and how the clustering of physiological responses relates to subjective emotional experience. Motivated by previous meta-analyses and reviews (Cacioppo et al., 2000; Hamm & Weike, 2005; Kreibig, 2010), it was expected that any clusters would map loosely onto valence (negative and positive) and arousal (high and low) dimensions.

4.2.2 Methods

All research procedures were approved by the Medical Sciences Ethics Committee (IDREC) – reference: R60697/RE004 and were in accordance with the revised 2013 Declaration of Helsinki. A total of 51 healthy participants (33 female; M_{age} = 24.88, SD_{age} = 7. 7.15) recruited from the university and the community sample pool took part in the study for either course credit or monetary compensation. Sample size was determined a priori based on a

power analysis which combined effect sizes from previous literature and from a pilot study (10 participants) to conduct simulations for mixed models (DeBruine & Barr, 2021).

4.2.2.1 Emotion Task

A total of 56 images were selected from the International Affective Picture System (IAPS, Lang et al., 2008), which are designed to elicit emotional responses. The complete list of stimuli is provided in SM (**8.3.2**). The stimuli were selected to cover a wide range of valence (according to norms provided in the stimulus set; M = 5.07, SD = 1.92, Range = 1.46 – 8.19 – on a nine-point scale) and arousal (M = 4.66, SD = 1.19, Range = 2.63 – 7.21). Colour versions of the images were used to preserve their vividness and naturalness, and image statistics (e.g., brightness, contrast) were computed and controlled for statistically in all relevant analyses.

Task structure was the same as in Experiment 6. Each trial started with a fixation cross of variable duration (ranging from 7 to 15s), followed by a presentation of the stimulus image for 6 seconds, after which participants provided their ratings for subjective valence using a slider ('How did you feel watching the image?') from '-10 (Extremely negative)' to '+10 (Extremely positive)'; and subjective arousal ('How intense was your emotional reaction?') from '-10 (Very calm)' to '+10 (Very agitated)' – see **Figure 4.12.** Participants were asked to report how they "actually" felt after viewing each stimulus, rather than how they thought they "should" feel.

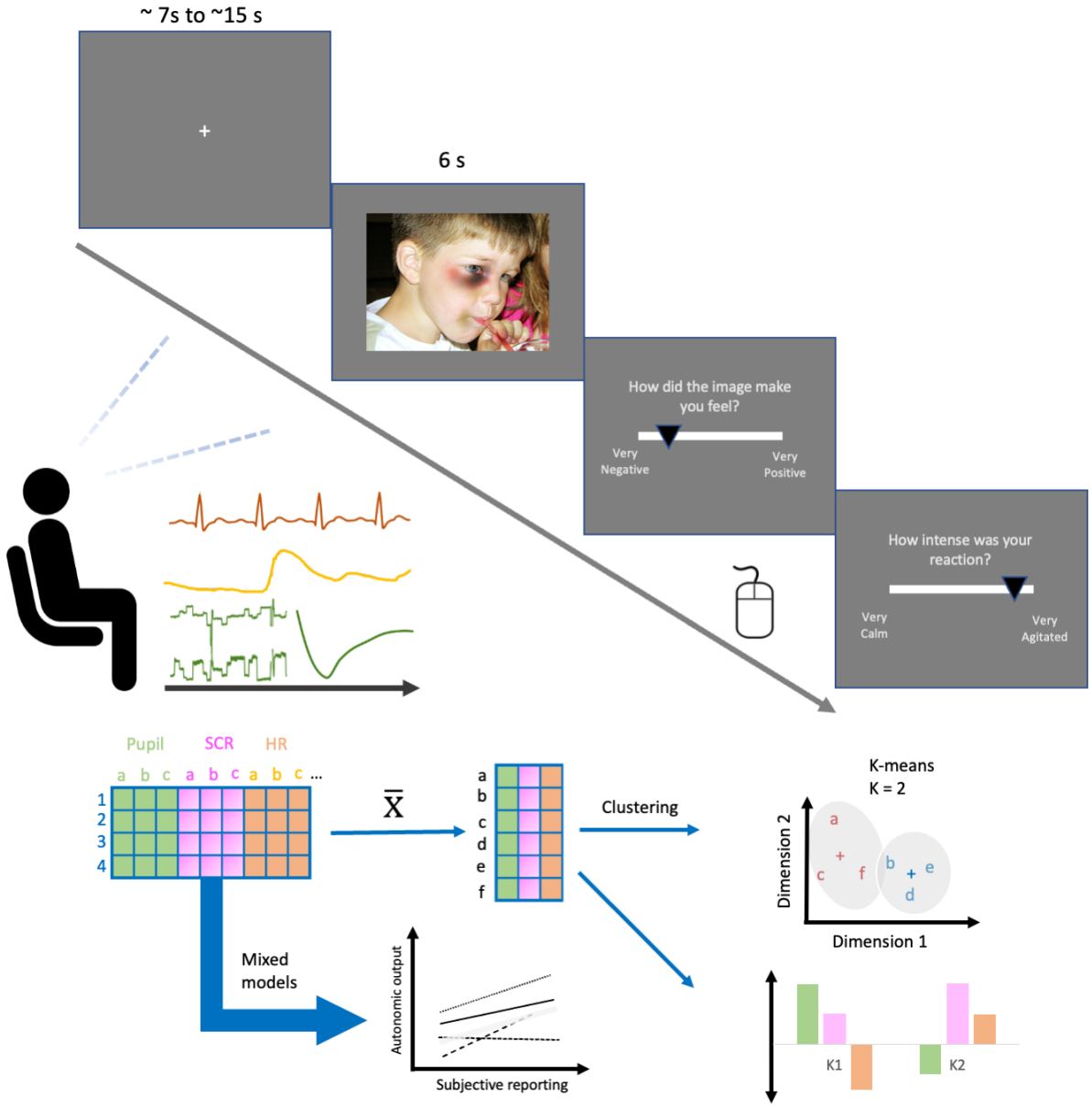

Figure 4.12 Schematics of Experiment 7 and analytical approach.

4.2.2.2 Physiological recording and processing

4.2.2.2.1 Eye movements and pupillometry

Eye movements and pupil changes were monitored using a GP3-HD (Gazepoint) eye-tracker sampling at 150 Hz, using the same procedures described in the previous experiment (Cuve, Stojanov, Roberts-Gaal, Catmur, & Bird, 2021). Blinks and invalid samples (pupil diameters outside the range of 2-10 mm) in the pupil data were interpolated linearly up to 150ms. A subtractive baseline correction was applied using an interval corresponding to one second before and one second after the stimulus onset (Geller, Winn, Mahr, & Mirman, 2020; Mathôt, Fabius, Van Heusden, & Van der Stigchel, 2018).

4.2.2.2.2 Skin conductance

Skin conductance responses (SCR) were recorded with the BIOPAC setup described in Experiment 6, and processed in Ledalab (Kaernbach, 2005) using the same processing steps described in Experiment 6. Responses were defined as phasic reactions detected 1 to 6 seconds after stimuli onset.

4.2.2.2.3 Heart rate

Heart rate (HR) was recorded via the BIOPAC system described in Experiment 6 and processed in using the same steps reported previously. A difference score was computed by subtracting mean heart rate after stimulus onset from average heart rate pre-stimulus (during the fixation cross), and this difference score was used for statistical analyses.

4.2.2.3 Statistical analyses

Linear mixed models were used to analyse trial-by-trial data with participant and stimulus random effects, using the R package lme4 (Bates et al., 2014). Approximate p-values were derived using the lmerTest package (Kuznetsova, Brockhoff, & Christensen, 2017). K-mean clustering was used to describe multivariate physiological reaction profiles across all

modalities (pupil, SCR and HR). The optimal number of clusters was identified by running the k-mean algorithm with $k = 2$ to $k = 10$, computing the within-cluster sum of squares (WCSS), a measure of the compactness of each cluster (lower values are better), and plotting the results using the elbow method (similar to a scree plot method). AIC and BIC were also used as additional criteria to validate *k*, and the final solution was evaluated using the amount of variance explained by the cluster solution as well as a silhouette score (ranging from -1 to 1 and describing the degree of cluster cohesion) with positive scores indicating better cluster separation and negative scores or a score of zero suggesting no detectable cluster. All cluster analyses were implemented in R and JASP and code is provided in https://github.com/hcuve/subj_obj_arousal_paper. Additional correlational analyses and analyses of variance (ANOVAs) were used to analyse aggregated data. Data associated with this study will be made available with publication at https://osf.io/vf9sg/.

4.2.3 Results

4.2.3.1 Concordance between physiological indices and subjective emotion responses

4.2.3.1.1 Pupil diameter and subjective emotion concordance

Subjective arousal on a trial-by-trial basis predicted changes in pupil size in comparison to baseline pupil size as expected (Estimate = .36, SE = .05, t = 7.48, p < .001), even after accounting for stimulus valence and brightness. There was no main effect of valence (Estimate = .03, SE = .04, t = 0.68, p = .50). As expected, there was a strong effect of brightness (Estimate = -.96, SE = 0.05, t = -17.76, p < .001) with brighter stimuli inducing greater pupil constriction and darker stimuli inducing greater pupil dilation. Additionally, there was a significant valence by arousal interaction (Estimate = .16, SE = .04, t = 4.14, p < .001), indicating an asymmetric relationship between subjective arousal and pupil change, with higher concordance between arousal and pupil size when participants reported feeling more positive after each stimulus (see **Figure 4.13-A & B**). These results could not be explained by eye-movements (fixation duration, blinks, and gaze loss) all of which were not predictive of subjective ratings (arousal and valence), nor moderated their relationship with pupil changes (see SM – **8.3.3**).

There is a possibility that the relationship between arousal and pupil size may evolve over time, as suggested by recent studies showing distinct temporal components for the pupil response to emotional states (Kinner et al., 2017). To investigate this, linear mixed models were fitted to predict pupil size from subjective ratings of arousal and valence, their interaction, and image statistics (brightness, contrast) at different time bins (n = 60, each bin being an average of 10 time points = 100 ms). Given the rapid (~ 1 second) pupillary light reflex (PLR) expected with visual stimuli, significant relationships between subjective

arousal, valence and pupil are only plausible after the PLR. Significant time clusters were defined as adjacent time bins where the corrected p value remained < .05 after the PLR.

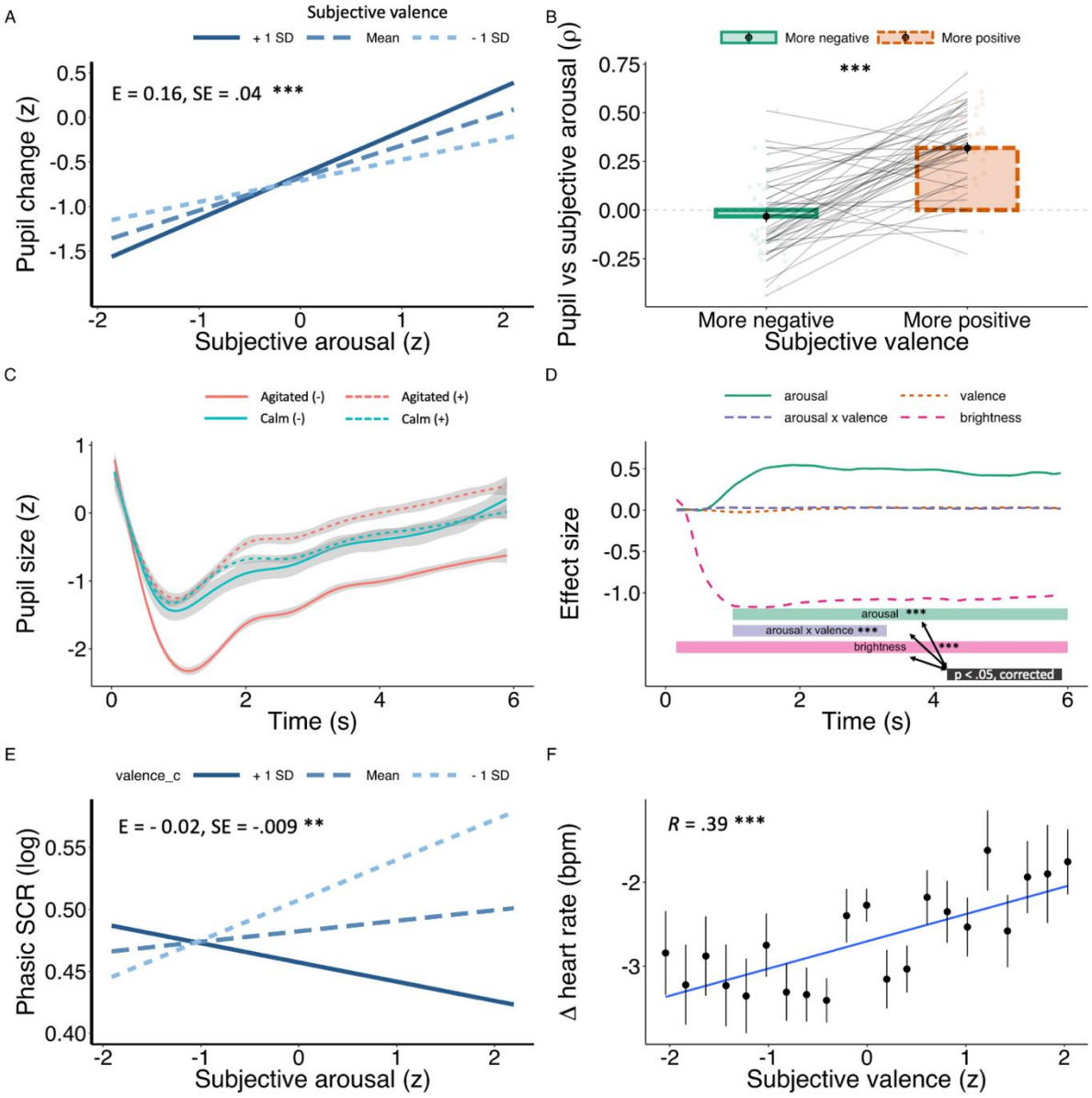

Figure 4.13 Summary of subjective and physiological experiences.

A. Subjective arousal and pupil showed tighter coupling on more positive self-reports (+ 1SD). B. Within-subject correlation between subjective arousal and pupil diameter for positive and negative valence ratings (median split was performed for visualisation purposes only). Each dot represents a unique participant. C. Time course of pupil diameter (median split) by arousal and valence. D. Time clustering analyses show that pupil changes are highly

influenced by brightness throughout stimulus viewing, while subjective arousal predicts pupil diameter changes after the early pupillary light reflex. Arousal and valence interact to predict pupil diameter changes at the start of the recovery period after this reflex. E. Illustration of the arousal and valence interaction from the mixed model showing the subjective arousal and skin conductance response (SCR) had a tighter coupling for negative but not positive viewing experiences. F. Heart rate tracked primarily valence, with negative stimuli inducing a larger negative change in heart rate in comparison to baseline. * $p < .05$. ** $p < .01$, *** $p < .001$.

The results of these analyses were consistent with the previous aggregated analyses (aggregated across time, not across participants or trials). Specifically, there was a large effect of brightness (Mean effect size$_{0.3\text{-}6\ s}$ = -1.02, $p < .001$, **Figure 4.13-D**). There was also a significant effect of subjective arousal, with more subjectively arousing stimuli associated with larger pupil dilation (Mean effect size$_{1\text{-}6s}$ = 0.45, $p < .05$, corrected). The interaction between subjective arousal and valence had a small but significant effect (Mean effect size $_{1\text{-}3s}$ = 0.03, $p < .05$, corrected), suggesting that early changes in pupil diameter following the light reflex are differently modulated by arousal and valence. Similar to the aggregated analysis, there was no significant effect of valence (Mean effect size = 0.02, $p > .05$, corrected) suggesting that pupil diameter is primarily associated with arousal. The size of the relationship between subjective emotion and pupil diameter is in line with previous observations showing that psychological modulation of pupil change is very modest, about ~ 1%, compared to the effect of brightness for example (Mathôt, 2018).

In summary, these results suggest that the relationship between subjective arousal and changes in pupil diameter is somewhat dependent on valence. Furthermore, while the visual

representation of the interaction shows tighter coupling between subjective arousal and pupil dilation changes for positive valence (**Figure 4.13 A**), a subset of participants showed the opposite effect, with subjective arousal and pupil diameter showing a tighter coupling for more negative subjective experiences. This suggests the presence of individual differences in the relationship between physiological reactivity and subjective arousal, which are typically overlooked in previous studies which rely only on aggregated (group-level) patterns of physiological activity and subjective experiences.

4.2.3.1.2 Skin conductance and subjective emotion concordance

Both subjective arousal (Estimate = 0.02, SE = 0.01, t = 2.60, p < .01) and valence (Estimate = -0.03, SE = 0.01, t = -2.51, p < .01) were significantly predictive of SCR individually, but not when included within the same model to predict event-related SCR. This suggest that the subjective arousal and valence effects on skin conductance are likely related to common variance in subjective valence and arousal ratings. Similar to the pupil diameter results, there was an interaction between subjective arousal and valence, suggesting that subjective arousal and SCR relation is also modulated by subjective valence (Estimate = - 0.02, SE = -.009, t = -2.77, p < .01). In contrast to the pupil diameter data, subjective arousal and SCR data were more concordant during negative valence states than positive states (see **Figure 4.13**).

4.2.3.1.3 Heart Rate and subjective emotion concordance

Unlike pupil diameter and SCR changes, heart rate was predicted by subjective arousal (Estimate = - 0.28, SE = 0.09, t = - 3.04, p < .01) and valence (Estimate = 0.29, SE = 0.08, t = 3.28, p < .01), such that heart rate decelerated when participants reported high arousal and negative emotion. However, when tested in the same model, only valence was significant (Estimate = 0.22, SE =0.001, t = 1.94, p < .05), such that more negative stimuli induced a

stronger heart rate deceleration whereas positive stimuli induced heart rate acceleration (see Figure 4.13-F).

4.2.3.1.4 Limited concordance of phasic physiological responses across modalities

It was also investigated how the different physiological modalities could be predicted from one another (convergent validity). Interestingly, while pupil diameter and skin conductance tracked subjective arousal in the previous analyses, they could only be predicted by heart rate significantly on a trial-by-trial basis. That is, there was a negative effect of HR on SCR (Estimate = -0.005, SE = 0.002, $p < .001$), whereas no effect of pupil diameter changes on SCR was significant (Estimate = -0.006, SE = 0.005, $p = 0.224$). Trial-by-trial fluctuation in pupil diameter were also predicted by heart rate difference (Estimate = 0.03, SE= 0.007, $p < .001$) but not by SCR (Estimate = -0.04, SE = 0.03, $p = .240$). It is worth noting, however, that even the significant estimates are very modest, which raises questions regarding the interchangeability of physiological modalities to measure trial-by-trial fluctuations in arousal. Even when aggregating the data across participants or trials there was no consistent relationship across physiological signals (see **Figure 4.14**). The only exception was a negative correlation between pupil change and heart rate difference (r = -.36, $p < .01$), that is, an increase in pupil diameter was related to HR deceleration when aggregating responses by trial and ignoring individual differences (that is, averaged across participants).

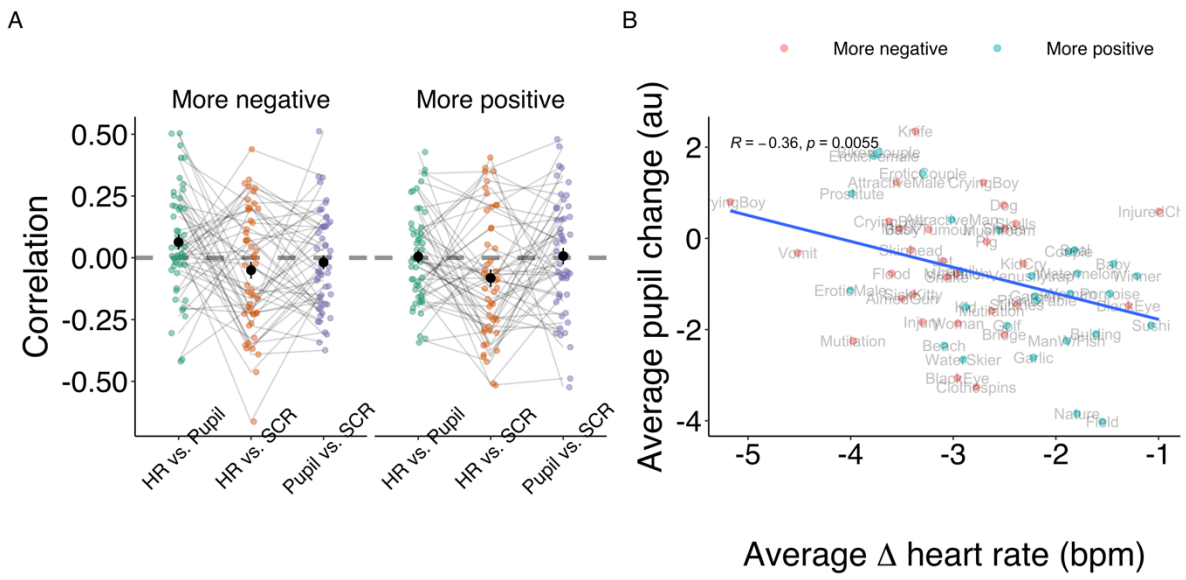

Figure 4.14 Illustration of average cross-correlations between autonomic modalities.

A. Average subject correlations between pupil, skin conductance response (SCR), and heart rate (HR). Each dot represents a participant and data is faceted by valence (median split). Data from the same participants are connected across conditions by lines. B. Illustration of the only significant aggregate correlation between physiological signals (note that data is aggregated by stimuli, across participants, removing individual differences).

4.2.3.1.5 Multivariate clusters of psychophysiological responses and subjective emotional experience

Another goal of this study was to attempt to determine whether multivariate clusters or 'profiles' of physiological signals are better predictors of emotional experience than signals from individual modalities (Berntson et al., 1991; Stemmler, 2004). While the previous section revealed limited concordance across physiological signals, it may be the case that emotional reactions are best characterised as a multivariate pattern of responses instead of set patterns across physiological modalities. Finding clusters that represent such multivariate

patterns, may help more readily translate findings between different physiological markers and their relation to subjective emotional experience.

The three indices used to select an optimal number of clusters (WCSS, AIC and BIC) converged on $k = 3$ clusters (see **Figure 4.15**). That is, combinations of changes in pupil diameter, SCR and HR could be grouped into three profiles. This solution explained 55% of the variance and had a silhouette score of .33, with positive values closer to 1 indicating perfect separation.

While not fully separable, these 3 clusters mapped loosely onto arousal and valence dimensions. Cluster 1 included trials with stimuli of mixed valence, including negative, positive and neutral stimuli, and was characterised by positive pupil diameter, negative HR and negative SCR changes. Cluster 2 was characterised by stimuli that could be described as arousing stimuli, including positive sexual stimuli and negative stimuli (e.g., mutilation). This cluster was characterised by positive negative diameter, positive HR, and negative SCR changes. Cluster 3 was characterised by predominantly pleasant and calming stimuli, generating increased pupil diameter, negative HR, and positive SCR changes. Clusters 1 and 3, therefore, suggest the strongest activation.

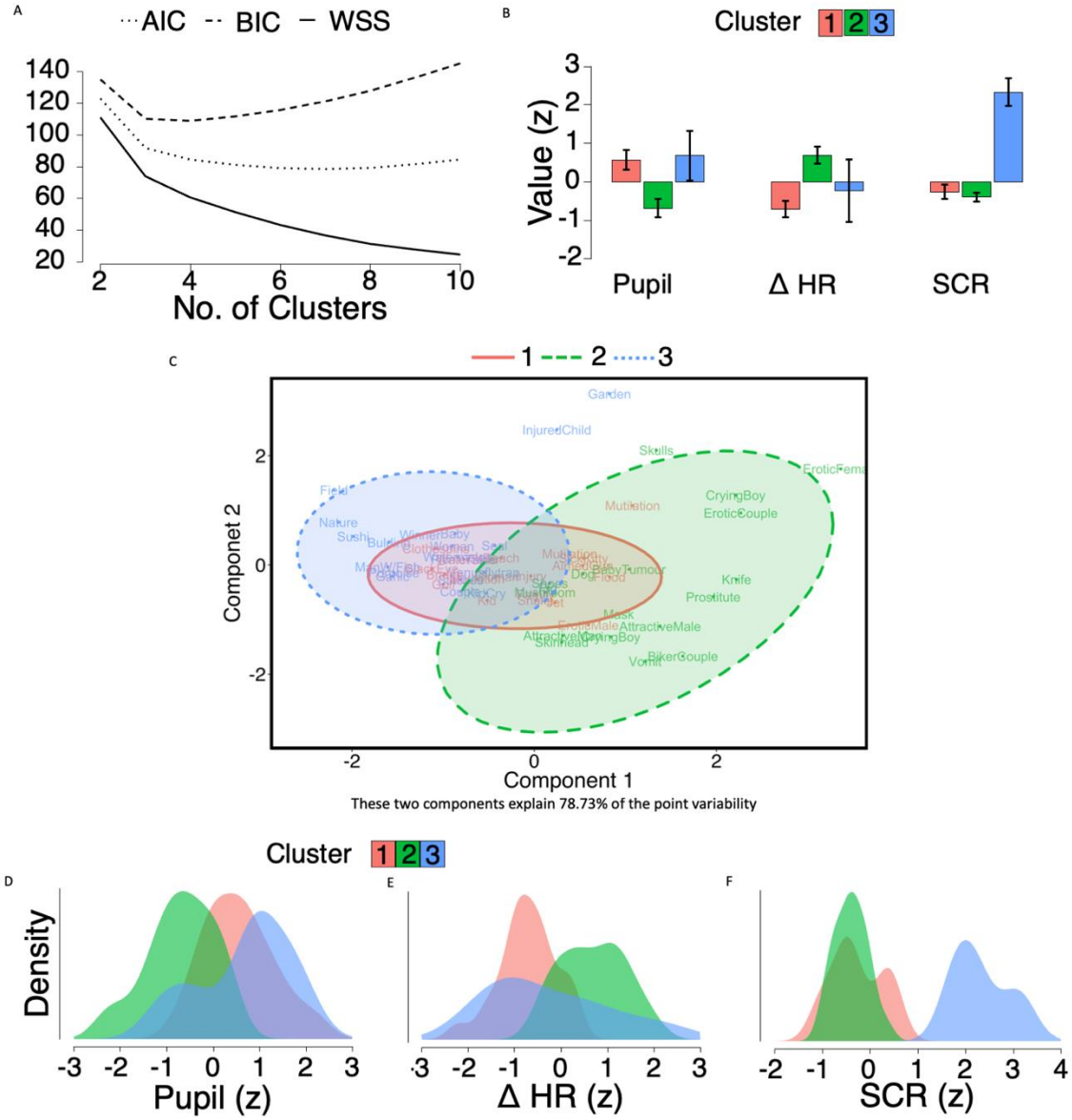

Figure 4.15 Cluster analyses results.

A. Elbow plot visualising the search for the optimal number of clusters across different methods. B. Cluster means for all psychophysiological predictors. C. Two-dimensional representation of the cluster solution. D, E, and F. Cluster densities for pupil diameter, heart rate change, and SCR respectively.

A two-dimensional PCA representation of the cluster solution is visualised in **Figure 4.15-C**, with the two dimensions explaining over 78% of point variability.

A 3-D visualisation of the multivariate cluster profile is also provided (**Figure 4.16**).

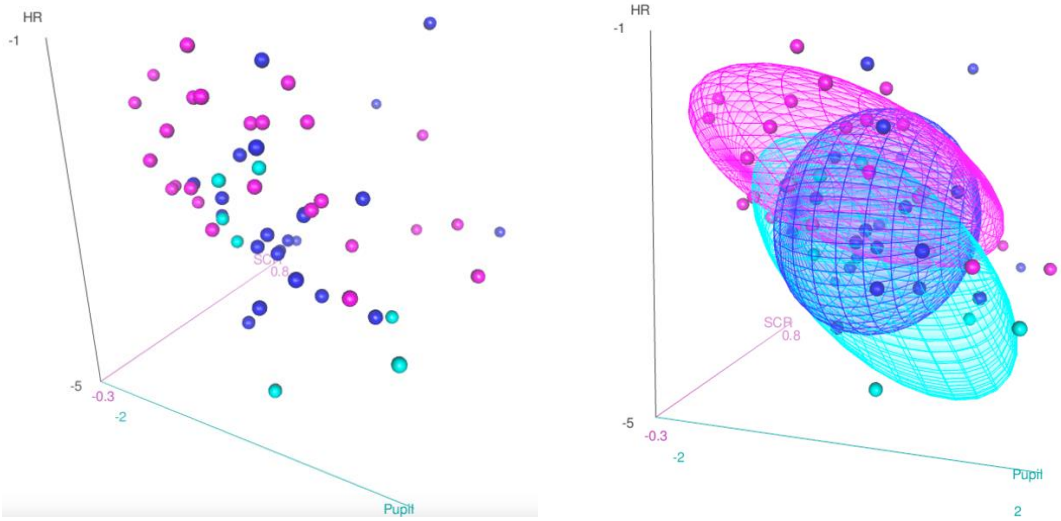

Figure 4.16 Scatter visualisation of the physiological profile for pupil diameter, skin conductance and heart rate.

The ellipsoids were grouped using the cluster obtained from the k-means analysis.

There were also significant differences in subjective ratings to each cluster pair (see **Figure 4.17**). Specifically, Cluster 2 had the highest positive ratings of subjective valence, and more ratings towards calm, whereas Cluster 3 had the lowest rating on valence (negative) and higher ratings for arousal (agitated) and Cluster 1 had mid scores along valence and arousal between Cluster 1 and 2.

In summary, data-driven clustering of physiological signals to emotionally inducing stimuli revealed 3 clusters that broadly mapped onto subjective arousal and valence dimensions, with negative and predominantly positive sexual (agitating) cluster, positive (calm) cluster, and a third cluster containing mixed/ambiguous stimuli. These clusters were characterised by a multivariate physiological profile such that physiological responses did not show a clear-cut pattern separating high and low arousal or positive and negative valence.

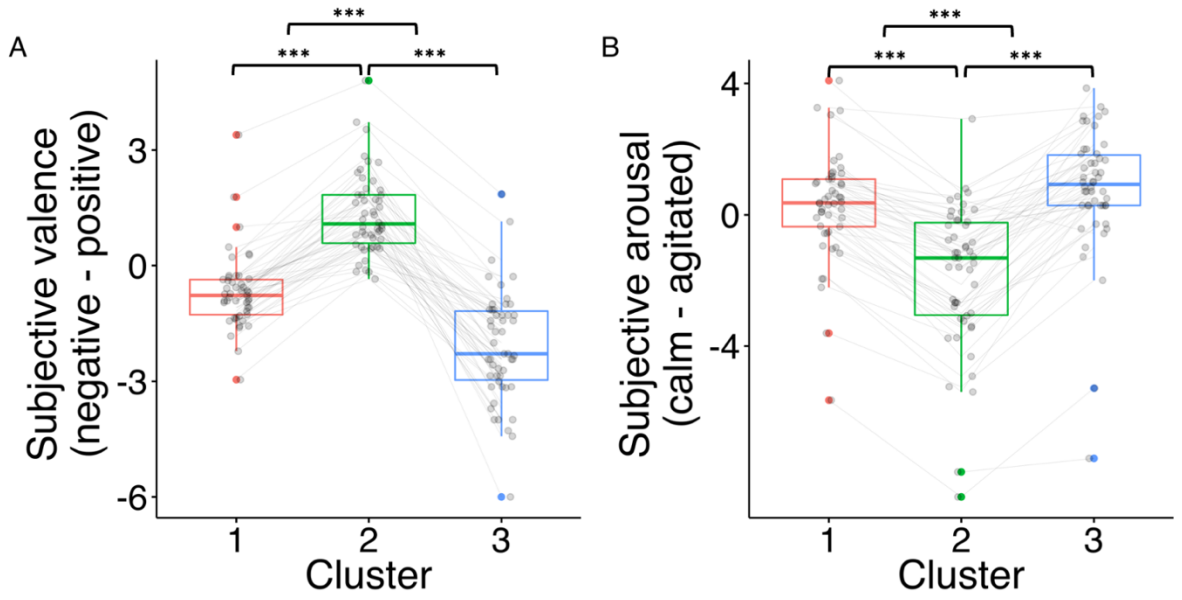

Figure 4.17 Subjective ratings by cluster.

Illustration of subjective valence and arousal ratings by the data-driven clusters of physiological responses. Obtained clusters map loosely into arousal and valence dimensions, from agitating (negative and positive sexual), mixed/ambiguous, and positive (calm). *** p < .001.

4.2.4 Discussion

The current study evaluated the relation between subjective emotional arousal, valence, and physiological reactions for three different physiological modalities: pupil diameter changes, skin conductance responses (SCR), and heart rate (HR). Analyses suggest that while all these modalities track subjective arousal to some degree, the relationship between subjective arousal and physiological responses is asymmetrically modulated by valence. Specifically, a small bias was observed for pupil diameter, such that the pupil responses tracked subjective arousal more closely for positively-valenced experiences and stimuli than for negatively-valenced stimuli. Conversely, there was a bias in the SCR and subjective arousal relationship with SCR scaling more consistently for negative subjective states. Finally, HR was primarily

modulated by valence with more negatively perceived stimuli inducing a stronger heart deceleration.

These group-level effects conceal individual differences in the relationship between emotional reactivity and subjective experience, as in all cases a smaller subset of participants exhibited the opposite effect to that observed at the group level. This finding highlights the importance of considering individual differences and is in line with suggestions that the relationship between physiological responses and emotional experience might be idiosyncratic – at least to a degree (Barrett, 2006; Picard, Vyzas, & Healey, 2001; Siegel et al., 2018).

The moderation of the association between physiological signals and subjective arousal by valence is incompatible with frequent assertions that such signals track arousal regardless of valence (Bradley et al., 2008; Collet & Vernet, 1997), but such moderation has been documented in several studies using other modalities, including blood pressure and other cardiovascular parameters (Cacioppo et al., 2000; Kreibig, 2010; Siegel et al., 2018), and were the subject of early theorising in the physiology of emotion literature (Taylor, 1991).

Additionally, the current results indicate limited coherence between the various physiological signals, with the pattern of concordance being non-significant or very modest across signals. In fact, only heart rate was consistently related to pupil diameter and SCR, with the other pairwise associations not significant. It is worth noting that these results are inconsistent with the few previous studies looking specifically at these three modalities simultaneously (Bradley et al., 2008) which showed some concordance across pupil diameter, SCR and HR changes. Nonetheless, the current results are in keeping with the conclusions of several reviews of studies over the past five decades on several autonomic modalities (Barrett, 2006; Cacioppo et al., 2000; Collet, Vernet-Maury, Delhomme, & Dittmar, 1997; Kreibig, 2010;

Norman et al., 2016; Schachter & Singer, 1962; Siegel et al., 2018). In previous studies, concordance was assumed from being able to replicate relationships with subjective arousal (Leuchs et al., 2019; Snowden et al., 2016), but mostly in aggregated data ignoring individual differences in both physiological reactivity and subjective experience. However, when analysed at the trial-by-trial level, coherence across physiological signals and in their relationship to subjective arousal was often small to non-existent (Barrett, 2006; Cacioppo et al., 2000; Leuchs et al., 2019) or with some concordance observed in tonic but not phasic responses (Leuchs et al., 2019; Wang et al., 2018).

Combined with earlier reports, the current data raise doubts about the interchangeable use of pupil diameter, SCR, and HR as indices of arousal, and the degree to which they can capture individual differences in emotional experiences, particularly across studies. The single use of different physiological signals to index subjective emotional experiences is likely to contribute to limited replicability of physiological partnering of emotion and mixed findings in both basic and translational (e.g., clinical) studies of the physiology of emotion. Researchers need to carefully consider whether the use of these measures is intended to capture important trial-by-trial fluctuations and individual differences, or simply aggregate patterns across participants and trials.

It is worth noting that there are a number of purely methodological issues which may contribute to heterogeneous results when attempting to relate physiological responses to emotional experience. These include psychometric factors related to how subjective emotional arousal is operationalised and measured (e.g., on an activation-deactivation spectrum, as emotional categories in an all or nothing manner, or as varying degrees of intensity), response biases and social desirability (where people respond as they think they are expected to do, rather than as they actually feel) (Barrett, 2006; Kreibig, 2010).

On another hand, peculiarities related to each physiological modality may pose an upper limit to how much one modality can be used to infer another, for example the fact that single innervated modalities like SCR can only increase relative to baseline, whereas dual innervated modalities like pupil can constrict and dilate (Berston et al., 1991). Additionally, it is likely that individuals do not always display a sizable physiological response to an emotional experience (at least not one detected by some measures like pupil diameter, SCR, and HR). Similarly, it is possible that certain emotional experiences are only accompanied by physiological changes in some modalities, but not others (Berntson et al., 1991; Kreibig, 2010). Some participants may display more phasic reactivity than others and even then, only in some modalities (as illustrated by non-responder participants, and no-response trials in SCR).

It has also been proposed that physiological responses are likely to include both non-specific emotional responses, undifferentiated emotional responses as well as specific emotional responses (Cacioppo et al., 2000; Stemmler, 2004), combined with the degree of downstream regulation of the ANS by the CNS, which does not affect all autonomic modalities equally, various response profiles are plausible (Berntson et al., 1991; Folkow, 2000; Jänig & McLachlan, 1992). This is especially pertinent when one considers the impact of emotion regulation in studies comparing physiological signals and emotional experience. The task used in the current study exposes participants to various types of stimuli with some being particularly uncomfortable to view. While it is believed that SCR and HR are not under direct voluntary control, several findings show cognitive effects on pupil diameter which alter (and dampen) expected arousal effects (Kreibig, 2010). This opens the possibility for regulatory and even mind-wandering processes to affect how well physiological responses capture subjective arousal as well as how they relate to one another. A lack of concordance across

physiological signals, therefore, does not necessarily imply no underlying relationship between autonomic states and subjective emotion.

The data-driven clustering approach revealed subgroups of physiological responses that could loosely be located at distinct points on valence and arousal dimensions. This and other multivariate approaches may therefore be particularly useful for unravelling the physiological profile of emotional experiences, particularly in light of the methodological difficulties discussed above. Relying on only one modality to quantify subjective emotional experiences is likely to lead to incomplete and inconsistent results when generalising to other modalities and other subjective experiences. By making use of all data, multivariate approaches allow one to build multivariate physiological profiles and align with conceptualisations of emotion and physiological responses as multidimensional state-space relationships (Berntson et al., 1991; Stemmler, 2004).

However, it is important that more efforts to characterise this autonomic state-space are made by integrating further modalities, and more importantly testing the robustness, quality and replicability of cluster solutions. More research and discussion of these questions will also inform about best practices and suitability of the various data-driven approaches to characterise this physiological state-space of emotion.

Finally, while clustering approaches seem worthwhile, it is important to acknowledge a number of limitations that are not easily addressable by standard algorithms. For instance, most clustering algorithms rely on aggregated data to find clusters, but these are not tailored for non-independence in data which typically is present in repeated-measures experiments (including multiple data points within participants). Ideal approaches would search for clusters within and across individuals making sure that attempts to map the physiological state-space do not fall into the same trap of overlooking individual differences that has

plagued the standard aggregated psychophysiology research on emotion. Hierarchical clustering methods might be particularly useful in this regard.

Materials availability

Data and code for this paper are available at:

https://github.com/hcuve/subj_obj_arousal_paper and https://osf.io/vf9sg/ respectively.

4.3 EXPERIMENT 8: Alexithymia, not autism, is related to atypical multivariate patterns of physiological response to emotion

Abstract

Atypical physiological responding is a core feature of theories of autism and alexithymia and is thought to drive atypical socioemotional functioning. Research on the exact profile of physiological responses associated with autism and alexithymia has, however, proved mixed, with findings highlighting complex patterns that are inconsistent with prevailing hypo and hyperarousal models. Given the high comorbidity between alexithymia and autism, the overlap of their proposed mechanisms is relevant for ongoing debates regarding whether atypical emotional responding in autism, reflect common, or distinct contributions of autism and alexithymia. The current study addressed these issues by investigating the contributions of both autism and alexithymia to physiological responses to emotions. The current approach capitalises on the multimodal recordings of physiological responding and characterisation of the multivariate physiological profiles, enabling a test of the discriminant value of physiological responses to *a priori*-defined groups (autism vs controls, high vs low alexithymia, high vs low autistic symptoms). Additionally, potential subgroups were explored in a data-driven fashion using clustering analyses. Consistent with the alexithymia hypothesis, results show that alexithymia and not autism, is related to atypical multivariate patterns of physiological responses to emotions, and reduced concordance between subjective emotion reports and physiological outputs. I argue that rather than supporting strict hypo or hyperarousal accounts of physiological responses to emotion, these results suggest an atypical mobilisation of physiological responses across various levels of autonomic signalling. This physiological mobilisation imbalance hypothesis is consistent with neuroimaging and psychophysiological findings indicating patterns of hypo and hyperactivation in pathways and systems implicated in emotion processing in autism and alexithymia. As such, the role of

physiological responding in autism and alexithymia needs to be viewed in terms of the functional consequences of various multivariate profiles of autonomic mobilisation during emotional responding.

Keywords: arousal, autonomic, autism, alexithymia, emotion, multivariate.

4.3.1 Introduction

Many influential theories of emotion argue that physiological responses are an integral part of, or necessary for, emotional states. As reviewed in Chapter 1, William James argued that all emotional states ("emotion proper") arise from our physiological responses to events in the environment (James, 1948). Evolutionary accounts propose that physiological responses to emotion-inducing events are a mechanism to mobilise metabolic processes optimising behavioural responses essential for survival or maintenance of the organism (e.g., approach or avoidance behaviours) (Kreibig, 2010; Stemmler, 2004a). Even constructivist views of emotion which argue that specific emotions are not associated with specific physiological responses, highlight the role of transient alterations of an organism's physiology as an important component of "core affect" (Barrett, 2006; Russell, 2003). Such theories argue that these transient alterations in physiological state are accessible to conscious experience via interoceptive processes (enabling perception of the physiological state of the body), and thus enable subjective judgments of the quality of those states in terms of valence (positive or negative) and degree of arousal.

The importance of physiological responses to emotional experience means that atypical physiological responses, or atypical awareness of those responses (i.e., atypical interoception), is thought to characterise several clinical conditions (Reitan, 1976, Doom & Gunnar, 2013, Murphy, Brewer, Catmur & Bird, 2017). Atypical responses are thought to

impact all aspects of emotion processing, including socioemotional processes such as the recognition of emotion in others and empathy (Bird & Viding, 2014; Decety & Jackson, 2004). Of particular relevance to the current study is that atypical physiological responding is a core feature of several theories of autism as discussed in Chapter 1 (Senju & Johnson, 2009; Tanaka & Sung, 2016) and of alexithymia (Panayiotou et al., 2018). In the next section, I will briefly review and recap the physiological mechanisms and arousal-based models of autism and alexithymia relevant for the current study.

4.3.1.1 Autism and physiological response during emotion

It has been suggested that atypical physiological responses to emotion-inducing stimuli may underlie many of the socioemotional and behavioural consequences of autism. Specifically, various arousal-based theories of autism (Cuve et al., 2018; Rogers & Ozonoff, 2005), posit that aberrant resting and/or phasic physiological activity in autism leads to several autistic symptoms. These theories can be separated into two main types: Hyperarousal and Hypoarousal theories which were discussed in Chapter 1, but are briefly reviewed here.

Hyperarousal theories argue that emotion regulation difficulties in autism result in heightened sensitivity to emotion stimuli (Cuve et al., 2018; Hadjikhani et al., 2018; Tanaka & Sung, 2016). It is argued that strategies designed to reduce the resultant physiological dysregulation cause problems processing emotions (e.g., recognising emotional facial expressions), atypical gaze patterns (e.g., a lack of eye contact), and stereotyped behaviours (Lassalle et al., 2017; Tanaka & Sung, 2016).

There has been some support for hyperarousal theories, including from studies linking atypical gaze to social stimuli with heightened autonomic activity as measured by skin conductance and pupil diameter in autistic individuals or individuals high in autistic traits (Cuve et al., 2018; Lydon et al., 2016; Top et al., 2018). Many studies have also documented

an increased tonic heart rate in autism compared to non-autistic individuals (Hollocks, Howlin, Papadopoulos, Khondoker, & Simonoff, 2014). Neuroimaging research has also produced some evidence for hyperarousal of key structures implicated in the expression and processing of emotions, particularly in subcortical systems (Hadjikhani et al., 2017; Kliemann et al., 2012; Monk, 2010).

Hypoarousal theories suggest disengagement in response to socioemotional stimuli in autism, and therefore, reduced physiological responses to emotion-inducing stimuli (Cuve et al., 2018; Rogers & Ozonoff, 2005). These theories are supported by evidence showing that social stimuli cause stronger physiological reactions in non-autistic individuals than in autistic individuals (Mestanik et al., 2017). Other studies have reported reduced autonomic reactivity to socioemotional stimuli in autism, particularly measured by SCR (Cuve et al., 2018; Mathersul, McDonald, & Rushby, 2013). Consistent with these reports, hypoactivation of the amygdala, the superior temporal sulcus and fusiform gyrus in response to socioemotional stimuli has been observed in autism (Hadjikhani et al., 2006; Harms et al., 2010; Kim et al., 2015).

In summary, while there is an abundance of evidence for atypical physiological responding in autism, the exact physiological profile of emotional responses has been difficult to establish. One way to resolve the mixed findings is to recognise the possibility is that hypoarousal and hyperarousal need not be mutually exclusive. Hypoarousal and hyperarousal may arise from different neural substructures and pathways implicated in emotion responding. For example, structures including the amygdala, insula and ACC (found to have atypical response profiles in autism), are not anatomically or functionally homogenous; thus hypoarousal and hyperarousal may be a product of different substructures or functional networks involving these areas (Adolphs & Baron-cohen, 2003; Cuve et al., 2018; Dalton et al., 2005; Dziobek,

Bahnemann, Convit, & Heekeren, 2010; Etkin et al., 2011; Hadjikhani et al., 2017; Saygin, Osher, Augustinack, Fischl, & Gabrieli, 2011).

A further possibility is the existence of multiple subgroups within the autistic population, characterised by different patterns of physiological responding (Cuve et al., 2018; Lydon et al., 2016; Rogers & Ozonoff, 2005). These subgroups may be a product of individual differences in a range of relevant variables and/or comorbid traits (such as anxiety), and it is alexithymia which is focussed on here (G Bird & Cook, 2013; Cuve et al., 2018).

4.3.1.2 Alexithymia and physiological response to emotion

As discussed in Chapter 1, an early focus of alexithymia research centred around somatic processes, including reports of atypical physiological responses to emotion and a reduced concordance between subjective experience and physiological responses (Nemiah, 1976; Porcelli & Taylor, 2018; Taylor, 2018). Under the "alexithymia hypothesis" of autism, alexithymia drives qualitatively distinct profiles of socioemotional functioning in autism. Of central interest to the current work is whether alexithymia is responsible for (at least some of) the atypical physiological responding seen in autism.

It has been claimed that there are two distinct subtypes of alexithymic individuals, each with a distinct profile of physiological responding. The first, sometimes referred to as Type I alexithymia (Bailey & Henry, 2007), is characterised by a failure to generate the typical physiological correlates of emotional experience. The second subtype is primarily characterised by deficits in interoception, such that physiological responding is typical, but the individual is either unaware of, or otherwise has an impaired perception of, that physiological arousal. As a consequence, subjective reports of emotion and physiological patterns of emotional response become 'uncoupled' (Brewer, Cook, & Bird, 2016, Papciak et al., 1985).

A number of studies have reported atypical physiological responses and/or interoceptive processing in alexithymia. In an interesting parallel with the literature on physiological responses in autism, both **hyporarousal and hyperarousal** have been reported. Consistent with a hypoarousal pattern of responses, alexithymia has been linked to reduced SCR responses during stressor tasks, while watching negative videos (Franz et al., 1999; Franz, Schaefer, & Schneider, 2003), and to arousing affective imagery (Constantinou, Panayiotou, & Theodorou, 2014; Panayiotou et al., 2018; Pollatos, Schubö, Herbert, Matthias, & Schandry, 2008).

Gaigg et al. (2018) provided one of the few studies to explore the contribution of alexithymia to physiological responses to emotions in autism. Using an emotion induction viewing task they reported that high levels of alexithymia in both autistic and neurotypical individuals correlated with lower levels of skin conductance response. Importantly, decoupling effects were also found, whereby alexithymic participants had decreased awareness of their own autonomic arousal, as demonstrated by a lower correlation between their physiological responses (SCRs) and their subjective arousal (Gaigg et al, 2018).

Hyperarousal has also been reported in alexithymia, with evidence from SCR (Nandrino et al., 2012), blood pressure (Waldstein, Kauhanen, Neumann, & Katzel, 2002) and heart rate to emotional situations (Luminet, Rimé, Bagby, & Taylor, 2004). Hyperarousal may have a specific temporal profile in alexithymia; Bogdanov et al., (2013) found larger SCRs to the onset of affective stimuli in alexithymia which dissipated over time.

Neuroimaging studies provide evidence consistent with both hypoarousal and hyperarousal. Consistent with findings of reduced autonomic responses, studies have reported disrupted transmission of interoceptive emotional information to the anterior cingulate cortex (ACC) (Goerlich & Aleman, 2018), and substructures within the ACC such as the ventral portions of

the ACC (proposed to be involved in the generation of emotional responses) and dorsal areas (implicated in the appraisal and expression of emotion) have been linked to hypoactivation in alexithymia (Etkin et al., 2011; Goerlich & Aleman, 2018; Miyake et al., 2012). Meta-analyses have demonstrated reduced activation of the dorsomedial PFC and the amygdala during both conscious and unconscious processing of emotions in alexithymia (Goerlich, 2018; Goerlich & Aleman, 2018), while both hyper- and hypo-reactivity of the insular cortex has been reported in alexithymic individuals when processing emotional stimuli (Bird et al., 2010; Silani et al., 2008; Velde et al., 2013).

In summary, studies of autonomic responses and neural patterns of activity support both hypoarousal and hyperarousal in alexithymia in response to emotion. This picture is surprisingly similar to that seen in autism, yet despite the high proportion of alexithymic individuals in the autistic population, very few studies of emotional physiological arousal in autism also measure and account for the effect of alexithymia. Conversely, most studies of arousal in alexithymia do not account for autistic traits. Determining the extent of any common and/or separate contributions of autism and alexithymia is necessary to inform ongoing debates regarding the nature of emotional difficulties in autism and alexithymia, as well as to inform clinical practice and assessment (Bird & Cook, 2013; Cuve, et al., 2021). This study, therefore, attempts to identify the impact of autism and alexithymia on physiological responses to emotional stimuli.

4.3.1.3 The need for multimodal recording and multivariate approaches

Early work on the autonomic nervous system and its involvement on emotion considered it an ‘all-or-nothing’ system, as such most studies relied on measurement of a single physiological signal such as SCR, blood pressure, cortisol, or pupil dilation with the expectation that one signal provided information about the state of the whole system in response to emotion

(Berntson et al., 1991; Stemmler, 2004, Normal et al., 2016, Kreibig, 2010). In Experiment 7 in the current chapter, it was highlighted that the 'all or nothing' characterisation is unfounded, as it is unclear how patterns of physiological activation translate across various autonomic signals. Instead, results suggest that recordings of multiple autonomic modalities signals should be used to obtain an multivariate response 'profile' across physiological responses consistent with views of physiological mobilisation in emotion (Berntson et al., 1991).

Such multivariate profiles can be used in a hypothesis-driven way – to determine whether apriori defined groups (such as autistics vs neurotypical individuals) vary in their response profiles – or in an exploratory data-driven manner to identify patterns of covariation across modalities, experimental conditions, or participant groups. Furthermore, data-driven clustering methods can also be used to discover subgroups based on multivariate profiles of physiological responses (Cacioppo, Tassinary, & Berntson, 2016). While rarely applied in physiological research of emotion, particularly in clinical applications such as the study of autism and alexithymia, multivariate approaches are common-used in neuroscience and genetics to differentiate groups, stimulus categories, experimental manipulations, genetic markers or functionally distinct neural regions (Cacioppo et al., 2016; Krishnan, Williams, McIntosh, & Abdi, 2011).

Although several multivariate approaches may be equally appropriate for different experimental designs, two particular approaches may be useful to highlight here and are relevant for the current study. The first is called Barycentric Discriminant Analyses (BADIA) (see also Section **4.3.2.4.3**), which is a PCA based discriminant analyses method that allows testing whether apriori groups (e.g. clinical vs non-clinical) can be reliably separated based on the identification of discriminant components that represents various features of variables

of interest (e.g. brain data, genetic data, psychological tests) (Hervé Abdi & Williams, 2010; Krishnan et al., 2011). What makes this method particularly useful in comparison to other discriminant analyses is that it can be used in cases where the features or sets of variables exceeds the number of observations (e.g., participants) (Abdi & Williams, 2010), which is a typical problem in physiological datasets.

The second method worth noting concerns data-driven clustering based on physiological responding. Although the existence of sub-groups (defined according to their physiological patterns of responding) is theorised within the autistic and alexithymic populations, the factors that define these subgroups at the non-physiological level are not understood. Data-driven clustering, for example, K-means algorithms can be used to explore the presence of subgroups without any apriori idea of variables that define those sub-groups or how many sub-groups there are (also see Experiment 7).

4.3.1.4 The current study

The current study examined the impact of autism and alexithymia on the physiological responses to emotion across multiple autonomic signals of arousal (pupil diameter, skin conductance, and heart rate). Based on the alexithymia hypothesis, it was predicted that any atypical physiological response to emotions would be driven by alexithymia and not autism.

A novel approach was used to characterise the multivariate physiological profiles of responses to emotions and their relationships to alexithymia and autism. It was hypothesised that alexithymia, not autism, would more robustly allow for subgroup differentiation based on profiles of physiological response.

4.3.2 Methods

All research procedures were approved by the Medical Sciences Ethics Committee (IDREC) – reference: R60697/RE004/5 and were in accordance with the revised 2013 Declaration of Helsinki. A total of 74 participants took part in the study, 58 were neurotypical (34 female; Group Mean age = 25, SD = 6.9), and 16 were autistic (female = 4, Group Mean age = 38.12 SD = 14.45).[6] To maximize data availability, exclusions for data quality and processing issues were made separately for each analysis and will be highlighted where appropriate. But on average 66 participants were available across all analyses[7].

4.3.2.1 Task

The same task described in Experiment 7 was used here which consisted of affective images selected from the International Affective Picture System (IAPS, Lang et al., 2008), designed to elicit emotional responses in observers - see **Figure 4.18.**

4.3.2.2 Physiological measures

Three main indicators of physiological activity were recorded, pupil diameter, skin conductance, and heart rate[8], recorded using the setup already described in Experiment 6 and 7 and all the same pre-processing steps described in Experiment 7 (**Physiological recording and processing**).

[6] 60% of this sample was also used in Cuve et al., 2021, but none of the analyses and results overlap.

[7] Data collection was conducted at two separate time points, in 2019 and early 2020 prior to the Covid pandemic, and in 2021.

[8] Most participants tested in 2021 were wearing masks and were instructed to attach the sensors on themselves, in 2019 the researcher was the one attaching the sensors and participants were unmasked. Informal piloting indicated no significant effects of masking on phasic markers of arousal and eye tracking data quality, and a full detailed analysis of these factors will be discussed elsewhere.

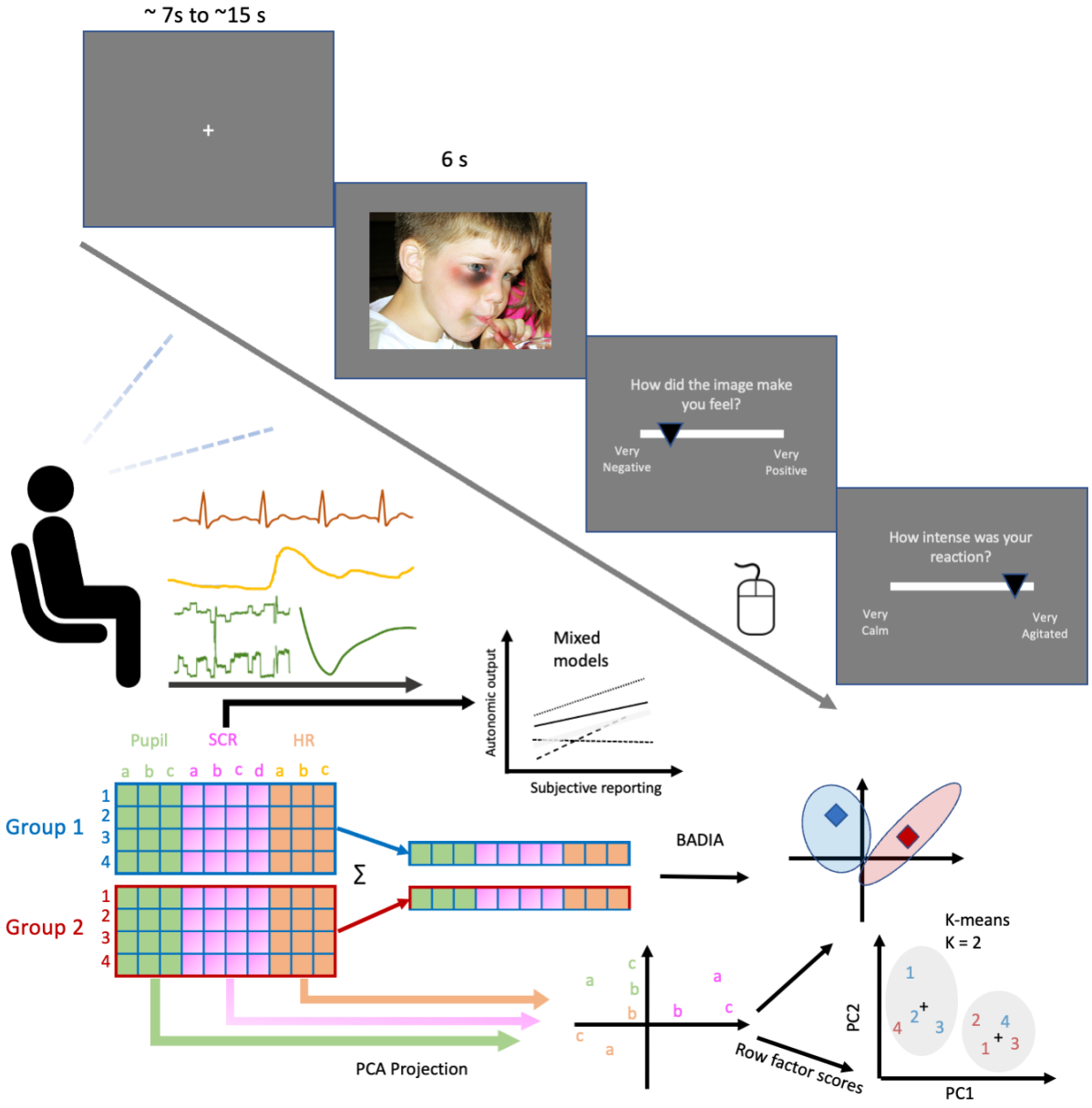

Figure 4.18 Schematics of Experiment 8 and the multivariate approaches.

Top panel represents the task. Bottom panel represents the analytical approach. BADIA relies on identifying PCA components that discriminate groups. An inferential PCA was also used to characterise physiological profiles and for data-driven clustering. Mixed models were used for univariate relationship analyses (e.g., separate analyses of each physiological signal and subjective rating, or between pairs of physiological signals).

A baseline corrected measure of pupil diameter with brightness effects regressed out (via residualisation) was used. For SCR, the *CDA PHASIC MAX* response was used as the phasic SCR response, which is defined as the maximum value of phasic activity detected 1 to 6 seconds after stimulus onset (Kaernbach, 2005). The SCR variable was log-transformed to correct for skewness (floor effect). Non-responses were indexed as zero and analyses with and without zero responding trials were conducted for sensitivity comparisons. A difference score subtracting average heart rate after stimulus onset from average heart rate pre-stimulus (fixation cross) was used for statistical analyses.

4.3.2.3 Psychometric assessments

The Toronto Alexithymia Scale – TAS-20, Autism Spectrum Quotient – AQ-28, Depression Anxiety and Stress Scale - DASS21 and Weschler Abbreviated Scale Intelligence Test – WASI, were used to assess alexithymia, autistic traits, anxiety and depression symptoms and IQ respectively. All these measures were described in detail in previous chapters.

4.3.2.4 Statistical analyses

4.3.2.4.1 Univariate analysis.

All analyses were conducted in R (R Core Team, 2013). Descriptive and correlational analyses are presented in SM **(8.3.4)** to characterise the sample and show aggregate patterns of physiological and subjective responding collapsed over the whole range of valence and participants' responses. Mixed models were implemented using lme4 (Bates, Mächler, Bolker, & Walker, 2014) to analyse the relation between physiological responses across modalities, alexithymia, autistic traits, and subjective ratings across valence and arousal dimensions. The different physiological modalities (pupil diameter, SCR, and HR) were analysed separately. The lmerTest package was used for inference tests on the mixed models (Kuznetsova et al., 2017). All mixed models were fitted with random intercepts for

participant and stimuli id, based on the assessment of random effect contributions (Bates, Kliegl, Vasishth, & Baayen, 2015).

4.3.2.4.2 Multivariate and cluster analyses.

To identify the multivariate profile of physiological responses, dimensionality reduction was used. Specifically, a Principal Component Analysis (PCA) was used on physiological responses for all participants – see **Figure 4.18**. To identify reliable components, an inferential PCA battery of tests implemented in the TinPosition package was used (Beaton, Rieck, & Abdi, 2013) based on permutation and bootstrap tests. Permutation tests on the features (variables) are used to determine which, if any, observed components are significant. Bootstrap ratio tests are conducted to identify the variables that significantly contribute to the variance of a component. Bootstrap ratios whose magnitude is larger than a critical value analogous to a z or t value (default = 2) are considered significant. The solution was used to describe the pattern of physiological responses and also as the basis for the discriminant analyses focused on comparing apriori defined subgroups (autism vs neurotypical; high vs low alexithymia) using BADIA, and for data-driven clustering of participants (without apriori specified subgroups) and dimensional analyses with alexithymic and autistic traits.

4.3.2.4.3 Barycentric Discriminant Analyses - BADIA

BADIA (Abdi & Williams, 2010) was used to explore whether apriori-defined groups, such as autistic vs controls (and high vs low alexithymia, and high vs low autistic traits) could be differentiated based on the multidimensional patterns of physiological responses. The BADIA analysis was performed using the packages TEXPOSITION and TINPOSITION (Beaton, Rieck, & Abdi, 2013; Beaton, Rieck, & Chin, 2013) in R. More details of this method are explained in the respective authors' papers, but the essential steps are:

1. *A priori* categories are defined as rows in the participants (rows) by variable (columns) matrix. Each category of interest is represented by the barycentre of its observations (i.e., the weighted average).
2. PCA is performed on the category by variable matrix.
3. A set of discriminant factor scores is given for the categories and another set of factor scores for the variables.
4. The original observations are projected onto the category factor space, providing a set of factor scores for the observations. The distance of each observation to the set of categories is computed from the factor scores, and each observation is assigned to the closest category.

A schematic illustration of the analysis is provided in **Figure 4.18**.

4.3.2.4.4 Data-driven clustering

K-means clustering was used to explore the existence of sub-groups based on multivariate profiles of physiological responses. The components extracted from the PCA were used for the k-means analyses rather than the raw variables to improve the signal-to-noise ratio for clustering. Furthermore, conducting clustering analyses on the PCA components allow for consistency of interpretation of results, allowing comparability of the various analyses employed (e.g., apriori groups based on BADIA vs. data-driven cluster based on k-means).

The optimal number of clusters was tested by running the k-mean algorithm with $k = 2$ to $k = 10$ and computing the within-cluster sum of squares (WCSS) and plotting the results using the elbow method (the selected model being the model with the lowest WCSS) as well as a silhouette score (ranging from -1 to 1) with positive scores indicating better cluster separation and negative scores or a score of zero suggesting no reliable clusters. All cluster analyses

were implemented in R. Analyses code is provided in https://github.com/hcuve/InteroStudy_Autism_alexithymia.

4.3.3 Results

Descriptive aggregate patterns and correlations are presented in Tables S-4.8-1 and S-4.8-2 for self-reports and physiological ratings.

4.3.3.1 Univariate results

4.3.3.1.1 Alexithymia, not autism, is related to reduced concordance between phasic pupil responses and subjective arousal

Phasic changes in pupil diameter were positively related to ratings of subjective arousal, with more arousing stimuli inducing stronger pupil dilation (Estimate = 0.11, SE = 0.04, $p < .001$), even after accounting for brightness. There was no direct effect of alexithymia on pupil diameter changes (Estimate = .06, SE = .12, $p = .064$). Nonetheless, there was an interaction between subjective arousal ratings and alexithymia (Estimate = -0.08, SE = .03, $p = .007$), suggesting that the concordance between subjective arousal and phasic pupillary change was moderated by alexithymia (see **Figure 4.19**). Slope analyses confirmed this conclusion, showing that the slope relating arousal to pupil diameter was significant for low (Estimate = 0.19, SE = 0.05, $t = 3.80$, $p < .01$) and average (Estimate = 0.11, SE = 0.04, $t = 2.81$, $p = .01$), but not for high levels of alexithymia (Estimate = 0.04, SE = 0.05, $t = 0.79$, $p = .430$).

Autistic traits did not significantly predict pupil diameter changes (Estimate = -0.14, SE =0.12, $p = .260$), nor interact with subjective arousal (Estimate = -0.054, SE = 0.03, $p = .090$), and results remained consistent when testing autism and alexithymia in the same model.

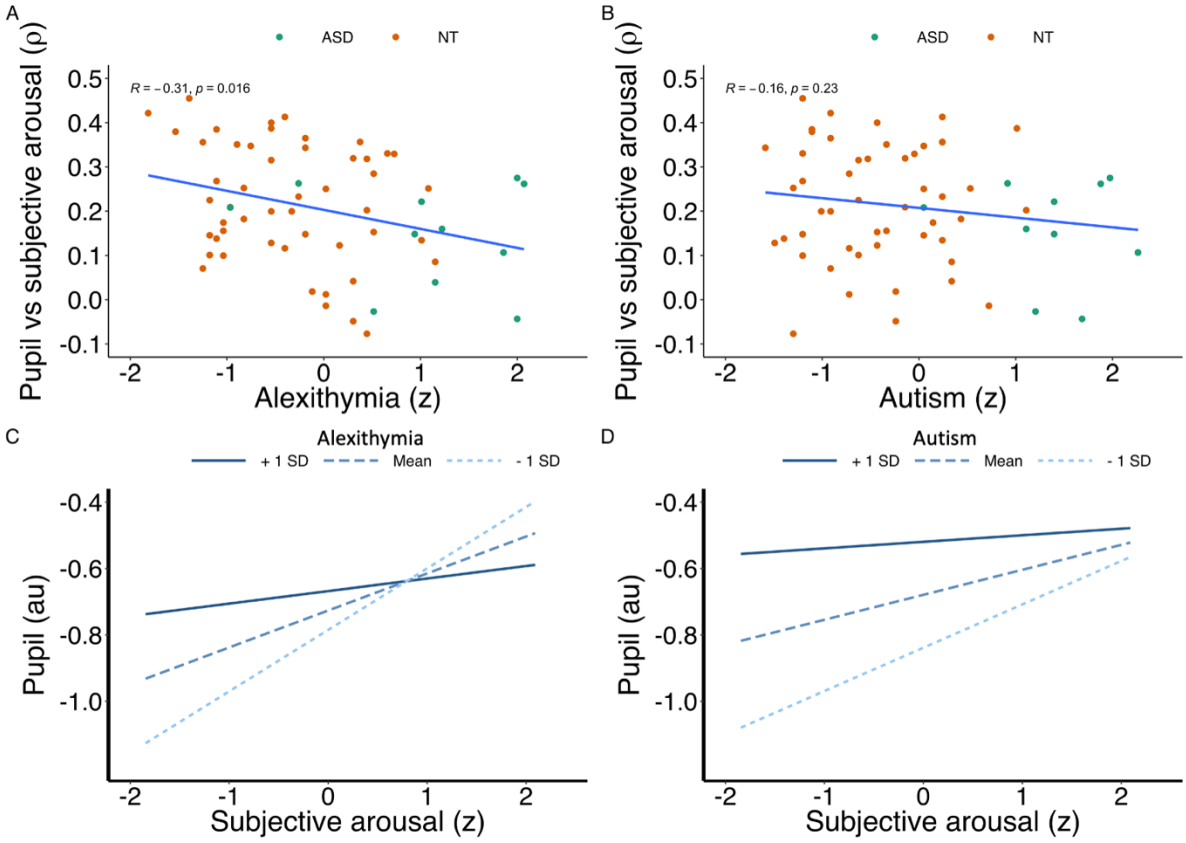

Figure 4.19 A visual summary of pupil diameter and subjective arousal analyses. There was reduced concordance between subjective arousal and pupil diameter for individuals higher in alexithymia (A and C), but not autism (B and D). Pupil values reflect baseline-corrected values.

4.3.3.1.2 Alexithymia, not autism, is related to reduced phasic SCRs to emotion-inducing stimuli

For SCR analyses only a significant effect of Alexithymia was observed, which predicted decreased phasic SCRs in response to stimuli (Estimate = -0.35, SE = .013, p = .029). There was no significant effect of subjective arousal (Estimate = 0.02, SE =0.02, p = .260), nor significant interactions with alexithymic traits (Estimate = -0.01, SE = .01, p = .340). There was also no main effect of autistic traits (Estimate = 0.03, SE = 0.13, p = .810), or interaction with subjective arousal on SCRs (Estimate= 0.0009, SE = 0.02, p = .096) – see Figure 4.20.

These results indicate a direct effect of alexithymia consistent with hypo-arousal to emotionally inducing stimuli, at least in terms of sympathetic activation as measured by SCR.

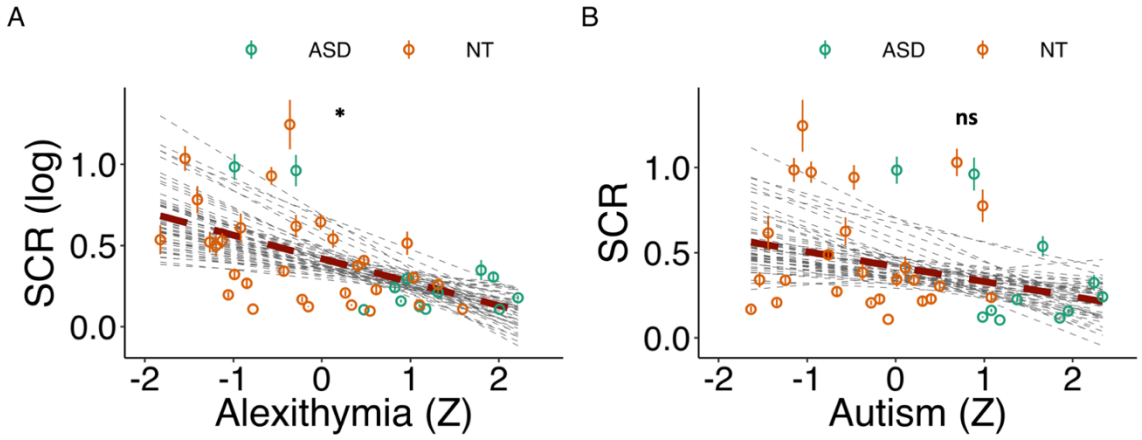

Figure 4.20 Illustration of mixed models results showing a significant effect of alexithymia.

* p < .05, ns = non-significant.

4.3.3.1.3 Alexithymia, not autism predicts phasic heart rate changes to emotion-inducing stimuli

Heart rate changes were predicted by a negative effect of subjective arousal, that is, arousing stimuli were related to a significant decrease in heart rate during stimulus viewing (Estimate = -0.41, SE = 0.12, $p < .001$). There was also a significant effect of alexithymia on heart rate change; increasing alexithymia was related to phasic heart rate deceleration (Estimate = -0.59, SE = 0.27, $p = .030$). There was also a significant interaction between subjective arousal and alexithymia (Estimate = -0.26, SE = 0.13, $p < .01$). A slope analysis revealed that this effect was driven by a significant negative slope relating heart rate to arousal for high (Estimate = -0.50, SE = 0.14, $t = -3.71$, $p < .001$), and average levels of alexithymia (Estimate = -0.29, SE = 0.10 , $t = -2.95$, $p < .01$), whereas it was not significant in low levels of alexithymia (Estimate = -0.07, SE = 0.12, $t = -0.60$, $p = .550$) – see **Figure 4.21**. These results remained significant when controlling for autistic traits, and there was no direct effect of

autistic traits on heart rate change (Estimate = 0.25, SE = 0.29, p = .390), nor significant interactions with subjective arousal rating (Estimate = -0.11, SE = 0.15, p = .450).

4.3.3.1.4 Summary of univariate results

In summary, the separate mixed models exploring the influence of autism and alexithymic traits and their relationship with subjective arousal and physiological responses suggested a consistent contribution of alexithymia across all physiological modalities. Nonetheless, rather than supporting general hypoarousal or hyperarousal theories, these results suggest a complex relationship between alexithymia and physiological responding. While the analysis of pupil diameter and SCR suggests reduced concordance between pupil diameter and subjective arousal, and physiological responding as measured by pupil diameter was linked to alexithymic traits, alexithymia was related to increased physiological responding as measured by heart rate changes. Therefore, these results confirm atypicalities in alexithymia with respect to physiological responses to emotions.

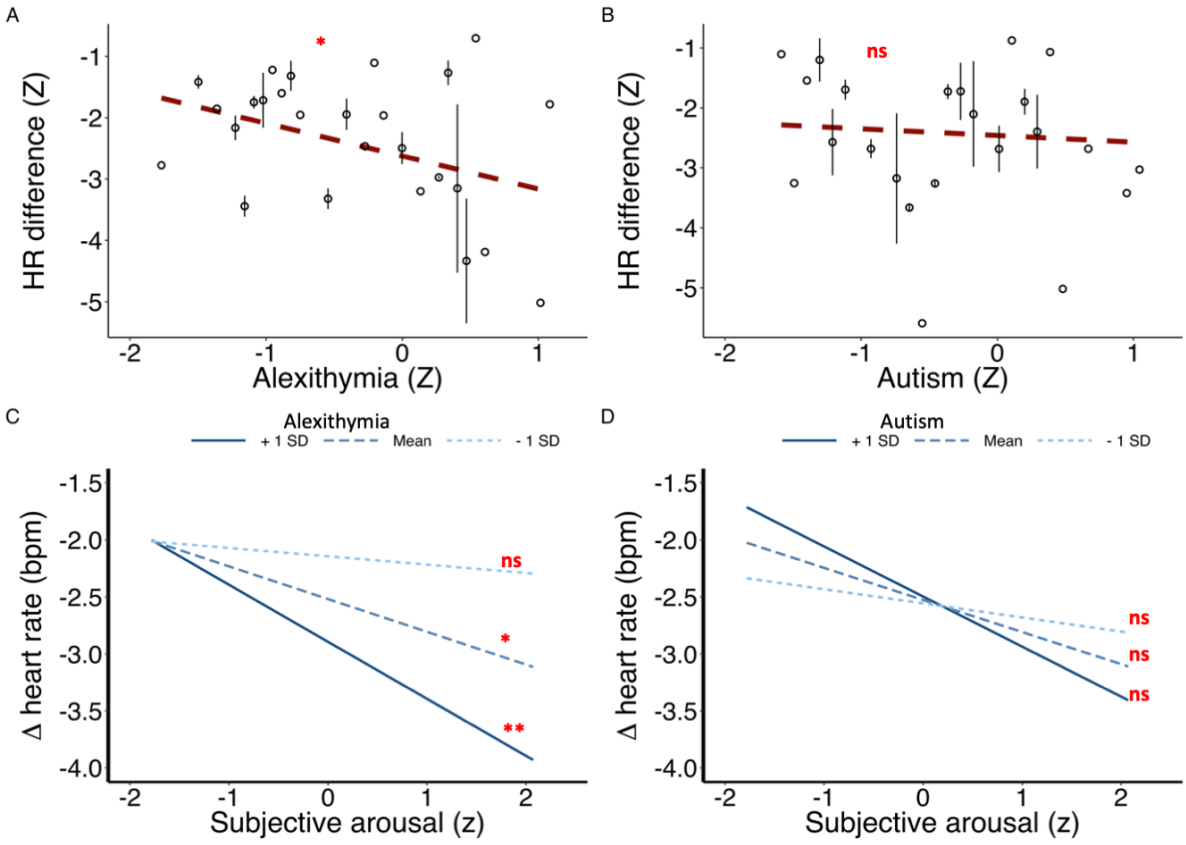

Figure 4.21 Visual illustration of results from the mixed models predicting phasic HR changes.

Alexithymia (A), not autism (B), was related to a negative change in heart rate during stimulus viewing. Alexithymia also interacted with subjective arousal ratings, with high but not low alexithymia levels being related to heart rate deceleration during emotional stimuli viewing (C), whereas no effect was observed for autistic traits (D). * $p < .05$, ** $p < .01$, ns = non-significant.

4.3.3.2 Exploring multivariate patterns of physiological responses

In this section, I present results exploring the multivariate profiles of physiological responses and test for the existence of sub-groups based on these profiles, including whether these profiles can separate groups defined *a priori*.

4.3.3.2.1 Dimensionality reduction of physiological responses

To make exploration of multivariate physiological profiles tractable, first, a dimensionality reduction approach using a Principal Component Analysis was applied to capture the covariation in the various physiological responses and reduce them into a subset of dimensions/components that best accounts for the observed response patterns, as well as to identify the most discriminant set of variables contributing significant variance to these components (Beaton, Fatt, & Abdi, 2014).

Inference tests on the PCA solution suggested an average of 2 to 5 significant components, with the first two components explaining the largest amount of variance. Component 1 explained 17.42% of the variance, $p = .001$, whereas component 2 explained 11.258% of variance, $p = .001$.

The easiest way to interpret the corresponding components is by looking at the variables and direction of the largest loadings in each component. Component 1 was characterised primarily by positive SCR coefficients, and negative HR coefficients (see **Figure 4.22**). Inspecting the stimuli loadings revealed that this component included physiological responses dominated by highly arousing stimuli, including sexual stimuli and injury (see Figure 4.22). Component 1 can be interpreted as an SCR and heart rate activation pattern, such that subjects with high scores on this dimension display the greatest SCR activation and negative heart rate change. Similarly, stimuli contributing more strongly to these components would show a similar pattern. Component 2 was characterised primarily by positive loadings of

pupil diameter and negative loadings of heart rate. Inspection of the stimuli loadings also suggested that highly arousing stimuli clustered together based on similar patterns of pupil diameter change, and some of these stimuli were the same as those that loaded highly on Component 1.

However, looking at the multidimensional scaling plots and correlation circle (see **Figure 4.23**) which plots the components and variables in terms of their similarity (closer together = more similar, B) contributed variance to the components (size of the dots – Figure 4.23-B, and proximity to outer circle –Figure 4.23-A) (Derek Beaton et al., 2014), it becomes apparent that pupil diameter patterns show the least covariation with the other responses, but are significantly explained by Component 2. Thus, this component appears to capture physiological responses that are distinct from the SCR and HR patterns in Component 1. Nonetheless, a few pupil diameter patterns contributed significantly to both components, and stimulus inspection revealed this to be driven primarily by positively-valenced stimuli.

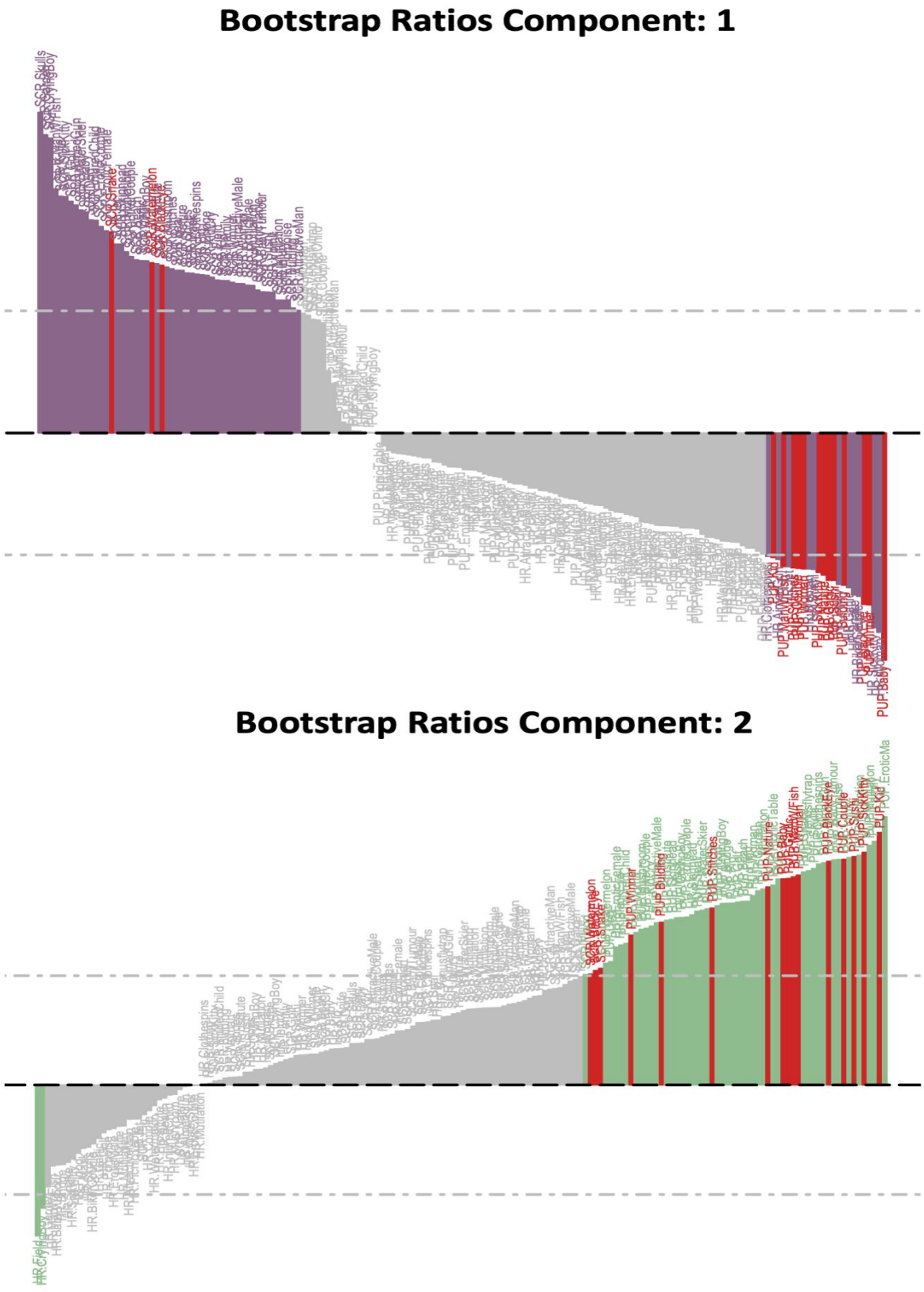

Figure 4.22 The bootstrap ratios for the first 2 components.

Gray bar indicates variables that did not significantly contribute to the components. Contributing variables (> 2 thresholds, indicated by dashed lines) are marked plum for Component 1 and green for Component 2. Red bars indicate features contributing to both dimensions.

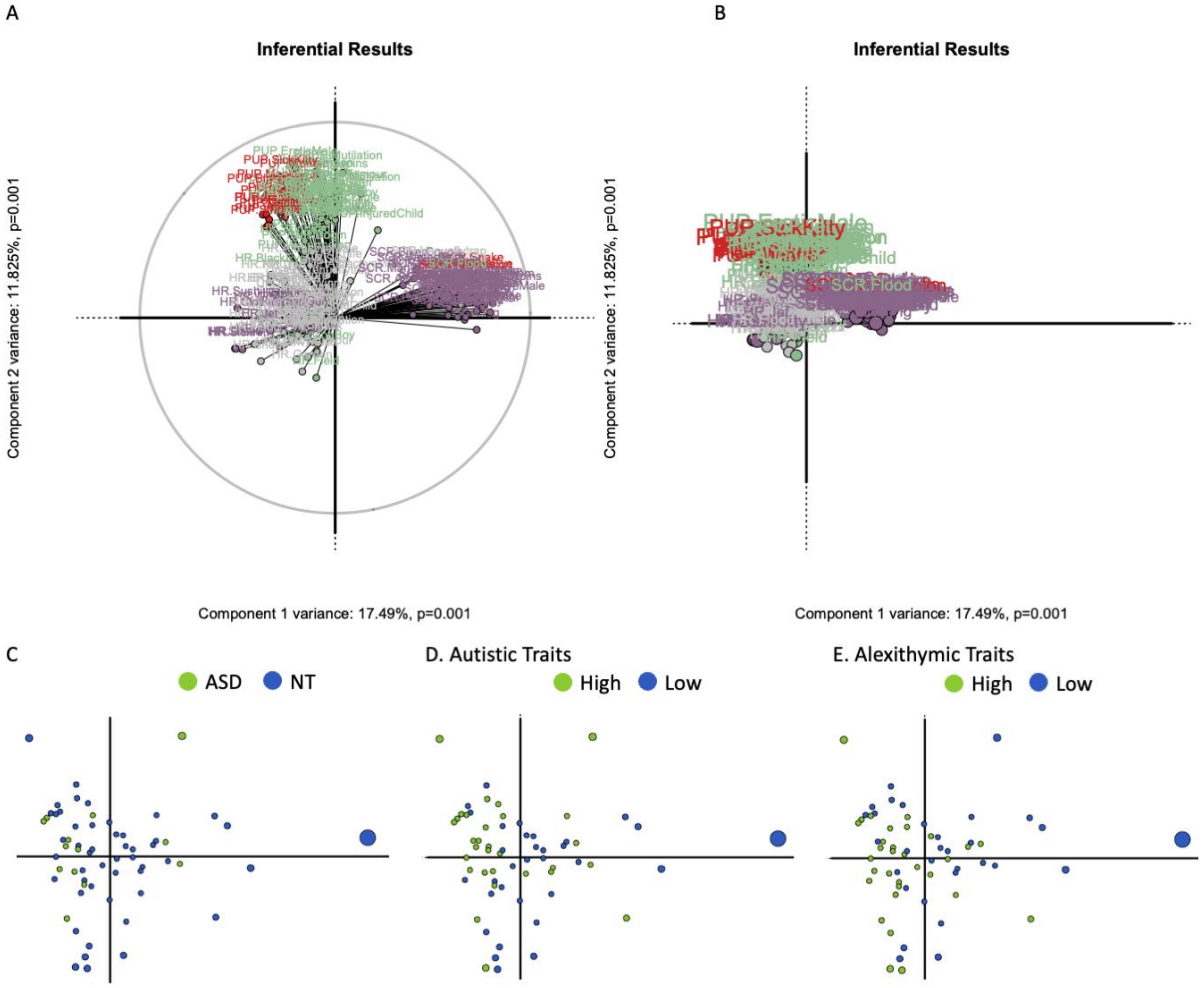

Figure 4.23 Principal component maps.

A. Unit circles showing how much the physiological responses are explained by each dimension (closer to outer circle means that variables are more strongly explained by their respective dimension): SCR and pupil diameter are more strongly explained by components 1 and 2, respectively. B. Component map for physiological responses. Gray indicates features that did not significantly contribute to the components. Plum-colored cluster

contributes to Component 1, green to Component 2, and red to both. Circle size is proportional to variance contributed to the component. C, D & E represent component maps with factor scores for participants split by autism and alexithymia, high vs low autistic traits, and high vs low alexithymia respectively.

4.3.3.2.2 PCA on aggregated data

Given the number of variables represented in the solution above, it may be difficult to absorb the general patterns. Therefore, a similar PCA was conducted on the aggregated data. This also allows exploration of potential trade-offs in statistical power which may be reduced when estimating a large set of variables as in the analysis above. The aggregation was performed by computing an average score for each modality per participant, for the 1st quartile, mean, and 3rd quartile of normative valence ratings for the IAPS stimuli (loosely reflecting negative, neutral/ambiguous, and positive valence). Although other criteria could be used, this was preferred for simplicity and given that categorical normative or ground truth data is not necessarily available for IAPS images.

Overall results were consistent with the ones reported above (see **Figure 4.24**) with the PCA solution suggesting between 2 to 5 components, and the first 2 components explaining the large amounts of variance (71% of the total variance). The first component explained 47% of the variance ($p < .001$), and the second component was also significant, explaining 28.79% of the variance ($p < .001$). Component 1 was characterised primarily by positive loadings for SCR and negative HR. Component 2 again emerged as a pupil diameter response component with positive pupil diameter loadings. Similar to the previous analyses, one set of pupil responses (to positive stimuli) contributed to both components.

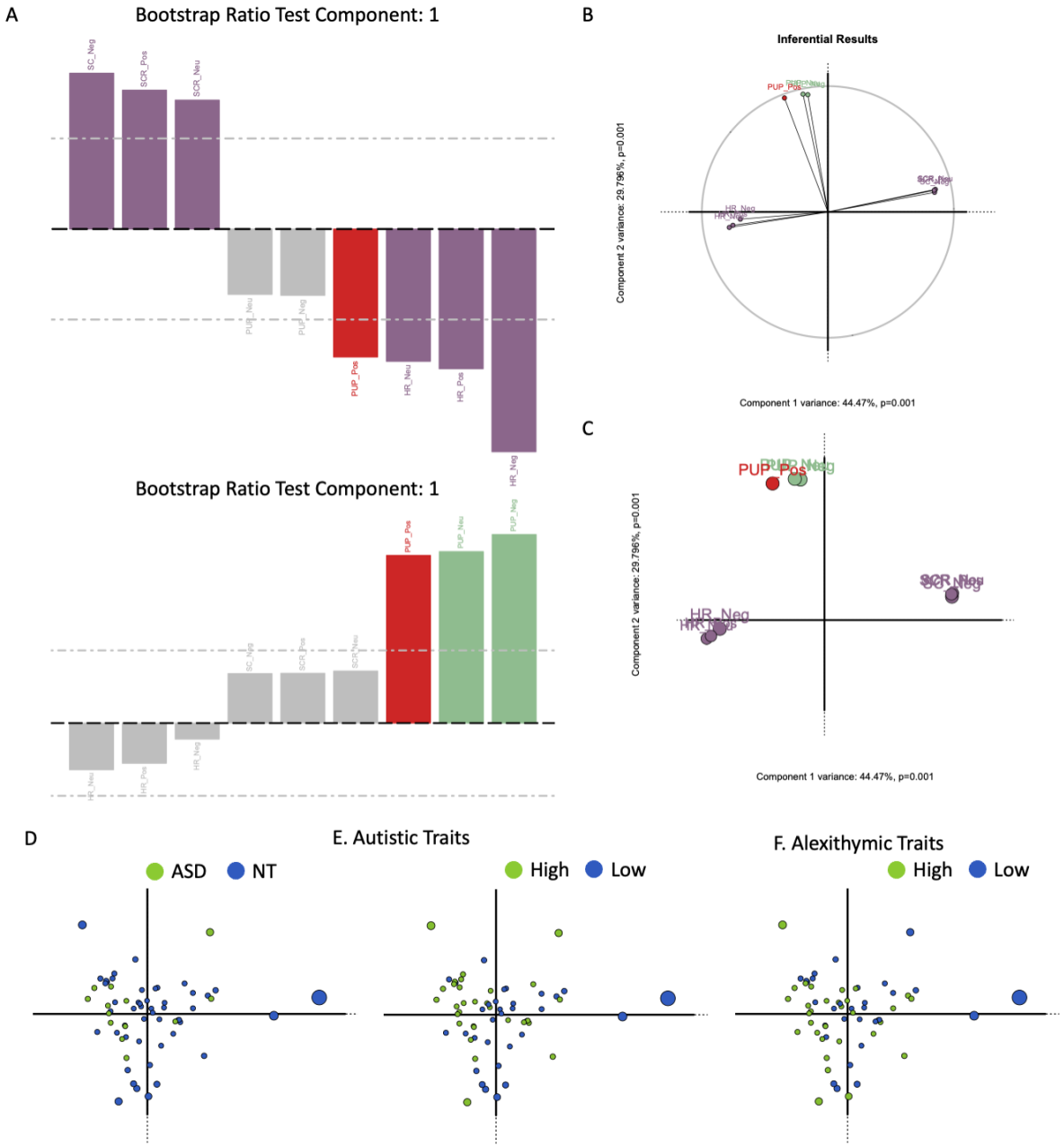

Figure 4.24 PCA results on aggregated data.

A. Bootstrap ratio test plot. B. Unit plot (correlation between variable and component). C. Correlation map based on factor scores by variables (physiological responses by valence). D, E, and F. Correlation map based on factor scores by subjects (physiological responses by valence). A visual summary of the PCA inference results based on aggregated factor scores. PUP = Pupil, HR = Heart rate, SCR = Skin Conductance Response, Neg = Negative, Neu = Neutral, Pos = Positive.

The contributions of each modality across the levels of valence, descriptively resemble the relative scaling of arousal by negative, neutral (ambiguous), and positive stimuli that were reported in in Experiment 7 including the tighter coupling of subjective arousal and pupil dilation for positive valence.

4.3.3.2.3 Summary of dimensionality reduction results

The dimensionality reduction of physiological responses revealed that most responses could be reduced to at least two components. Component 1 represents a pattern of coactivation between SCR and HR as well as pupil responses to primarily positive valenced stimuli. Component 2 reflects primarily pupillary arousal that does not covary with other physiological modalities apart from responses to some positively valenced stimuli. These results suggest differences in the degree of contributions across modalities, such that not every autonomic modality contributed equally to the components.

4.3.3.2.4 Clustering participants based on multivariate physiological profiles

Two main approaches were employed to explore whether participants could be clustered based on their physiological profiles. First, a barycentric discriminant analysis (BADIA) was used based on *a priori*-defined groups (Abdi & Williams, 2010). Second k-means clustering on the PCA components was employed to explore the possibility that subgroups may not necessarily conform to any *a priori* definitions.

Testing apriori group categories based on physiological data

After 1000 permutations, the BADIA method suggested a single vector solution, and the reliability of group assignment (R^2) and omnibus inertia were not significant (p = .900), nor for high vs low autistic traits (based on median split) (p = .946) or high vs low alexithymic traits (p = .144). These results therefore suggest that the separation of *a priori* defined groups based on specific multivariate profiles was not reliable. As such, these results falsify the

hypothesis that participants would be able to be differentiated via their pattern of physiological responses on the basis of alexithymia or autism subgroups.

Data-driven clustering of participants based on physiological responding

Following the test of *a priori* subgroupings based on physiological profiles, I proceeded to test data-driven clustering. A similar approach to the one described in the Experiment 7 was employed here, but using the components from the first PCA (see: 4.3.3.2.1 Dimensionality reduction of physiological responses page: 241) as features to the k-mean analysis to improve signal-to-noise ratio. The ideal number of clusters (from $k = 2$ to $k = 10$) was selected based on the silhouette score for each solution. The silhouette value is a measure of cohesion (how similar an object is to its own cluster) and separation (compared to other clusters) with 1 indicating perfect separation and values closer to zero indicating deficient solutions. The k-means results suggested an optimum of 2 clusters with a silhouette score of .42. This method was also consistent with the results from the elbow method, which is similar to the scree plot method used for PCA or factor analysis. It uses the within-cluster sum of squares errors (WCSS), a measure of the compactness of each cluster (lower values are better). The validation of the optimal number of clusters is made considering the size of each step, with only the largest differences being considered important to select the number of clusters. So, in the current case, the largest drop in WCSS occurs on the second cluster (K2, WCSS = 319.57) - see **Figure 4.25**.

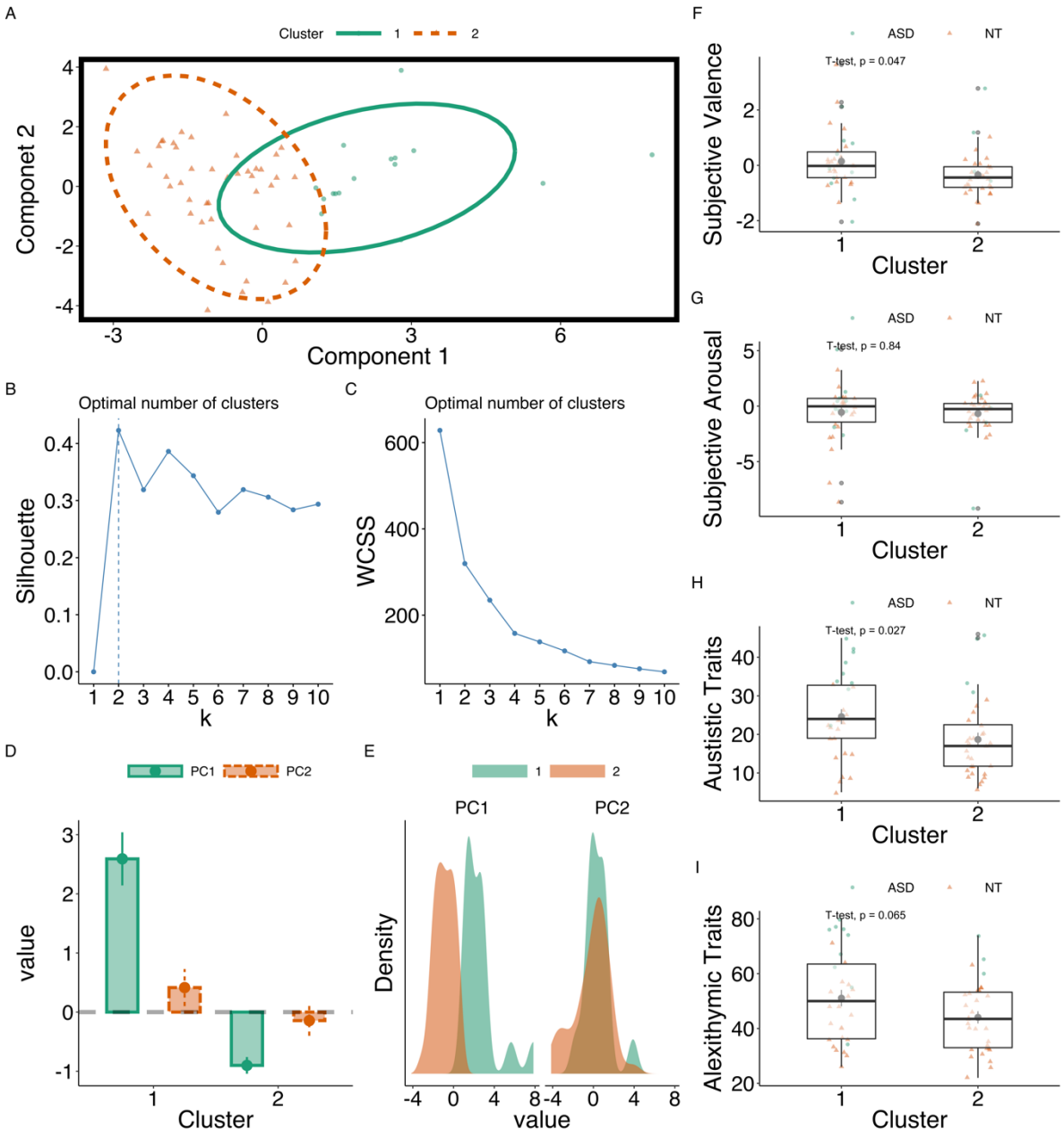

Figure 4.25 Illustration of the K-mean cluster results based on the multivariate physiological scores.

A, B & C. Illustration of selection criteria of optimal *k*. D & E. Descriptive patterns for the clusters. F-I. Comparison of self-reports based on k-mean clusters.

Nonetheless when comparing the respective clusters (groups) in terms of autistic and alexithymic trait scores, there was a trend for higher autistic and alexithymia scores on Cluster 1 compared to cluster 2 (Autistic traits: t = 2.26, df = 59.137, p = .026; Alexithymia: t = 1.88, df = 54.21, p = .065). When looking at how these clusters differed in terms of subjective ratings, there was also a significant difference in average valence ratings, with cluster 1 showing slightly more positive ratings than cluster 2 (t = 2.02, df = 62.53, p = .047) but no difference in average subjective arousal (t = 0.203, df = 62.407, p = .839).

4.3.3.2.5 Dimensional analyses

One critique from previous research on physiological responding in alexithymia and autism was the overreliance on subgrouping, which varies depending on the criteria for subgrouping (e.g., median split, diagnoses, or cut-off scores) and ignores individual differences to a large extent. Indeed, the failure to discriminate *a priori* groups based on the BADIA method, as well as the fact that the k-mean clusters can only be differentiated by very small group differences on alexithymia and autistic traits might indicate high variability within each group, which may reduce statistical power for group-based analyses with the current sample size. So, I also explored how autism and alexithymia related to the PCA factor scores (participant scores/position in the multivariate physiological space). These scores essentially rank different participants on the components that explain the multivariate pattern of physiological activation. For example, Component 1 is dominated by positive SCR loadings and negative HR loadings. High scores for one subject on this component would indicate that they exhibited these patterns (high SCR and strong HR deceleration).

Indeed, correlational analyses indicated that alexithymia was negatively related to Component 1 (SCR and HR component, and pupil responses to positive valenced stimuli) ($r_{(61)}$ = -.46, p = .001), whereas no relationship was observed for Component 2: pupil

responding ($r_{(60)}$ = 0.02, p = .830). There was a similar significant trend for autistic traits with Component 1 ($r_{(60)}$ = -.25, p = .048) and no relationship with Component 2 ($r_{(60)}$ = -0.06, p = .630). However, the alexithymia relationship with Component 1 remained significant even after accounting for autistic traits (r(61) = -0.31, p = .014), whereas the autistic traits relationship with Component 1 became non-significant when controlling for alexithymia ($r_{(60)}$ = 0.02, p = .880) – see **Figure 4.26.** These correlational analyses suggest that despite the failure to discriminate *a priori* groups based on physiological profiles, alexithymia is in fact related to atypical physiological responding profiles particularly in the pattern of SCR and HR coactivation.

4.3.3.2.6 Summary of multivariate results

A dimensionality reduction of physiological response profiles suggested two main components representing covarying patterns of SCR, HR (and a subset of pupil responses), and another component primarily dominated by pupillary arousal. While the discriminant analysis based on *a priori*-defined groups did not prove to be able to differentiate groups reliably based on physiological patterns, both data-driven clustering and dimensional analyses indicate that alexithymia is related to atypical physiological responding, ranking lower on a dimension representing covarying SCR, HR, and pupil diameter changes.

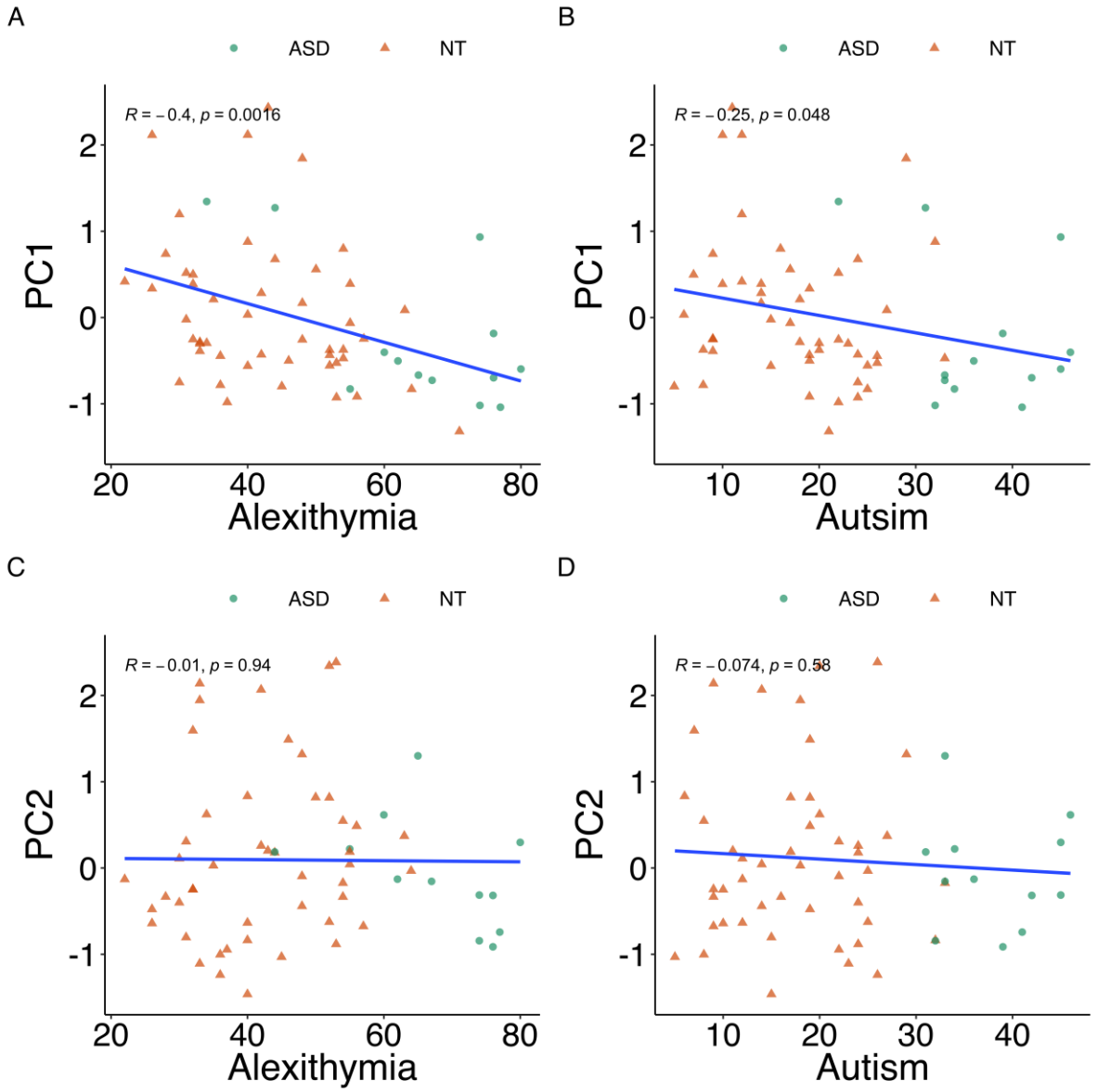

Figure 4.26 Scatterplots for correlational analyses between alexithymia, autistic traits, and physiological profile scores.

PC1 = Component 1 (primarily SCR and HR deceleration), and PC2 = Component 2 (primarily pupil response).

4.3.4 Discussion

Hypotheses regarding atypical socioemotional functioning in autism and alexithymia have implicated hypo-and hypoarousal profiles of physiological responding as possible

mechanisms. Empirical evidence has proved mixed, however, with most studies failing to explore potential multivariate profiles of physiological mobilisation to emotion. The current study explored the potential role of alexithymia and autism in disrupting links between multiple signals of physiological arousal and emotion. A novel approach was used that capitalises on multimodal recordings and building multivariate response profiles to test differentiation of participants according to autism and alexithymia, or to identify subgroups within those populations.

Results show that alexithymia is associated with atypical multivariate patterns of physiological responses. Consistent with the alexithymia hypothesis, alexithymia, not autism, was related to atypical skin conductance, phasic heart rate changes, and reduced concordance between subjective reporting and pupil dynamics. As such, these findings suggest that alexithymia may disrupt emotional responding via physiological and interoceptive routes, and that varying profiles of physiological responses to emotions in autism may be driven by alexithymia.

These findings are consistent with several studies implicating atypical physiological responses in alexithymia in general (Panayiotou et al., 2018) and in autistic participants in particular (Bird et al., 2010; Gaigg et al., 2018; Lassalle et al., 2019). Nonetheless, rather than supporting general hypoarousal or hyperarousal, both the univariate and multivariate analyses paint a complex picture regarding the profile of physiological responding. While alexithymia was related to hypoarousal in terms of sympathetic outputs (SCR), it was also associated with HR deceleration during the emotion task. In a previous study, it was shown that the latter is in fact a marker of activation, particularly to negative emotions (see Experiment 7).

One way to view the current results is that hypoarousal and hyperarousal tendencies in alexithymia may be modality specific. While the suggestion of diverging activation pattern

across autonomic signals might sound counterintuitive, such patterns are not unlikely if one considers that the autonomic system is not anatomically or functionally uniform. Influential models of physiological responding have outlined a number of constraints which may drive diverse patterns of physiological responding. For example, some organs that are modulated by the ANS are subject to the dual influence of both sympathetic and parasympathetic activation (e.g. cardiac responses, due to dual innervation), which may result in multidimensional profiles of responding (Berntson et al., 1991) when compared to singly innervated organs (most of the electrodermal system). Furthermore, interactions between autonomic pathways and CNS open up the possibility for modulation of autonomic responding by higher-level systems (Folkow, 2000; Hilton, 1975; Jänig & Häbler, 2000; Leuchs et al., 2019) which need not affect different autonomic outputs (and different arousal signals) equally.

One question that must be addressed if one accepts the above, however, is if there are divergent hypo- and hyper-arousal states across various autonomic signals, how do these drive subjective experiences and behavioural processes in a coherent manner? One possibility is that the subjective impact of varying levels of hyper and hypoarousal across autonomic systems depends on the degree to which the different modalities can be considered interoceptive. At present, the degree to which the various physiological signals can be construed as interoceptive (i.e., available for conscious perception) is still unclear.

Any difference in the degree to which arousal signals are available for interoception is particularly important for research investigating decoupling between physiological arousal and subjective experience in alexithymia - especially when different degrees of decoupling are observed across physiological signals. The degree of attention paid to the various physiological signals may be relevant in explaining such differences, especially if modulated

by emotional states. For example, fear and anxiety may promote attention to heart rate changes, while anger, sadness, or disgust may promote attention to body temperature and stomach sensations (Oka, 2015; Rausch et al., 2003). The implication of these points is that the various tests of concordance between physiological responses and emotional experience reported in the literature may be incomplete, requiring multivariate characterisations of physiological responses across subjective experiences.

4.3.4.1 The physiological mobilisation imbalance hypothesis

It is useful to discuss how the findings linking alexithymia to atypical multivariate patterns of physiological responding may inform theorising in alexithymia, and the implications for understanding autism and other clinical conditions. The current findings suggest that alexithymia is related to an imbalance in the mobilisation of physiological processes in reaction to emotional stimuli. Therefore, rather than looking at the mere direction of individual outputs (SCR, HR, pupil) or searching for general hypo or hyperarousal tendencies, the patterns of coherence (or lack thereof) of the mobilised responses may be what defines alexithymia. Alexithymia might be taken as a (series of) imbalanced physiological mobilisation profiles, marked by insufficient or incomplete activation at various levels of physiological responding caused by aberrant autonomic control. As such, the physiological mobilisation imbalance hypothesis could reconcile the various mixed findings including inconsistencies in the direction of physiological responses across signals reported in alexithymia (Panayiotou et al., 2018) and, as a result of high rates of alexithymia in the autistic population (Cuve et al., 2018; Lydon et al., 2016). The physiological mobilisation account may also explain the mixed evidence from neuroimaging studies, where hypoactivation and hyperactivation of various structures and pathways implicated in emotion has been reported in autism and alexithymia (Cuve et al., 2018; Goerlich & Aleman, 2018; Philip et al., 2012; Senju & Johnson, 2009b).

The crucial tests of the physiological mobilisation imbalance hypothesis have yet to be done. In particular, more studies exploring multivariate patterns of physiological responses are needed to characterise the normative profiles of physiological mobilisation to emotion, and in those with autism and alexithymia. Despite many years of research, multivariate profiles of emotion responding have not been sufficiently mapped due to the reliance on single modality recordings and univariate tests (Friedman et al., 2014; Norman et al., 2016; Stephens, Christie, & Friedman, 2010). Larger sample size studies are also needed, given the potential high degree of individual variability of physiological and subjective responses to emotion.

4.3.5 Conclusion

Results show that alexithymia, and not autism, is related to atypical multivariate patterns of physiological responses to emotions, and the degree of concordance between subjective emotion and physiological responding. However, rather than supporting strict hypoarousal or hyperarousal accounts, these results suggest an atypical mobilisation of physiological responding across various levels of autonomic responding. As such, physiological responding needs to be viewed in terms of the functional consequences of various multivariate profiles of autonomic mobilisation during emotional responding. These results call for a rethinking of models of emotional responding in alexithymia and autism, and highlight that alexithymia, not autism may drive atypical emotion responding.

Materials availability

Code and data are available at: https://github.com/hcuve/InteroStudy_Autism_alexithymia and https://osf.io/vf9sg/ respectively.

4.4 What comes next?

Chapter 4 contributed new insights to the study and characterisation of physiological responses involved in emotional experience. It provided an additional test of the specificity of autism and alexithymia in socioemotional deficits and consistent with the alexithymia hypothesis, results implicated alexithymia in atypical autonomic mobilisation to emotions. In Chapter 5, I turn the attention to a question that is central to the logical coherence of the alexithymia hypothesis and examine whether there is a potential overlap between autism and alexithymia at a construct level and what the implications of this are for theory and assessment

5 CHAPTER 5 - ARE AUTISM AND ALEXITHYMIA DISTINCT?

An edited version of this chapter has been published as:

Cuve, H. C., Murphy, J., Hobson, H., Ichijo, E., Catmur, C., & Bird, G. (2021). Are Autistic and Alexithymic Traits Distinct? A Factor-Analytic and Network Approach. *Journal of Autism and Developmental Disorders*, 1-16.

Abstract

Despite the heterogeneity in autism, socioemotional difficulties are often framed as universal. Increasing evidence, however, suggests socioemotional difficulties may be explained by alexithymia, a distinct yet frequently co-occurring condition. If, as some propose, autistic traits are responsible for socioemotional impairments, then alexithymia may itself be a symptom of autism. The work in this chapter aimed to determine whether alexithymia should be considered a product of autism or regarded as a distinct condition. Using factor-analytic and network approaches, the work in this chapter provides evidence that alexithymic and autistic traits are distinct. I argue that: 1) models of socioemotional processing in autism should conceptualise difficulties as intrinsic to alexithymia; and 2) assessment of alexithymia is crucial for diagnosis and personalised interventions.

Keywords: autism, alexithymia, factor, network, separation.

5.1 Introduction

It is well-recognised that autism is a highly heterogeneous condition (Martinez-Murcia et al., 2017; Mottron & Bzdok, 2020), and this heterogeneity is particularly apparent in socioemotional functioning (emotion recognition and emotional reciprocity). Despite assertions that socioemotional difficulties are a 'hallmark' of autism (Du Bois, Hobson, & Hobson, 2014; Guastella et al., 2010), these claims are often based on indirect evidence - such as impaired theory of mind or a claimed lack of prosocial behaviour – thought to rely on emotion recognition and affect sharing (Ben-Shalom, Belmonte, Gaigg, Bowler, 2021; Gaigg, 2012). As discussed throughout this thesis , despite the widespread acceptance of this view, direct studies of socioemotional processing in autism have produced highly mixed findings - for a review see Cuve, Gao, & Fuse (2018) or Uljarevic & Hamilton (2013) - suggesting that socioemotional impairments are far from universal in autism.

Appeals to the heterogeneity of autism do not *explain* these mixed findings, rather they just provide a redescription of the variability across autistic individuals (note: I use the word autistic to refer to individuals with autism as this terminology is preferred by the autistic community, Kenny et al., 2015). In contrast, a body of work suggests that heterogeneity with respect to socioemotional processing within the autistic population may be systematic and explained by co-occurring alexithymia which is more prevalent in the autistic population (approximately 50%) than in the general population (Bird & Cook, 2013; Cook, Brewer, Shah, & Bird, 2013; Kinnaird, Stewart, & Tchanturia, 2019; Trevisan, Bowering, & Birmingham, 2016).

As discussed in Chapter 1, the increased prevalence of alexithymia in the autistic population is not specific to autism, but is observed in numerous other psychiatric conditions (Hobson et

al., 2020a; Hobson, Brewer, Catmur, & Bird, 2019; Taylor, Parker, Bagby, & Bourke, 1996; Westwood, Kerr-Gaffney, Stahl, & Tchanturia, 2017). Alexithymia is therefore argued to be neither necessary nor sufficient for an autism diagnosis.

Throughout this thesis I reviewed and provided evidence in support of the 'alexithymia hypothesis' – the idea that, where observed, socioemotional deficits in autism are due to co-occurring alexithymia and not autism. For the alexithymia hypothesis to be logically coherent, autism and alexithymia must be distinct. However, others have considered alexithymia to be a symptom or consequence of autism (Gaigg, 2012; Quattrocki & Friston, 2014). Under this view, alexithymia would be yet another characteristic of autism which shows variability within the autistic (and general) population, albeit a characteristic which covaries with socioemotional functioning (such that autistic symptoms cause some individuals to be alexithymic and have poor emotion recognition and low levels of empathy, while other autistic individuals are unaffected in these domains). Understanding whether alexithymia and autism are distinct, or whether alexithymia is a symptom or product of autism is therefore important for theoretical reasons.

There are also clinical reasons to ascertain whether alexithymia and autism are distinct, particularly in relation to autism assessment, diagnosis and treatment. If the emotional difficulties in autism are in fact due to alexithymia, and alexithymia is distinct from autism, then an assessment of alexithymia is required when diagnosing autism to ensure that a full picture of the patient's strengths and weaknesses is obtained, and their needs addressed. This scenario may also require a rethinking of diagnostic protocols; evidence suggests that alexithymia increases the likelihood of an autism diagnosis at least two-fold (Berthoz & Hill, 2005; Hobson et al., 2020b). If alexithymia and autism are indeed separable, then diagnostic

protocols may need revision to account for the fact that not all autistic individuals will show socio-emotional problems and yet they may still struggle with restricted interests and communication more broadly. Furthermore, autistic individuals who exhibit good socioemotional functioning (due to an absence of alexithymia) may not be referred for assessment, or receive a diagnosis, if the presence of good socioemotional functioning is deemed to preclude an autism diagnosis.

Given the absence of objective markers (e.g. genetic or brain markers), autism and alexithymia are operationalised using questionnaires or interviews to identify diagnostic behaviours, symptoms or traits. Two extensively used measures of autism and alexithymia are the AQ-50 (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001) and TAS-20 (Bagby, Taylor, & Parker, 1994) respectively which were used in previous chapters. The AQ-50 measures autistic traits across a number of dimensions (*social skills, communication, imagination, attention to detail* and *attention switching*), while the TAS-20 measures three facets of alexithymia; *difficulties identifying and describing one's own emotions*, and *externally oriented thinking*. Both the AQ-50 and TAS-20 were designed to be used with both clinical and non-clinical samples. In order to explore whether alexithymia is a product of autism (i.e. that a common factor underlies autistic and alexithymic traits, where that common factor may be autism itself), or whether autism and alexithymia traits are distinct, we examined the overlap between alexithymic and autistic traits as measured by the TAS-20 and AQ-50. The focus on the measurement level is based on three reasons: 1) these measures operationalise the constructs into measurable traits; 2) given their frequency of use, potential overlap between these measures has practical implications for research and clinical practice, and 3) they are compatible with prevailing models of autism and alexithymia as traits that exist to varying degrees in the general population.

Two main approaches were used to examine the overlap between these measures: dimensionality reduction and a network approach. Dimensionality reduction was addressed with a joint exploratory factor analysis of the AQ-50 and TAS-20, with additional testing of confirmatory, theoretically-driven models of the covariance between dimensions of autism and alexithymia in an independent sample. This approach allows competing models of the relationship between autistic and alexithymic traits (i.e. the common vs distinct latent factor models) to be formally contrasted.

A network approach was also used, which allows investigation of complex relationships between variables. This approach builds on systems theories of psychopathology, which attempt to explain relationships between different symptoms and the frequent comorbidity seen in psychopathology (Borsboom & Cramer, 2013). Underlying this approach is the view that psychopathology (and mental health) is a dynamic system, where all nodes (symptoms or traits) can influence other nodes in the system (network), and these dependencies can be quantified. For instance, even if completely separable at the latent dimensional level, autism and alexithymia may influence one another causally. To illustrate, difficulties identifying feelings might lead to difficulties socialising, or difficulties with communication might make it difficult to describe feelings, leading to strong dependencies between autistic and alexithymic traits.

In Experiment 9 a joint exploratory factor analysis was conducted of the AQ-50 and TAS-20 items in a group of neurotypical individuals as well as in a group of clinical participants with autism and other conditions. Networks using both AQ-50 and TAS-20 items for both groups (N = 931) were also estimated. In Experiment 10, additional data from 849 new participants was utilised to conduct a confirmatory factor and network analysis based on the results of

Experiment 9, before pooling the data for comparable samples across studies (N = 1571) for confirmatory analyses. Despite the positive association between alexithymic and autistic traits (Kinnaird et al., 2019), the shared variance is often small (less than 30%). Therefore, it was hypothesised that both approaches would separate autism and alexithymia, suggesting they are distinct conditions and reflect distinct underlying latent causes.

5.2 EXPERIMENT 9. Exploratory Factor and Network Analysis of Autism and Alexithymia Constructs

5.2.1 Methods

5.2.1.1 Participants

Data was gathered from 1138 participants recruited for a larger project. There was an especially large number of non-binary (individuals identifying as neither male nor female) autistic participants, a proportion thought to be non-representative of the autistic population as a whole (Murphy et al., 2020). As a consequence, analyses were conducted both with and without this subset of participants, with consistent results. After accounting for missing data, the full set of participants reported here comprised 931 (50% female) participants, of whom 522 reported no mental health conditions. Of the remaining 409 participants self-reporting a clinical diagnosis, 122 reported a diagnosis of autism, 287 reported another clinical diagnosis (63 depression and anxiety, 34 depression, 22 anxiety, 20 gender dysphoria), and the remaining 148 reported other conditions and combinations of two or more conditions, (e.g., a mix of eating disorders, personality disorders, ADHD, OCD, and substance use). The inclusion of clinical participants, particularly those with autism, ensured that the full range of autism and alexithymia traits was captured. However, while a proportion (approximately 35%) of participants reporting an autism diagnosis were recruited from a volunteer database with independent confirmation of their diagnosis, the majority of participants were recruited online and their diagnosis could not be confirmed. As the clinical sample was heterogeneous,

with autistic people on average reporting three other co-occurring conditions, all clinical participants were grouped together. The average age of the participants was 29 years (SD = 12.03). The clinical group was slightly older (M_{age} = 30.73, SD = 11.29) than the neurotypical group (M_{age} = 28.45, SD = 12.26, $t_{(717)}$ = 2.30, p < .02, *d* = 0.19).

5.2.1.2 Instruments

Given the focus of this work on exploring measures of autism and alexithymia using psychometric approaches, I will recap the key aspects relating to the two questionnaires used here, the AQ-50 and TAS-20.

Autism Spectrum Quotient – AQ-50

The AQ-50 assesses levels of autistic traits. It was originally thought to have five dimensions: *social skills (SS), communication (COM), imagination (IMG), attention to detail (ATD)* and *attention switching (AS).* Items are scored on a four-point scale (maximum score 200 as there are 50 items). Confirmatory studies of the factor structure have been inconclusive, psychometric properties are, however, acceptable (Ruzich et al., 2015). In the current sample, using the original five factor structure, internal reliability ranged from .66 to .83 for individual subscales, and was .89 for the entire scale.

Prior to jointly estimating the factor and network structures for both questionnaires, a confirmatory factor analysis (CFA) was conducted on the AQ-50 to test the original factor structure (Baron-Cohen et al., 2001) as well as several other proposed factor structures (English, Gignac, Visser, Whitehouse, & Maybery, 2020; Hoekstra, Bartels, Cath, & Boomsma, 2008). The original five factor structure underperformed compared to more parsimonious solutions (see **8.4.3 CFA of Individual** Measures in SM), which is

consistent with previous reports that the AQ-50 contains redundancies that do not improve measurement precision (Lundqvist & Lindner, 2017). In the total sample the average AQ score was 112.52 (SD = 20.84), with the clinical group reporting higher autistic traits (M = 122, SD = 24.30) than the neurotypical group (M = 108.87, SD = 18.06, $t_{(720)} = 13.3$, $p < .001$, $d = .61$).

Toronto Alexithymia Scale – TAS-20

The TAS-20 assesses levels of alexithymic traits. The original structure included 3 factors: *difficulties identifying feelings* (DIF), *difficulties describing feelings* (DDF) and *externally-oriented thinking* (EOT). Each item was scored on a five-point scale (maximum score 100 as there are 20 items). The psychometric properties of the TAS-20 have been consistently reported as adequate to excellent (Sekely, Bagby, & Porcelli, 2018). In the current sample, the internal reliability of the TAS-20 was .87 for the total scale and ranged from .66 to .86 for individual subscales. The CFA on the factor structure of the TAS-20 was best fitted by the originally proposed three-factor solution plus a method factor for reversed items (Bagby, Parker, & Taylor, 2020; Preece et al., 2020). In the total sample, the average alexithymia score was 49.11 (SD = 12.23), with the clinical group reporting higher levels of alexithymia (M = 53.02, SD = 24.30) than the neurotypical group (M = 47.62, SD = 18.06, $t_{(720)} = 5.4$, $p < .001$, $d = 0.25$).

5.2.1.3 Statistical analyses

5.2.1.3.1 Exploratory Factor Analysis (EFA)

An EFA was estimated jointly for both the AQ-50 and TAS-20 using a minimum residual estimation. Because the AQ-50 and TAS-20 items are on different scales, the EFA used the correlation (rather than covariance) matrix.

Factor extraction and rotation

A parallel analysis and an oblique - *promax* rotation was used motivated by previous positive correlations between TAS-20 and AQ-50 scores (Poquérusse, Pastore, Dellantonio, & Esposito, 2018), which was also observed in the current sample ($r_{(720)} = .62$, $p < .001$). Given the large number of variables, a cut-off of .4 was used for factor loadings. Fit indices (LTI, RMSEA) were used to assess the overall factor solution. Group-specific analyses provided similar results and are included in the SM (**8.4.1.1** Exploratory Factor Analysis (EFA) in SM).

Assumption checks

Multivariate normality was assessed by plotting the distribution of all variables. Factorability assumptions were assessed using the Kaiser-Meyer-Olkin (KMO) test and Bartlet test for sphericity (BTS). All items had Measure of Sampling Adequacy (MSA) > .5, ranging from .6 to .98, overall MSA = .93. Similarly, the BTS was also significant, $X^2_{(2415)} = 24415.70$, $p < .001$. This indicates that the item covariance matrix can be simplified using a reduced number of factors.

5.2.1.3.2 Network Analyses

In psychological networks, relationships between symptoms or traits are estimated as undirected networks by means of partial correlations between all variables. The following concepts are required for interpretation: nodes, edges and centrality. Symptoms/traits are termed *nodes*, and the connections between these symptoms/traits are termed *edges*. Nodes (symptoms/traits) can be described in terms of their *centrality*, a measure of how strongly connected a node is to all other nodes. Nodes with more connections are more central, and are

traditionally understood as critical points of influence on other nodes (i.e. changes in a more central node will affect a greater number of other nodes in comparison to a less connected node). The average centrality indicates the interconnectedness of the network. Edges, the connection between two nodes, can be described in terms of their *strength*, which is the size of regularised partial correlation between two nodes conditioned on all other nodes. Thus, two nodes that make an edge are dependent after controlling for all other nodes they are related to in the network (Epskamp & Fried, 2018).

The main advantage of the network approach over the factor approach is that it offers an alternative to the nearly-equivalent and non-unique factor solution problem (van Bork et al., 2017). Importantly, the Gaussian Graphical Models (GGM) used for estimating undirected networks are typically equivalent to the latent factor approach (Golino & Epskamp, 2017), but are uniquely identified, that is, there is only one structure that represent the relation between variables (Epskamp, 2020), whereas multiple equivalent covariance structures can represent the same factor solution. The network approach can therefore provide converging evidence for whether autism and alexithymia are distinct.

Network estimation

A joint network for AQ-50 and TAS-20 items was estimated using a GGM which uses a graphical Lasso regularization method based on Extended Bayesian Information Criteria to minimise spurious connections (Friedman, Hastie,& Tibshirani, 2008; Epskamp & Fried., 2018). Clinical and neurotypical networks as well as a joint network with all data were estimated. The goal was not to interpret specific nodes or edges because questionnaires usually include multiple items that tap onto the same dimension, all with some measurement error. Additionally, the feasibility and validity of specific interpretations like this with

networks of this size are currently debated (Castro, Ferreira, Castro, & Rodrigues, 2019; Fried & Cramer, 2017). Instead, the focus was on assessing the overall structure of the network to test the central question of whether autism and alexithymia are distinct. To visualise the networks, the walktrap algorithm was used, which allows detection of clusters of items (traits) in exploratory graphical analysis akin to the dimensions of factor analysis (Golino & Epskamp, 2017).

Network and node description and inference

The validity of network metrics is dependent upon how stable the network is, since, like any other statistical test, differences may be due to chance and sensitive to statistical power. Bootstrapped 95% confidence intervals (CIs) around edge weights were computed along with centrality stability coefficient (CSC). CSC estimates range from 0-1, with a CSC > .5 indicative of a stable network (Epskamp, Borsboom, & Fried, 2018; Fried et al., 2018). Edge weights difference tests were also conducted to compare specific connections, and centrality difference tests to compare centrality metrics within the networks.

In addition, network centrality was assessed based on *strength* as it is considered to be the most reliable estimate of centrality (Epskamp et al., 2018). In line with recommendations (Haslbeck & Fried, 2017) shared variance of each node with its neighbours was computed using the *mgm* package in R, to assess the absolute level of interconnectedness. This metric can be understood in terms of predictability of one node by other nodes in the network.

Network comparison

To compare networks across different samples, a similarity measure was computed by correlating the ordered edge weights from both networks (Fried et al., 2018). Second, a

Network Comparison Test was used (Van Borkulo & Boschloo, 2017), a permutation-based test which allows comparison of networks on three aspects: network invariance, edge invariance and global strength. The network structure invariance analyses test for a difference in overall structure (rather than individual connections) between two networks. The edge invariance test tests the null hypothesis that all edges are exactly identical in two networks. Edge invariance was tested using Bonferroni-corrected pairwise comparison tests to examine how many edges differed across the networks. The third test - global strength comparison - tests the null hypothesis that both networks have the same degree of absolute interconnectedness. Because of the large number of nodes and edges estimated in the joint network for autism and alexithymia traits, which may reduce statistical power, the three steps above were repeated for a network analysis based on factor scores derived using the original factor structures for these questionnaires (see Factor Score Networks in SM - **8.4.1.2**) which yielded results consistent with those reported in the main text.

5.2.2 Results

5.2.2.1 Exploratory Factor Analysis

Removed items

For the TAS-20, three items did not reach the factor loading threshold. All items belonged to the EOT subscale. For the AQ-50, 23 items failed to reach the factor loading threshold. The majority belonged to the attention-related factors of the AQ-50 scale (*AS* and *ATD*; see **Table 8.9** in SM for details).

Factor loadings

Factor reduction suggested solutions ranging from 5 to 8 factors, where autism and alexithymia traits loaded on entirely separate factors with a final solution of 6 factors (see

Table 5.1). The first factor contained items assessing *social interests and abilities (SOC)* from the AQ-50 and explained about 9% of the variance. The second factor contained only TAS-20 items focused on *identifying* and *describing feelings and sensations (FEE)* and explained 8% of the variance. The third factor contained items assessing *flexibility (FLX)* in behaviour and interests, mostly consisting of *communication* and *attention switching* items from the AQ-50 and explained 5% of the variance. The fourth factor contained *externally-oriented thinking (EOT)* items from the TAS-20 and explained 4.5% of the variance. The 5th factor exclusively contained items belonging to the *imagination (IMG)* subscale of the AQ-50, and explained 3.6% of the variance. The final factor explained only 2.7% of the variance and contained items belonging to the *attention to detail (ATD)* subscale of the AQ-50.

The final solution explained 34% of the variance, and showed an acceptable fit (RMSEA = 0.042, TLI = 0.834). As the first extracted factor explained less than 30% of the total variance, this suggests that the solution does not represent a unidimensional latent measure (Slocum, 2011).

Table 5.1 Factor loadings.

		Factor						
Items	**Description**	1	2	3	4	5	6	**UN**
TAS_1	I am often confused about what emotion I am feeling.		0.755					0.383
TAS_2	It is difficult for me to find the right words for my feelings.		0.769					0.332
TAS_3	I have physical sensations that even doctors don't understand.		0.429					0.756
TAS_4	I am able to describe my feelings easily.		0.671					0.402
TAS_6	When I am upset, I don't know if I am sad, frightened, or angry.		0.65					0.509
TAS_7	I am often puzzled by sensations in my body.		0.51					0.635
TAS_8	I prefer to just let things happen rather than to understand why they turned out that way.				0.451			0.747

TAS_9	I have feelings that I can't quite identify.		0.799				0.404
TAS_10	Being in touch with emotions is essential.			0.529			0.642
TAS_11	I find it hard to describe how I feel about people.		0.529				0.547
TAS_12	People tell me to describe my feelings more.		0.453				0.641
TAS_13	I don't know what's going on inside me.		0.737				0.393
TAS_14	I often don't know why I am angry.		0.492				0.632
TAS_15	I prefer talking to people about their daily activities rather than their feelings.			0.533			0.599
TAS_17	It's difficult for me to reveal my innermost feelings, even to close friends.		0.414				0.596
TAS_18	I can feel close to someone, even in moments of silence			0.404			0.762
TAS_19	I find examination of my feelings useful in solving personal problems.			0.492			0.701
AQ_1	I prefer to do things with others rather than on my own.	0.506					0.725
AQ_3	If I try to imagine something, I find it very easy to create a picture in my mind.				0.602		0.709
AQ_6	I usually notice car number plates or similar strings of information.					0.547	0.502
AQ_7	Other people frequently tell me that what I've said is impolite, even though I think it is polite.		0.501				0.662
AQ_8	When I'm reading a story, I can easily imagine what the characters might look like.				0.653		0.586
AQ_11	I find social situations easy.	0.836					0.275
AQ_13	I would rather go to a library than a party.	0.661					0.636
AQ_14	I find making up stories easy.				0.651		0.606
AQ_15	I find myself drawn more strongly to people than to things.	0.506					0.629
AQ_16	I tend to have very strong interests which I get upset about if I can't pursue.		0.533				0.644
AQ_17	I enjoy social chit-chat.	0.82					0.406
AQ_18	When I talk, it isn't always easy for others to get a word in edgeways.		0.48				0.814
AQ_22	I find it hard to make new friends.	0.74					0.497
AQ_23	I notice patterns in things all the time.					0.33	0.485
AQ_26	I frequently find that I don't know how to keep a conversation going.	0.546					0.465
AQ_29	I am not very good at remembering phone numbers.					0.558	0.71
AQ_34	I enjoy doing things spontaneously.	0.547					0.623

AQ_35	I am often the last to understand the point of a joke.		0.42			0.713
AQ_38	I am good at social chit-chat.	0.787				0.359
AQ_39	People often tell me that I keep going on and on about the same thing.		0.611			0.634
AQ_40	When I was young, I used to enjoy playing games involving pretending with other children.			0.45		0.761
AQ_44	I enjoy social occasions.	0.895				0.362
AQ_46	New situations make me anxious.	0.547				0.54
AQ_47	I enjoy meeting new people.	0.763				0.471
AQ_49	I am not very good at remembering people's date of birth.				0.522	0.75
AQ_50	I find it very easy to play games with children that involve pretending.			0.429		0.774

Notes: 1: Social skills (SOC); 2: Feelings and sensations (FEE), 3: Flexibility (FLX); 4: Externally oriented thinking (EOT), 5: Imagination (IMG) and 6: Attention to detail (ATD); Uniqueness (UN).

Factor characteristics

All extracted factors showed small to strong positive intercorrelations, except for *ATD* which showed small positive to negative correlations with the other factors (see **Figure 5.1**). The presence of correlations that are close to zero, or negative, again suggests that the extracted solution is unlikely to be unidimensional.

Reliability analyses

Internal reliability for the factors was computed separately for both groups (neurotypical and clinical). In the neurotypical sample, for the alexithymia items, reliability scores were: *FEE* α = 0.89 [.88, 0.91], *EOT* α = 0.64 [0.59, 0.69], and global reliability α = 0.87 [0.88, 0.90]. For the autism subscales, reliability was as follows: *SOC* α = 0.9 [.89, 0.91], *FLX* α = 0.65 [0.61, 0.7], *IMG* α = 0.67 [0.63, 0.72], *ATD* α = 0.65 [0.6, 0.69], with global reliability α = 0.84 [0.82, 0.86]. The reliability for all items combined (across both scales) was α = 0.9 [0.89,

0.91]. For the clinical group, reliability scores were similar, with alexithymia subscale reliability ranging from .50 to .91, autism scales ranging from .65 to .93, and global reliability for both scales α = 0.93 [0.92, 0.95].

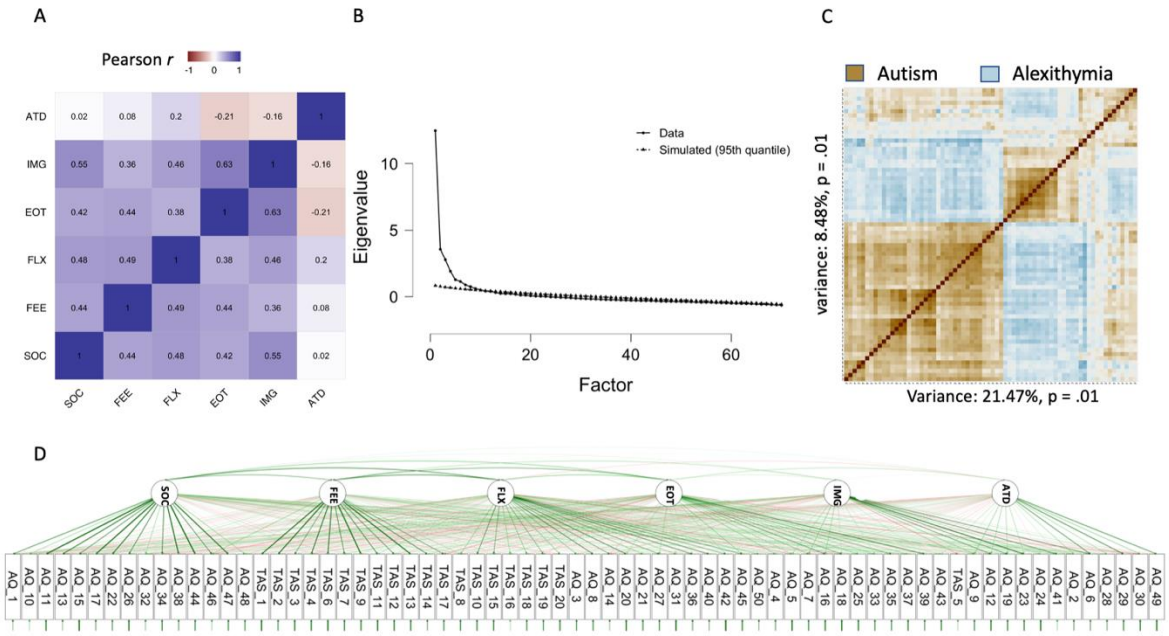

Figure 5.1 Extracted factors, clusters and factor correlations.

Notes. A: Heatmap of factor intercorrelations: most factors showed small to moderate positive correlations, apart from ATD. SOC = Social Skills; FEE = Feelings and sensations; FLX = Flexibility; EOT = externally oriented thinking, IMG = Imagination, ATD = attention to detail. B: Scree plot of the factor solution. Solid line represents real data, dashed line depicts simulation from parallel analysis suggesting a 5 to 7 factor solution. C. A PCA based clustering representation autism and alexithymia traits. C. Path diagram: strongest connections for each factor contain either autism or alexithymia traits, not both.

Exploratory Factor Analysis: Results summary

The results of the exploratory factor analysis were incompatible with the idea that alexithymia is a product of autism or that it reflects the same condition. Results did not support a single latent factor, and alexithymia and autism traits (i.e. TAS-20 and AQ-50

items) loaded onto entirely separate factors. The factor solution was reliable, and the model had an acceptable fit.

5.2.2.2 Network Analyses

Network estimation

The estimated networks for the neurotypical, clinical and combined groups are visualised in **Figure 5.2.** Descriptively, the estimated networks produced on average 7 clusters, which separated autism and alexithymia items in a similar manner to the factor analysis. All networks were largely comparable, and so for brevity I will focus on the neurotypical network as it is better powered and less heterogeneous, and will be used for replicability analyses in Experiment 10. In this network, Cluster 1 included mostly *attention to detail* items from the AQ-50. Cluster 2 included AQ-50 items which tended to be those excluded from the final solution in the factor analysis, made up of a mixture of items from *attention switching* to *communication*. Cluster 3 perfectly aligned with the *feelings and sensations* factor extracted in the factor analysis consisting of TAS-20 items only. Similarly, Cluster 4 aligned perfectly with the *social interests and abilities* factor extracted in the factor analysis, made up exclusively of AQ-50 items. Cluster 5 included the *EOT* factor of the TAS-20 and Cluster 6 included the *imagination* traits from the AQ-50. The clusters had no overlap of autism and alexithymia traits, consistent with the suggestion that the two conditions are distinct.

Network stability

Network stability was assessed by randomly dropping cases (participants) and nodes (traits) and computing correlation coefficients for centrality indices with the original sample.

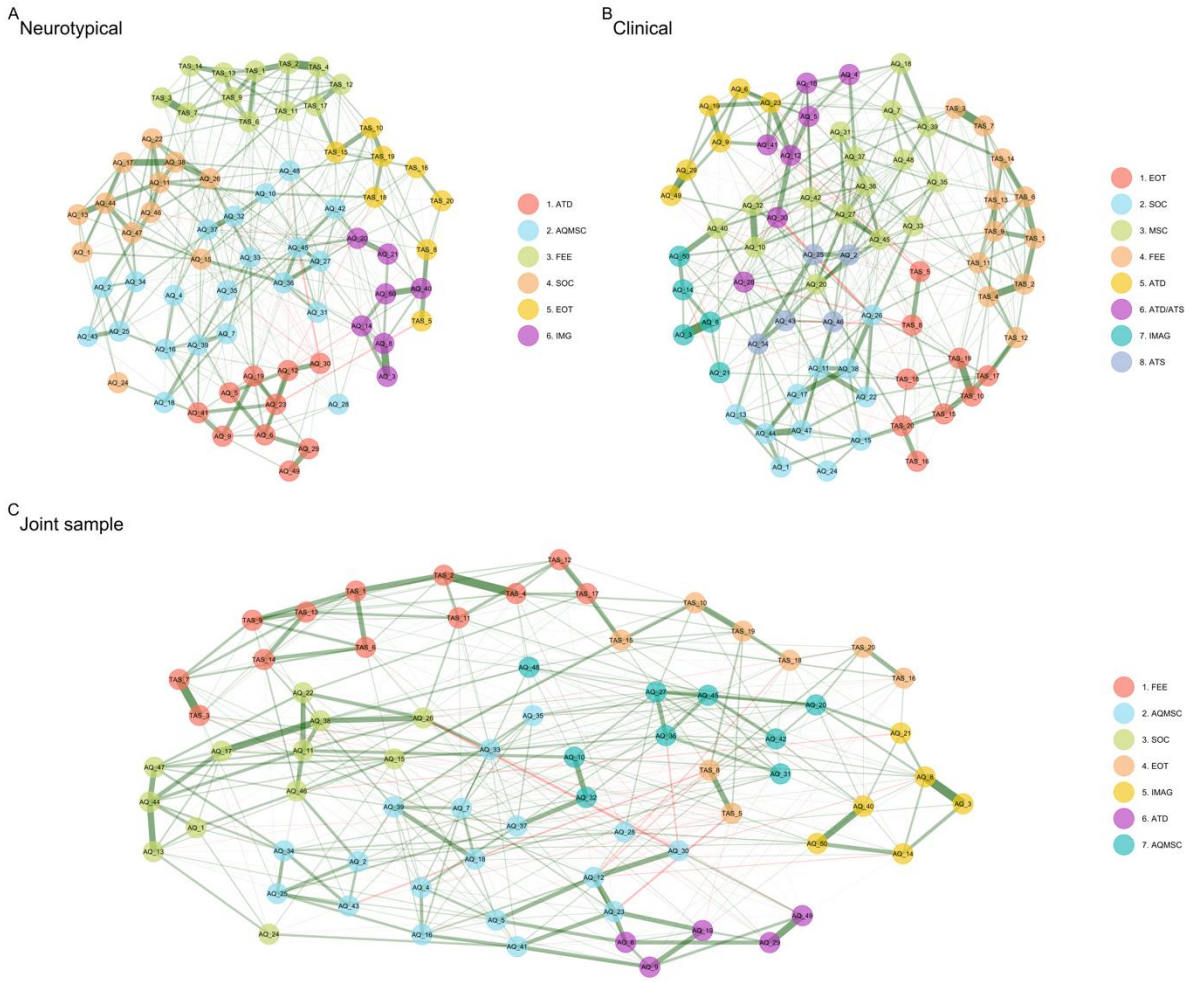

Figure 5.2 Exploratory Graph Networks for Alexithymia and Autism Traits.

Notes. Each colour represents a 'cluster' of connected items within the network. All networks separated autism and alexithymia into different clusters, consistent with the results of the Exploratory Factor Analysis. FEE = Feelings and sensations; AQMSC = Miscellaneous autistic traits including social, communication and imagination; ATD = Attention to detail, ATS = Attention switching, SOC = Social skills and interests, COM = Communication; EOT= Externally oriented thinking, IMAG = Imagination.

These results showed that the neurotypical and jointly-estimated network were reliably estimated, with a CSC of .52 and .59 respectively, greater than the recommended cut-off of .5.

However, the clinical network was unstable, with a CSC of .13, likely due to reduced statistical power, and therefore a factor network was estimated which was sufficiently powered and produced results consistent with those reported above (see Factor Score Network Analysis in **8.4.1.2** SM).

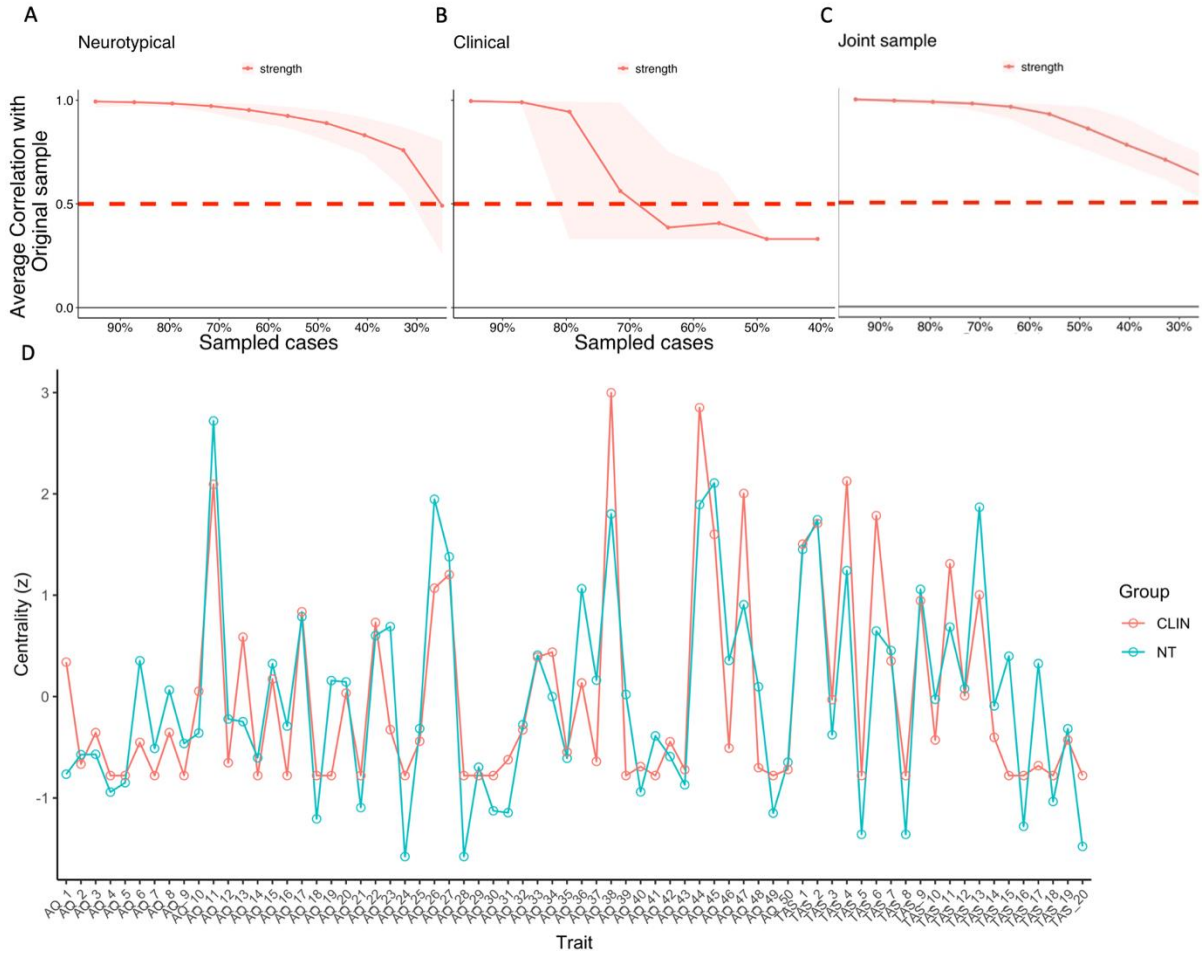

Figure 5.3 Network Stability Plots.

Notes. A shows the correlation stability coefficient (the average correlation between the full sample and a sub-sample created through resampling - y axis) as a function of the percentage of cases (participants) retained in the sub-sample (x axis). The neurotypical network was more reliable than the clinical network. B. Centrality plot showing standardised node strength (the degree of interconnectedness of a trait/symptom). Clin = Clinical sample; NT = Neurotypical sample.

Network comparisons

Because of the large number of items it is not feasible to focus on interpretation of specific nodes and edges. Instead, centrality estimates were computed and are visualised in **Figure 5.3.** Centrality order was highly correlated across networks, .82 for *clinical* vs. neurotypical, and .70 for *clinical* vs *joint sample*; and .89 for *neurotypical* vs *joint sample*. This means that the order of the most central (interconnected) items was relatively consistent across networks. Node predictability (an index of network connectivity) was higher in the *clinical* (.47) than *neurotypical* (.20) network, with nodes sharing on average 34% of variance (i.e., the amount variance in ratings for each autistic or alexithymic trait, was explained by neighbouring nodes).

The correlation between edge weight matrices (the strength of trait connections) was .42 for *clinical* vs *joint network*, .82 for *neurotypical* vs *joint network* and .6 for *clinical* vs. *neurotypical*. These values indicate relatively strong similarity across networks.

For the Network Comparison Test, the null hypothesis of structural invariance, that is, that both networks (clinical and neurotypical) are identical, was not rejected ($M = .25$, $p = .64$). There were also no significant differences in global strength between neurotypical and clinical networks ($S = 21.84$, $p = .33$), indicating that they have a similar degree of interconnectedness. When testing for edge invariance (i.e. that each pair of node connections are equivalent across networks), none of the edges reached significance after Bonferroni correction. As stated above, network estimation for the clinical group and the group comparison may be underpowered given the large number of traits in the network, which also impacts the sensitivity of the NCT. Therefore, therefore analyses 1 to 3 were repeated using factor scores rather than individual items scores. Overall, the results were consistent. These results suggest that neurotypical and clinical networks can be considered structurally

identical, and the separate clustering of alexithymia and autism variables is consistent with the suggestion that they are distinct conditions.

Network Analysis Results summary

The results of the network analysis were consistent with the EFA in that autistic and alexithymic traits were separated into distinct clusters, and the nature of those clusters broadly mapped onto the factors identified in the factor analysis. The neurotypical only, clinical, and joint networks were largely comparable, as were networks constructed on factor scores to guard against low statistical power.

5.2.3 Discussion

Both the factor and network analyses suggested that autistic and alexithymic traits cluster separately, despite being positively correlated. The factor analysis suggested separate factors made up of exclusively autistic or alexithymic traits. The explained variance from each factor, and factor intercorrelations, suggests a multidimensional solution rather than a unitary structure. Networks of both items and factor scores were consistent with the factor analysis and supported strong independence of autism and alexithymia. The results of Experiment 9 therefore support the claim that autism and alexithymia are distinct.

5.3 EXPERIMENT 10. Confirmatory Factor and Network Analysis

Experiment 10 aimed to confirm the factor and network structures estimated in Experiment 9, specifically the separation of autism and alexithymia dimensions at both a latent level and in terms of the relationship between traits in a joint network. Based on Experiment 9, for the confirmatory factor analysis it was predicted that a factor structure of autistic and alexithymic traits as separate latent underlying constructs would fit the data better than a unitary factor

structure. For the network analysis, it was hypothesised that alexithymic and autistic traits would cluster separately, and expected to replicate the network structure from Experiment 9.

5.3.1 Methods

5.3.1.1 Participants

A total of 849 (70% female) neurotypical participants completed the AQ-50 and TAS-20 questionnaires (see Methods in Experiment 9). Participants were on average 28 years old (SD = 9.67) and did not differ significantly from the neurotypical sample in Experiment 9 in terms of age ($t_{(1362)}$ = .455, p = .65), or alexithymia scores ($t_{(1369)}$ = 1.23, p = .22). Experiment 10 participants scored slightly higher than neurotypical participants in Experiment 9 ($t_{(1369)}$ = -2.84, p = .005, d = -.16) on the AQ-50, but lower than the clinical group in Experiment 9 ($t_{(1069)}$ = -.6.33, p < .001, d = 0.52). TAS-20 and AQ-50 showed a medium-sized positive correlation ($r_{(847)}$ = .48, p < .001), lower than in Experiment 9 (z = -3.62, p < .001).

5.3.1.2 Confirmatory Factor Analyses

A confirmatory factor analysis was conducted in R using the lavaan package (v.0.6-6). Eight models were fit to distinguish between unitary or distinct factor structure(s) underlying autistic and alexithymic traits as measured by the AQ-50 and the TAS-20, respectively. Full model details are given in SM (Model Specification in **8.4.2.1** SM) but three families of models were tested (See **Figure 5.4**).

Models in Group A were based on the six-factor solution obtained in the joint factor analysis of the AQ-50 and TAS-20 in Experiment 9. Models in Group B were based on the original factor structures for each questionnaire (5 factor solution for the AQ-50: *social skills*, *communication*, *imagination*, *attention to details* and *attention switching* (Baron-Cohen et al.,

2001), and 3-factor solution for TAS-20: *difficulties identifying feelings*, *difficulties describing feelings* and *externally oriented thinking* (Bagby, Parker, & Taylor, 1994). Models in Group C were based on the best performing factor solutions identified in meta-analyses and reviews (English et al., 2020), which were also the best preforming models for the individual scales in Experiment 9 (see **8.4.3 CFA of Individual** Measures– SM). The fitted models included three factors for the AQ (*social*, *communication* and *attention*), and four factors for the TAS-20 (*DIF*, *DDF*, *EOT* and a method factor for reversed items; Preece et al., 2020; Watters, Taylor, Ayearst, & Michael Bagby, 2016) - see **Figure 5.4.**

Within each family of models, the following models were compared: 1) distinct correlated factors; 2) autism factors and alexithymia factors are driven by distinct latent causes (i.e. autism and alexithymia, respectively); or 3) a common latent factor gives rise to autism and alexithymia.

5.3.1.2.1 Model assessment and comparison

Fit indices including CFI, TLI and RMSEA were used to assess model properties. A Likelihood Ratio Test was used to compare nested models and AIC and BIC were used in addition to fit indices for non-nested models.

5.3.1.3 Network Analysis

Network analysis was conducted as in Experiment 9. To confirm the results of Experiment 9, the network obtained in Experiment 9 was compared to that obtained using data from Experiment 10, and also confirmed using data pooled across Studies (N = 1571).

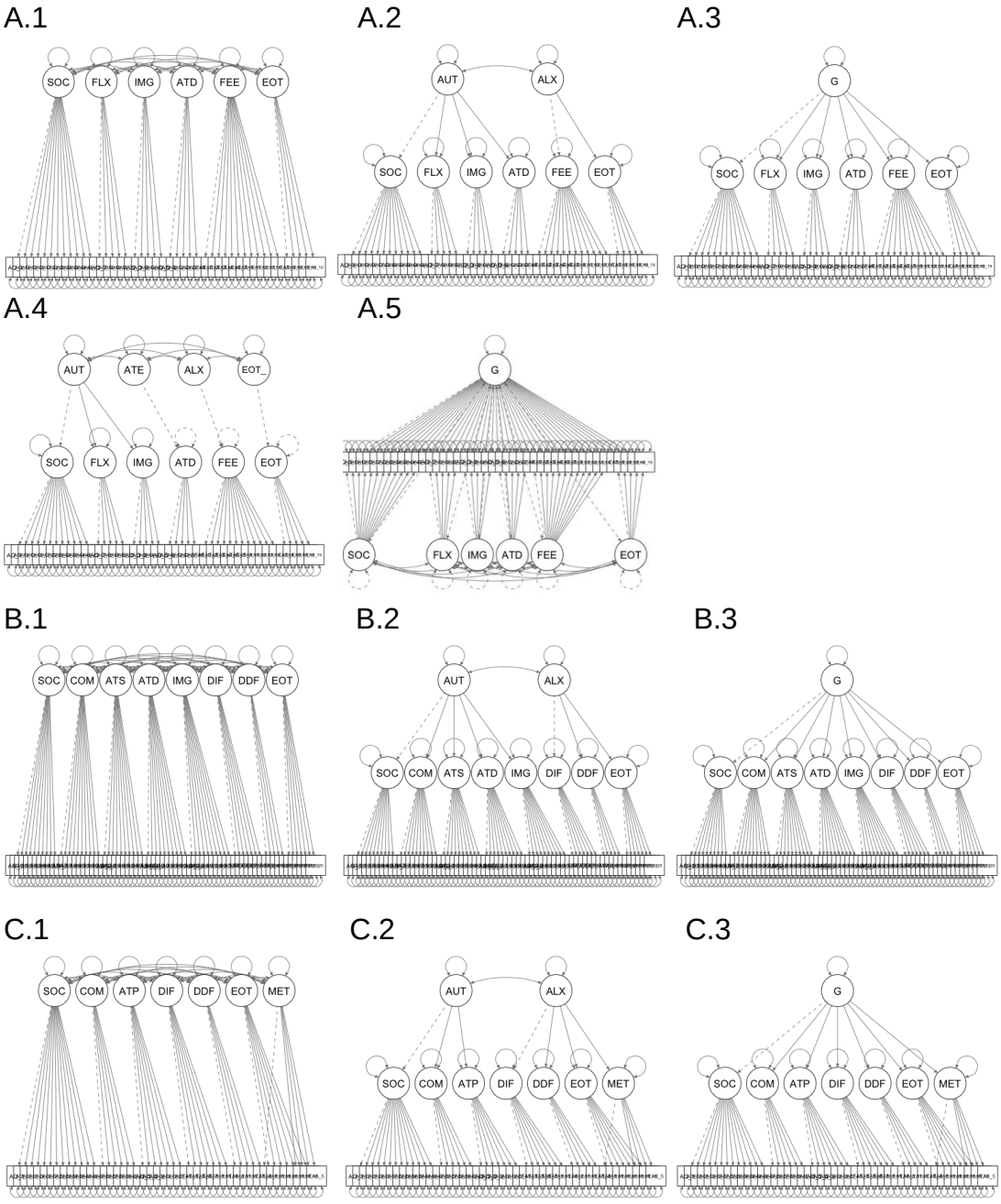

Figure 5.4 Graphical representation of confirmatory models

Notes. Representation of the models fitted in the confirmatory factor analysis of the AQ-50 and TAS-20. A models were based on Exploratory Factor Analysis solution in Experiment 9, B models were based on the original factor structures of each questionnaire and C models were based on proposed alternative solutions to the original factor structures.

5.3.2 Results

5.3.2.1 Confirmatory Factor Analysis

Results were consistent across all three model families. In each group, the best performing model was the one in which the factors of autism and alexithymia were separate, or with separate latent causal factors. Summary statistics of model comparisons (where appropriate) are provided here, and full details of fit indices and model comparisons are provided in SM (**Table 8.11**).

From Group A the best performing model was Model A.1, which contained a six-factor correlated solution. The model fit was acceptable (CFI = 0.80, and RMSEA of .05 90% CI (.53, .57), p < .001). Neither model A.2, ($\chi^2_{(8)}$= 99.53, p <.001) nor A.3 provided a better fit to the data ($\chi^2_{(9)}$= 104.44, p<.001). Model A.4 showed poor fit and model A.5 failed to converge.

From Group B, the best performing model (B.1) specified distinct correlated factors for autism and alexithymia, as opposed to second-order models (B.2 and B.3). However, Group B models showed poor fit indices and were therefore not considered further.

From Group C, model C.1, specifying a seven-factor solution with no higher-order terms, was the best performing model. This model showed acceptable fit with CFI of 0.841, RMSEA of .50 [.48,.52], ns. Model C.1 outperformed model C.3 ($\chi^2_{(14)}$ = 141.83, p < .001). Model C.2 showed negative variances and therefore was not considered further.

For Model A.1 (and all best-fitting models in each group), all items showed significant positive factor loadings, with standardized coefficients ranging from .1 to .83 (see **Table 8.11** in SM). There were also significant positive correlations among 4 of the 6 factors (*social skills and interests*, *feelings and sensations*, *flexibility* and *imagination*, ranging from

.11 to .48). This indicates that participants' scores are likely to correlate positively on those dimensions. However, as shared variance for all cases is less than 20%, it suggests that there is little overlap in the measured constructs. In sum, these results support the proposal that autism and alexithymia are distinct.

5.3.2.2 Confirmatory Network Analysis

Network estimation

Figure 5.5 depicts the network estimated in Experiment 10, and the jointly-estimated network derived from Studies 1 and 2. As can be seen, in Experiment 10, as in Experiment 9, autism and alexithymia traits clustered separately. There were five clusters identified in Experiment 10, rather than the six identified in Experiment 9. Nonetheless, alexithymia clusters were mostly the same as in Experiment 9, with *feelings and sensations (FEL)* items clustering together (Cluster 1). However, unlike in Experiment 9, three EOT items clustered with the *FEL* items, with the rest of the EOT items clustering together (Cluster 5).

AQ items produced three clusters: Cluster 2 contained a mixture of social, communication scales and imagination items, Cluster 3 consisted mainly of the *attention-related* items identified in Experiment 9, and Cluster 4 mostly contained items assessing *social skills*. Overall, alexithymia and autism clusters showed positive correlations, within and between clusters, but the autism cluster contained some negative correlations from the autism miscellaneous Cluster 2 to the attention-related Cluster 3. The neurotypical network from Experiments 9 and 10 also produced a similar network. Across studies, alexithymia clusters were more consistent than autism clusters.

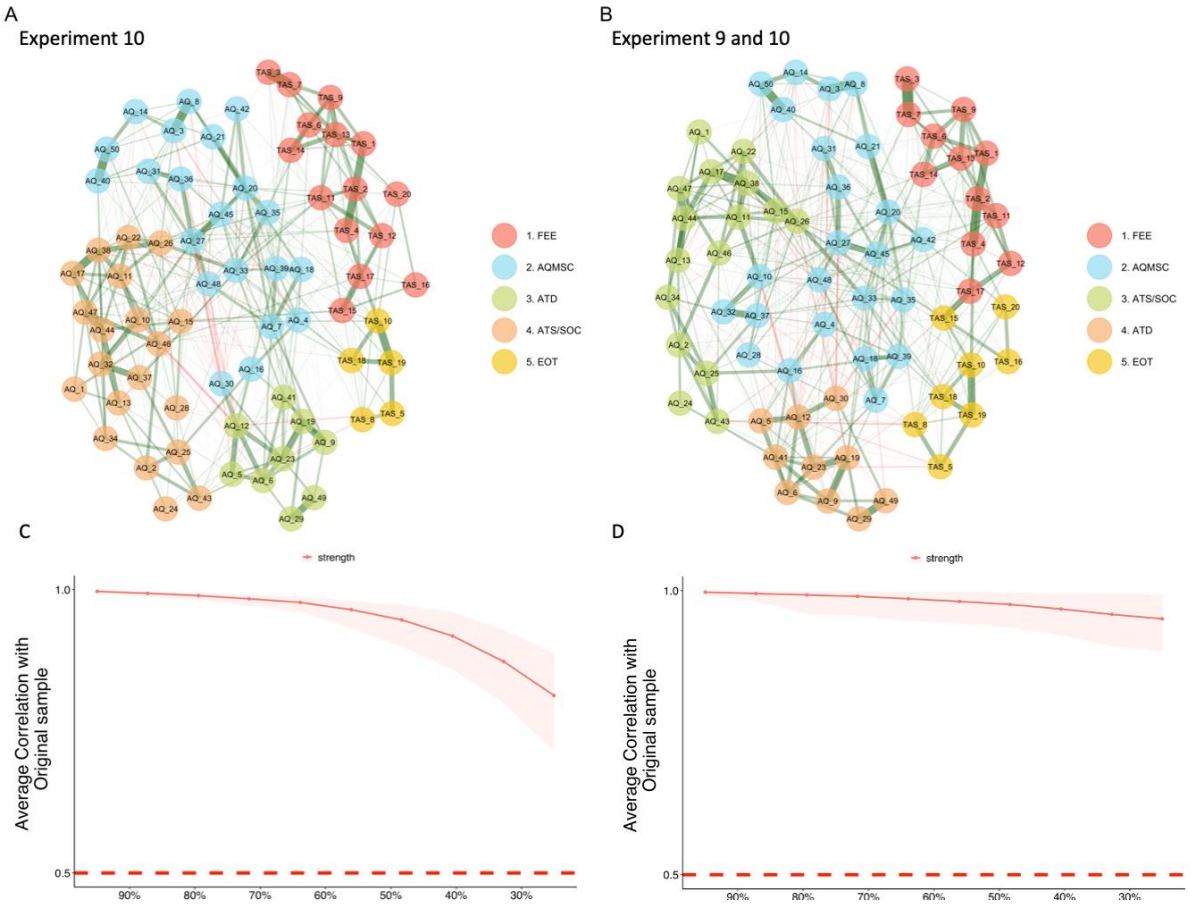

Figure 5.5 Estimated networks – Experiment 10

Notes. Network from Experiment 10 (A) and pooled network combining neurotypical samples from Experiment 9 & 10 (B). Each colour represents a cluster of traits. C & D show the correlation stability coefficient - the correlation of centrality indices between the full sample and sub-samples across various sub-sample sizes. Values > .5 suggest stable and reliable networks. FEE = Feelings and sensations; AQMSC = Miscellaneous autistic traits including social, communication and imagination; ATD = Attention to detail; ATS = Attention switching; SOC = Social skills and interests, EOT= Externally oriented thinking.

Network inference and stability

Centrality measures were in general consistent with Experiment 9 and are presented in SM (**8.4.2.2**). Network bootstrapping demonstrated that the network was stable with a CSC of .67 (greater than the recommended value of .5).

Network comparison

There was a strong similarity between the network structures from Experiment 9 & 10, with a correlation between edge weight matrices of .83. The NCT indicated no significant differences in network invariance (structure) M = 0.16, p = .37, nor in global strength (the average strength of the connections) S = 3.78, p = .84. There was also only 1 significant difference in edge invariance in the network (less than 1%). Together, these results suggest that the estimated networks are largely similar. As in Experiment 9, networks estimated using factors were also consistent with a separation of alexithymia and autism (See **8.4.2.2** in SM).

5.3.3 Discussion

Experiment 10 confirmed that alexithymic and autistic traits are best characterised as distinct. In the CFA, models specifying a common latent structure fit the data poorly. Of note is that models that defined separate higher-order factors (of autism and alexithymia) did not fit the data significantly better than a model with no higher order factors (i.e. where each sub-factor of the AQ-50 and TAS-20 was independent). Results of the network analysis replicated across studies; autism and alexithymia traits again clustered separately, supporting the claim that autism and alexithymia are distinct conditions.

5.4 General Discussion

Socioemotional difficulties have long been considered a hallmark of autism (APA, 2013; Guastella et al. 2010; Du Bois et al. 2014), but it has recently been argued that any socioemotional difficulties in the autistic population are caused by co-occurring alexithymia (Bird & Cook, 2013). For this account to be logically coherent, autism and alexithymia must be distinct conditions, yet it has been claimed that alexithymia is a product of autism (Quattrocki and Friston 2014, Gaigg, 2012; Ben-Shalom et al., unpublished communication). This series of studies therefore sought to examine whether alexithymia should be considered a consequence of autism, or distinct from it. Results support the argument that alexithymia and autism are distinct. Experiment 9 used factor analytic and network approaches to assess responses to the most widely-used self-report measures of autism and alexithymia and found distinct autism and alexithymia factors and clusters. Experiment 10 used confirmatory methods to show that all models assigning a unitary latent factor common to autistic and alexithymic traits fitted the data poorly in comparison to both multidimensional models and a model specifying distinct latent sources of covariance for autism and alexithymia factors.

Network analyses again supported the independence of autism and alexithymia.
The results from both studies are consistent with previous reports showing double dissociations between effects of autism and alexithymia (Bernhardt et al., 2013; Desai et al., 2019; Mul et al., 2018). The independence of autism and alexithymia has important implications for research and clinical practice. For research, results suggest we need to rethink models that attempt to account for emotional difficulties in autism without considering the role of alexithymia. Although autism and alexithymia are not the same construct, the increased prevalence of alexithymia in autism may be crucial for understanding increased vulnerability to emotional problems (e.g., poor emotion regulation) in autism. For

clinical practice, these results suggest a need for assessment of socio-emotional abilities in general, and alexithymia specifically, when working with autistic individuals.

The assessment of the AQ suggests that the measurement of autistic traits needs improvement. While the key dimensions of alexithymia were reliably identified across analyses, the same was not true for autism using the AQ, a finding consistent with previous studies (English et al., 2020).

The focus on the measurement level in this study (using the AQ-50 and TAS-20) represented a practical solution to the conceptual problem of potential autism/alexithymia overlap, but it could be considered a limitation of the study. Rather than using self-report questionnaires, symptom/trait severity could be assessed using diagnostic interviews or performance on objective tests (cognitive, affective, etc).

Another potential limitation is the use of a cross-sectional adult sample in this study. Future studies could use dynamic network models based on longitudinal data which could inform causal models of how autistic and alexithymic symptoms are related (Epskamp, Waldorp, Mõttus, & Borsboom, 2018). Developmental studies of this kind would be especially useful, allowing the relationship between alexithymic and autistic traits to be tracked over time. Such work would also allow the exploration of the multiple possible developmental routes for alexithymia outlined by Hobson et al (2019), particularly whether alexithymia may be causally related to language impairments in a sub-sample of individuals.

It should also be acknowledged that the clinical sample included in this study was not exclusively of autistic individuals with an independently-verified diagnosis, and included a higher proportion of female participants than might be expected in such a sample. The

average IQ of the samples included in the study may also be considered to be not representative of the autistic population as a whole (Chiang, Tsai, Cheung, Brown & Li, 2014; Bishop, Farmer, Thurm, 2015). As such, it is possible that results may differ in a representative sample of individuals diagnosed with autism.

5.5 Conclusion

Across two studies and using factor analytic and network analyses it was shown that alexithymia and autism are distinct, though they frequently co-occur. Consideration of alexithymia is therefore likely to aid research into the socioemotional abilities of individuals with autism, and to contribute to diagnosis and treatment in clinical practice.

Supplementary Materials: **8.4 Chapter 5. Supplementary Materials**

Open Materials: https://osf.io/5fn8b/

5.6 What comes next?

This chapter investigated whether autism and alexithymia reflect distinct constructs and provided strong evidence of separation of the two conditions at a latent level as well as in the level of their interactions. This result is central to the logical coherence of the alexithymia hypothesis and substantiates the importance of accounting for alexithymia in socioemotional processes in autism. In the next and final chapter, I summarise the key findings of this thesis and draw conclusions, theoretical implications and directions for future research.

6 CHAPTER 6. GENERAL DISCUSSION

In this thesis, I presented a series of experiments that explored the mechanisms underlying socioemotional symptoms in autism. Specifically, this research investigated whether atypical socioemotional processes in autism could be explained by atypical social attention, atypical arousal, deficits in the use of priors, or individual differences in alexithymia; in line with several theoretical models introduced in (Chapter 1- **Theoretical frameworks).** Arguably, most of these models emerged as an attempt to explain specific features of autism, for example, reduced eye contact, reduced emotion recognition, inflexible behavioural tendencies, and hypo-hypersensitivity to sensory stimulation. In the various studies from **Chapters 2** to **Chapter 5**, I explored how these models account for atypical visual social attention, disruption in the experiential aspects of emotion associated with physiological responding, and atypical modulation of gaze behaviour by priors. At the same time, the work presented in this thesis highlighted some consistencies that transcended specific topics and models, with implications for understanding typical socioemotional processes.

This chapter will therefore provide a summary of the various findings and draw conclusions across chapters to offer an integrated account that informs theories of typical and atypical socioemotional processes. Understanding the nature of socioemotional deficits in autism and the mechanisms by which they arise is the prerequisite for translational applications centred around improving assessment and interventions of socioemotional deficits. Therefore, implications for clinical practice will also be highlighted, including future directions to guide research that may ultimately inform translational applications.

Chapters 2 and 3 explored the specificity of autism and alexithymia influences on atypical social attention and emotion processing abilities relevant to face-to-face interactions. **Chapter 4** investigated the physiological and interoceptive mechanisms of emotional responding, and how these may be affected by autism and alexithymia in line with theories of physiological mobilisation in emotion and arousal-based accounts of autism and alexithymia. **Chapter 5** explored the overlap of autism and alexithymia constructs and considered the implications for assessment and subtyping approaches in the face of individual differences and comorbidity.

In the next sections, a thematic summary is provided organising the key findings of the empirical chapters, a summative discussion of implications for theory, research, and clinical practice, future directions, followed by a general discussion.

6.1 VISUAL MECHANISMS OF EMOTION PROCESSING IN AUTISM

Chapters 2 and 3 investigated the outstanding question of whether social attention is atypical in autism, and, if so, whether this reflects alexithymia influences as proposed by the alexithymia hypothesis, or atypical top-down effects as suggested by Bayesian accounts of autism. This work was primarily motivated by the fact that despite atypical eye gaze constituting a key feature of descriptions of autism, eye-tracking studies have provided surprisingly mixed evidence concerning patterns of gaze allocation in autism (Papagiannopoulou et al., 2014). Nonetheless, atypical gaze is key to several models of autism, which implicate atypical gaze behaviour in empathy deficits and socioemotional reciprocity (Hadjikhani et al., 2018; Senju & Johnson, 2009a).

A novel task and methodological approach were used to investigate task modulation effects of social attention, as well as to capture the complex spatiotemporal dynamics of eye movements during emotion processing described in terms of gaze predictability (entropy) and how gaze evolves over time. Additionally, the influence of alexithymia was explored, in line with the alexithymia hypothesis which argues that individual differences in alexithymia may underlie varying profiles of social attention in autism.

6.1.1 Findings

Experiment 1. Moving beyond the traditional aggregate descriptions of gaze allocation to face stimuli, data from this study demonstrated the novel finding that atypical gaze behaviour in autism reflects influences of alexithymia on the spatiotemporal dynamics of visual exploration of emotional information in dynamic face cues. This study highlighted that alexithymia resulted in reduced predictability (increasing entropy) of gaze behaviour, and reduced modulation of visual exploration strategies by task goals and emotion-related priors. As such these results support the alexithymia hypothesis, the view that alexithymia, and not autism, is the primary driver of atypical attention to socioemotional stimuli. Furthermore, the patterns described by entropy metrics of gaze exploration and reduced modulation effects, suggest that the top-down modulation mechanisms proposed in Bayesian models of autism, in fact, better describe the influence of alexithymia on the visual exploration of emotions. These findings, therefore, suggest that the mixed results regarding the nature of social attention deficits in autism may reflect unaccounted effects of alexithymia and a failure to account for task modulation effects on gaze behaviour. These results also indicate that the effects of alexithymia are not limited to perceptual difficulties or difficulties in identifying feelings, also affecting executive attention processes relevant to social processing.

While **Experiment 1** provided strong evidence regarding atypical modulation of gaze behaviour by task and emotion-relevant priors in alexithymia, it did not allow for an equally sensitive test of processing of sensory information related to stimuli dynamics. Bayesian accounts of vision argue that deficits in visual processing might arise from atypical processing of sensory evidence, atypical priors, or their interactions. The stimuli used in **Experiment 1** were limited in their dynamic range of sensory information (they were subtle expressions), which is not representative of the rich dynamic variation in communicative face actions (Jack & Schyns, 2015). Therefore, a proper characterisation of the spatiotemporal dynamics of facial expressions was needed to properly consider how visual information relevant to facial expressions shapes visual processes and compete or interact with top-down priors to guide social attention and perceptual processes. In **Experiment 2 (Chapter 3),** building on the methodological approach from **Experiment 1**, a novel approach was developed to quantify and analyse spatiotemporal dynamics of facial expressions and investigate the impact of these dynamics on typical perceptual judgments of emotion. Results from **Experiment 3**, show that the dynamic properties of facial expressions influence the variability and consistency of perceptual judgments of emotion in observers. This study substantiates the view that these spatiotemporal properties of facial expressions can be used to describe sensory evidence of communicative facial cues, as these dynamics are intrinsically linked to the precision with which various emotional signals are conveyed via face movements. In **Experiment 4 (Chapter 3),** this novel approach to quantify facial expression dynamics is combined with the task modulation paradigm from **Experiment 1**, to provide an additional test of the influence of alexithymia and autism on the modulation of gaze behaviour by task goals, emotion-related priors and stimulus-related dynamics. A novel gaze allocation estimation task was devised to measure social attention unobtrusively in online settings while participants viewed and responded to a wide range of dynamic facial

cues. Results from **Experiment 4** replicated results obtained in **Chapter 2,** showing alexithymia effects on atypical task modulation of gaze allocation to facial regions (eyes, nose, and mouth). This study further indicated that alexithymia was related to an atypical influence of spatial-kinematics of facial expressions on gaze allocation and emotion judgements. These results confirmed the importance of top-down mechanisms to atypical social gaze and emotion processing in alexithymia, but also implicate specific stimulus dynamics influences on emotion processing and social attention. As such these results confirm the contention that atypical socioemotional processing in autism is primarily related to individual differences in alexithymia as suggested by the alexithymia hypothesis, and highlight specific top-down and bottom-up processing mechanisms which are affected by alexithymia.

6.1.2 Theoretical implications

The implications of the results from **Chapters 2 and 3** are twofold. First, findings of task modulation effects on gaze indicate a simple but crucial aspect of visual social attention, which is that social gaze should be evaluated in terms of modulation by task-and-emotion-relevant priors and sensory information rather than absolute patterns of gaze allocation. There is likely no one single universal pattern of gaze that reflects typical social attention to socioemotional information. Instead, gaze reflects the dynamic processing of complex changing information in the world and as such needs to be optimised to solve specific task goals. This explains why unique patterns of gaze allocation are typically only seen in the processing of static facial expressions (Schurgin et al., 2014), and are less consistent to dynamic facial expressions (Yitzhak, Pertzov, Guy, & Aviezer, 2020). This seems intuitive in hindsight, considering that the dynamics of diagnostic information in facial expressions vary

over time (Jack et al., 2014). As such models of social attention should incorporate stimuli-driven influences on gaze behaviour and top-down modulation by task- and stimulus-relevant goals and knowledge.

Results also contribute important details to the emerging alexithymia hypothesis. It seems clear that the characterisation of atypical eye gaze as a core feature of autism needs to be revised to account for the consistent contribution of alexithymia on how gaze strategies are recruited to solve many relevant social processing tasks. The influence of alexithymia on social attention and emotional processes is likely to result from a combination of predictive abnormalities and atypicalities in sensory processing. While the Bayesian accounts tend to contrast these two positions, it's likely that in practice atypicalities in processing sensory aspects of social stimuli may lead to problems learning relevant priors to guide future social attention processes. For example, some studies have implicated alexithymia in the atypical perception of biological motion or metacognitive aspects involving confidence in perceptual ratings of biological motion (Borhani et al., 2016; Rigby, Jakobson, Pearson, & Stoesz, 2020). As such, atypicality in the processing of biological motion in alexithymia may emerge early in development and lead to impaired learning of stimulus-related priors for dynamic cues of emotion, which likely involve representations of biological motion (Furl et al., 2020).

6.1.3 Future directions

6.1.3.1 Testing for precision, congruency, and describing priors

As discussed at the end of **Chapter 3**, formal implementations of Bayesian models of visual exploration may further clarify the source of atypical modulation of social attention by priors and stimulus-related dynamics. For example, approaches relying on dynamic manipulation of the precision of sensory information (e.g. precision of spatiotemporal dynamics of facial

expressions), as well as precision of emotion and task-relevant priors, are needed to test whether atypical social attention reflects imprecise priors, lack of priors, or a failure to update priors. Similarly, precision in spatiotemporal dynamics might vary with emotional expressions, such that some emotions may be more consistently characterised by a certain profile of spatial-kinematics than others.

Exploring the effect of (in)congruency of emotion cues may help test whether alexithymia effects on gaze reflect atypical prediction errors. Both prior updating and prediction errors are likely key to modulate social attention during face-to-face interactions. For example, in conversational interactions subjects need to infer the emotional states of others, predict mental states (e.g. for humour understanding), or judge the degree of interest of the target in the conversation. Similarly, observers might learn idiosyncrasies associated with the expression dynamics of certain people, and therefore use more precise priors (specific to the people they are interacting with) when attending to and interpreting facial cues. Prior updating through tracking of prediction errors is therefore likely the mechanism through which typical social attention is deployed and maintained in successful face-to-face interactions.

Finally, it seems important for future research to explore the specific nature of priors about emotion-related information like facial expressions in alexithymia, for example, whether participants' expectations of spatiotemporal stimuli dynamics are idiosyncratic or systematic within alexithymic individuals. A recent study in this direction allowed participants to manipulate spatial-kinematics of facial expressions to study internal representations of expression dynamics for autistic and non-autistic participants and found no differences when alexithymia was accounted for (Connor Tom Keating, Sowden, & Cook, 2021). Similar approaches analysing the direct impact of alexithymia could therefore reveal whether

representations of spatial-kinematics aspects of facial expressions are atypical in alexithymia and how so, and this may provide insights about gaze exploration depending on the nature of priors associated with dynamic facial cues. Other approaches could also use spatiotemporal caricatures (Furl et al., 2020), or data-driven methods such as reverse correlation and generative models of facial expressions to test whether the perceptual representation of emotion based on spatiotemporal dynamics are invariant in alexithymia (Jack et al., 2014; Jack, Garrod, Yu, Caldara, & Schyns, 2012).

6.1.3.2 Towards interactive assessment of gaze behaviour

While the current study used several tasks and a wide range of face stimuli to assess social attention and emotion processing, the gaze behaviour was not measured during interactions. The degree to which non-interactive gaze generalises to real-world face-to-face interactions remains unclear. However, several recent studies suggest that even implied, or anticipated face-to-face interaction may have important consequences in social attention (Gobel, Kim, & Richardson, 2015; Roy S Hessels, 2020; Laidlaw, Foulsham, Kuhn, & Kingstone, 2011). While the approaches to study dyadic gaze can vary considerably, and some sacrifice important aspects of internal validity, for example by using gaze data of a quality that precludes fine-grained assessment of spatiotemporal dynamics of gaze, recent approaches have been developed which allow for relatively controlled interactions in the lab (e.g. dual eye-tracking) while allowing for the interactive nature of face-to-face communication to unfold (Hessels, Cornelissen, Hooge, & Kemner, 2017).

While the work in this thesis shows a robust contribution of alexithymia to social attention and emotion processing compared to autism, it is still possible that a pure autism phenotype (without alexithymia) may be related to atypical gaze in social interactions, for example due

to social anxiety (Hessels et al., 2017; White, Maddox, & Panneton, 2015). As such, future studies should disentangle whether social anxiety effects on gaze are distinct from the more complex social attention deficits linked to alexithymia, which involve atypical modulation of gaze resulting in inefficient visual exploration due to both stimulus factors and top-down effects on gaze. These studies should also explore how dyadic interactions or implied and anticipated interaction modulate gaze behaviour while accounting for alexithymia and social anxiety in autism studies.

Furthermore, dyadic studies should explore whether gaze modulation effects generalise to interactive settings accounting for task factors (or interaction goals), individual difference factors, and stimulus factors. Some of the approaches used in this thesis can also aid these studies by allowing, for example, the quantification of stimulus dynamics (face movements) and estimating influence on gaze behaviour in a more objective and sensitive manner, for example through a combination of face tracking using video and or motion tracking in addition to eye-tracking – see a suggested setup in **Figure 6.1.**

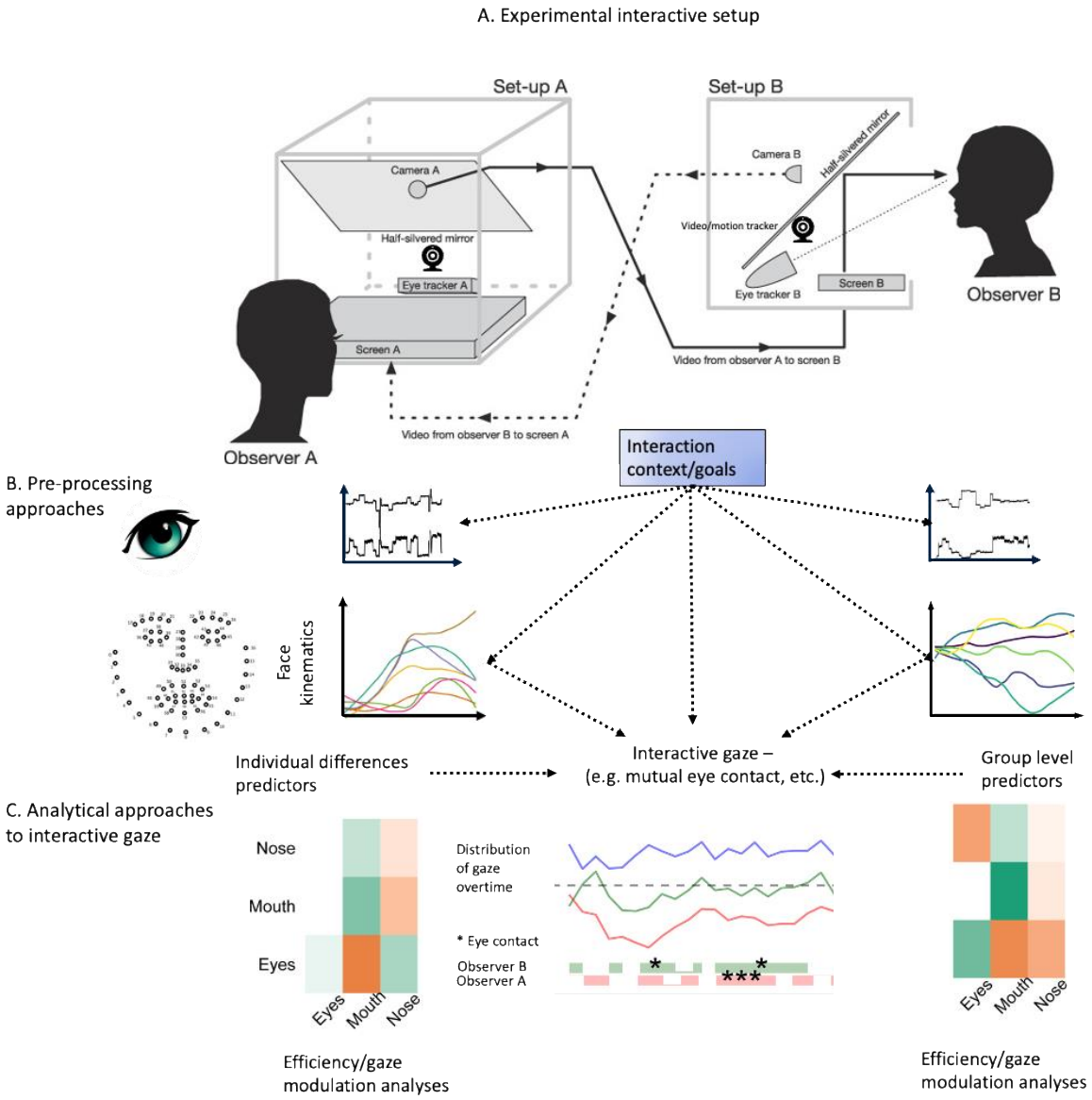

Figure 6.1 Interactive approaches to study social attention.

A: Dual eye-tracking setup validated by Hessels et al., 2017, that allows for interactive eye-contact in a relatively controlled experimental setting. Adapted to include face-tracking using motion or video recording and offline processing (for example using openFace (Baltrušaitis & Robinson, 2016). B & C describe potential analytical approaches based on methods used in this thesis and studies of dyadic gaze interactions.

6.1.3.3 Atypical development of social attention and gaze modulation

As discussed in **Chapters 2** and **3**, the effects of priors on social attention likely reflect experience with previous interactions and probably emerge during development. Children are likely to learn how to track people's emotional and mental states and to learn that various verbal and non-verbal cues (e.g. facial expressions) are more or less informative depending on the situation or the type of social inference they need to make. Stimulus-driven modulation of gaze is likely to emerge earlier during perceptual development and to be supported by the innate sensitivity of the visual system to biological motion (Pavlova, 2012), whereas task-specific gaze modulation or emotion-related priors effects on gaze may emerge later with interactive experience (Gredebäck, Fikke, & Melinder, 2010; Triesch, Teuscher, Deák, & Carlson, 2006). Developmental studies are therefore needed to investigate when does gaze differentiation by stimuli and priors - and their interactions – emerge, and how this developmental trajectory might be linked to alexithymia and autism risk factors.

6.2 PHYSIOLOGICAL AND INTEROCEPTIVE MECHANISMS OF EMOTION IN AUTISM AND ALEXITHYMIA

Chapter 4 set out to investigate the experiential components of emotion which are thought to be atypical in autism and alexithymia. Specifically, arousal-based models of autism claim that atypicality in physiological and interoceptive regulation dynamically modulate socioemotional processes (e.g. experience of emotion, empathy, social attention, emotion recognition) (Cuve et al., 2018; Lydon et al., 2016). Conceptual limitations with prevailing views of physiological mobilisation during emotion, and methodological limitations related to quantification and analyses of physiological response profiles, meant that the exact nature of physiological responding to emotions in autism and alexithymia had not been identified previously.

To address these issues, experiments in **Chapter 4** capitalised on multimodal recording of multiple autonomic signals using a well-established emotion-induction task, combined with novel multivariate approaches to describe the profiles of physiological responses to emotion in autism and alexithymia.

6.2.1 Findings

Experiment 6 in **Chapter 5** revealed that contrary to traditional views of physiological responses to emotions, there is limited coherence between different autonomic signals. This study further explored a novel application of multivariate approaches to analyse multimodal recordings and characterise profiles of physiological responses. Results obtained from this approach showed that physiological responses to emotions reflect at best, multivariate profiles of autonomic mobilisation, which may not be apparent in relationships between emotions and individual autonomic signals. Nonetheless, data-driven clustering, showed that these multivariate profiles of physiological mobilisation loosely mapped onto arousal and valence dimensions.

Building on this approach, **Experiment 7 in Chapter 5** showed that atypical responding to emotions is primarily driven by alexithymia in both autistic and neurotypical participants. These findings also highlight for the first time that this atypicality is best described as a multivariate profile of incomplete or imbalanced physiological mobilisation to emotion, as opposed to generalised hypo- or hyper-arousal. Specifically, alexithymia was related to reduced coactivation of SCR and HR responses, as well as coactivation of pupil responses to positive stimuli. Furthermore, pupil responses to negative stimuli exhibited a unique pattern which did not reflect coactivation of other autonomic modalities (cardiac and electrodermal).

Autism diagnosis and autistic trait predictors did not significantly explain participants' position in the multivariate space representing the profile of physiological responses, nor were they significantly related to univariate patterns of physiological responses to emotion. As such, these results support the view that alexithymia may disrupt socioemotional functioning in autism via physiological and interoceptive routes.

6.2.2 Theoretical implications

The finding that alexithymia is related to atypical multivariate patterns of autonomic mobilisation to emotions has important implications for models of emotions that consider physiological correlates a key component of emotion processes. It shows that single modality recordings and univariate tests are suboptimal for specificity questions, e.g. mapping physiological correlates to subjective emotional experiences or to clinical predictors.

For theories of autism and alexithymia, these results call for revision of the arousal-based accounts of atypical emotional processes. First, atypical physiological responding seems to be more robustly related to alexithymia, which may suggest that subgrouping within autism may be related to various profiles of physiological responding driven by alexithymia. However, this work also calls for a rethinking of alexithymia formulations, particularly in terms of physiological and interoceptive mechanisms. In light of the results from **Experiment 7** and **8** in **Chapter 5**, and the physiological mobilisation imbalance hypothesis, most putative arousal patterns used to describe potential subgroups based on physiological responding in alexithymia reflect only partial or incomplete information about the nature of autonomic co-activation and interoceptive signalling. That is, hypo-or hyperarousal may reflect modality-specific responding rather than generalised physiological co-activation to emotions. For

interoceptive accounts of alexithymia, these results require a reconsideration of decoupling or concordance tests of physiological and subjective arousal experiences.

Furthermore, it seems not only possible but highly probable that interoceptive deficits result from atypical physiological mobilisation. Modern models of interoception argue that active inferential and predictive processes are key in interoception (Barrett, 2017; Ramstead, Wiese, Miller, & Friston, 2020). As such, one possibility is that imbalances in physiological mobilisation make internal states inherently imprecise, and it is this imprecision that makes interoceptive integration difficult to realise as it interferes with predictive mechanisms that underlie interoceptive processes (interoceptive attention, interoceptive awareness).

Similarly, a recent alexithymia theory proposes a multi-route model of alexithymia, where one possible route involves atypical language processes (Hobson, Brewer, Catmur, & Bird, 2019). The idea that language may be relevant to alexithymia seems obvious given the implication of language in many emotion processes (Lindquist, MacCormack, & Shablack, 2015). Furthermore, studies of acquired alexithymia following brain insult show more severe alexithymia in patients with language problems (e.g. aphasic patients) (Hobson et al., 2018). One interesting suggestion is that atypical involvement of language in alexithymia may also result from uncertain or imbalanced physiological profiles to emotional experiences during development. Most lay language for emotion is bounded to physiological attributes, for example, people burn with rage, stomach drops with fear or anxiety, and so on. Accordingly, atypicality in language related to emotion might reflect inconsistent associations between physiological states, emotional episodes and verbal attributes for these experiences. This could explain difficulty describing not only emotions but also bodily sensations and somatic experiences in alexithymia (Taylor, Parker, Bagby, & Acklin, 1992). This may also be related to beliefs about bodily activation to emotions (Nummenmaa, Glerean, Hari, & Hietanen,

2014). For example, a recent study suggested that self-reported bodily activations for emotions in alexithymia are more diffuse and less localised in high compared to low alexithymic participants (Lloyd et al., 2021). Therefore, physiological mobilisation imbalance may explain interoceptive and language features associated with alexithymia. A simple illustration of the link between physiological mobilisation imbalances and interoceptive processes, as well as the potential link with language, is presented in **Figure 6.2**

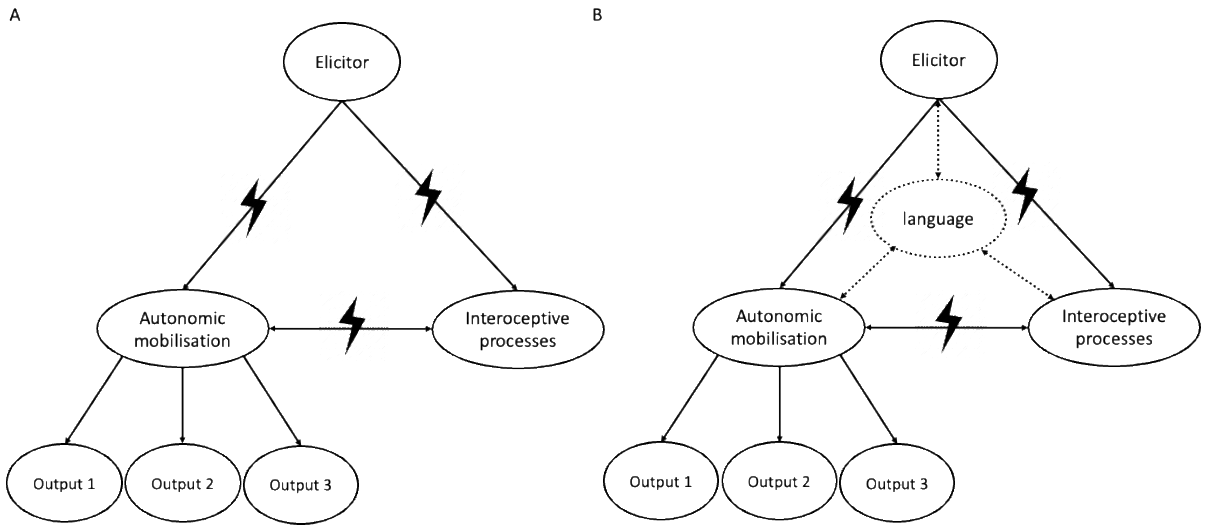

Figure 6.2 Illustration of two ways in which alexithymia may influence atypical physiological mobilisation and interoceptive processes.

Panel B shows how the same model can explain atypical language involvement in emotional processes in alexithymia.

6.2.2.1 Implication for research and potential translation

One of the key goals of alexithymia research in recent years has been to identify objective markers of the condition moving beyond self-reports (Gaigg et al., 2018; Hickman, Keating, Ferrari, & Cook, 2021; Panayiotou et al., 2018). Profiles of physiological responding have the potential to provide an objective marker of alexithymia. The current work, however, highlights that arousal- and interoception-based models of alexithymia may be overly

simplistic and fail to consider different modes of physiological mobilisation. In principle, however, replication of multivariate physiological mobilisation profiles could pave the way to define physiological mobilisation imbalance as an objective feature of alexithymia. Larger samples will be needed, however, to test the predictive power and validity of subtyping approaches based on physiological profiles of autonomic mobilisation to emotions.

6.2.3 Future directions

6.2.3.1 Mapping multivariate modes of autonomic activation

In line with the physiological mobilisation hypothesis advanced in Chapter 4, atypical mapping of autonomic activation and emotional states likely happens during development. Developmental studies are therefore needed to explore the trajectory of physiological mobilisation and interoceptive mapping implicated in emotional responses in alexithymia. In particular, studying key developmental periods where hormonal and physiological imbalances are more likely (e.g. adolescence) may allow causal connections between physiological mobilisation and atypical socioemotional functioning. Cross-sectional and developmental studies can also be used to reveal whether related processes such as language influence the development of conceptual representations of physiology/body and subjective experiences and whether they vary in various age groups. Furthermore, these studies should explore the specificity of bodily activations to emotional and non-emotional states (e.g. hunger, tiredness) given the relationship between alexithymia and altered interoception to emotional and somatic states (Murphy, Brewer, Catmur, & Bird, 2017).

6.2.3.2 Exploring causal links between autism and alexithymia

One important question for future research concerns the reason for the increased prevalence of alexithymia in autism. One possibility is that neuromodulatory imbalances associated with autism (e.g. in neural excitation and inhibition) predispose the physiological mobilisation imbalances relevant to emotion processes affected by alexithymia. One potential direction to establish causal mechanisms would be to explore transient modulation of cortical excitation or inhibition, for example by using TMS or TDCS paradigms, and exploring whether mobilisation of arousal responses to subsequent emotion induction approximates alexithymia profiles.

6.2.3.3 Bayesian accounts of physiological and interoceptive signalling

In **Chapter 1**, the Bayesian account of autism is presented in contrast to the other models including the alexithymia hypothesis and arousal-based models, yet these models are not necessarily mutually exclusive. As shown throughout this thesis, most results suggest a primary influence of alexithymia, and in the domain of visual processing, alexithymia affected processes that are typically implicated by the Bayesian hypothesis, such as reduced modulation of social attention by priors. One interesting question is whether predictive aspects of the Bayesian hypothesis could account for atypical physiological and interoceptive processes. Recent theories of interoception have argued that interoceptive processes and physiological mobilisation during emotion can be accounted for by predictive processes described by active inference models (Barrett, 2017). For example, psychopathology might reflect failures of physiological mobilisation resulting from atypical generative models of the internal and external milieu, given a model of both the external world and the internal states (interoception) (Barrett, Quigley, & Hamilton, 2016; Seth & Friston, 2016; Smith, Badcock, & Friston, 2021). Formal application of computational Bayesian accounts of interception and

physiological mobilisation may therefore be useful to investigate whether and how, for example, imbalances in physiological mobilisation affect precision of autonomic signalling and interoceptive processes.

6.3 THE OVERLAP BETWEEN AUTISM AND ALEXITHYMIA

The two studies in **Chapter 5** investigated the construct overlap and separation between autism and alexithymia. In prevailing views of autism as a multifaceted condition, alexithymia may be considered a symptom or a consequence of autism (Gaigg, 2012; Quattrocki & Friston 2014, Ben-Shalom et al., 2021, unpublished communication). Given that the assessment of autism and alexithymia relies predominantly on self-reports, a pre-requisite for the alexithymia hypothesis is that autism and alexithymia can at least be differentiated in the operationalisation of traits and symptoms included in assessment measures. Chapter 5, therefore, explored whether autism and alexithymia reflect distinct underlying latent factors. An exploratory and confirmatory factor analytical approach was employed, allowing for model-free and constrained models of the latent factors underlying the covariance of alexithymia and autism symptoms. Recent developments in network psychometric methodologies were also used to evaluate the nature of interactions of autism and alexithymia symptoms in clinical and non-clinical samples.

6.3.1 Findings

Experiment 9 and **10 in Chapter 5** tested a large sample of autistic and non-autistic participants, and results substantiated the alexithymia hypothesis, showing that emotional dimensions of alexithymia reflected separate latent underlying factors from the social dimensions of autism. Confirmatory models assigning a common underlying latent influence

on autism and alexithymia symptoms fitted the data poorly, whereas models explicitly specifying separate latent influences on autistic and alexithymia-related symptoms, provided the best fit to the data. These analyses also indicated that, whereas alexithymia dimensions were consistent with the theoretical models and always identifiable, autism-related dimensions did not necessarily follow the proposed multifaceted models of autism underlying common psychometrics measures. Network analyses, which assume causal dependencies between symptoms in line with the network theory of psychopathology (Borsboom & Cramer, 2013) showed a clear pattern of separation between autism and alexithymia dimensions, such that when all patterns of interactions and dependencies were accounted for, alexithymia and autism cluster separately.

6.3.2 Theoretical implications

The implication of the results from this chapter can be summarised in three points. First, these results suggest that theories of autism that view emotion components and alexithymia as intrinsic to autism (a consequence of autism) need to be revised. Despite the increased prevalence of alexithymia in autism, analysis of the latent construct profile as well as the interaction of manifest aspects of autism and alexithymia suggest that they reflect distinct conditions. Therefore, given the importance of alexithymia in socioemotional functioning demonstrated throughout this thesis and other studies of the alexithymia hypothesis, emotional difficulties in autism need to be reconceptualised as causally linked to alexithymia.

Second, these results have implications for models of autism and alexithymia underlying the psychometric development of tests. Particularly, the measurement model of autism is likely ill-fitted to capture the nuance of emotional problems that are linked to alexithymia (difficulties identifying and describing feelings, somatic experiences). As such, assessment of

alexithymia needs to be routine in clinical practice and research focused on how emotional processes are influenced by autism or individual differences in autistic traits.

Third, and related to the previous point, these results have important implications for assessment and diagnosis, as failure to capture key aspects of emotional functioning related to alexithymia may result in an incomplete characterisation of patients' strengths and needs, including failures to refer patients for diagnosis. It's important to note that this thesis does not suggest that autistic non-alexithymic individuals have less severe difficulties or that they are less disserving of intervention, as people may still strangle with other aspects of autism, including rigidity in behaviour or restricted interests which detrimentally impacts the quality of life, well-being and social relationships.

6.3.3 Future directions

6.3.3.1 Data-driven approaches to subtyping autism and alexithymia

Most of the research on autism and alexithymia comorbidity relies on apriori definitions, based on cut-off scores on self-report measures. However, it's clear that these criteria only provide a loose description of these groups such that they are not necessarily homogeneous - or at least not homogeneous enough to provide accurate subtyping based on profiles of socioemotional functioning (emotion processing abilities, interoceptive abilities, physiological responding). As such, more comprehensive approaches are needed for subtyping. Future studies could combine and extend approaches such as the one used in **Chapters 4** and **5** to test for apriori subtypes or discover subtypes in a data-driven fashion. Rather than separating groups based on summary scores, discriminant and factor analytical approaches could be used to explore differences in the full pattern of autistic and alexithymic traits. This research may be particularly informative for identifying key symptoms and

clusters of symptoms that drive subtyping. Additionally, information regarding physiological responding and emotion processing from experimental paradigms could also be integrated to develop sensitive and reliable classification or subtyping algorithms, which may be used for assessment and diagnosis.

6.3.3.2 Development of more robust psychometric assessments for alexithymia and autism

As discussed throughout this thesis, the interest in alexithymia in autism research is relatively recent and was more strongly driven by the work on the alexithymia hypothesis (Bird & Cook, 2013; Brewer et al., 2016; Hobson et al., 2019). As such, the majority of studies in autism have not accounted for alexithymia. The same is true for alexithymia research, while views that alexithymia may constitute a vulnerability for psychology are not new, most of the research and development of the alexithymia construct and measures happened independently from developments in psychopathology, and neurodevelopmental conditions such as autism have been particularly underrepresented. This means that conceptual redundancies are likely to be reflected in measures of autism and alexithymia. For example, while **Chapter 5** showed independence of autism and alexithymia, it is the case that the communicative component of autism shows the strongest associations with the feelings components of alexithymia. Similarly, the externally oriented thinking in alexithymia besides being related with the other alexithymia components was also related to the imagination components of autistic traits. These associations make it hard to match participants such that high autistic traits tend to be associated strongly with alexithymia in standard alexithymia and autism measures. While this likely reflects the factual nature of this relationship, it complicates tests of independence of autism and alexithymia traits, as such tests require relative statistical independence of these constructs. As such, the development of measures of autism and alexithymia that are more

"pure" or specific to the autistic and alexithymic phenotype seems particularly relevant. Some of the data in this thesis, particularly in **Chapter 5**, could be used to support the development of novel measures based on existing measures such as the AQ50 and TAS-20.

6.4 GENERAL CONCLUSIONS, FUTURE DIRECTIONS, AND LIMITATIONS

6.4.1 Moving beyond micro-theories and theories of everything in autism

This thesis explored approaches that can be considered micro-theories, in the sense that they are very targeted to specific features of autism. Nonetheless, micro theories of autism, while driving much progress thus far, may have begun to outlive their usefulness. Conversely, some theories of autism, particularly domain-general accounts such as the Bayesian hypothesis, try to be 'theories of everything' in autism, explaining both social and non-social aspects of the condition. Given the multifaceted nature of how autism is manifested, as well as the likely multiple aetiological routes for autism and alexithymia, it may be best to aim for a theory that is parsimonious enough to provide a domain-general account of these conditions, yet specific enough to account for multiple profiles of functioning across several domains (cognitive, affective, physiological).

This thesis provides a compelling case that the alexithymia hypothesis first advanced by (Bird & Cook, 2013) has some ingredients to achieve a unified account, not necessarily of all features of autism, but at least of the socioemotional functioning aspects of autism and psychopathology where emotional aspects are implicated. While this thesis certainly does not provide a complete test of the alexithymia hypothesis, it highlights several mechanisms that need to be incorporated into a formal update of the alexithymia hypothesis.

Ultimately, the alexithymia hypothesis should highlight the aetiological routes for alexithymia. This wasn't explored in this thesis but speculating on the grounds of the results observed involving physiological components, this should include likely neurobiological predisposition (due to genetic reasons or brain maturation) to imbalances in autonomic control and interoceptive regulation. In line with the physiological mobilisation imbalance hypothesis advanced in Chapter 4, atypicality in physiological modulation is likely to interfere with responses to exteroceptive information in two ways. The first is by disrupting automatic modes of responding, and the second is by interfering with learning and storing associations between physiological responses and the external state of the world, resulting in top-down impairments in how models of internal states (interoception) and external word influence predictions about exteroceptive input.

The atypical mapping of physiological mobilisation to interoceptive processes is likely to result during development from previous experience, and therefore bias integration of interoceptive and exteroceptive states. In summary, the alexithymia model should include vulnerability to physiological imbalances, causal links between atypical physiological mobilisation, and imprecision of models of the internal milieu (interoception). These processes are also likely to influence cognitive and executive processes relevant to social and non-social functioning (e.g. social attention, motion processing). **Figure 6.3** provides a preliminary sketch of such a model and the mechanisms discussed above including a suggestion of the neurobiological underpinnings.

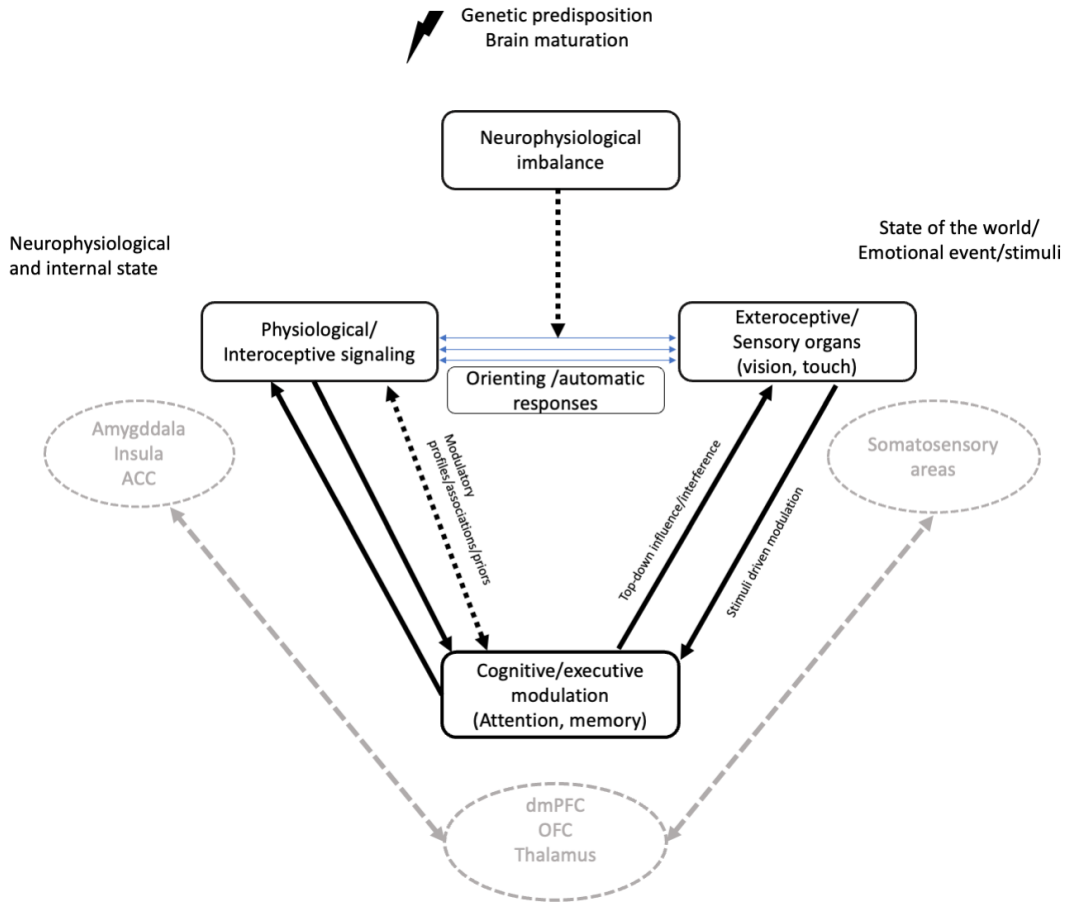

Figure 6.3 Schematic illustration of mechanisms that may be implicated in atypical socioemotional processes in alexithymia

6.4.2 GENERAL LIMITATIONS AND FUTURE DIRECTIONS

6.4.2.1 Sample

The clinical samples used throughout the experiments covered in this thesis are of high functioning autistic individuals. As such, it's unclear how these results generalise to the vast majority of autistic individuals with more severe cognitive impairments. The development of tasks that do not rely on verbal ability is likely to aid the study of such populations. Furthermore, given the focus of alexithymia influences on autism, it's unclear whether these results generalise to other clinical conditions where alexithymia is also highly prevalent. Only

comparative studies and meta-analyses including various disorders comorbid with alexithymia will inform about potential interactive effects that alexithymia may have with specific conditions. Additionally, the sample sizes for the autistic population particularly for the eye-tracking and psychophysiology studies was relatively small, often due to exclusions related to data-quality. Therefore, replication studies with larger samples of autistic participants are needed.

6.4.2.2 Assessment of alexithymia

Another key limitation of the studies discussed in this thesis is that the assessment of alexithymia relied primarily on self-report using the TAS-20. A more comprehensive assessment of alexithymia should ideally involve a multi-method approach, combining self-reports with clinical interviews, and third-party observer reports (Taylor, Bagby, & Luminet, 2000). This is particularly important given the correlation between alexithymia and general mental health symptoms including depression and anxiety (Grabe, Spitzer, & Freyberger, 2004).While in all studies, depression and anxiety effects were controlled for, it's unclear to which degree, high alexithymic participants in the samples used here reflect a "pure" alexithymia phenotype. Furthermore, the cut-offs used for alexithymia were proposed only as suggestive discriminant values over 30 years ago (Taylor et al., 1988). It's unclear how sensitive this cut-off remains, as no population norms exist. Future reviews and meta-analyses could attempt to estimate the distribution of alexithymia scores in the general population and derive population norms for several age groups, and clinical conditions.

6.4.2.3 Cross-sectional approaches

The methodology employed in this thesis is cross-sectional. As such, the results do not speak directly to causal mechanisms underlying socioemotional processes. However, longitudinal and developmental research can greatly aid the understanding of alexithymia influences in the emergence of atypical socioemotional processes. On the physiological and interoceptive basis of alexithymia, pharmacological interventions could be used in typical participants to affect patterns of mobilisation across autonomic modalities, for example by using drugs to target certain autonomic pathways.

6.4.2.4 Stimuli

The current research was limited to primarily visual or audio-visual stimuli (faces, evocative images, videos). As such, it's unclear whether some of the effects observed here may reflect modality-specific effects of alexithymia (e.g., to visual affective stimuli). As such, more research should explore other types of stimuli for social attention and emotion induction, these could include voices (emotional vocalisations), or tactile emotional stimuli (e.g. cutaneous stimulation) to explore whether effects of alexithymia are generalised across these classes of stimuli.

6.5 Conclusion

Across several experiments, this thesis provides one of the most extensive tests of the alexithymia hypothesis of autism. The evidence overwhelmingly supports the view that key socioemotional processes thought to be linked to autism (social attention, emotion recognition, and experiential aspects of emotion) are in fact driven by alexithymia. These results provide insights relevant to understanding the documented mixed evidence regarding

socioemotional processes in autism, and provide several methodological and conceptual advances for understanding and studying typical socioemotional processes.

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8 APPENDICES

8.1 Chapter 2. Supplementary Materials

A **B**

Figure 8.19 Illustration of AOI discretisation method.

A. Illustration of OpenFace facial landmarking (Baltrušaitis et al., 2016) on a sample stimulus used in the study (Yitzhak et al., 2017). This set was chosen as it contains subtle emotional displays which helps to avoid the recognition ceiling effects typically observed with high intensity expression datasets (Krumhuber, Kappas & Manstead, 2013). B. Segmentation of AOIs using the Limited Radius Voronoi Tessellation method (LVRT method). Gaze points are assigned by finding the minimum Euclidian distances from facial landmarks at every frame. Each AOI corresponded to approximately 200 pixels in radius (approximately 4° of visual angle; see Hessels et al., 2018 for more details on the method).

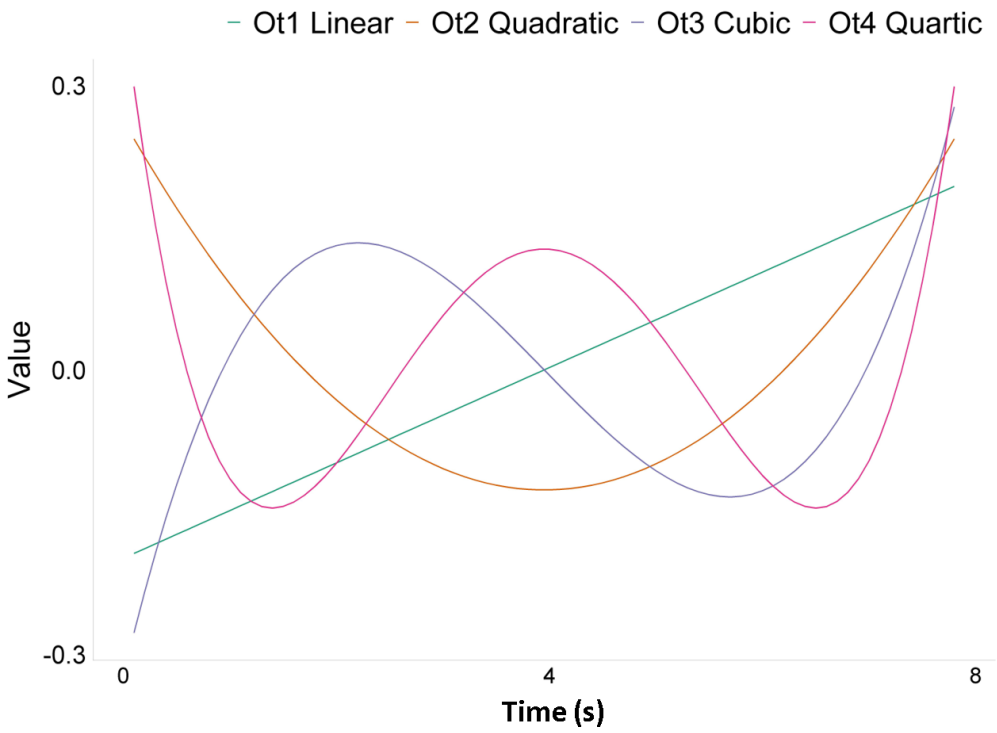


Figure 8.2 Fourth-order orthogonal (ot) time polynomials.

Fourth-order orthogonal (ot) time polynomials were created based on the stimulus viewing window (7.8 seconds) and were used to model the non-linear trajectory of gaze points within each AOI over time. These polynomials were included as fixed effects as well as random slopes for participant and trial id (video stimulus). Additionally, the interactions between the polynomials and the other fixed effects of interest (autism, alexithymia, and condition) were also modelled.

8.1.1 Participants

A total of 90 participants were originally tested, 15 of whom took part in a pilot study and 75 in the final study. Out of 75 participants in the final study 5 were excluded due to eye problems (e.g., severe cataract or extreme correction) or data quality issues, leaving a final sample with 70 participants whose data were analysed. Statistical simulations for mixed models based on pilot data revealed that this sample size was sufficient to achieve 80% statistical power for our analyses. Autistic participants had previously been diagnosed by an independent clinician and took part in an Autism Diagnostic Observation Schedule (ADOS) interview administered by one of the authors, in a separate session. As is typical for autistic adults with high IQ scores, not all met the total score threshold for autism on the ADOS, although all had clinical diagnoses and met cut-off on the Autism Spectrum Quotient – AQ-50.

8.1.2 Model building and model comparison

For timecourse analyses, *free-gaze* fitted models have the form:

*Eye gaze proportion (elog) ~ 1 + Alexithymia * ot (overall effect of time - linear) + ot2 (quadratic) + ot3 (cubic) + ot4 (quartic) + (1 + ot1 + ot2 + ot3 | Participant) + (1 + ot1 + ot2 | Video id).*

Where 1 is the overall intercept, *Alexithymia* and o*t1* to o*t4* terms are fixed effects, *participant* and *video id* are random intercepts. *1+ ot* terms represent random slopes for *participants* and *video id*.

Condition models have the form:

*Eye gaze proportion (elog) ~ 1 + Condition (reference = free-gaze) * Alexithymia (Group coded as -5(autistic participants – ASC); +5 (neurotypical participants – NT); Autistic traits)*

** (ot1+ot2+ot3+ot4)+ (1 + ot1 + ot2 + ot3 + ot4 | Participant) + (1 + ot1 + ot2 + ot3 | Participant) + (1 + ot1 + ot2 | Video id).*

For the rest of the analyses (entropy and aggregated fixation), models have the form:

SGE/GTE (Shannon and conditional entropy in normalised bits) ~ 1+ Condition + Alexithymia (or Group or Autistic traits) + (1 | Participant) + (1 |Video id) for free-gaze and alexithymia (see **Table 8.3** in SM for model building and comparison).

For model comparisons, each model of interest, for example, the alexithymia model containing alexithymia, condition, and its interactions with the polynomial time terms would be compared against the nested model without the effect of interest or its interactions (see Table 8.3 Model building and comparison). The drop function in lmerTest package was also used to test for significant terms and the results were equivalent to the method above. For clarity of presentation, whenever models of interest performed better than random effects only, then results will normally be reported in relation to the simpler model (nested) not containing that term (e.g., alexithymia*condition vs. condition). This reflects the fact that if, for example, the condition model performs better than random effects only model, and the alexithymia by condition model outperforms the condition model, then it also outperforms the random effects model. To test the relative influence of alexithymia and autism on gaze patterns, models containing alexithymia and autism were also directly contrasted in a jointly fitted model which produced standardised estimates. Finally, although not a formal test, the anova function was used to compare the models based on AIC information criterion.

Descriptive and correlational data are presented in the tables below.

Table 8.1 Descriptive aggregated data

Variable	NT *N = 45*		ASC *N = 25*	
	M	SD	M	SD
Fixations				
Fix dur (eyes)	3457	1737.6	3307	1593.6
Fix count (eyes)	7.72	3.92	6.97	3.30
Fix dur (mouth)	1736.6	1217.6	1746.9	1253.5
Fix count (mouth)	3.39	1.95	3.55	2.52
Entropy				
SGE	.59	.12	.61	.11
GTE	.28	.03	.29	.04
ER Accuracy	.81	.08	.71	.14

Notes. Fix dur (ms) = Fixation duration, calculated as the average of the total fixation durations to the AOI. Fix count = Fixation count, calculated as the average of the total number of fixations to the AOI. SGE = Stationary gaze entropy. GTE = Gaze transition entropy in normalised bits. ER = Emotion recognition.

Table 8.2 Zero-order correlations for aggregated data

Variable	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*
1. TAS20	1												
2. AQ28	**.40***	1											
3. DASS21	**.42***	**.56*****	1										
4. IQ	.19	.02	.09	1									
5. Age	.38	.24	.18	.27	1								
6. ER Acc	-.35	-.20	-.23	-.05	**-.5****	1							
7. SGE	.16	-.08	.11	-.07	-.02	-.04	1						
8. GTE	.12	-.02	.18	-.06	-.06	0	**.92*****	1					
9. Fix dur eyes	-.22	.05	-.02	.05	-.13	.09	**-.57*****	**-.41***	1				
10. Fix dur mouth	.12	-.05	0	-.05	.09	-.14	.32	.27	**-.75*****	1			
11. Fix count eyes	-.22	-.13	-.18	.03	-.11	.13	**-.4****	**-.35****	**.78*****	**-.69*****	**1**		
12. Fix count mouth	.20	-.09	.01	-.08	.10	-.10	.34	.21	**-.75*****	**.84*****	**-.59*****	1	
13. Sacc amp	.26	-.02	.27	-.05	.07	-.26	.30	.27	.01	.08	.05	.19	1

Notes. * $p < .05$; ** $p < .01$; *** $p < .001$. All correlations are Bonferroni-corrected. ER Acc = Emotion recognition accuracy; Fix dur = Fixation duration; Sacc amp = Saccade amplitude; GTE = Gaze transition entropy; SGE = Stationary gaze entropy.

Time course analysis

Model comparisons

For the free-gaze condition, the alexithymia model outperformed the autism model (ΔAIC = 63), and the autistic traits model (modelling autistic traits as a continuous variable ΔAIC = 67; see table below). Similarly, for the condition comparisons when directly contrasting the ML fitted models, the alexithymia model significantly outperformed the autism model (ΔAIC = 937) and the autistic traits model (ΔAIC = 1671).

Table 8.3 Model building and comparison

Sampling Units		N total obs = 509061 N subjects = 70; N videos = 42										
Model specification	**Model name**	**Nested / alternative**	**Fixed Effects added**		**Random Effects**		**Model fit**				**LRT against nested**	
					Subjects	**Items**	**AIC**	**BIC**	**LL**	**df**	**df**	***X²***
Random Effects												
RE	Null	-	-		Intercepts, t to t^3 poly slopes	Intercepts, t to t^2 poly slopes	1913907.6	1914152.7	- 956931.8	28		
Fixed Effects												
FE main effects	ots	Null	ot1+ot2+ot3 +ot4		Intercepts, t to t^3 poly slopes	Intercepts, t to t^2 poly slopes	1881964	1882320	-940950	32	4	721.68 ***
FE two-way interactions	Cond x ots	ots	Cond* (ot1+ot2+ ot3+ot4)		"	"	1881606	1882130	-940756	47	15	387.97 ***
FE three-way interactions	Group x Cond x ots	Cond x ots	Group* Cond* (ot1+ot2+ ot3+ot4)		"	"	1908570	1909205	-954228	57	10	ns[a]

FE three-way interactions	AQ x Cond x ots	"	AQ* Cond* (ot1+ot2+ ot3+ot4)		"	"	1909304	1909939	-954595	57	35	4629.60***
FE three-way interactions	Alex x Cond x ots	"	TAS* Cond* (ot1+ot2+ ot3+ot4)		"	"	1907633	1908268	-953760	57	35	6300.53***
FE three-way interactions	Alexsplit x Cond x ots	"	TASsplit* Cond* (ot1+ot2+ ot3+ot4)		"	"	1908073	1908708	-953980	57	0	496.58 ***

Sampling Units		N total obs = 509061 N subjects = 70; N videos = 42										
Model specification	**Model name**	**Nested / alternative**	**Fixed Effects added**		**Random Effects**		**Model fit**				**LRT against nested**	
					Subjects	**Items**	**AIC**	**BIC**	**LL**	**df**	**df**	***X²***
Model Comparisons (Three-way Interaction Models of Interest)												
FE three-way interactions	Alex x Cond x ots	Group x Cond x ots	TAS* Cond* (ot1+ot2+ ot3+ot4)		Intercepts, t to t^3 poly slopes	Intercepts, t to t^2 poly slopes	1907633	1908268	-953760	57	0	936.65 ***
FE three-way interactions	Alex x Cond x ots	AQ x Cond x ots	TAS* Cond* (ot1+ot2+ ot3+ot4)		"	"	1907633	1908268	-953760	57	0	1670.93 ***
FE three-way interactions	Alexsplit x Cond x ots	Group x Cond x ots	TASsplit* Cond* (ot1+ot2+ ot3+ot4)		"	"	1908073	1908708	-953980	57	0	496.58***
FE three-way interactions	Alexsplit x Cond x ots	AQ x Cond x ots	TASsplit* Cond* (ot1+ot2+ ot3+ot4)		"	"	1908073	1908708	-953980	57	0	1230.9***
FE	Alexsplit	TAS x	TASsplit*		"	"	1908073	1908708	-953980	57	0	ns

three-way interactions	x Cond x ots	Cond x ots	Cond* (ot1+ot2+ ot3+ot4)									

Sampling Units		N total obs = 509061 N subjects = 70; N videos = 42										
Model specification	**Model name**	**Nested / simpler (or alternative) Model**	**Fixed Effects added**		**Random Effects**		**Model fit**				**LRT against nested**	
					Subjects	**Items**	**AIC**	**BIC**	**LL**	**df**	**df**	X^2
Additional Models with Control Variables												
FE three-way interactions + Main effect	Alex x Cond x ots + Group	Cond x ots	TAS* Cond* (ot1+ot2+ ot3+ot4)+ Group		Intercepts, t to t^3 poly slopes	Intercepts, t to t^2 poly slopes	1907635	1908281	-953760	58	36	6300.7 ***
FE three-way interactions + Main effect	Alex x Cond x ots + AQ	"	TAS* Cond* (ot1+ot2+ ot3+ot4)+ AQ		"	"	1907635	1908282	-953760	58	36	6300.57 ***
FE three-way interactions + Main effect	Alex x Cond x ots + DepAnx	"	TAS* Cond* (ot1+ot2+ ot3+ot4)+ DASS		"	"	907635	1908282	-953760	58	36	6300.5 ***
FE three-way interactions + Main effect	Alex x Cond x ots+ Age	"	TAS* Cond* (ot1+ot2+ ot3+ot4)+ Age		"	"	1908073	1908708	-953980	57	35	5860.5 ***
FE three-way interactions + Main effect	Alex x Cond x ots + IQ	"	TAS* Cond * (ot1+ot2+ ot3+ot4)+		"	"	1907633	1908279	-953759	58	36	6302.7 ***

			IQ									
Sampling Units		N total obs = 509061 N subjects = 70; N videos = 42										
Model specification	**Model name**	**Nested / alternative**	**Fixed Effects added**		**Random Effects**		**Model fit**				**LRT against nested**	
					Subjects	**Items**	**AIC**	**BIC**	**LL**	**df**	**df**	***X²***
Additional Models with Control Variables												
FE three-way interactions	DepAnx x Cond x ots	Cond x ots	DASS* Cond* (ot1+ot2+ ot3+ot4)		Intercepts, t to t^3 poly slopes	Intercepts, t to t^2 poly slopes	convergence warning – Recalculation of hessian gradient > tolerance					
FE three-way interactions	Age- Cond x ots	"	Age* Cond* (ot1+ot2+ ot3+ot4)		"	"	convergence warning – Recalculation of hessian gradient > tolerance					
FE three-way interactions	IQ x Cond x ots	"	IQ* Cond* (ot1+ot2+ ot3+ot4)		"	"	convergence warning – Recalculation of hessian gradient > tolerance					

Notes.

Alex – Alexithymia (measured with TAS20)
Alexsplit – Alexithymia, dichotomised by median spit
AIC – Akaike Information Criterion
AQ – Autistic traits measured by AQ28
BIC – Bayesian Information Criterion
Cond – Conditions (*free-gaze, emotion recognition, cued and intensity judgment conditions*)
DepAnx – Depression and anxiety measured by DASS21
df – degrees of freedom
FE – Fixed effects
IQ – Intelligence Quotient measured by WASI
LL – LogLikelihood
LRT – Likelihood Ratio Test
X^2 – Chi-square
RE - Random effects
ots – orthogonal time polynomials (up to the quartic)
Poly – Polynomials

Note that the LRT test (LL, chi-square) is provided for all models for completeness, yet they are only valid for nested comparisons. In non-nested cases, the assessment of the models relied on AIC and joint estimated standardised estimates.

Alexithymia x polynomial effects – Time course analysis

Free-gaze: There was a main effect of the quartic polynomial, indicating that a quartic function best describes eye gaze over time (Estimate = -.54, SE = .15, $p < .001$). This reflects the pattern of small gaze changes between different AOIs and reduced eye attention at the beginning and end of the time window. Alexithymia interacted with the cubic (Estimate = -.27, SE = .04, $p < .001$), and quartic terms (Estimate = .23, SE = .04, $p < .001$), suggesting that the rate of gaze changes differs throughout the timecourse as a function of alexithymia, with highly alexithymic individuals reaching near asymptote after reduced attention to the eyes early in the trial.

Condition vs free-gaze baseline: Alexithymia had a significant effect on the quadratic polynomial term for the *emotion recognition* (Estimate = -.45, SE = .05, $p < .001$) and *intensity judgement* (Estimate = -.27, SE = .05, $p < .001$) but not the *cued* conditions (Estimate = .06, SE = .05, ns). There was also a significant interaction between alexithymia and condition on the cubic term for the *emotion recognition* (Estimate = .49, SE = .05, $p < .001$), *cued* (Estimate = .32, SE = .05, $p < .001$) and *intensity judgement* conditions (Estimate = .31, SE = .05, $p < .001$). Finally, the interaction between alexithymia and condition was also significant for the quartic time term, showing that relative to *free-gaze*, there was a significant interaction effect during the *emotion recognition* (Estimate = -.45, SE= .05, $p < .001$), *cued* (Estimate = -.37, SE = .05, $p < .001$) and *intensity judgement* conditions (Estimate = -.37, SE = .05, $p < .001$).

Post-hoc tests on each condition separately revealed that alexithymia still predicted reduced eye gaze throughout the timecourse for all conditions including the *emotion recognition* (Estimate = -.16 [95% CI: -.49 - -.09], SE = .004, $t = -2.81$, $p < .001$), *cued* (Estimate = -.20

[95% CI: -.49 - -.12], SE = .04, t = -3.25, p < .001) and *intensity judgement* (Estimate = -.13 [95% CI: -.49 - -.03] ; SE = .004, t = -2.19, p < .001) conditions.

8.1.3 Control and additional analyses

Time course analyses excluding the first .05 seconds

There is a valid concern that the fast orientation to eyes in the first few milliseconds, may make it so that one requires high-order polynomials to describe the timecourse of eye gaze proportions appropriately, even if the measure at this period may be relative and unreliable. The visualisation and analyses below excluded the first 500 ms. Overall, there is still an effect of alexithymia and condition on both the offset and eye gaze and the overall shape that describes eye gaze allocation overtime, with marked non-linearities in free-gaze compared to recognition, cue and intensity conditions.

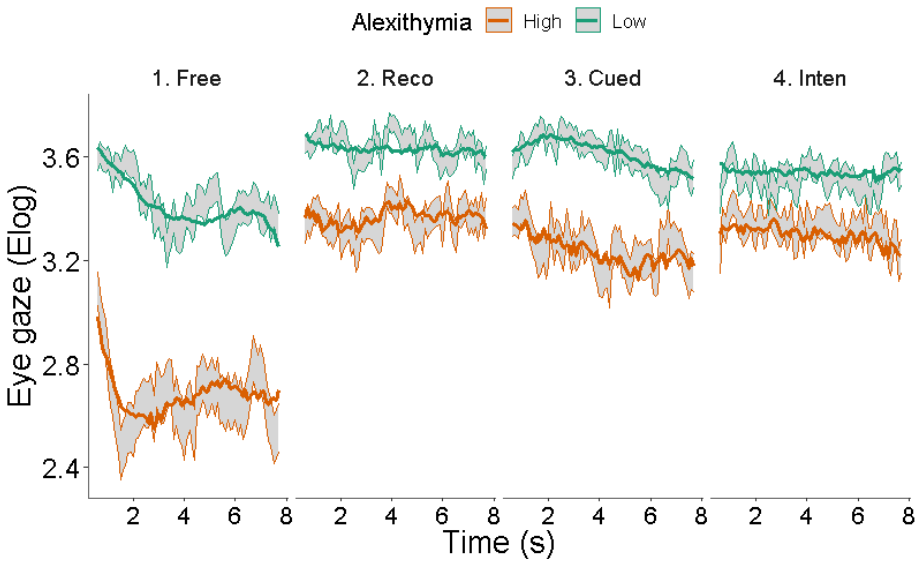


Figure 8.3 Time course model of eye gaze without the first 500ms.

Alexithymia predicted reduced eye gaze overtime and the nonlinear shape of eye gaze as a function of condition. Alexithymia was used as a continuous predictor; the split high vs low is done for visualisation purposes.

The same analyses were repeated while excluding the first second, and the results were consistent with the main difference being that the model did not need a quartic term to be well fitted, nonetheless, the higher order terms (quadratic and cubic) provided improved fit compared to only the linear term (χ^2 = 376, ΔAIC = 353 p < .001).

8.1.3.1 Timecourse for the mouth AOI

Free-gaze condition: Effects of alexithymia and autism

For the free-gaze condition, none of the models of interest (alexithymia, autism diagnosis or autistic traits) improved model fit compared to the polynomial model, which outperformed the random effects only model in predicting the timecourse of attention to the mouth region (χ^2 = 476, ΔAIC = 31, p < .001). Therefore, only the polynomial model was interpreted.

This model indicates that mouth gaze trajectory in the free-view condition was described well by fourth-order terms. There was a significant effect of the linear term (Estimate = .84, SE= .34, p = .02), indicating an increase of attention to the mouth over time, and a negative effect of the quadratic term (Estimate = -.71, SE = .28, p = .01), indicating that attention to the mouth is low at the start and at the end of the stimulus, and a significant effect of the quartic term (Estimate = .80, SE = .13, p < .001) indicating changes of direction throughout the timecourse of mouth gaze. No other effects were significant.

Condition effects relative to the free-gaze baseline: Effects of alexithymia and autism

As with eye gaze, the alexithymia model outperformed the condition model (χ^2 = 13909, ΔAIC = 13869, p < .001). Alexithymia also provided improved fit over the autism diagnosis

model (ΔAIC = 12953) and the autistic traits model (ΔAIC = 13550), in predicting how the timecourse of gaze to the mouth was changed by task. The alexithymia model revealed a significant interaction between alexithymia and condition on the quartic term during the *intensity judgement* condition only (Estimate = -.03, SE = .08, $p < .001$).

The time-based cluster analysis of the mouth gaze timecourse suggested only two small significant time windows at the extremes of the stimulus window, spanning the first 300 ms and the last 800 ms. The bootstrapping procedure indicated that these clusters were not significant ($p > .05$). This suggests that taking into account individual differences in alexithymia or autism and autistic traits might result in overfitting for the mouth gaze timecourse, and that simpler models including task manipulations and polynomials likely suffice.

Entropy

The distribution of entropy was zero inflated, i.e., continuous apart from a spike (a second mode) in zero. This resulted from trials with no gaze transitions (e.g., where only one region of the face was looked at) leading to zero entropy. Therefore, these models were refitted with zero truncated Poisson distribution as well as excluding the zero cases, and results remained unchanged.

Entropy was used to assess the level of complexity underlying the distribution of fixations and the efficiency of scanning behaviour. To confirm that entropy measures were meaningful, the relationship between entropy and a standard gaze metric, fixation duration, was assessed. Longer fixations should, in general, produce minimal entropy, due to a less complex pattern of fixations. This is because longer fixations should be inversely related to the chance of transitioning between multiple gaze targets. As expected, fixation duration correlated

negatively with SGE (r = -.82, $p < .001$) and GTE (r = - .31, $p < .01$). This relationship was mainly driven by fixation duration to eyes with SGE (r_{eyes} = -.57, $p < .001$) and GTE (r_{eyes} = -.41, $p < .05$), whereas fixation duration to the mouth was not significantly related to SGE (r_{mouth} = .32, ns) or GTE (r_{mouth} = .34, ns). Following a visual inspection, however, it became evident that the relationship between fixation duration and entropy is non-linear (see Figure 8.4).

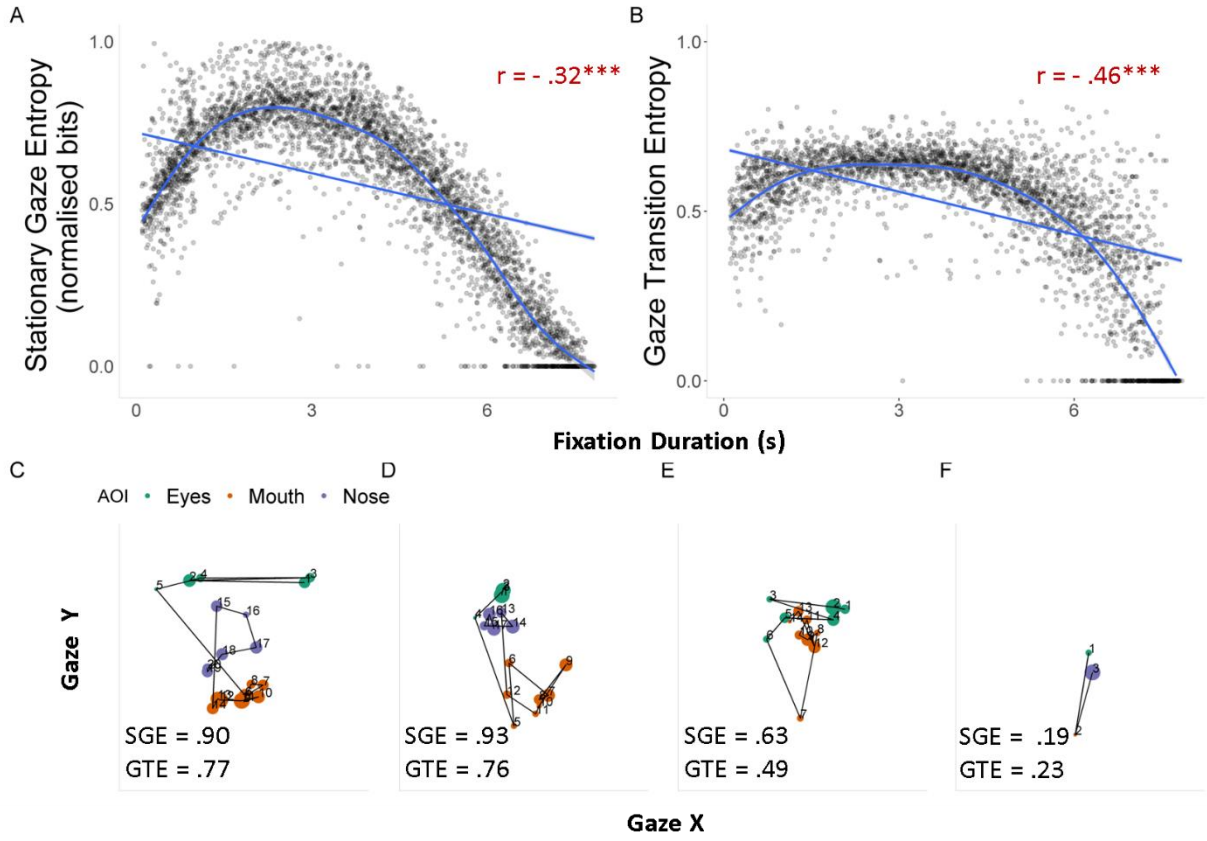


Figure 8.4 Entropy checks.

A & B. Scatterplots indicate a non-linear yet largely negative relationship between entropy and fixation duration. C & F. Ordered sample scan paths and entropy scores (in normalised bits) for a sample participant (NT) viewing a happy expression in the emotion recognition condition. D & E. Ordered sample scan paths and entropy scores for an autistic participant viewing disgust and anger expressions in the emotion recognition and cued conditions. The

size of the circles represents duration of the fixation, with larger circles indicating increased fixation duration. High values of entropy indicate increased fixation dispersion (SGE) or transitions (GTE), which suggest increased randomness and reduced predictability in gaze behaviour. Entropy data include fixation to all areas of interest (eyes, nose, and mouth).

Like longer fixations, shorter fixations also lead to reduced entropy. This pattern was not anticipated but suggests shorter total fixation durations also imply less chance of transitioning between different state spaces. Overall, however, these checks suggest that the entropy metrics are appropriately capturing the dispersion of gaze data as well as the complexity of transitions.

SGE & GTE model comparisons

When directly contrasting ML fitted models for SGE, the alexithymia model provided a better fit than the autism diagnosis (ΔAIC = 21) and the autistic traits (ΔAIC = 14) models. Similarly, for GTE, alexithymia provided a better fit than the autism diagnosis (ΔAIC = 12) and autistic traits (ΔAIC = 5) models, indicating that alexithymia is a better predictor of the complexity of fixation dispersion and transitions.

8.1.3.2 Aggregated fixation analyses – mouth AOI

Fixation count was calculated as the average of the total number of fixations to an AOI per trial. Fixation duration was calculated as the sum of the durations of all fixations to an AOI per trial.

Free-gaze condition: Effects of alexithymia and autism

For mouth fixations during the *free-gaze* condition, none of the models provided a significant improvement in fit compared to the random effects only model, or relative to one another, therefore, they were not further interpreted.

Condition effects relative to the free-gaze baseline: Effects of alexithymia and autism

For the condition analysis, model selection indicated that all models had a worse fit than the baseline null models (χ^2 = 6.5, ΔAIC = -2, ns) and so these higher-level models were also not further interpreted.

Fixation duration

Free-gaze condition: Effects of alexithymia and autism

None of the regressors improved model predictions for duration of mouth fixations in the *free-gaze* condition beyond the random effects models. Therefore, none of these models were interpreted further, as this suggests that duration of mouth fixations is not explained beyond variability in participants and stimuli.

Condition effects relative to the free-gaze baseline: Effects of alexithymia and autism

For the analysis including all conditions, fixation duration to the mouth was best predicted by the autism model when compared to the alexithymia model (ΔAIC = 8). However, compared to condition and random effects models the autism model was significantly penalised for complexity under the BIC criteria, which suggests overfitting. This suggests that experimental manipulations may be a better predictor of mouth fixation duration. The

condition model (χ^2 = 51.15, ΔAIC = 45, p < .001) indicates a significant decrease in fixation duration to mouth for the *recognition* (Estimate = -.11, SE = .02, p < .001), and *intensity judgement* conditions (Estimate = -.09, SE = .02, p < .001) but not the *cued* condition (Estimate = -.01, SE = .1, p = .60).

8.1.3.3 Depression and Anxiety

The autistic group had a range of other clinical traits and symptoms that were not the focus of this study, particularly depression and anxiety (as measured by the DASS21), as is typically observed in autistic populations (Lugo-Marín et al., 2019). To check for any confounding effect of these traits, a model with DASS scores was fitted and compared to the alexithymia model. The results showed that the alexithymia model outperformed the Depression-Anxiety-Stress model in terms of fit (ΔAIC = 9975), with small but significant effect on eye gaze (Estimate = -.28, SE = .06, p < .001), compared to a non-significant effect of Depression-Anxiety-Stress (Estimate = -.02, SE = .06, ns). Crucially, effects could not have been driven by depression and anxiety in the autistic group, as the strength of the correlation between alexithymia and DASS scores was similar and non-significant in both groups separately (r_{autism} = .10, ns; r_{NT} = .18, ns; **see also separate analyses by group below).** DASS scores were therefore not further considered, to prevent overparametisation and overfitting of models. Although future studies with larger ASC samples should attempt to achieve full orthogonality of clinical predictors, it is worth noting that this usually comes at the cost of representativeness and generalisability.

8.1.3.4 Age

A further control analysis was conducted to ensure that age was not a contributing factor in the eye gaze results as the autistic group, although matched to the NT group in terms of IQ and gender, was older on average. This issue was addressed in two ways. First, a separate model for age was fitted and model selection once again favoured alexithymia (χ^2 = 11629; AIC = 11629, $p < .001$). Similarly, when jointly estimated, alexithymia showed a larger and significant effect (Estimate = -.32, SE = .06, $p < .001$) than age (Estimate = .07, SE = .06, ns). Second, a variation of the main timecourse analysis was conducted using a subsample of the groups selected to match on age. This sample had 37 NT (M_{age} = 27.68, SD = 6.21) and 15 ASC (M_{age} = 29.76, SD = 6.03; $F_{(1,50)}$ = 1.12, MSE = 39.45, p = .30) participants. Using this subsample, results were consistent with the main analysis, the alexithymia model outperformed all models, including age (ΔAIC = 6660; **see also separate analyses by group)**.

8.1.3.5 Separate analyses by group

It may also be argued that comparing the autism model (based on diagnosis) to alexithymia is not sensitive to variance in autistic traits or symptom severity, and that trait or symptom severity models may be better predictors of eye gaze than a binary diagnosis variable. As detailed in the main text, autistic trait models usually performed worse than or similar to the autism diagnosis model which were usually eliminated in a refitted backwards selection when estimated jointly with alexithymia. However, to guard against potential confounds of group differences in score ranges and variance, timecourse analyses were re-fitted separately for each group, while controlling for autistic traits and alexithymia in neurotypical controls and the autistic group separately. In addition to these analyses, the effect of ADOS scores was also examined in the autistic group. Results were consistent with previous analyses. As

shown in Table 8.4 Model comparison by group**.** alexithymia outperformed all models in predicting eye gaze behaviour in both autistic and neurotypical control individuals.

Table 8.4 Model comparison by group

Group	Condition	AQ	ADOS	Alexithymia
Autism				
Δ -2LL	742491	738507	738290	737317
X^2	3102	881	216.94	972.64
ΔAIC	3073	841	217	973
ΔBIC	2920	640	217	972
$p < .001$	***	***	***	***
Neurotypicals				
Δ -2LL	__	1197091	__	1196668
X^2 *(1)*	__	548.34	__	422.27
ΔAIC	__	508	__	423
ΔBIC	__	294	__	422
$p < .001$	__	***	__	***

Notes. All models were fitted with an interaction with the fourth-order polynomials and random effects for participants and video. As the polynomials capture the non-linear trajectory of gaze behaviour, including additional predictors for different models (e.g., autism, alexithymia, and condition) allows for testing whether these predictors provide an improved fit and predict differences over time. Note that likelihood tests are only valid for nested models. Non-nested model comparisons are restricted to AIC and BIC comparisons only.

8.1.3.6 Additional data quality checks

In a series of additional control analyses, it was confirmed that the results reported above could not be explained by differences in data quality parameters such as precision, measured as the RMS of successive gaze samples ($t_{(1,68)} = 1.44$, ns), nor fixation kinematics assessed by the standard deviation in point of gaze indicating the relative dispersion or stability of each fixation ($F_{Group(1,68)} = 0.11$, ns, $F_{Condition\ (2.14,145)} = 1.11$, ns, $F_{Group*Condition(2.14,\ 145)} = 0.57$, ns).

8.2 Chapter 3. Supplementary Materials

8.2.1 Experiment 2

Table 8.5 Full Model Details – Experiment 2

	DV: Face Action		
Predictors	*Estimates*	*CI*	*p*
(Intercept)	1.48	-0.20 – 3.17	0.084
ot1	1.23	-4.06 – 6.53	0.648
ot2	-1.53	-4.42 – 1.35	0.297
ot3	3.14	1.20 – 5.09	**0.002**
Emotion1	-0.36	-4.12 – 3.41	0.853
Emotion2	-20.61	-24.37 – -16.85	< **0.001**
Emotion3	22.75	18.98 – 26.51	< **0.001**
Emotion4	-18.26	-22.02 – -14.50	< **0.001**
Emotion5	-8.50	-12.26 – -4.74	< **0.001**
Stimulus set1	2.94	1.26 – 4.62	**0.001**
Stimulus type1	2.06	0.38 – 3.74	**0.016**
ot1 * Emotion1	6.61	-5.22 – 18.45	0.273
ot1 * Emotion2	-56.94	-68.77 – -45.10	< **0.001**
ot1 * Emotion3	65.43	53.60 – 77.27	< **0.001**
ot1 * Emotion4	-53.62	-65.46 – -41.79	< **0.001**
ot1 * Emotion5	-25.57	-37.41 – -13.74	< **0.001**
ot2 * Emotion1	6.41	-0.04 – 12.85	0.051
ot2 * Emotion2	21.62	15.18 – 28.06	< **0.001**
ot2 * Emotion3	-24.25	-30.69 – -17.81	< **0.001**
ot2 * Emotion4	20.00	13.56 – 26.44	< **0.001**
ot2 * Emotion5	7.78	1.34 – 14.22	**0.018**
ot3 * Emotion1	-4.96	-9.31 – -0.61	**0.025**

ot3 * Emotion2	-9.64	-13.99 – -5.29	< **0.001**
ot3 * Emotion3	10.13	5.78 – 14.48	< **0.001**
ot3 * Emotion4	-5.53	-9.88 – -1.19	**0.013**
ot3 * Emotion5	-4.34	-8.69 – 0.00	0.050
ot1 * Stimulus set1	5.19	-0.10 – 10.49	0.054
ot2 * Stimulus set1	-4.15	-7.03 – -1.27	**0.005**
ot3 * Stimulus set1	3.06	1.11 – 5.00	**0.002**
Emotion1 * Stimulus set1	-9.91	-13.67 – -6.15	< **0.001**
Emotion2 * Stimulus set1	-10.95	-14.71 – -7.18	< **0.001**
Emotion3 * Stimulus set1	18.02	14.26 – 21.78	< **0.001**
Emotion4 * Stimulus set1	-7.37	-11.13 – -3.61	< **0.001**
Emotion5 * Stimulus set1	-3.90	-7.66 – -0.13	**0.042**
ot1 * Stimulus type1	1.83	-3.46 – 7.12	0.499
ot2 * Stimulus type1	-3.66	-6.54 – -0.78	**0.013**
ot3 * Stimulus type1	2.39	0.45 – 4.34	**0.016**
Emotion1 * Stimulus type1	-0.46	-4.22 – 3.30	0.812
Emotion2 * Stimulus type1	-4.07	-7.84 – -0.31	**0.034**
Emotion3 * Stimulus type1	6.93	3.17 – 10.69	< **0.001**
Emotion4 * Stimulus type1	-5.29	-9.05 – -1.53	**0.006**
Emotion5 * Stimulus type1	-1.92	-5.68 – 1.84	0.318
Stimulus set1 * Stimulus type1	0.98	-0.70 – 2.66	0.254
ot1 * Emotion1 * Stimulus set1	-27.87	-39.70 – -16.03	< **0.001**
ot1 * Emotion2 * Stimulus set1	-25.12	-36.96 – -13.29	< **0.001**
ot1 * Emotion3 * Stimulus set1	47.54	35.70 – 59.37	< **0.001**
ot1 * Emotion4 * Stimulus set1	-14.79	-26.63 – -2.96	**0.014**
ot1 * Emotion5 * Stimulus set1	-12.62	-24.46 – -0.79	**0.037**
ot2 * Emotion1 * Stimulus set1	9.33	2.89 – 15.78	**0.005**

ot2 * Emotion2 * Stimulus set1	15.24	8.80 – 21.68	< **0.001**
ot2 * Emotion3 * Stimulus set1	-23.60	-30.05 – -17.16	< **0.001**
ot2 * Emotion4 * Stimulus set1	10.93	4.49 – 17.37	**0.001**
ot2 * Emotion5 * Stimulus set1	4.04	-2.41 – 10.48	0.219
ot3 * Emotion1 * Stimulus set1	-6.27	-10.62 – -1.93	**0.005**
ot3 * Emotion2 * Stimulus set1	-10.46	-14.80 – -6.11	< **0.001**
ot3 * Emotion3 * Stimulus set1	7.15	2.80 – 11.49	**0.001**
ot3 * Emotion4 * Stimulus set1	-6.43	-10.78 – -2.09	**0.004**
ot3 * Emotion5 * Stimulus set1	1.26	-3.08 – 5.61	0.569
ot1 * Emotion1 * Stimulus type1	4.06	-7.78 – 15.89	0.502
ot1 * Emotion2 * Stimulus type1	6.65	-5.18 – 18.48	0.271
ot1 * Emotion3 * Stimulus type1	0.34	-11.50 – 12.17	0.955
ot1 * Emotion4 * Stimulus type1	0.73	-11.11 – 12.56	0.904
ot1 * Emotion5 * Stimulus type1	3.82	-8.02 – 15.65	0.527
ot2 * Emotion1 * Stimulus type1	5.97	-0.48 – 12.41	0.070
ot2 * Emotion2 * Stimulus type1	19.36	12.92 – 25.80	< **0.001**
ot2 * Emotion3 * Stimulus type1	-24.67	-31.11 – -18.23	< **0.001**
ot2 * Emotion4 * Stimulus type1	20.53	14.09 – 26.97	< **0.001**
ot2 * Emotion5 * Stimulus type1	8.90	2.45 – 15.34	**0.007**
ot3 * Emotion1 * Stimulus type1	-1.71	-6.06 – 2.63	0.440
ot3 * Emotion2 * Stimulus type1	-7.74	-12.09 – -3.39	< **0.001**
ot3 * Emotion3 * Stimulus type1	10.28	5.93 – 14.63	< **0.001**
ot3 * Emotion4 * Stimulus type1	-7.20	-11.54 – -2.85	**0.001**
ot3 * Emotion5 * Stimulus type1	-5.79	-10.14 – -1.45	**0.009**
ot1 * Stimulus set1 * Stimulus type1	-3.13	-8.43 – 2.16	0.246
ot2 * Stimulus set1 * Stimulus type1	-4.31	-7.19 – -1.42	**0.003**
ot3 * Stimulus set1 * Stimulus type1	3.18	1.23 – 5.12	**0.001**

Emotion1 * Stimulus set1 * Stimulus type1	-1.35	-5.11 – 2.41	0.482
Emotion2 * Stimulus set1 * Stimulus type1	-4.31	-8.07 – -0.55	**0.025**
Emotion3 * Stimulus set1 * Stimulus type1	4.61	0.85 – 8.37	**0.016**
Emotion4 * Stimulus set1 * Stimulus type1	-2.61	-6.37 – 1.15	0.173
Emotion5 * Stimulus set1 * Stimulus type1	-1.36	-5.12 – 2.41	0.480
ot1 * Emotion1 * Stimulus set1 * Stimulus type1	4.97	-6.86 – 16.81	0.410
ot1 * Emotion2 * Stimulus set1 * Stimulus type1	2.22	-9.62 – 14.05	0.714
ot1 * Emotion3 * Stimulus set1 * Stimulus type1	-5.03	-16.87 – 6.80	0.405
ot1 * Emotion4 * Stimulus set1 * Stimulus type1	4.68	-7.15 – 16.52	0.438
ot1 * Emotion5 * Stimulus set1 * Stimulus type1	-3.53	-15.37 – 8.30	0.559
ot2 * Emotion1 * Stimulus set1 * Stimulus type1	9.63	3.19 – 16.07	**0.003**
ot2 * Emotion2 * Stimulus set1 * Stimulus type1	14.95	8.50 – 21.39	< **0.001**
ot2 * Emotion3 * Stimulus set1 * Stimulus type1	-21.84	-28.28 – -15.39	< **0.001**
ot2 * Emotion4 * Stimulus set1 * Stimulus type1	11.13	4.69 – 17.58	**0.001**
ot2 * Emotion5 * Stimulus set1 * Stimulus type1	2.47	-3.97 – 8.91	0.452
ot3 * Emotion1 * Stimulus set1 * Stimulus type1	-2.90	-7.25 – 1.45	0.191
ot3 * Emotion2 * Stimulus set1 * Stimulus type1	-10.50	-14.85 – -6.15	< **0.001**

ot3 * Emotion3 * Stimulus set1 * Stimulus type1	6.50	2.15 – 10.84	**0.003**
ot3 * Emotion4 * Stimulus set1 * Stimulus type1	-6.94	-11.28 – -2.59	**0.002**
ot3 * Emotion5 * Stimulus set1 * Stimulus type1	1.39	-2.96 – 5.74	0.530

Random Effects

σ^2	52.79
$\tau_{00\ stim\_id}$	140.08
$\tau_{00\ stim\_id.1}$	136.14
$\tau_{11\ stim\_id.ot1}$	1347.26
$\tau_{11\ stim\_id.ot2}$	362.23
$\rho_{01\ stim\_id.ot1}$	0.90
$\rho_{01\ stim\_id.ot2}$	-0.78
ICC	0.78
$N_{\ stim\_id}$	192
Observations	7680
Marginal R^2 / Conditional R^2	.73 / .94

Notes. ot: polynomials. Stimulus set (1 = Low prototypicality; 2 = High prototypicality).

Stimulus type (1 = Morphed, 2 = Original) Emotion (Reference = Anger, 1 = Disgust, 2 = Fear,

3 = Happiness, 4 = Sadness, 5 = Surprise).

8.3 Chapter 4. Supplementary Materials

8.3.1 Experiment 5 & 6

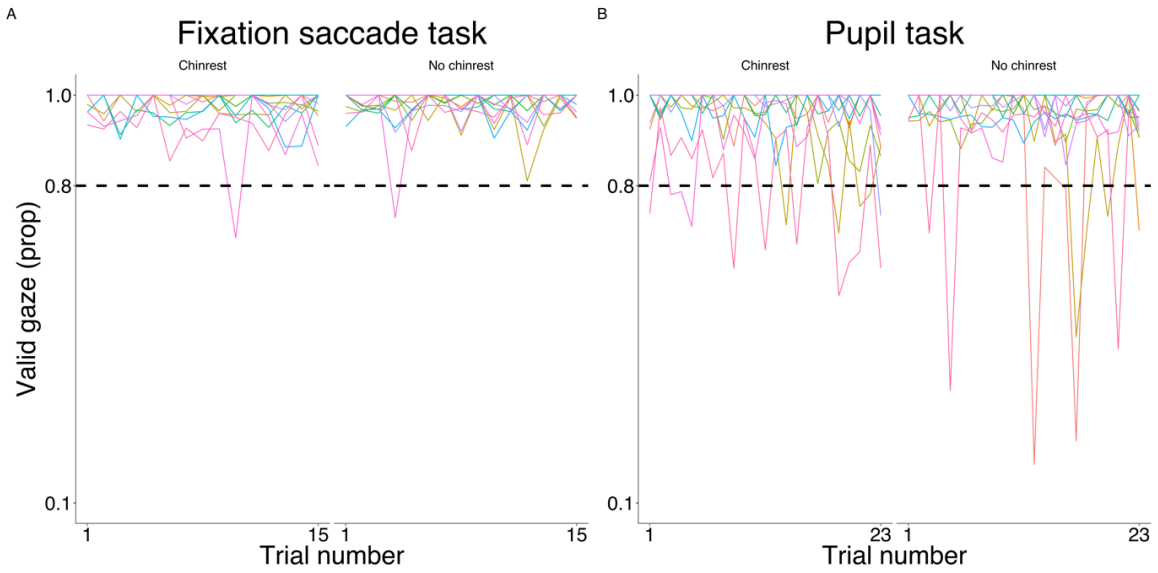


Figure 8.5 The proportion of valid gaze across conditions (chinrest, no-chinrest) and tasks (Fixation-Saccade, PLR task).

Coloured lines represent data loss over trials for each participant.

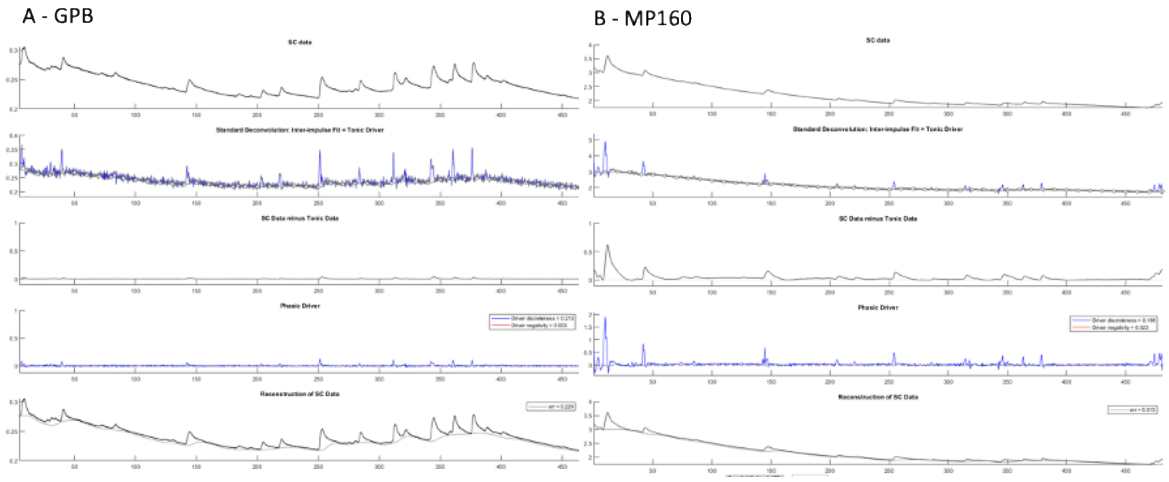


Figure 8.6 Sample plots for GPB and BIOPAC.

Data from one representative participant in an equivalent portion of the task (the first block) from the Gazepoint Biometrics System (GPB) and the BIOPAC MP160.

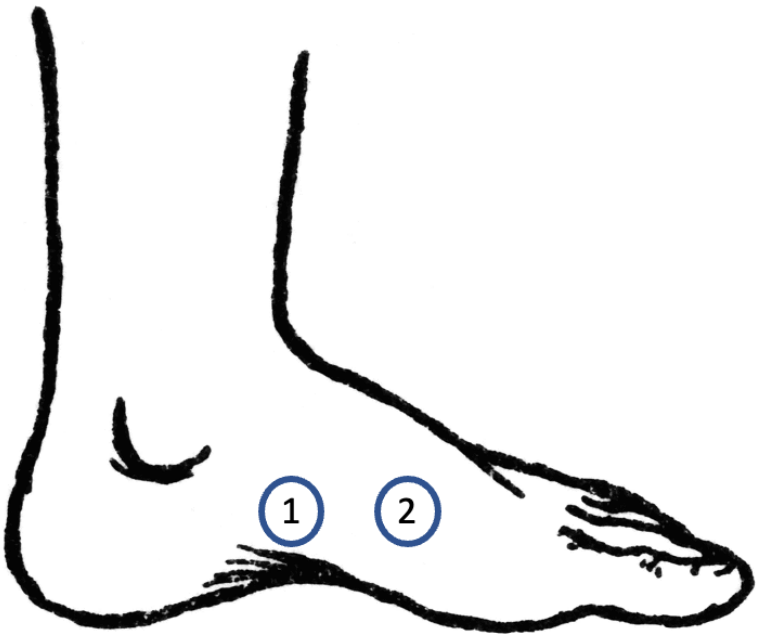


Figure 8.7 Diagram demonstrating where the BIOPAC foot sensors were attached.

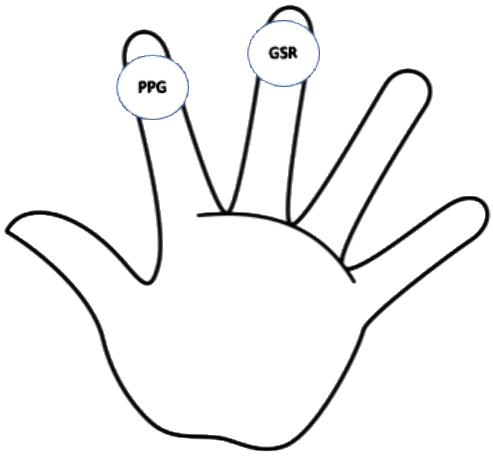


Figure 8.8 Diagram of the Gazepoint biometrics recording setup.

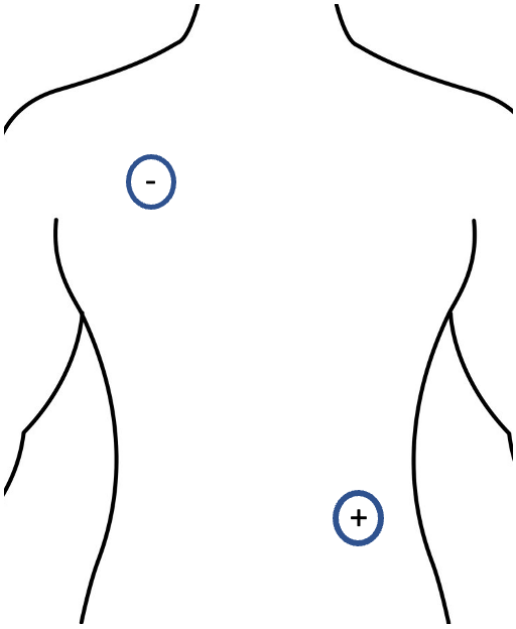


Figure 8.9 Diagram demonstrating where the BIOPAC torso sensors were attached.

8.3.2 List of stimuli

1019.jpg; 1302.jpg; 1350.jpg; 1440.jpg; 1920.jpg; 2035.jpg; 2071.jpg; 2156.jpg; 2301.jpg; 2345.jpg; 2374.jpg; 2392.jpg; 2400.jpg; 2457.jpg; 2550.jpg; 2770.jpg; 2900.jpg; 3010.jpg; 3069.jpg; 3103.jpg; 3130.jpg; 3170.jpg; 3185.jpg; 3301.jpg; 4230.jpg; 4311.jpg; 4530.jpg; 4573.jpg; 4574.jpg; 4631.jpg; 4800.jpg; 5040.jpg; 5199.jpg; 5530.jpg; 5665.jpg; 5725.jpg; 5760.jpg; 5836.jpg; 6250.jpg; 6555.jpg; 7026.jpg; 7038.jpg; 7052.jpg; 7325.jpg; 7354.jpg; 7477.jpg; 8200.jpg; 8312.jpg; 8330.jpg; 9320.jpg; 9440.jpg; 9472.jpg; 9561.jpg; 9622.jpg; 9800.jpg; 9926.jpg

8.3.3 Experiment 7

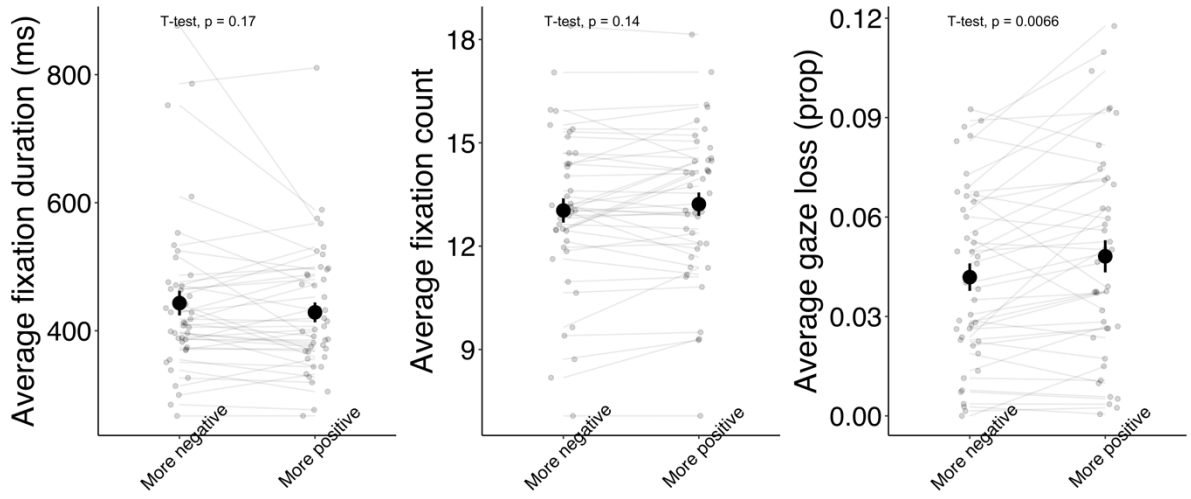


Figure 8.10 Gaze visualisations and analyses suggest no notable relations between gaze metrics and valence.

8.3.4 Experiment 8

Table 8.6 Descriptive values for aggregated self-reports and physiological data

	NT		**ASD**	
	M	SD	M	SD
TAS	42.32	11.22	63	14.69
AQ	17.28	7.09	35.6	6.60
Age	24.70	6.05	29.82	7.67
WASI	28.65	14.53	33.2	12.59
SCR (log)	0.51	0.39	0.40	0.35
Heart rate difference	-2.64	1.42	-2.14	1.70
Pupil	-0.72	1.05	-0.40	0.80
Arousal	-0.83	2.09	-0.94	3.32
Valence	0.08	0.93	0.19	1.35

Table 8.7 Correlation of aggregated self-reports and physiological data

Variable	1	2	3	4	5	6	7	8	9
1. TAS	—								
2. AQ	.641 ***	—							
3. SCR	-.338 *	-.180	—						
4. HR	-.023	.103	-.449 ***	—					
5. Pupil	.142	.156	-.107	.143	—				
6. Arousal	.036	-.023	.005	.122	-.010	—			
7. Valence	-.064	.048	-.181	.168	.059	-.442 ***	—		
8. Age	-.125	.225	-.194	.492 ***	.163	.139	.199	—	
9. WASI	.304 *	.539 ***	-.115	.108	.191	-.177	-.051	.407 **	—

Notes. * $p < .05$, ** $p < .01$, *** $p < .001$

8.4 Chapter 5. Supplementary Materials

Table 8.8 Descriptive measures for Experiment 9 & 10

	Experiment 9 N = 522		Experiment 10 N = 849			
Variable	Mean	SD	Mean	SD	t	p
Age	28.46	12.28	28.19	9.67	0.46	ns
DIF	16.20	5.72	15.01	6.09	3.60	***
EOT	18.20	4.28	18.74	4.72	-2.14	*
DDF	13.22	4.53	13.05	4.77	0.68	ns
SS	20.75	5.66	21.57	5.45	-2.69	**
AS	24.17	4.75	24.74	4.48	-2.24	*
ATD	24.59	4.98	24.87	5.23	-0.98	ns
COM	20.18	5.06	20.34	5.18	-0.56	ns
IMG	19.20	4.45	20.16	4.70	-3.74	***
TAS-20	47.62	11.57	46.80	12.49	1.22	ns
AQ-50	108.88	18.06	111.68	17.47	-2.84	**

Notes. DIF = Difficulties identifying feelings; EOT = Externally oriented thinking; DDF = Difficulties describing feelings; SS = Social skills; ATD = Attention to detail; AS = Attention switching; COM = Communication; IMG = Imagination; TAS-20 = Toronto Alexithymia Scale - 20; AQ-50 = Autism Spectrum Quotient Scale – 50; * $p < .05$, ** $p < .01$, *** $p < .001$.

8.4.1 Experiment 9

8.4.1.1 Exploratory Factor Analysis (EFA)

Table 8.9 List of all AQ-50 and TAS-20 items

Item	Description
TAS_1	I am often confused about what emotion I am feeling.
TAS_2	It is difficult for me to find the right words for my feelings.
TAS_3	I have physical sensations that even doctors don't understand.
TAS_4	I am able to describe my feelings easily.
TAS_5*	I prefer to analyse problems rather than just describe them.
TAS_6	When I am upset, I don't know if I am sad, frightened, or angry.
TAS_7	I am often puzzled by sensations in my body.
TAS_8	I prefer to just let things happen rather than to understand why they turned out that way.
TAS_9	I have feelings that I can't quite identify.
TAS_10	Being in touch with emotions is essential.
TAS_11	I find it hard to describe how I feel about people.
TAS_12	People tell me to describe my feelings more.
TAS_13	I don't know what's going on inside me.
TAS_14	I often don't know why I am angry.
TAS_15	I prefer talking to people about their daily activities rather than their feelings.
TAS_16*	I prefer to watch "light" entertainment shows rather than psychological dramas.
TAS_17	It's difficult for me to reveal my innermost feelings, even to close friends.
TAS_18	I can feel close to someone, even in moments of silence
TAS_19	I find examination of my feelings useful in solving personal problems.
TAS_20*	Looking for hidden meanings in movies or plays distracts from their enjoyment.
AQ_1	I prefer to do things with others rather than on my own.
AQ_2*	I prefer to do things the same way over and over again.
AQ_3	If I try to imagine something, I find it very easy to create a picture in my mind.
AQ_4*	I frequently get so strongly absorbed in one thing that I lose sight of other things.
AQ_5*	I often notice small sounds when others do not.
AQ_6	I usually notice car number plates or similar strings of information.
AQ_7	Other people frequently tell me that what I've said is impolite, even though I think it is polite.
AQ_8	When I'm reading a story, I can easily imagine what the characters might look like.
AQ_9*	I am fascinated by dates.

AQ_10*	In a social group, I can easily keep track of several different people's conversations.
AQ_11	I find social situations easy.
AQ_12*	I tend to notice details that others do not.
AQ_13	I would rather go to a library than a party.
AQ_14	I find making up stories easy.
AQ_15	I find myself drawn more strongly to people than to things.
AQ_16	I tend to have very strong interests which I get upset about if I can't pursue.
AQ_17	I enjoy social chit-chat.
AQ_18	When I talk, it isn't always easy for others to get a word in edgeways.
AQ_19*	I am fascinated by numbers.
AQ_20	When I'm reading a story, I find it difficult to work out the characters' intentions.
AQ_21	I don't particularly enjoy reading fiction.
AQ_22	I find it hard to make new friends.
AQ_23	I notice patterns in things all the time.
AQ_24	I would rather go to the theatre than a museum.
AQ_25	It does not upset me if my daily routine is disturbed.
AQ_26	I frequently find that I don't know how to keep a conversation going.
AQ_27*	I find it easy to "read between the lines" when someone is talking to me.
AQ_28*	I usually concentrate more on the whole picture, rather than the small details.
AQ_29	I am not very good at remembering phone numbers.
AQ_30*	I don't usually notice small changes in a situation, or a person's appearance.
AQ_31*	I know how to tell if someone listening to me is getting bored.
AQ_32*	I find it easy to do more than one thing at once.
AQ_33*	When I talk on the phone, I'm not sure when it's my turn to speak.
AQ_34	I enjoy doing things spontaneously.
AQ_35	I am often the last to understand the point of a joke.
AQ_36*	I find it easy to work out what someone is thinking or feeling just by looking at their face.
AQ_37*	If there is an interruption, I can switch back to what I was doing very quickly.
AQ_38	I am good at social chit-chat.
AQ_39	People often tell me that I keep going on and on about the same thing.
AQ_40	When I was young, I used to enjoy playing games involving pretending with other children.
AQ_41*	I like to collect information about categories of things (e.g. types of car, types of bird, types of train, types of plant, etc.).
AQ_42*	I find it difficult to imagine what it would be like to be someone else.
AQ_43*	I like to plan any activities I participate in carefully.
AQ_44*	I enjoy social occasions.

AQ_45	I find it difficult to work out people's intentions.
AQ_46	New situations make me anxious.
AQ_47	I enjoy meeting new people.
AQ_48	I am a good diplomat.
AQ_49	I am not very good at remembering people's date of birth.
AQ_50	I find it very easy to play games with children that involve pretending.

Notes. * denotes excluded items in the joint EFA.

Table 8.10 Factor loadings for joint EFA

	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5	Uniqueness
TAS_1		.894				.359
TAS_2		.839			.413	.307
TAS_3		.540				.663
TAS_4		.713			.484	.332
TAS_5						.915
TAS_6		.778				.454
TAS_7		.658				.583
TAS_8						.848
TAS_9		.900				.386
TAS_10						.678
TAS_11		.625				.486
TAS_12		.489			.426	.617
TAS_13		.849				.415
TAS_14		.619				.610
TAS_15						.675
TAS_16						.887
TAS_17					.439	.645
TAS_18						.863
TAS_19						.751
TAS_20						.888
AQ_1	.642					.663
AQ_2						.761
AQ_3						.839
AQ_4						.767
AQ_5						.708
AQ_6				.640		.578
AQ_7						.711
AQ_8			.490			.754
AQ_9				.533		.705
AQ_10						.640
AQ_11	.823					.275
AQ_12				.598		.609
AQ_13	.713					.575
AQ_14						.833
AQ_15	.538					.599
AQ_16						.709
AQ_17	.832					.417
AQ_18						.820
AQ_19				.547		.686
AQ_20			.633			.616

	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5	Uniqueness
AQ_21						.940
AQ_22	.686					.496
AQ_23				.712		.483
AQ_24						.879
AQ_25						.674
AQ_26	.542					.462
AQ_27			.684			.479
AQ_28						.843
AQ_29						.903
AQ_30						.851
AQ_31			.435			.773
AQ_32			.409			.698
AQ_33						.602
AQ_34	.593					.596
AQ_35						.710
AQ_36			.622			.545
AQ_37						.663
AQ_38	.786					.315
AQ_39						.662
AQ_40			.424			.783
AQ_41				.561		.660
AQ_42			.512			.694
AQ_43						.815
AQ_44	.900					.316
AQ_45			.636			.463
AQ_46	.491					.615
AQ_47	.810					.406
AQ_48						.677
AQ_49						.962
AQ_50						.804

Notes. Applied rotation method is *promax*. Factor 1 = Social skills (SOC); Factor 2 = Feelings and sensations (FEE); Factor 3 = Flexibility and imagination (FLEX); Factor 4 = Externally oriented thinking (EOT); Factor 5 = Attention to detail (ATD).

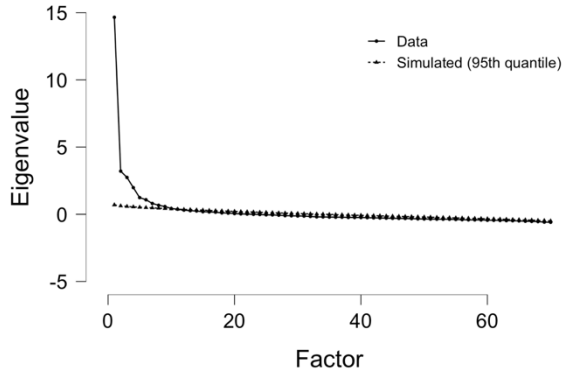


Figure 8.11 Scree plot of the joint sample EFA.

8.4.1.2 Network Analysis

Analysis of all items

Centrality

A few *social skills* (autism) and *feelings and sensation* (alexithymia) items showed the highest centrality, indicating that these items were strongly connected with other terms in the network (e.g., AQ_11, AQ_45, AQ_26, AQ_44, TAS_13, AQ_38, TAS_2, TAS_1). Conversely, a mix of *attention to detail* (autism), *imagination* (autism), and *externally oriented thinking* (alexithymia) items had standardised centrality below zero, indicating that they were very weekly connected nodes (e.g., AQ_24, AQ_28, TAS_20, TAS_5, TAS_8, TAS16, AQ_18, AQ_49).

A. Neurotypical B. Clinical

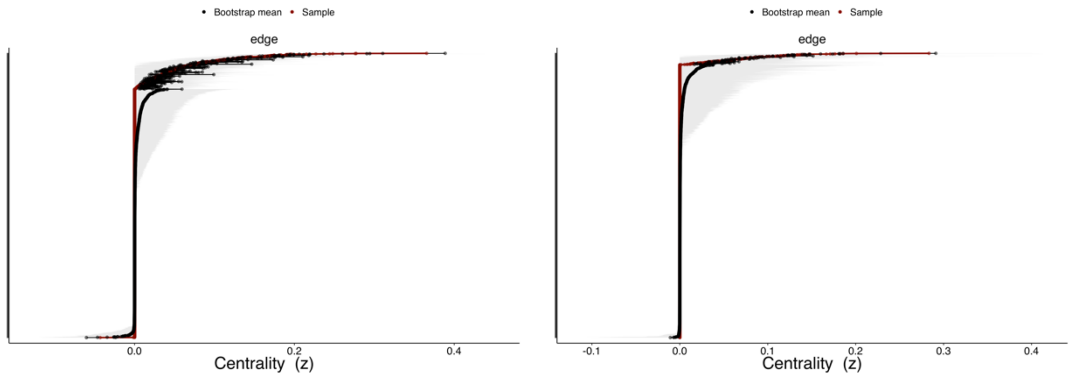


Figure 8.12 Bootstrapped confidence intervals for edge strength for neurotypical and clinical groups respectively.

Edges with CI including zero are not different.

Factor score network analysis

Network estimation

The estimated networks using factor scores are visualised in **Figure 8.13.** As in the analyses with all items, the stronger connections appear within autism and alexithymia factors, with generally positive correlations across these clusters. Both networks are highly similar with the strongest edges emerging between difficulties describing and identifying feelings (DDF - DIF) with a regularised partial correlation (RPC) of .47 for neurotypicals and .51 for clinical participants, whereas all other RCPs between DDF or DIF and autism factors were < .15. Similarly, communication and social skills (COM – SOC) showed the strongest RCP – .42 and .44 for the neurotypical and clinical groups respectively. Notably, one edge is slightly stronger in the neurotypical sample network, namely EOT – IMG (RCP = .21) compared to the clinical sample (RCP = .12).

Network inference

Centrality metrics are displayed in **Figure 8.13.C**. The correlation between centrality measures in ordered nodes was generally high, .81 for clinical *vs.* neurotypical networks, .96 for clinical *vs.* full sample, and .92 for NT vs. full sample. Whereas *communication* (COM) and *social skills* (SOC) were the most central factor traits in both networks, *difficulties identifying feelings* was more central in the neurotypical network compared to the clinical network. *Attention to detail* (ATD), *externally oriented thinking* (EOT) and *imagination* (IMG) were consistently low in centrality.

A. Neurotypical B. Clinical C

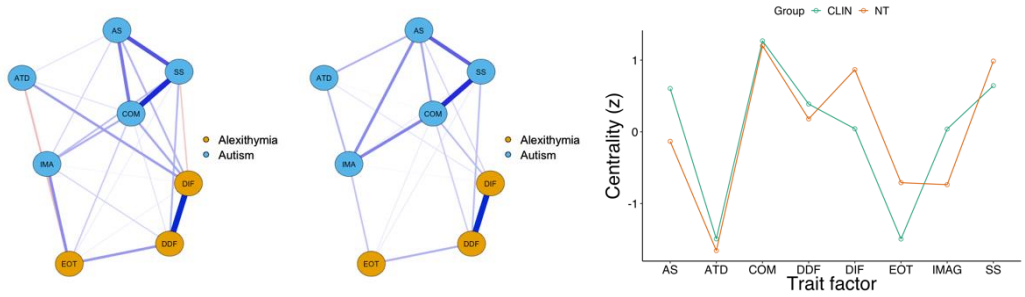


Figure 8.13 Estimated networks using factor scores.

A & B. Estimated networks using factor trait scores. C. Standardised centrality for all factors traits. As in the network based on individual items, the node predictability for factors was relatively high, with neighbouring nodes sharing approximately 41% of the variance for the NT group, and 52% for the clinical group. Node predictability was very strongly correlated with centrality (.94 for NT and .99 for the clinical groups).

Network stability

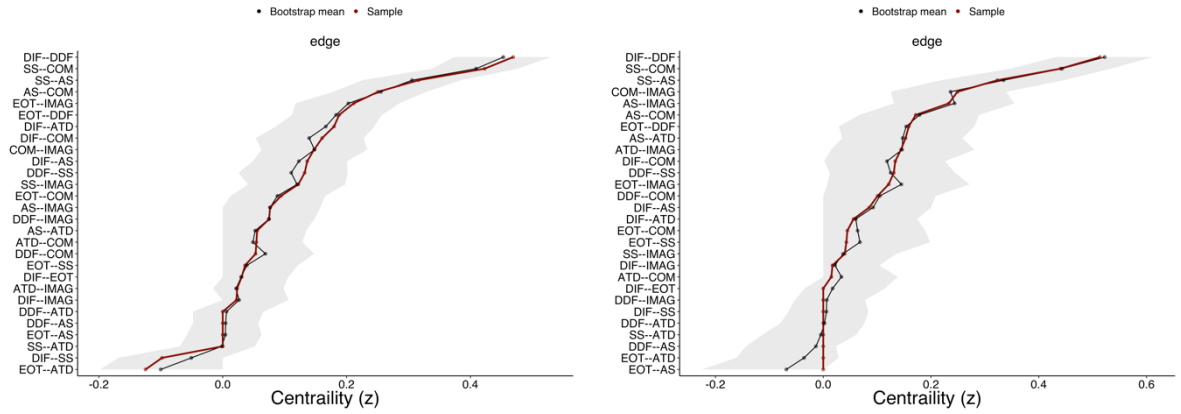
The correlation stability coefficient (CSC) was .75 for the NT group and .67 for the clinical group, both values above the recommended .50 cut-off. This indicates that the network structures were robust and stable (see **Figure 8.14 Network stability and inference**). Edges *social and communication* (SOC-COM) and *difficulties identifying and describing feelings* (DIF-DFF) were significantly stronger than all other edges, but they did not differ.

Network comparison

Edge weights were highly similar across networks with very high correlations (NT – CLIN = .86, NT – full sample = .97 and CLIN vs full sample = .91). The network invariance test was significant (M = 0.295, $p < .001$) but there were no differences in global strength ($Strenght_{NT}$ = 2.944286, $Strenght_{CLIIN}$ = 3.14; S = 0.20, $p = .63$) nor in individual edges at $p < .05$ (Bonferroni corrected). These results suggest that networks are similar.

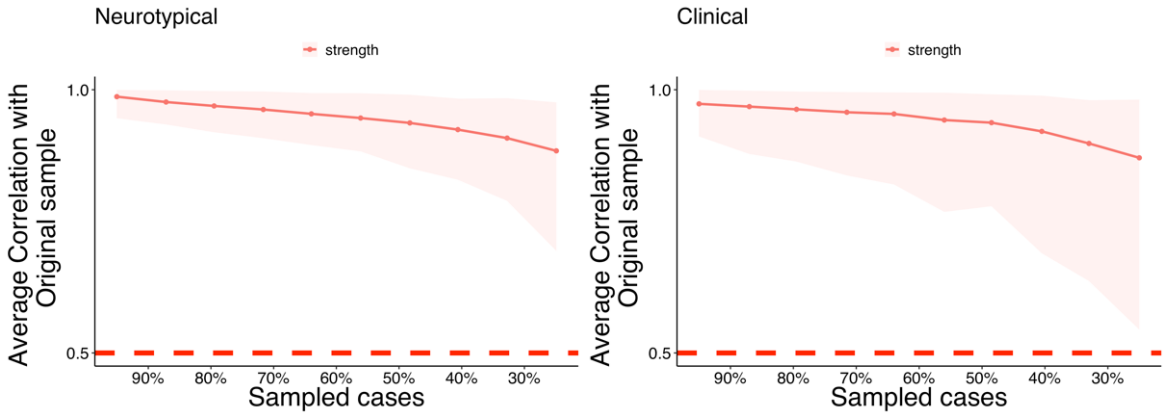
A. Neurotypical

B. Clinical



C

D



E

F

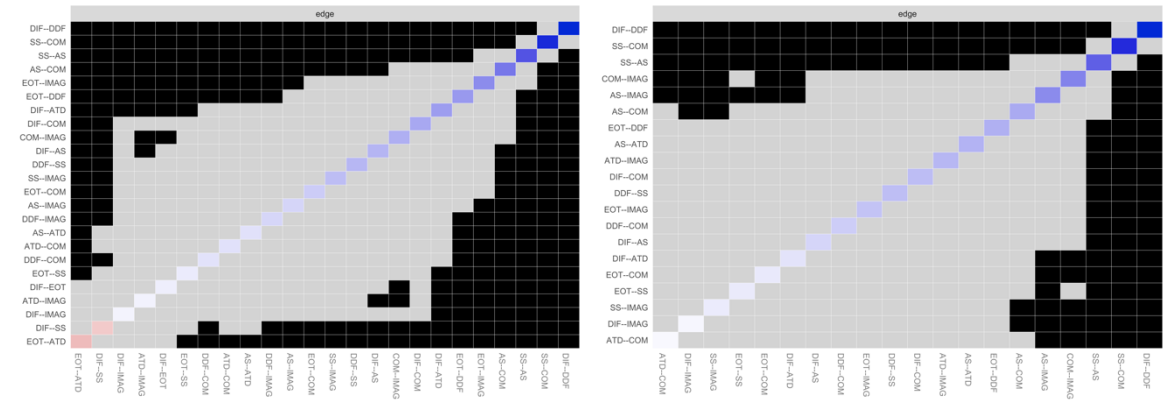


Figure 8.14 Network stability and inference.

Network stability and inference. A & B. 95% bootstrapped CI around edge weights. Overlapping pairs CIs and CIs including zero are not significant. C & D. The correlation stability coefficient (y-axis) per dropped cases (x-axis). All networks were estimated reliably.

E & F. Edge difference plot, comparing edge pairs (strength of pairwise trait connections). Darker squares represent significant ($p < .05$) difference between the pairwise edge comparisons. Within-alexithymia and within-autism connections are significantly stronger than alexithymia-autism connections.

8.4.2 Experiment 10

8.4.2.1 Confirmatory Factor Analysis: Model specification

Model A. Four versions of this model were tested based on the results from Experiment 9 which suggested a six-factor solution: *social skills (SOC), feelings and sensations (FEL), flexibility (FLEX); externally oriented thinking (EOT), imagination (IMG), attention to detail (ATD).*

Model A.1. Included only the six factors with no second-order factors. This model tests the hypothesis that the facets (factors) of autism and alexithymia are distinct.

Model A.2. Included the six factors and two second-order terms for alexithymia and autism. This model tests the hypothesis that while autistic traits and alexithymia may be correlated, they reflect separate underlying latent dimensions.

Model A.3. Included the six factors and one higher order factor. This model tests the hypothesis that all autism and alexithymia traits are a product of a single casual latent factor (which may be autism itself).

Model A.4. Similar to A.1, but *ATD* and *EOT* were included as separate second order-factors, as these items cluster less consistently than other aspects of autism and alexithymia.

Model A.5. Was a bi-factor model, a variation of the general models, where a general factor explains the variance of observed trait symptoms orthogonally to specific factors.

Model B. Based on the factor structures proposed by the questionnaire authors - the 5 factor solution for the AQ-50: *social skills*, *communication*, *imagination*, *attention to details* and *attention switching* (Baron-Cohen et al., 2001) and 3-factor solution for TAS-20: *difficulties identifying feelings*, *difficulties describing feelings* and *externally oriented thinking* (Bagby, Parker, et al., 1994). Three versions of this model were fitted.

Model B.1. Included the individual factors (8) and no second-order factors.

Model B.2. In addition to the first-order terms in B.1, two correlated second-order factors for autism and alexithymia were included.

Model B.3. A model specifying a common latent second-order factor underlying all alexithymia and autism factors.

Given that previous literature suggests that the original factor solutions for the TAS-20 and AQ-50 often underperform in comparison to alternative models (as also observed in Experiment 9), additional models were fitted (Model C) using the best performing alternative solutions identified in meta-analyses and review studies (English et al., 2020). These were also the best preforming models of the individual scales in Experiment 9 (see section CFA of Individual Measures below). The fitted models included three factors for the AQ (*social*, *communication* and *attention patterns*), and four factors for the TAS-20 (*DIF, DDF, EOT and a method factor for reversed items*) (Preece et al. 2020; Watters et al. 2016).

Model C.1. This model specified a correlation between all 6 factors and no second-order factor (like A.1. and B.1.).

Model C.2. Included two second-order correlated factors for autism and alexithymia.

Model C.3. Included one single latent common second-order factor.

An illustration of the fitted models is visualised in **Figure 5.4** in the main text.

Table 8.11 Confirmatory factor analysis: Fit indices and model comparisons

Model	df	AIC	BIC	X^2	X^2diff	DF	p	RMSEA	CFI	CTI
MODEL A										
Model A.1	887	99783.15	100271.8	3169.427	NA	NA		.055[.53,.57]***	.81	.79
Model A.2	893	99870.02	100330.2	3268.297	98.87	6	***	.56[.54,.58]***	.79	.79
Model A.3	895	99866.68	100317.4	3268.957	0.66	2	ns	.56[.54,.58]***	.79	.79
ModelA.4	896	99869.59	100315.5	3273.872	4.91	1	*	.56[.54,.58]***	.79	.79
Model A.5	849	99673	100342	2983.6	1			.054[.52,.057] ***	.81	.79
MODEL B										
Model B.1	2249	155524.2	156311.7	7973.631	NA	NA		.055[.053, .56]***	.67	.66
Model B.2	2268	155574.1	156271.5	8061.51	87.88	19	**	.055[.054, .56]***	.67	.66
Model B.3	2269	156041	156733.6	8530.41	468.90	1	***	.057[.056, .058]***	.64	.63
MODEL C										
Model C.1	964	103680.4	104235.5	2977.54	NA	NA		.050[.048,.052]ns	.85	.83
Model C.2	977	103718	104211.3	3041.05	63.51	13	***	.050[.048,.052]ns	.84	.83
Model C.3	978	103794.3	104282.9	3119.364	78.31	1	***	.051[.049,.053]ns	.84	.83

Notes. df = Degrees of freedom for the model; AIC and BIC = Akaike and Bayesian information criteria, respectively. These can be used to compare models directly (lower value models are preferred). X^2 = Chi-square test for each model solution; X^2 diff = The difference in chi-square relative to the nested model, this comparison is only valid for nested models. RMSEA = Root Mean Squared Error of Approximation, the p-value indicates whether RMSEA is below the recommended cut-off. CFI (Comparative Fit Index) and TLI (Tucker-Lewis Index) are goodness of fit measures, values > .80 are generally considered acceptable. * $p < .05$, ** $p < .01$, *** $p < .001$

Table 8.12 Descriptive statistics for all items

Variable	Mean	SD	Min	Max
AQ_1	2.46	0.90	1	4
AQ_3	1.86	0.90	1	4
AQ_6	2.40	1.09	1	4
AQ_7	1.75	0.94	1	4
AQ_8	1.88	0.89	1	4
AQ_11	2.37	0.96	1	4
AQ_12	2.95	0.94	1	4
AQ_13	2.21	1.02	1	4
AQ_14	2.37	1.02	1	4
AQ_15	2.20	0.89	1	4
AQ_16	2.50	0.95	1	4
AQ_17	2.26	0.95	1	4
AQ_18	2.03	0.93	1	4
AQ_22	2.27	0.98	1	4
AQ_23	2.53	0.98	1	4
AQ_26	2.32	0.98	1	4
AQ_29	2.45	1.06	1	4
AQ_34	2.11	0.89	1	4
AQ_35	1.88	0.93	1	4
AQ_38	2.27	0.98	1	4
AQ_39	2.08	0.98	1	4
AQ_40	1.89	0.94	1	4
AQ_44	1.92	0.89	1	4
AQ_46	2.88	0.93	1	4
AQ_47	2.11	0.92	1	4
AQ_49	2.49	1.11	1	4
AQ_50	2.23	1.02	1	4
TAS_1	2.40	1.18	1	5
TAS_2	2.72	1.31	1	5
TAS_3	1.72	1.09	1	5
TAS_4	2.72	1.18	1	5
TAS_6	2.27	1.24	1	5
TAS_7	2.00	1.16	1	5
TAS_8	2.27	1.12	1	5
TAS_9	2.38	1.24	1	5
TAS_10	1.98	1.00	1	5
TAS_11	2.35	1.20	1	5
TAS_12	2.25	1.29	1	5
TAS_13	2.16	1.22	1	5
TAS_14	2.09	1.24	1	5

TAS_15	2.81	1.23	1	5
TAS_17	3.00	1.44	1	5
TAS_18	2.12	1.09	1	5
TAS_19	2.34	1.12	1	5
SOC	27.37	7.42	12	48
ATD	12.81	3.20	5	20
FLEX	10.24	2.89	5	20
IMG	10.23	3.05	5	20
FEL	28.06	9.84	12	59
EOT	11.51	3.46	5	23

Joint confirmatory factor analysis of autism and alexithymia

Table 8.13 Factor loadings

Factor	Item	Est	SE	z	p	std (all)
Social	AQ_1	1	0			0.327
Social	AQ_11	2.49	0.269	9.239	< .001	0.765
Social	AQ_13	1.516	0.193	7.842	< .001	0.437
Social	AQ_15	1.551	0.186	8.347	< .001	0.516
Social	AQ_17	2.452	0.266	9.224	< .001	0.759
Social	AQ_22	2.185	0.244	8.944	< .001	0.656
Social	AQ_26	2.19	0.245	8.943	< .001	0.656
Social	AQ_34	1.27	0.164	7.739	< .001	0.423
Social	AQ_38	2.748	0.293	9.369	< .001	0.83
Social	AQ_44	2.294	0.248	9.234	< .001	0.763
Social	AQ_46	1.606	0.193	8.314	< .001	0.51
Social	AQ_47	2.27	0.248	9.152	< .001	0.729
Feelings	TAS_1	1	0			0.759
Feelings	TAS_2	1.167	0.049	23.829	< .001	0.799
Feelings	TAS_3	0.534	0.043	12.371	< .001	0.436
Feelings	TAS_4	0.806	0.045	17.779	< .001	0.614
Feelings	TAS_6	0.934	0.047	19.705	< .001	0.674
Feelings	TAS_7	0.664	0.045	14.665	< .001	0.513
Feelings	TAS_9	1.045	0.047	22.22	< .001	0.751
Feelings	TAS_11	0.872	0.046	18.97	< .001	0.651
Feelings	TAS_12	0.783	0.05	15.618	< .001	0.544
Feelings	TAS_13	0.994	0.046	21.53	< .001	0.73
Feelings	TAS_14	0.806	0.048	16.786	< .001	0.582
Feelings	TAS_17	0.757	0.057	13.328	< .001	0.468
Flexibility	AQ_7	1	0			0.587
Flexibility	AQ_16	0.578	0.08	7.199	< .001	0.336
Flexibility	AQ_18	0.782	0.085	9.154	< .001	0.467
Flexibility	AQ_35	0.643	0.081	7.982	< .001	0.384
Flexibility	AQ_39	1.034	0.101	10.27	< .001	0.585
EOT	TAS_8	1	0			0.293
EOT	TAS_10	2.062	0.299	6.894	< .001	0.68
EOT	TAS_15	1.33	0.23	5.778	< .001	0.356
EOT	TAS_18	1.734	0.263	6.583	< .001	0.522
EOT	TAS_19	2.205	0.321	6.861	< .001	0.648

Imagination	AQ_3	1	0			0.62
Imagination	AQ_8	0.938	0.082	11.406	< .001	0.588
Imagination	AQ_14	0.798	0.085	9.378	< .001	0.436
Imagination	AQ_40	0.658	0.077	8.572	< .001	0.389
Imagination	AQ_50	0.941	0.089	10.557	< .001	0.514
ATD	AQ_6	1	0			0.686
ATD	AQ_12	0.732	0.062	11.807	< .001	0.583
ATD	AQ_23	0.868	0.071	12.218	< .001	0.661
ATD	AQ_29	0.38	0.06	6.324	< .001	0.268
ATD	AQ_49	0.202	0.061	3.303	< .001	0.136

Notes. Est = Estimate; SE = Standard error; z = Standardised estimate; p = p-value.

Table 8.14 Latent factor correlations for autism and alexithymia - Joint CFA

Factor 1	Factor 2	Correlation	p
SOC	FEL	.400	< .001
SOC	FLEX	.242	< .001
SOC	EOT	.310	< .001
SOC	IMG	.488	< .001
SOC	ATD	.111	.009
FEL	FLEX	.396	< .001
FEL	EOT	.380	< .001
FEL	IMG	.212	< .001
FEL	ATD	.128	.003
FLEX	EOT	.263	< .001
FLEX	IMG	.232	< .001
FLEX	ATD	.371	< .001
EOT	IMG	.343	< .001
EOT	ATD	.076	.124
IMG	ATD	-.094	.063

Notes. SOC = Social skills; FEL = Feelings and sensations; FLEX = Flexibility; EOT = Externally oriented thinking; IMG = Imagination; ATD = Attention to detail.

8.4.2.2 Network Analysis

Analysis on all items

Network inference

Centrality metrics are plotted in **Figure 8.15**. Centrality order was strongly related for both Experiment 9 & 10 in neurotypical samples, with a correlation of .87. One *feelings and sensation* item and a few *social skills*, *communication* and *imagination* items from the AQ showed the highest centrality strength, indicating that these items were highly interconnected with other items in the network (e.g., TAS_2, AQ_38, AQ_20, AQ_44, AQ_27). On the other hand, a mix of attention to detail and imagination (autism), and EOT (alexithymia) items had standardised centrality below zero, indicating that they were very weekly connected nodes (e.g., AQ_24, TAS_16, AQ_28, TAS_20, TAS_8, TAS_5, AQ_14).

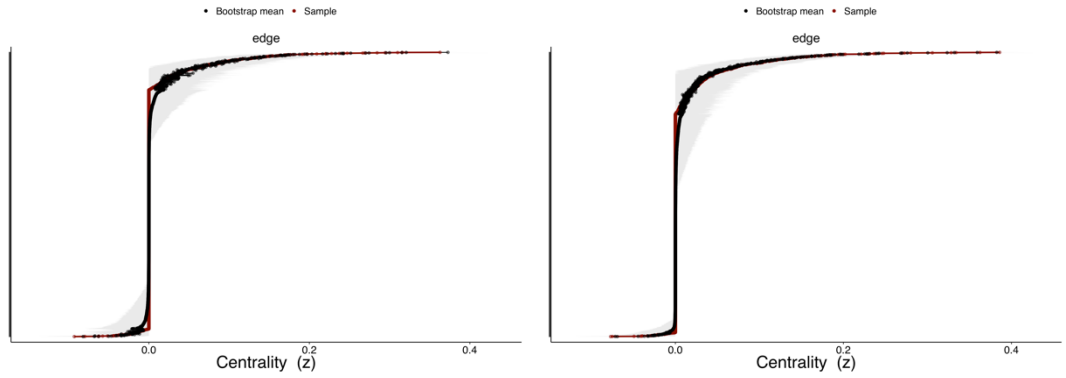
Node predictability for Experiment 10 sample was .35, that is, there was on average 35% of shared variance between neighbouring nodes which highlights the level of influence of one node on another assuming that all other nodes are connected to it. This was an increase of 7% compared to the NT network estimated in Experiment 9. For the full sample combining both Experiment 9 (NT sample only) and Experiment 10, the results were similar (38% of shared variance) which suggests that the difference in sample size did not affect the reliability of predictability estimates drastically. Similar to Experiment 9, there was a strong correlation between node centrality and the predictability (.92) – the more connected a node was, the more predictable it was. This was similar for the full network estimation with both samples combined (.87).

A

Experiment 10

B

Experiment 9 & 10



C

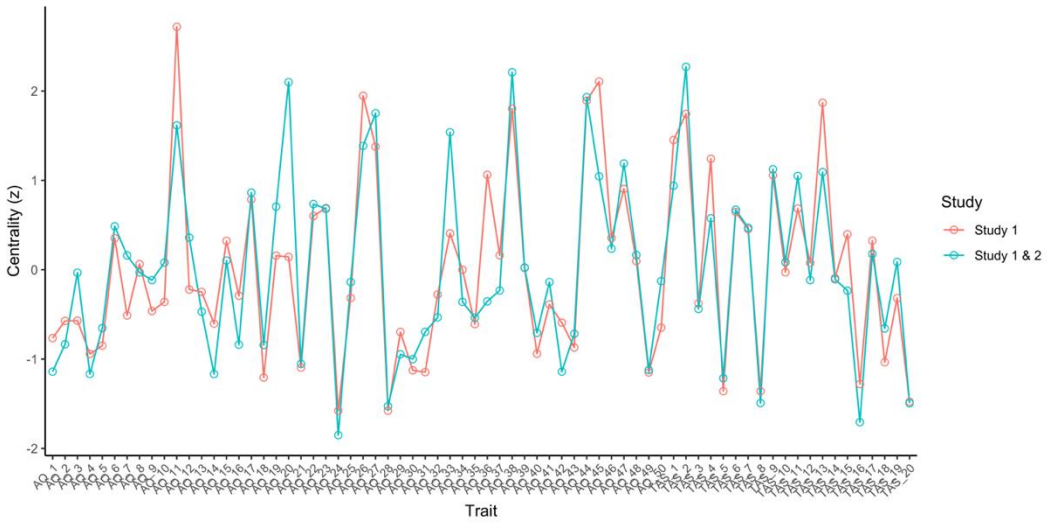


Figure 8.15 Centrality plots.

A & B. Bootstrapped CIs around edge weights. C. Standardised centrality strength for all items.

Factor networks

Network estimation

The estimated networks based on factor scores are displayed in **Figure 8.16.** As in Experiment 9, strong connections were observed between the alexithymia nodes DIF-DDF (RCP = .51), and DDF-EOT (RPC = .25), compared to connections to autism nodes (RCP < .10). Within the autism nodes, replicating Experiment 9, strong connections were seen between COM-SS (RPC = .47), and COM-AS (RPC = .28), with ATD showing the weakest connections to other nodes (< .10). This pattern was also consistent with the joint network combining neurotypical samples from Experiment 9 and 10.

A B C

Experiment 10 Experiment 9 & 10 (study 1 and 2)

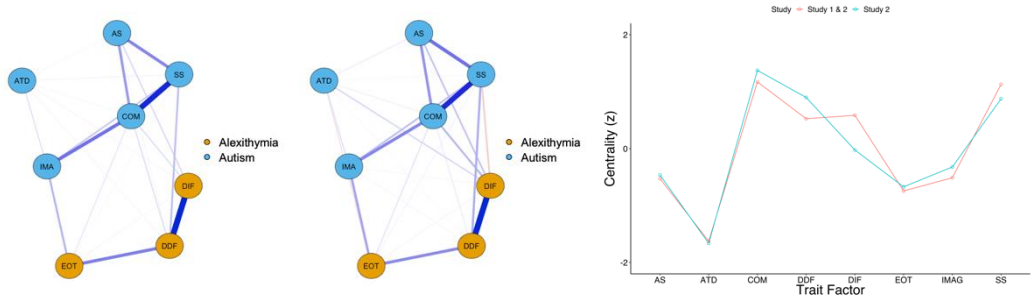


Figure 8.16. Factor score networks.

A. Estimated networks based on factor scores for Experiment 10. B & C. Estimated networks based on the neurotypical samples from Experiment 9 & 10 (N = 1371). Similar to Experiment 9, communication, social skills (autism) and difficulties with feelings (alexithymia) showed the highest centrality (highly connected nodes).

Network inference

Node centrality is visualised in **Figure 8.16.** Node centrality estimated in Experiments 9 & 10 was highly related, with a correlation of .88. As in Experiment 9, communication appeared as the most central node in the network followed by difficulties describing feelings and social skills. Conversely, attention to detail was the least central node followed by externally oriented thinking. The average shared variance between neighbouring nodes was 38% for the Experiment 10 sample and 13% for the full sample. As in Experiment 9, there was a strong correlation between node predictability and how central (connected) a node was. This correlation was .98 for the Experiment 10 sample and .68 for the joint network.

Network stability

The Experiment 10 network was stable, with a CSC of .75, and the joint network had a CSC of .74. Trait connections within alexithymia and autism were significantly stronger than between alexithymia autism connections (see **Figure 8.17** A & B). As in Experiment 9, DIF-DDF and SS-COM edges had the highest strength, differing significantly from all other edges but not differing from each other (see **Figure 8.17**).

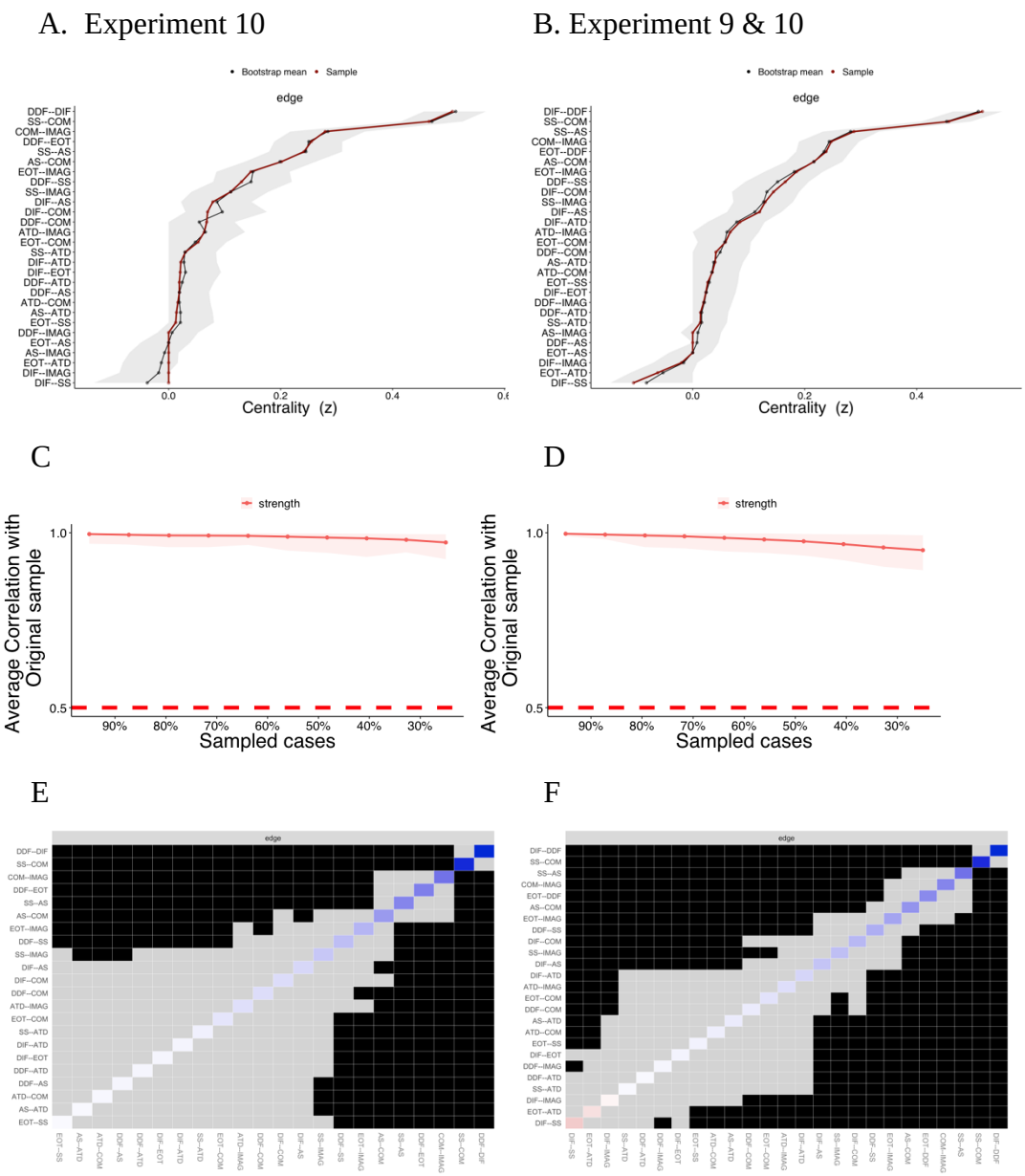


Figure 8.17 Network stability.

A & B. 95% bootstrapped CIs around edge weights.

C & D. Correlation stability coefficient. All networks were estimated reliably. E & F. Edge difference plot, comparing edge pairs (strength of pairwise trait connections). Darker squares represent a significant (p <.05) difference between the pairwise edge comparisons. As in Experiment 9, trait connections within alexithymia and autism were significantly stronger than between alexithymia and autism connections.

Network comparison

Experiment 9 & 10 networks were highly similar in structure, with a correlation of the edge weight matrixes of .88. The formal NCT showed no significant differences in network structure (M = 0.15, p = .05), but the network from Experiment 9 showed stronger average connectivity (3.48), than the network from Experiment 10 (2.82), S = .66, p = .01. There were no differences in individual edge invariance. Overall, though, the separation of autism and alexithymia items and factors was replicated.

8.4.3 CFA of Individual Measures

Autism Spectrum Quotient (AQ-50)

Three models were fitted based on the previous confirmatory studies of the AQ-50.

Model A.1. This model fitted the originally proposed five-factor structure with no second-order terms (Baron-Cohen et al., 2001).

Model A.2. This model fitted a three- factor solution (*social skills, attention* patterns and *communication*) suggested to outperform competing models and provide a more parsimonious measure (Russell, 2012; English et al., 2020).

Model A.3. This model is a popular variation of the original AQ-50 factor structure specifying second order factors for all social related abilities, and a separate factor for attention patterns (Hoekstra et al., 2011).

Overall, Model A.2, with only a three-factor solution and no second-order factors was the best performing model (see **Table 8.15**).

Table 8.15 Model comparison for the AQ-50 CFA models

Model	Df	AIC	BIC	Chisq	Chisq diff	Df diff	CFI	LTI	RMSEA	p
Model A.2	296	85956.04	86243.32	2184.29	NA	NA	.84	.82	.07 [.06, .07]	***
Model A.1	1165	171541.75	172116.32	8402.08	6217.79	869***	.63	.61	.07 [.06, .07]	***
Model A.3	1170	171539.82	172088.26	8410.14	8.06	5ns	.63	.61	.07 [.06, .07]	***

Notes. * $p < .05$, ** $p < .01$, *** $p < .001$.

Toronto Alexithymia Scale (TAS-20)

We compared our models to the original proposed structure for the TAS-20 (Bagby et al., 1994), measuring *difficulties identifying feelings* (DIF), *difficulties describing feelings* (DDF) and *externally-oriented thinking* (EOT).

Model A.1. This model included the three factors with no second-order factor.

Model A.2. This model included a higher-order general factor as a latent cause for the specific factors.

Model A.3. This model was a variation of A.1., with an inclusion of the method factor for the reversed scored items.

A bi-factor model was also tested but it did not converge.

Model A.1. was the best performing model (see **Table 8.16**).

Table 8.16 Model comparison for the TAS-20 CFA models

Model	Df	AIC	BIC	Chisq	Chisq diff	Df diff	CFI	LTI	RMSEA	p
Model A.1	149	74153	74367	1193.8	NA	NA	.88	.86	.07 [.06, .08]	***
Model A.2	152	75131	75329	2177.5	983.71***	3	.76	.73	.10 [.09, .10]	***
Model A.3	150	77692	77954	1129.9	-1047.56ns	8	.89	.87	.07 [.06, .07]	***

Notes. * $p < .05$, ** $p < .01$, *** $p < .001$.