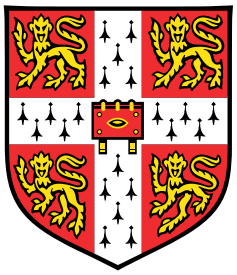
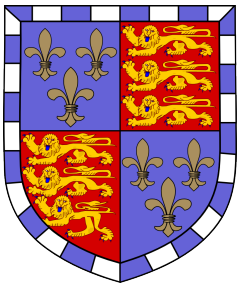


Investigating Sensory Prediction in Autism Spectrum Conditions



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The world is woven all of dream and error
And but one sureness in our truth may lie-
That when we hold to aught our thinking's mirror
We know it not by knowing it thereby.
For but one side of things the mirror knows,
And knows it colded from its solidness.
A double lie its truth is; what it shows
By true show's false and nowhere by true place.
Thought clouds our life's day-sense with strangeness, yet
Never from strangeness more than that it's strange
Doth buy our perplexed thinking, for we get
But the words' sense from words- knowledge, truth, change.
We know the world is false, not what is true.
Yet we think on, knowing we ne'er shall know.

FERNANDO PESSOA, SONNET XXVI

Declaration

I hereby declare that except where specific reference is made to the work of others, the contents of this dissertation are original and have not been submitted in whole or in part for consideration for any other degree or qualification in this, or any other university. This dissertation is my own work and contains nothing which is the outcome of work done in collaboration with others, except as specified in the text and acknowledgements. This dissertation contains fewer than 65,000 words and has fewer than 150 figures.

Johanna J. S. Finnemann
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S.D.G. בס"ד

Abstract

Autism is the umbrella term for a family of neurodevelopmental disorders characterised by heterogeneous clinical presentations affecting social interaction, communication, sensory atypicalities and restricted and repetitive behaviours (DSM 5). The idea of a unifying explanation that can account for the range of cognitive, behavioural and neurological features associated with autism is attractive, but as of yet no such cognitive or physiological underpinnings have been identified.

However, the last years have seen a growing interest in using approaches within the nascent field of the predictive processing framework to explore the potential causal role of aberrant prediction for the autistic phenotype. While hypothesised differences in predictive abilities have demonstrated some explanatory power for symptoms of psychotic spectrum disorders, empirical investigations into autism are still sparse.

In this thesis I follow up on the theoretical work about difficulties with expectation generation in autism with three empirical studies on prediction in perception and sensory processing (Chapters 2-4). My results did not support the idea of autism as a generalised disorder of prediction; however better phenotyping in future work might help to tease apart some of the variability observed in the autism group.

Furthermore I also examined the psychometric properties of two widely-used self-report questionnaires assessing autistic traits and schizotypy (Chapter 5). If latent traits are not measured equivalently across clinical and non-clinical populations, this could have implications for studies using high self-reported traits in healthy participants as proxies for the clinical condition as well as for correlational studies.

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Nomenclature

In recognition of the fact that there is no ubiquitous consensus about terminology referring to autism [272], I would like to preface my thesis by stressing that I will be using people-first and identity-first language interchangeably without intending to imply anything about the nature of autism as either a secondary attribute or part of someone's identity. Furthermore I will use the term Autism Spectrum Condition (ASC) instead of Autism Spectrum Disorder (ASD) in order to recognise both the disabling as well as potentially beneficial aspects of autism. However both the DSM-5's as well as the ICD-11's taxonomy refer to Autism Spectrum Disorders and my participants all carry a clinical diagnosis of ASD.

Acronyms / Abbreviations

ADHD Attention Deficit Hyperactivity Disorder

ADOS Autism Diagnostic Observation Schedule

ANOVA Analysis of Variance

ASC Autism Spectrum Condition

CFA Confirmatory Factor Analysis

DIF Differential Item Functioning

DSM-5 Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition

EBIC Extended Bayesian Information Criterion

EFA Exploratory Factor Analysis

ERP Event-related Potential

FEP Free Energy Principle

IB Intentional Binding

ICC Item Characteristic Curve

ICD-11 International Classification of Diseases, 11th Edition

JND Just Noticeable Difference

LASSO Least Absolute Shrinkage and Selection Operator

ML Maximum Likelihood

MMN Mismatch Negativity

MRF Markov Random Field(s)

NMDA N-methyl-D-aspartate

OCD Obsessive Compulsive Disorder

PCA Principal Component Analysis

RDoC Research Domain Criteria (US NIMH)

SCZ Schizophrenia

SD Standard Deviation

SEM Structural Equation Modelling

SEP Sensory Evoked Potential

SE Standard Error

SoA Sense of Agency

SR Sensory Reactivity

SSRI Selective Serotonin Reuptake Inhibitors

WCC Weak Central Coherence

Chapter 1

Of Models and Men

1.1 A Map and its Territory

> *A map is not the territory it represents, but, if correct, it has a similar structure to the territory, which accounts for its usefulness.*

ALFRED KORZYBSKI, SCIENCE AND SANITY

In psychiatry we are used to mapping mental landscapes onto clinical categories which are abstractions away from the complex, messy real world that does not (evidently) offer natural boundaries. These categories derive their usefulness from their probabilistic relationship with treatment response, time course of illness, symptom clusters, aetiology or other shared properties. Their utility hinges on their purpose, some maps might be too coarse for the questions asked, some describe too much of the naturally occurring variation. Some might simply be mapping the wrong features; it would not be of much use to me as a hiker to have a detailed road atlas of the Lake District and all its petrol stations, but equally a driver would not benefit from a map showing all the trig points even if the maps are at the same scale.

The thought that all these categories are man-made, not 'true' on a higher level, has created unease among some researchers. Efforts such as the US National Institute of Mental Health's Research Domain Criteria (RDoC) which aims to understand human behaviour and neurobiology along a continuum from normal to abnormal irrespective of diagnostic categories, are seen as a response to the taxonomic conundrum in psychiatry. Yet they never aimed to replace or supersede diagnostic systems (NIMH [398]), but were intended as frameworks to open up new research avenues that could focus on 'fundamental genetic, neurobiological,

behavioral, environmental, and experiential components' without the need to refer to clinical entities. However at the end of the day this (to me) is just a different approach to cutting the cake. Symptoms or 'components' are still abstractions away from the individual's behaviour, experience, personal and cultural circumstances; an artificial clustering of what is currently perceived as similar to behaviour observed in others. Dimensional approaches invariably rely on such symptoms or traits to be measured and compared. This is not a criticism of these approaches; we do need to look beyond our category-driven clinical approach of simply dichotomising abnormal and normal.

It is fine, yes, inevitable, that our parsimonious approximations and maps will be incomplete, sometimes even 'wrong' in the eyes of our successors (as the history of psychiatry has demonstrated). Maps after all are also just a reflection of the observer's perception. What we need to keep in mind is that our maps are not the landscape, we should not succumb to the fallacy of misplaced concreteness. Therefore I would like to start by laying out some thoughts about autism, its (current) definition and how it connects to the wider nosological landscape. Furthermore I will also talk about theories and models attempting to explain the 'components' of autism and how these theories relate to the experimental work I have undertaken.

1.2 Autism: Carving Nature at its Joints?

> *Und wir müssen uns daran erinnern, daß das, was wir beobachten, nicht die Natur selbst ist, sondern Natur, die unserer Art der Fragestellung ausgesetzt ist.*[1]
>
> WERNER HEISENBERG, PHYSICS AND PHILOSOPHY

One of the main diagnostic frameworks in psychiatry, the DSM-5, defines autism as a neurodevelopmental disorder characterised by 'persistent deficits in social communication and social interaction across multiple contexts [...] (and) the presence of restricted, repetitive patterns of behavior, interests or activities' (American Psychiatric Association [9]) and the newly published ICD-11 echoes this description (Organisation [407]).
Despite numerous changes to the diagnostic criteria since the autistic syndrome was first

[1] "[...] and we have to remember that what we observe is not nature in itself but nature exposed to our method of questioning."

described by Kanner ('(the) fundamental disorder is the children's inability to relate themselves in the ordinary way to people and situations from the beginning of life' [261]) and Asperger ("Wir wollen zeigen, daß die Grundstörung der Autistischen Psychopathen eine Einengung der Beziehungen zur Umwelt ist, daß die Persönlichkeit dieser Kinder von da her zu begreifen ist, daß sie von da aus "durchorganisiert" ist. [...] Am unmittelbarsten aber muß sich das Wesen dieser psychopathischen Kinder dartun, wenn man sie in ihrem Verhalten zu anderen Menschen betrachtet." [18])[2], the reported interpersonal deficits have remained central to the understanding and classification of autism. Social difficulties are thought to be at the heart of the condition by the majority of clinicians and researchers - past and present (Wing and Gould [606], Baron-Cohen et al. [31], Klin et al. [284], Constantino et al. [92]). Some of the major changes in the conceptualisation of the condition include a reassessment of the extent to which autistic children also present with language delay/the absence of language ('gross deficits in language development' were a necessary diagnostic symptom in the DSM III, 1980) and intellectual impairments (Lotter [334]). The most recent edition of the DSM also added that symptoms may only start to manifest once 'social demands exceed limited capacities' during development or that they may be 'masked by learned strategies in later life' (American Psychiatric Association [9]) which sheds an interesting light on a diagnosis that (to date) is still based on behavioural assessments.

Yet comparing diagnostic boundaries in the DSM or ICD classification systems over time only captures part of the shift in people's perception of autism and excludes what some have called 'unacknowledged discontinuities and irregularities [in] narrative[s] of autism'(Verhoeff [584]). The change in diagnostic threshold has not only caused an increase in diagnosis in people who would not have been identified as autistic in the early days (Lenoir et al. [321]), but increased awareness has also led to diagnostic substitution, particularly with learning disabilities (Shattuck [504], Nassar et al. [390]) and language disorders (Bishop et al. [49]). Other sociological factors driving a potential increase in diagnoses include better service provision and funding (Gurney et al. [201], Leonard et al. [322]) and changing perceptions of autism as a preferable diagnosis to other disorders (Wehling et al. [596]). In the adult population a high rate of misdiagnoses with personality disorders has been reported with about 37% of diagnosed ASC cases being reassigned to other categories under best clinical judgement (Kästner et al. [298]). This taken together with the overdetermination of most symptoms that make up autism (Simms [510], Tebartz van Elst et al. [548]), for some

[2] 'We want to demonstrate that the essential abnormality in autism is a disturbance of the lively relationship with the whole environment. We claim that this disturbance explains all peculiarities shown by autistic individuals. [...] However, the nature of these children is revealed most clearly in their behaviour towards other people.', translation taken from Frith [174].

undermines the plausibility and validity of autism as a distinct category. Either way we are still very far from being able to relate changing diagnostic thresholds and symptom clusters to the underlying structures in nature. Diagnostic boundaries are often fixed in places where the consensus agreement would deem the behaviour of an individual to deviate significantly from a hypothetical 'prototype', yet with autism spectrum conditions there is currently no agreement what the 'prototype' would look like. While some advocate for 'severe autism' (often synonymous with co-morbid intellectual and language impairments) as being the core manifestation of the condition (Dover and Couteur [136]), others maintain that co-morbid impairments should be purposefully ignored when looking for core features of autism thus considering 'high-functioning' individuals the best test case of 'pure' autism (Vivanti et al. [586]). Clinically the prototype might even depend on what individual clinicians have seen and experienced which in turn affects diagnostic practices and results in variable diagnoses (Posserud et al. [437], Lord et al. [330], Hayes et al. [222]).

Beyond discussions about diagnostic nosology and the current limitations of research attempting to bridge the explanatory gap between biology, cognition, behaviour and qualia, autism research has also undergone a paradigm shift with the increasing influence of the neurodiversity movement not only at a societal, but also research level (Silberman [507], Baron-Cohen [33]). In the tradition of the anti-psychiatry movement, the social model of disability and based on to Deaf and LGBTQ+ rights movements, the psychopathological character of autism is questioned and replaced with the view that autistic cognition is different, but not deficient or disordered (Harmon [218], Blume [57], Ortega [408], Fenton and Krahn [150]). While this debate is not the focus here, it is still important to acknowledge different perspectives and be mindful of the fact that psychiatric research is rarely observer-independent or removed from personal intuitions and attitudinal stances (Sisti et al. [514]).

It is beyond the scope of this work to examine the issues of this epistemological hamster wheel, but it is important to be mindful of the fact that the following ideas and empirical investigations are necessarily constrained and guided by contemporary definitions and conceptualisations of autism which inevitably are subject to change. In the absence of biomarkers or other unique identifiers, circular inference is inevitable, yet I still hope to add a few pieces to the body of knowledge at large.

1.2.1 Sensorimotor Functioning in Autism

> *Ich sitze in meinem Zimmer im Hauptquartier des Lärms der ganzen Wohnung. Alle Türen höre ich schlagen, durch ihren Lärm bleiben mir nur*

> *die Schritte der zwischen ihnen Laufenden erspart, noch das Zuklappen der Herdtüre in der Küche höre ich. [...] Schon früher dachte ich daran, bei den Kanarienvögeln fällt es mir von neuem ein, ob ich nicht die Türe bis zu einer kleinen Spalte öffnen, schlangengleich ins Nebenzimmer kriechen und so auf dem Boden meine Schwestern und ihr Fräulein um Ruhe bitten sollte.*[3] ”

FRANZ KAFKA, GROSSER LÄRM

Differences in sensory processing have been reported as an associated feature of autism from the very beginning of its recognition as a diagnostic and clinical entity: ‘At about the same age, he began to spin toys and lids of bottles and jars by the hour. [...] He would watch them and get severely excited and jump up and down in ecstasy. Now he is interested in reflecting light from mirrors and catching reflections. When he is interested in a thing, you cannot change it.’ (Kanner [262]). Although earlier editions of the DSM noted persistent preoccupations with parts of objects and repetitive motor behaviours as diagnostic features, an explicit reference to sensory hyper- and hyposensitivities has only been included in its most recent edition. Estimates of sensory reactivity (SR) in autism vary, but according to Leekam et al. [318] and others (Baker et al. [24], Tomchek and Dunn [559]) more than 90% of autistic children have sensory differences which tend to persist into adulthood and occur irrespective of overall cognitive ability. Hyperreactivity to sound and touch are commonly reported (Futoo et al. [178], Khalfa et al. [276], Elwin et al. [138], Gomes et al. [190]), but all other modalities can be equally affected by hyper- or hyporeactivity. Parents report a vast array of behaviours associated with sensory abnormalities including excessive smelling or touching of objects (or extreme avoidance of certain textures or smells), a blunted response to heat, cold or pain and a fascination with spinning objects and strobe lights (Dickie et al. [131], Leekam et al. [318]). Importantly, sensorimotor symptoms are positively correlated with other core aspects of autism (Baker et al. [24], Kern et al. [274], Mosconi et al. [377]) and global sensory dysfunction can interfere significantly with activities of daily life such as eating, sleeping, schooling, employment and participation in leisure activities (Hochhauser and Engel-Yeger [230], Jasmin et al. [253], Baker et al. [24], Ashburner et al. [16], Rogers and Ozonoff [466]).

[3] ‘I sit in my room, the headquarter of noise of the entire apartment. All the doors I hear slamming, and by such noise I am spared only the continuous footsteps between them – still I hear the stove’s door shutting in the kitchen. [...] I had thought of that earlier, and now with the canaries it occurs to me again, whether I should open the door a crack and, like a snake, slither into the next room and beg my sisters and their governess on the ground floor for some quiet.’

Motor mannerisms supposedly related to sensory processing differences, such as hand or finger flapping, rocking and walking on tiptoes - again while having been noted since the first descriptions - have now entered collective researchers' consciousness as being worth investigating in their own right instead of being written off as meaningless behaviours or co-morbid symptoms of intellectual disability. In conjunction with these stereotypies, more general differences in motor cognition and action control are also widely reported in autism spectrum conditions, including gait differences (Rinehart et al. [462], Kohen-Raz et al. [290]), clumsiness and abnormal posturing (Ghaziuddin et al. [186], Manjiviona and Prior [349], Rinehart et al. [460], Hallett et al. [211]), difficulties with gross motor (Berkeley et al. [45], Staples and Reid [527]) and fine motor skills (Beversdorf et al. [47]), abnormalities in developmental motor milestones (Teitelbaum et al. [549], Provost et al. [441]), movement planning differences (Mari et al. [351], Rinehart et al. [460] and Rinehart et al. [461], Hughes [241]), difficulties with anticipatory motor control (Whyatt and Craig [602], Schmitz et al. [485]), and gestural impairments (Dewey et al. [128]). Rates of atypical motor control are variable, but reported to be as high as 80% (Green et al. [195]) and believed by some to be a cardinal feature of ASC (Fournier et al. [158]). Speculated underlying neural substrates of these motor differences include the cerebellum (Becker and Stoodley [41], Pierce and Courchesne [430], Traut et al. [560], Hallett et al. [211]), basal ganglia (Qiu et al. [446], Rinehart et al. [462]) and thalamus (Rinehart et al. [462]). The cerebellum in particular has been extensively implicated in autism using behavioral, neuroimaging, and post-mortem analyses (Courchesne et al. [101], Courchesne et al. [98], Rogers et al. [467], Hampson and Blatt [213], Courchesne et al. [99], Pierce and Courchesne [430]). Striking are also reports of comorbidity with catatonic-like states (Wing and Shah [607], Dhossche et al. [129], Hare and Malone [217]) which tend to emerge during puberty or early adulthood and involve slowing of movements, akinesia and difficulties with initiating movements. As other symptoms associated with catatonia such as echolalia, mutism and posturing/catalepsy are also common in autism, only a sudden change or onset of other symptoms later in development should raise concerns about the additional presence of catatonia. However at a sub-clinical level, inertia is a frequently reported problem by autistics and carers alike and has been called 'autism's deepest, most fundamental, most massive handicap.' (p.86, Clara Claiborne Park, in Schopler and Mesibov [487]). The contribution of motor and sensory difficulties to 'inertia' are not well explored as it has historically been seen as a manifestation of difficulties with decision making, time-parsing, anxiety, depression or executive functioning (Luke et al. [339], Hoerl et al. [234], Bogdashina [58]).

While early accounts and cognitive theories of autism saw the social difficulties as the core deficit of the condition, more recent attempts have turned their attention to the sensory aspects of autism (Pellicano [418]) which might causally precede the socio-communicative differences (Donnellan et al. [135], Kapp [265], Simmons et al. [509], Lafleur et al. [302]). The majority of recent theories that reframe autism as a perceptual condition explicitly or implicitly invoke narratives that cast the brain as a perpetuous model maker and reviser. At this point, it is important that we draw back to explore this general supposition:

1.3 The Brain as a Probabilistic Prediction Machine

> *Admitamos lo que todos los idealistas admiten: el carácter alucinatorio del mundo. Hagamos lo que ningún idealista ha hecho: busquemos irrealidades que confirmen ese carácter. [...] 'El mayor hechicero (escribe memorablemente Novalis) sería el que se hechizara hasta el punto de tomar sus propias fantasmagorías por apariciones autónomas. ¿No sería ése nuestro caso?'. Yo conjeturo que así es. Nosotros (la indivisa divinidad que opera en nosotros) hemos soñado el mundo. Lo hemos soñado resistente, misterioso, visible, ubicuo en el espacio y firme en el tiempo; pero hemos consentido en su arquitectura tenues y eternos intersticios de sinrazón para saber que es falso.*[4]
>
> JORGE LUIS BORGES, AVATARES DE LA TORTUGA

The mappings between the state of the world 'out there' and the voltage changes in sensory receptors it produces are many-to-many. The signals we receive to construct a representation of the world are ambiguous, incomplete, indirect, underdetermined, noisy and opaque: Many different causes can incite a neuron to fire and one cause can produce different firing patterns depending on how we access its sensory properties. And yet our (subjective) perceptual experience is transparent and - for the most part - monosemous.

[4] 'Let us admit what all idealists admit: the hallucinatory nature of the world. Let us do what no idealist has done: seek unrealities which confirm that nature. [...] "The greatest magician (Novalis has memorably written) would be the one who would cast over himself a spell so complete that he would take his own phantasmagorias as autonomous appearances. Would not this be our case?" I conjecture that this is so. We (the undivided divinity operating within us) have dreamt the world. We have dreamt it as firm, mysterious, visible, ubiquitous in space and durable in time; but in its architecture we have allowed tenuous and eternal crevices of unreason which tell us it is false.'

It is assumed that we owe this continuity and coherence of our grasp of the world to 'controlled hallucination'; our subjective experience is the result of guesses about the hidden causes in the world modulated by the incoming sensory signals.

The idea that the brain is not simply a passive vessel transforming sensory input into representations of the world is by no means novel. From the 10th century writings of Ibn al-Haytham [7] to Kant's Critique of Pure Reason [263], sensory information was noted to be only meaningful when it is distinguished from and compared to prior and consecutive information and subjected to the current state of knowledge about the world.

The recent recast of these ideas in more computational terms has generated a lot of interest within psychiatry with claims about its usefulness in understanding the causal mechanisms of various psychopathologies (Moore [376], Adams et al. [2]), its implications for the development of diagnostic tests (Petzschner et al. [428], Haker et al. [210]) and treatment options (Chekroud [79], Guo et al. [200]).

When perusing the recent literature on this topic it may seem as if the theoretical frameworks presented therein are identical, yet it is wiser to see them as a family of approaches with roots in different areas of science, from reinforcement learning to philosophy, vision and artificial intelligence[5] (Hinton and Zemel [229]). The most widely discussed theory of hierarchical Bayesian inference has its roots in early theoretical and modelling papers emerging from vision science (Rao and Ballard [448], Lee and Mumford [317], Srinivasan et al. [526], Dayan et al. [121], Ullman [566]). The ideas about perceptual inference in vision were later incorporated into a bigger framework in which the multilayer architecture arises naturally out of a principle of free-energy minimisation (Friston [166, 165]), shifting the problem from one of mapping relationships to one of metabolic efficiency. The Fristonian model also specifies that rather than processing actual sensory signals, all that is encoded in the nervous system are predictions and prediction errors (=deviations of the stimulus from the prediction) with higher levels passing down predictions (inter-regional feedback connections) and lower levels passing prediction errors back up (inter-regional feedforward connections). This occurs within a hierarchical dynamic model so that the output of one level acts as the input to the next, creating a bidirectional cascade of optimisation steps. In order to minimise (future) prediction errors (=variational free energy), the brain either modifies a probabilistic

[5] Unfortunately the terminology used to refer to these theories of brain function is lacking some clarity owing to the fact that names of specific theories ('predictive coding' and 'Free Energy Principle' by Friston and Kiebel [168], 'predictive processing' by Clark [85], 'Bayesian brain' by Knill and Pouget [287], Yuille and Kersten [620], and 'the predictive mind' by Hohwy [236]) are conflated with more general descriptions of the basic concept. In what follows I will use predictive coding, predictive processing etc. synonymously to refer to a broad framework arguing for a hierarchical brain mechanism of prediction error minimisation. Specific frameworks will be named as such when and where relevant.

generative model of the world (i.e. by learning) or acts to change the channels of its sensory input, an idea often referred to as 'active inference'.

1.3.1 Friston's Free Energy Principle

Predictive coding has been called the 'most complete framework to date for explaining perception, cognition, and action in terms of fundamental theoretical principles and neuro-cognitive architectures' (Seth [499]) and is based on the notion that hypotheses about the hidden properties and causes in the world are generated and updated in a Bayesian way:

Let y be the (sensory) data and θ a set of model parameters. According to Bayes theorem the (posterior) likelihood of the parameter values given the data is proportional to the prior likelihood of the model multiplied by the likelihood of the data given the model divided by the marginal likelihood of the data:

$$P(\theta|y) \propto \frac{P(y|\theta)P(\theta)}{P(y)} \tag{1.1}$$

where $P(y)$ can be computed using the law of total probability as $P(y) = \sum P(y|\theta)P(\theta)$ when y_i are mutually exclusive events[6]. $P(\theta)$ is the prior probability (of this set of parameters) and can represent various forms of prior knowledge or beliefs about states of the world such as object shapes, illumination, likely co-occurrences of objects or other people's goals.

Another key element of the Bayesian brain hypothesis is that information is represented as probability density functions (presumably through neuronal population coding, see Glaser et al. [187]), rather than as discrete values[7] - the advantage of which presumably is that information can be passed on without the need to commit to a particular interpretation too early on. However continuous random variables cannot be summed over, so the marginal likelihood, or 'evidence' has to be expressed as an integral:

$$P(y) = \int_{\theta} p(y|\theta)p(\theta)d\theta \tag{1.2}$$

This integral over all the model parameters can become difficult to compute[8] and thus prior and likelihood distributions are frequently fixed as Gaussian (as it changes a variational calculus problem to an optimisation problem that can be solved via gradient descent) even

[6] In mathematical terms sometimes called a 'disjoint set' or in an experimental context the 'sample set'.

[7] This is in contrast to classic information theory as proposed by Shannon ([503]).

[8] Which is partly why Bayesian approaches have only recently started to flourish as computerised solutions to integrals have become more feasible.

though real-world applications often call for non-Gaussian distributions (Zhu et al. [625], Otworowska et al. [409]).

There are two main approaches to estimating intractable integrals in Bayesian inference: 1) stochastic approximations through sampling numerical approximations (i.e. Markov chain Monte Carlo methods) and 2) variational Bayes, which is a generalisation of Laplace's method (i.e. finding an analytical proxy $q(y,\theta)$ to the original probability density function $p(y)$). Friston's 'Free Energy Principle' (FEP) is based on the assumption that the brain employs variational Bayes to infer the distal, hidden causes of sensory input.

A crucial step towards deriving a free energy term (that is to say 'quantifying the average surprise') is to measure the information gain from q to p, sometimes also called the relative entropy[9] of p with respect to q. This is equivalent to assessing how well the probability function q approximates p[10] and can be calculated using Kullback-Leibler divergence metric:

$$D_{KL}(P||Q) = \int_{-\infty}^{\infty} P(x_i)(logP(x_i) - logQ(x_i))dx \tag{1.3}$$

A set of $Q_i(y_i,\theta_i)$ is found that minimises KL divergence. In Friston et al. [169] the variational free energy F is given as

$$F(\tilde{s},\tilde{a},\tilde{\lambda}) = L(\tilde{s},\tilde{a},\tilde{r}) + D_{KL}[q(\tilde{\psi}|\tilde{r})||p(\tilde{\psi}|\tilde{s},\tilde{a},\tilde{r})] \tag{1.4}$$

where $\tilde{\psi}$ are the external system elements and $\tilde{\lambda}$, $\tilde{r}$ are the units of the internal representation with $\tilde{s}$ and $\tilde{a}$ being the sensing and acting units respectively (which make up the Markov blanket that separates internal units from external ones). Friston calls $L(\tilde{s},\tilde{a},\tilde{r})$ a 'free energy-like term' or the 'thermodynamic free energy' which is the same as the negative sum of the logarithm of the probability of the states of the internal and Markov blanket units: $-\sum ln(p(\tilde{s},\tilde{a},\tilde{r}))$.

According to Friston [167], F can also be expressed as the expected Gibbs energy minus the entropy of the variational density:

$$F(s,a,\lambda) = E_q[G(\Psi,s,a,\lambda)] - H[q(\Psi|\mu)] \tag{1.5}$$

linking it more explicitly to thermodynamic operations.

[9] In information-theoretic terms, entropy is the expected self-information/average surprise when sampling a random variable and as such limits the shortest possible average length of a lossless compression. Knowing this lower bound on compression, relative entropy then quantifies how much information is lost when the parameterised approximation is substituted for the original function.

[10] But not how well p approximates q - information gain is not symmetric!

In exact Bayesian inference (where D_{KL} can attain the value zero when F is minimised with regard to the internal states), variational free energy is equivalent to the Langrangian free-energy like term L, whereas in approximations it supplies the upper bound on the surprisal of the sensory data. Applying these general principles to the brain and assuming a hierarchical computational structure where the output of one level serves as input to the next, deep pyramidal cells are thought to take the role of prediction neurons, while superficial pyramidal cells correspond to error detection units (see Figure 1.1).

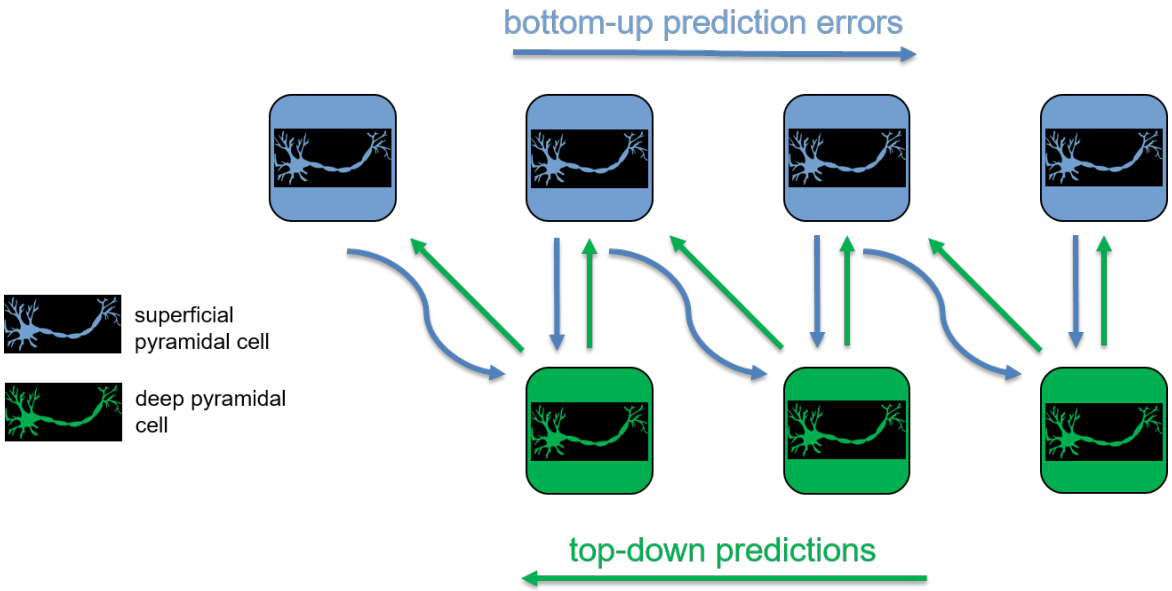


Figure 1.1 Hierarchical Prediction in the Brain

1.3.2 Alternative Approaches and Common Questions

As previously mentioned, predictive processing theories - while largely overlapping at the computational level (i.e. competency/performance) - have subtle, but important differences in their algorithmic formulation, hypothesised neuronal implementation and by extension: explanatory power[11].

According to Friston [166], the FEP should 'enable one to write down equations that predict the dynamics of synaptic activity (encoding expected states), synaptic efficacy (encoding expected parameters) and neuromodulation of synaptic gain (encoding expected precision)'. This seems difficult in practice as a lot of the (hyper)parameters[12], can still be implemented

[11] But explanatory power should not be mistaken for explanatory flexibility.
[12] Such as the precision around estimates.

and estimated in various different ways (Kogo and Trengove [289]). Buckley et al. [71] and Turkheimer et al. [564] list further limitations of the implicit assumptions of FEP at the theoretical as well as implementation level.

Furthermore there is no empirical data to suggest that the method employed by the brain to estimate the normalisation constant (=evidence) in Bayesian inference is variational Bayes rather than a stochastic sampling method. In fact simulations of stochastic spiking neurons carrying out probabilistic inference in general graphical models show a lot of promise even in large, complicated networks (Pecevski et al. [415]), but neurophysiological evidence in vivo is similarly lacking (Knill and Pouget [287]). The question of how and whether our brain performs marginalisation over non-Gaussian classes of distributions is therefore still open to debate. However as some have pointed out, FEP might still be more constrained than unprincipled appeals to Bayesian models and as such fills a gap in the modelling literature (Spratling [524]).

While in the Fristonian approach computations about the distal/hidden causes and properties are derived based on probabilistic inference, other approaches such as structural coding use descriptive complexities K (van der Helm [576]). Thus while the FEP seeks to minimise prediction errors, or - differently put - the surprisal (or entropy[13] as the long-term average of surprisal) of $-logp(y|\theta)$, structural coding aims to minimise the $\sum(K(\theta)+K(y))$ to achieve a stable state [14]. To put it in classical information theoretical terms: FEP is based entirely on the probability of the information y whereas structural coding also takes into account the internal structure of y.

Another difference between the multiple accounts concerns the conception of priors and their origins: Where priors come from or alternatively how they are acquired has been discussed in Bayesian approaches for some time (Suppes [541]) and is of particular interest to the nativism debate (Colombo [88]). According to Clark [84], predictive processing can 'operate anywhere along the nativist empiricist spectrum' (although he seems to lean more towards an empiricist 'bootstrapping heaven' himself: Clark [85]) and Tenenbaum [550] goes even further in claiming that the Bayesian approach makes dichotomies such as nativism vs. empiricism meaningless.

FEP takes a more specific approach: Given the hierarchical structure, the model can make use of parametric empirical Bayes (Kass and Steffey [267]), i.e. make its own priors based on the random fluctuations that act independently at each level (Friston and Stephan [170]).

[13] Note that neither Friston's 'free energy' not Shannon's 'entropy' correspond to their thermodynamic homonyms.

[14] Fristonians claim that 'complexity is the divergence between the variational density and prior beliefs about hidden states'.

Yet whatever the explanation for the emergence of priors, a few questions still remain - in the words of Seriès and Seitz [498]: 'How fast do priors change over time? Are there limits in the complexity of the priors that can be learned? How do an individual's priors compare to the true scene statistics? Can we unlearn priors that are thought to correspond to natural scene statistics? Where and what are the neural substrate (*sic*) of priors?' In order to address these questions, the authors make an explicit distinction between 'structural' and 'contextual' priors, the former being based on long-term, implicitly learned statistical regularities in the environment (which affect all perceptions of the stimulus or its properties in question) whereas the latter can be acquired or manipulated rapidly in the lab and are not generalisable to other contexts. However the distinction is once again not as clear-cut as the authors would like, as even supposedly 'structural' priors like the bias that lightsources are positioned above can still be modulated in a short time period (Adams et al. [4]).

The debate about priors also touches on a more philosophical area of Bayesian reasoning as it has been asserted that conditionalisation requires at least some propositions to be true or in the words of C.I. Lewis 'If anything is to be probable then something must be certain.' (Irving Lewis [249], p.186). In fact the validity of Bayesian inference itself is questioned by some, irrespective of whether probabilities are subject to epistemic or aleatoric uncertainty (Halpern [212]).

Lastly, skeptics of predictive processing theories of the brain may point out that predictability of stimuli can lead to both, attenuation of the intensity of the stimulus as well as sharp-tuning of its properties. The answer to this conundrum lies in the conflation of two intertwined processes, prediction (expectation) and attention (Summerfield and Egner [537]). While attention has been shown to amplify or sharp-tune neuronal responses (Lakatos et al. [305]), expectations shape the gain modulation of the prediction error signal and are observable in attenuative processes (Schröger et al. [488], Lange [308]). The two are evidently not completely separable and their interactive mechanisms and underlying neurobiology still need to be delineated. Kogo et al. ([289]) noted that end-stopped cells fulfill the role of error neurons when they signal the unpredicted end of a line segment in Rao and Ballard's [448] predictive coding model, whereas they function as prediction units in Kapadia et al.'s [264] work. Within the visual cortex, both repetition suppression (of prediction errors due to predictability) and repetition enhancement (=sharp-tuning due to accurate predictions) have been found to co-occur in the same region, but in different neuronal populations with different connectivity and latencies (de Gardelle et al. [122]). Evidence for separate prediction and error neurons outside of the visual cortex are even more parsimonious (Heilbron and Chait [225]). For a suggested neurocircuitry of FEP see Bastos et al. [37].

Ergo there is still an explanatory gorge between theoretical models and the behavioural and neurophysiological studies that invoke predictive processing: Event-related potential (ERP) components often relate to surprise or novelty: mismatch negativity in oddball tasks (Näätänen et al. [403]), the N400 for semantically unexpected stimuli (Broderick et al. [68], Kutas and Hillyard [296]) and unexpected visual stimuli eliciting the N170 (Robinson et al. [465]). Functional imaging has contributed to the literature with papers on repetition suppression[15] (Auksztulewicz and Friston [20], Summerfield et al. [539]) and demonstrated visual context influencing V1 responses (Smith and Muckli [518], Kok et al. [291]).

To sum up, despite the fecundity of the predictive coding framework regarding its proposed applications, both the empirical data and the candidate interpretations lack the unequivocality needed to link the two. Nevertheless, these ideas have found fertile ground in autism research, as the following section describes.

1.4 Prediction in Autism

> *While at the Center I encountered many curious phantasmagoria called 'theories'. I attempted to hold a seminar on what constitutes a good theory but was warned that the topic was explosive!*
>
> ROSS ASHBY, DIARY

On the back of studies exploring the possible causal role of aberrant prediction in other disorders such as psychosis (Corlett et al. [93], Murray et al. [386], Fletcher and Frith [153]), the last years have seen a growing interest in using approaches within the nascent field of predictive processing to investigate differences in the cognitive, perceptual and neural processes in autism spectrum conditions (Qian and Lipkin [445], Pellicano and Burr [419], Sinha et al. [512], Lawson et al. [315], Van de Cruys et al. [571], Rosenberg et al. [470]). Given the plethora of clinical accounts detailing difficulties with adapting to change and intolerance of uncertainty in children with autism[16], it seems plausible to conjecture that difficulties with expectation generation are at the core of many autistic symptoms. Table 1.1 summarises a few recent theories addressing sensory and learning atypicalities in autism.

[15] Although so far single unit recordings do not confirm this mechanism, see Vinken et al. [585].

[16] 'It was as if the children were constantly generating rules about how things should be based on how they were when they happened to come across them. A walk taken along a certain route one day had to be taken the same way every time after that.' (Silberman [507], p.178)

1.4.1 Attenuated Priors, Memoisation, Weak Central Coherence, Reduced Habituation and other Exploits into Autistic Cognition

Pellicano and Burr [419] proposed that individuals with autism exhibit an attenuation of Bayesian priors in perception, a diminished effect of prior knowledge on the processing of ambiguous sensory information, perhaps as a consequence of excessive endogenous neural noise. However, it has been pointed out that Bayesian modelling cannot explain atypicalities of autistic perception that are due to differences in how posteriors are calculated from the prior and likelihood function which are fixed (Teufel et al. [554]), and furthermore that reduced sensory noise would lead to similar patterns of perceptual performance as the ones predicted by 'hypopriors' (Brock [65]).

Qian and Lipkin ([445]) grounded their theory not explicitly in the predictive coding world, but instead approached the issue from a machine learning perspective. In their view, autism is characterised by a bias towards lookup table (LUT) learning, whereas nonautistic individuals rely more heavily on interpolation (INT) learning. Whereas the former is designed to learn and store local, precise bits of information without any ability or need for generalisation, the latter is equipped to tackle problems that require abstraction, context-dependent learning, recognition of regularities and prediction generation. They conclude that a LUT learning strategy in our noisy, complex world would lead to sensory overloads, inefficient processing of information and resulting problems with executive function and adaptation.[17]

This echoes earlier cognitive theories of autism such as the weak central coherence theory (WCC) which described autistic perception as being biased towards piecemeal processing at the expense of global form or meaning (Frith [173]). Empirical evidence for WCC are findings of superior performance on the block design and embedded figures tasks (Shah and Frith [501], Jolliffe and Baron-Cohen [256], Shah and Frith [500]), difficulties with using linguistic context to disambiguate words (Jolliffe and Baron-Cohen [255], Happé [215]) and decreased susceptibility to illusions (Happé [214]).

Later revisions of the WCC include the Enhanced Perceptual Functioning account of autistic perception (Mottron and Burack [381]) which reiterated the core idea of WCC while asserting that the bias towards locally oriented processing is not necessarily accompanied by a deficit in global processing. The Reduced Generalisation hypothesis, another response to WCC (Plaisted [433]) on the other hand is probably the closest relative to Qian and Lipkin's approach: It pointed out the failure of previous accounts in specifying whether

[17] Although this processing style has also been linked to some of the exceptional abilities observed in some individuals with autism, see the 'veridical mapping' theory by Mottron et al. [380].

the mechanism was acting on perceptual, attentional or conceptual processes (provided they are as separable as assumed) and rooted its assertions in statistical learning theories such as Estes [145]. According to the Reduced Generalisation theory, the core deficit of autistic cognition might lie in a reduced processing of shared features across stimuli leading to better detection of unique features, but difficulties with transfer learning. As the degree of similarity between target and distractor items in visual search should increase search time, a lack of slowing in autistic individuals during conjunctive search may be interpreted as supporting this theory (Plaisted et al. [434]). Autistics also have a reduced perceptual learning effect (i.e. they don't benefit from pre-exposure) concurrent with better discrimination of novel stimuli (Plaisted et al. [434]). Proposed mechanisms for enhanced discriminatory abilities are reduced habituation and increased lateral inhibition (Plaisted [433]). Reduced behavioural and neurophysiological habituation have repeatedly been reported in autism (altered neural and behavioural habituation: Tam et al. [546], reduced habituation to numerosity: Turi et al. [563], reduced behavioural habituation to novel stimuli: Vivanti et al. [587]) with the most consistent findings for (high level) social stimuli (Congiu et al. [89], Pellicano et al. [420], Ewing et al. [148]). Only habituation deficits to social (and not non-social) stimuli were found to correlate with sensory reactivity (SR) (Lawson et al. [313]), whereas tactile defensiveness was not related to reduced habituation (Baranek and Berkson [28]). Contextual modulation in low-level vision as measured via the computational concept of divisive normalisation is intact in autism (Van de Cruys et al. [573]). According to a study by Pellicano et al. [421], categorisation deficits might give rise to impairments in habituation. However this argument risks being circular given the earlier literature on reduced generalisation.

On a neural level, reports of local over-connectivity[18] and long-range under-connectivity in autistic brains (O'Reilly et al. [410], Cherkassky et al. [81], Courchesne and Pierce [100], Anderson et al. [10], Keown et al. [273], Barttfeld et al. [35], Horwitz et al. [238], Just et al. [259], Wass [595])[19] as well as disrupted connectivity in mouse models of autism (Cho et al. [82], Nagode et al. [388]) have inspired speculations about divergent signal-to-noise ratios in ASC. Belmonte et al. [42] hypothesised that overconnected networks leak too much noise whereas underconnected networks filter out too much of the signal which leads to the perceptual and cognitive symptoms outlined by WCC and its derivatives. Autistic teenagers and adults exhibit wider auditory filters than age and IQ-matched controls (Plaisted et al. [435]) which according to Belmonte et al. support the idea that individuals with

[18] Particularly for sensorimotor networks, see Oldehinkel et al. [405]

[19] But see Supekar et al. [540], Dajani and Uddin [112], Khan et al. [278] for contradicting findings.

ASC experience a delay or attenuation of context-dependent response selectivity similar to the developmental delay seen in animals reared in conditions that impair functional maturation (Chang and Merzenich [77]). This 'unconditional plasticity' underpins - according to Belmonte et al. - the context blindness and particular learning style with which autism has been associated. More recently, hyper-plasticity and hyper-reactivity of local neural networks were thought to lead to 'hyper-functionality' (manifesting in superior perception, attention and memory) in a theory known as the 'intense world syndrome' (Markram et al. [352], Markram and Markram [353]).[20] This enhanced reactivity, according to the authors, may be facilitated by insufficient inhibitory control and/or hyperexcitability of neuronal circuits (Markram et al. [352]). An alternative formulation of the same idea is found in accounts of higher levels of endogeneous noise in autism (Simmons et al. [508]) which is thought to be facilitated by pathophysiology of the inhibitory interneuron system (perhaps due to a sparsity of inhibitory Purkinje cells, see [516, 97, 39]). Decreased inhibition additionally explains the high comorbidity of epilepsy in autism (Reilly et al. [453], Maski et al. [356]).[21] And sensory gating deficits are more pronounced in children with comorbid epilepsy (Sprengers [525]).

The excitatory-inhibitory (E/I) imbalance hypothesis of autism usually assumes hyperexcitation and/or reduced inhibition (albeit sometimes localised to particular regions (Belmonte et al. [42])), but so far electrophysiological evidence is mainly found in the animal model literature (Yizhar et al. [616]) whereas the imbalance seems to be undetectable in children with ASC (van de Lagemaat et al. [575]). A reduction in the metabolite ratio of creatine and GABA has been reported for motor and auditory areas in children with ASC using magnetic resonance spectroscopy (Gaetz et al. [180]) and differences in GABA levels in sensorimotor areas correlate with observed differences in tactile dynamic detection thresholds in autism (Puts et al. [443]). Behavioural evidence stems mainly from studies reporting increased inter-trial variability in ASC (Dinstein et al. [133], Haigh et al. [209], Milne [364]). Electrophysiological measures that aim to show decreased signal-to-noise ratios are reduced visual evoked potentials (Weinger et al. [598]) and reduced event-related desynchronisation (Ewen et al. [147]). At the same time it seems physiologically implausible to assume that the E/I balance would not vary between cortical regions (Nelson and Valakh [395]) and thus it is not 'straightforward to define a single physiological measurement that accurately captures

[20] However scientific support for a general functional enhancement across these domains in autism is sparse and the conjectured treatment options that include sensory deprivation in childhood should be approached with caution.

[21] Interestingly the presence and absence of epilepsy elicits different neural phenotypes among children with autism, such that individuals with epilepsy show higher amplitudes for MMN and P300 compared to controls, whereas autistic children without epilepsy exhibit a dampening of these ERPs (Vlaskamp [588]).

the balance between excitation and inhibition.' (Nelson and Valakh [395]). Contrasting with the accumulating reports supporting high endogeneous neural noise in autism, it has also been pointed out that lower levels of endogenous noise could lead to similar symptomatology (Davis and Plaisted-Grant [119]). Furthermore low endogeneous neural noise can explain the unique pattern of sensory discriminatory strengths and weaknesses observed in ASC (Bertone et al. [46]) and account for the lower rate of transitions between different neural states (e.g. alternations of percepts during binocular rivalry, see Robertson et al. [464], Freyberg et al. [162]).

None of these theories explicitly referred to hierarchical prediction error processing as postulated in the Fristonian framework.

1.4.2 Back to Friston: High/Aberrant Precision of Sensory Prediction Errors?

Other accounts place the deviation in the processing hierarchy not at the level of the priors, but at the hyperparameters, specifically the precision around prediction errors (Van de Cruys et al. [572], Lawson et al. [315]). While inflexible precision estimates on sensory prediction errors would also result in context insensitivity, the underlying neural dynamics and predictions of each theory differ: Under the aberrant precision account of autism, cognition and behaviour will be disproportionally affected under extremely volatile conditions which differs from theories that assume a uniform deficit in expectation generation. Indeed certain priors such as gaze priors seem to be unaffected in autism (Pell et al. [417]) as are certain ways of implicit learning (Nemeth et al. [396]) and motion prediction for objects [555].

In keeping with theories about impaired metacognition in autism (Williams et al. [604], Grainger et al. [194]), aberrant precision in volatile environments might reflect a difficulty with distinguishing the learnable from the unlearnable (Van de Cruys et al. [571]). Physiologically, overfitting in volatile environments by autistic adults is thought to be modulated by cholinergic and noradrenergic signalling (Lawson et al. [314]). For others, tight bounds on the precision of sensory input have the capacity to explain a range of symptoms not just within one individual, but also the heterogeneity across individuals: The aberrant predictive processing may take place at varying neuronal levels in different individuals leading to different behavioural and cognitive outcomes (Haker et al. [210]). This view is also supported by Sinha et al. [512] who called autism a 'disorder of prediction' while pointing out that his theory does not imply a uniform or universal predictive impairment across individuals. The example given by Sinha et al. concerns a differential impact of impairments in temporal

prediction in the millisecond and multisecond range for language and social interaction respectively.

The appeal of such theories in dealing with the pervasive heterogeneity of autistic conditions is simultaneously their shortcoming. If such varying outcomes and behaviours can be accounted for under the same framework, what is its empirical value and prediction generating ability? Predictive processing as a purely 'computational' explanation also has its shortcomings: even if Bayesian predictions accord with the behavioural output, it cannot explain why (Brock [65]) and how (van Boxtel and Lu [570]) this behaviour comes about.

Table 1.1 Cognitive and Neurophysiological Autism Theories of Aberrant Prediction and Context Insensitivity

Authors	Mechanism	Physiology	Evidence	Predictions
[471]	increased excitation/inhibition ratio	increased glutamatergic signaling, reduced GABAergic signalling, delayed synapse maturation	increased rates of epilepsy, polymorphism associated with synaptic functioning	poorly functionally differentiated cortex positive response to anti-convulsant treatment
[572, 571, 315]	high and inflexible precision on sensory input	abnormal acetylcholine (ACh) and norepinephrine (NE) neuromodulation, monoamines, oxytocin	intact implicit learning and generalisation[396]	priors relying on simple mean estimation (without noise/uncertainty) are intact
[352, 353]	hyper-reactivity and hyper-plasticity of local neuronal circuits	early brain stem injury	valproic acid model of autism	hyper-perception, hyper-attention, hyper-memory, hyper-emotionality, increased distractor processing
[454, 455]	enhanced perceptual capacity		enhanced visual search performance	
[119]	low endogeneous neural noise	low tonic & high phasic noradrenergic activation	low binocular rivalry transition rates, enhanced visual search	reduced generalisation

1.4.3 Strange Bedfellows: (Prediction in) Schizophrenia/Schizotypy and Autism

Before being applied to autism, research into psychosis and schizotypy constituted fertile ground for the burgeoning theory of predictive processing. If perceptions and beliefs are the result of recursive inferences, then 'false' perceptions and beliefs in the form of hallucinations and delusions must be the result of aberrant inferences (Fletcher and Frith [153]). The strongest evidence for differences in how the causes of sensory inputs are deduced in individuals with schizophreniform disorders originates in the dopaminergic theory of psychosis: Pharmacologically, the majority of antipsychotics block dopamine D2 receptors (Seeman and Kapur [495]) whereas amphetamines and other psychoactive drugs which act on dopaminergic pathways can induce symptoms similar to those of psychosis (Lieberman et al. [326], Angrist and Kammen [11]). Furthermore pathophysiology of the dopamine system in psychotic patients has been found in imaging studies as well as post-mortem histologies (Zakzanis and Hansen [621], Seeman and Niznik [496], Kegeles et al. [268]). Thus the dopaminergic mechanisms that have been shown to underlie reward (and by extension prediction error) processing in associative learning (Schultz et al. [492], Schultz [491, 490]) provide a unifying framework for linking aberrant dopaminergic function and the positive symptoms in schizophrenia. Hallucinatory experiences can be induced in healthy people by administering NMDA-antagonists such as ketamine (which affect both glutamatergic and dopaminergic systems) causing aberrant prediction error processing (Corlett et al. [94]).

As with neurocognitive theories of autism, the details of the various theories unfold with minor descriptive differences - whereas some may see the core symptom as a difference in saliency attribution (Kapur [266], Heinz and Schlagenhauf [226]), others call it a 'prediction error disequilibrium' leading to different levels of severity and chronicity (Yamashita and Tani [614]), a misestimation of error size (and not a deficit in the generation of prediction errors, see Todd et al. [558]), an inappropriately high expectation of noise (Hohwy [236], p.58), high precision on noisy information (Stuke et al. [534]) or (localised) decreased precision of (some) prior beliefs relative to sensory data (Sterzer et al. [531]).

On a behavioural level - while not all findings are without contradiction - the majority of evidence in favour of prediction error deficits comes from decreased mismatch negativity signals (MMNs) in schizophrenia (McCleery et al. [360]), attenuated prepulse inhibition (Braff et al. [61])[22], gating and habituation deficits (Braff et al. [62])[23] and difficulties with

[22] Autistic individuals on the other hand display an increase in prepulse inhibition (Madsen et al. [344]).

[23] A direct comparison of sensory gating in autism and schizophrenia reported intact performance in autism, but attenuated P50 responses in schizophrenia (Magnée et al. [347]).

reward processing (Gold et al. [188]). Imaging studies have revealed attenuated 'prediction error signals' in the prefrontal cortex (Corlett et al. [95]), caudate, thalamus, insula and amygdala (Gradin et al. [193]) and midbrain, striatum and limbic systems (Murray et al. [386]).

Autism, a term initially used to describe withdrawal into an inner fantasy world in schizophrenia (Bleuler [56]), was borrowed by Kanner [261] and Asperger [18] to describe their case studies who exhibited social withdrawal and bizarre behaviour reminiscent of symptoms observed in patients with schizophrenia. While these two diagnoses are now clearly delineated from each other, researchers still have an interest in the potential commonalities and overlap of behavioural, neural and genetic markers. Impairments of social cognition and co-occurring atypical neural activation (Pinkham et al. [432], Couture et al. [102]) as well as difficulties with metacognition in schizophrenia (Lysaker et al. [342]) all echo similar findings in autism. Frith [171] noted, that even when the ease with which one can see cognitive and affective similarities across psychiatric disorders are taken into account, both conditions share a core deficit in what they termed 'second order representations': The ability to have knowledge about knowledge. Whereas in autism this deficit is most apparent in a basic mentalising deficit, individuals with schizophrenia might draw incorrect inferences about their knowledge of the world or other people's intentions (leading for example to paranoid delusions). The reason for the discrepant symptom manifestations lies in the different age of onset; whereas individuals with schizophrenia acquired a stable perception of the world and other people prior to the onset of the condition, autistic individuals experience a stable and enduring deficit in representing second-order beliefs. Although research in the last 20 years has refuted the idea that autism is characterised by a total and enduring absence of the ability to ascribe mental states to others (Scheeren et al. [482]), the basic tenet of Frith's hypothesis that similar underlying deficits in intentionality processing and metacognition give rise to both schizophrenia and autism, is still a frequently supported idea.

In a dimensional approach in healthy controls, a common 'social disorganisation phenotype' has been found to load onto both measures of schizotypy and autism with dissociable factors of perceptual oddities (schizotypy) and rigidity (autism) respectively (Ford and Crewther [156]). However the incidence of schizotypy in autism seems to be multidimensional (Gadow [179]) and the traits that appear to mediate schizotypy in autism include alexithymia (Russell et al. [476]), but not depression or anxiety (Mealey et al. [362]).

Autism and schizophrenia also seem to share some etiological risk factors (Sullivan et al. [536]) as well as rare small chromosomal variants (microdeletions or duplications) (Rapoport et al. [449], Hoeffding et al. [231], McCarthy et al. [358]) and copy number

variations (Levinson et al. [323]). Genetic variants implicated in altered synapse formation have been reported for both conditions (Kenny et al. [271], Liu et al. [328]) and a parental history of psychotic disorder is a risk factor for autism (King and Lord [282], Larsson et al. [310]). Affective psychoses are particularly common among autistic individuals themselves compared to diagnoses of schizophrenia (Larson et al. [309]).

Taken together this has led some researchers to believe that schizophrenia might be 'on the autism spectrum' and may be overrepresented at the higher-functioning end (King and Lord [282]). Analogously, a higher incidence of autistic-like traits and autism diagnoses is found in individuals with psychosis (Kincaid et al. [281]). Several research groups have also reported positive correlations between schizotypy and autistic traits in the general population (Louzolo et al. [335]), but Russell-Smith et al. [478] note that the specific underlying impairments leading to these self-reported traits seem to differ.

However when scrutinising the overlap in symptom descriptions between the two diagnoses, the scope for misdiagnoses seems evident: Social deficits, restricted interests and reduced affect display (which are all common symptoms in ASC) might easily be mistaken for psychotic disorders (Cochran et al. [86]). There is some indication that symptoms such as formal thought disturbances which are usually associated with psychosis-proneness are not predictive of psychosis in autism but rather seem to be an indication of autism severity (Eussen et al. [146]). Follow-ups with small samples of autistic individuals with co-morbid psychotic disorders found that the (mis)diagnosis of psychosis was very likely a result of moving to adult psychiatric services where clinicians might be less familiar with the presentation of autism and/or a need for a diagnosis other than autism to justify inpatient care (Van Schalkwyk et al. [579]).

With the most common genetic variations, single nucleotide polymorphisms, autism and schizophrenia show only low correlations compared to other disorders (Cross-Disorder Group of the Psychiatric Genomics Consortium et al. [110]). More generally speaking autism common variants in genome-wide association studies do not seem to be linked to general childhood psychopathology (Riglin et al. [459]). The different developmental time-course of schizophrenia and autism has also been used to argue against a true overlap between the conditions: Whereas schizotypal traits might be seen as a risk factor for developing psychosis later in life, autistic traits could at most be a risk factor cross-generationally. Furthermore while autistic symptoms tend to become more attenuated with age - the same prognosis can't be made for schizophrenia.

On the other side of the debate are researchers supporting a conceptualisation of schizophrenia that places it at the diametrically opposite end of the diagnostic spectrum to autism

(Crespi et al. [108]). According to this hypothesis, psychotic disorders are characterised by brain undergrowth whereas autism is the result of dysregulated overgrowth leading to different cognitive and behavioural profiles: While autism frequently features deficits in mentalising abilities, diminished imagination and creativity and a preference for processing local stimuli over global ones, psychotic disorders often involve 'hypermentalising' in the form of paranoia, loose association formation rather than literalness and global processing advantages (Crespi and Badcock [107]). Higher autistic traits in individuals with schizophrenia seem to result in fewer mentalising errors and better overall functioning which is interpreted as supporting the diametric model (Vaskinn and Abu-Akel [580]). Psychometric investigations into the common factor of social difficulties in schizophrenia and autism reveal opposite loadings for autistic and (positive) schizotypal features (Dinsdale et al. [132]), however so far opposite profiles of autistic versus schizotypal traits have not been found in the general population (Russell-Smith et al. [477]). Under the auspices of this theory, autism and schizophrenia are often interpreted as being inverse aberrations of predictive processing: While schizotypal traits predispose to overly strong reliance on (inaccurate) priors[24], autistic perception undervalues priors in favour of sensory information (Tulver et al. [562], Aru et al. [15]).

1.5 Aims of this Dissertation

Bearing in mind the sparsity of empirical inquiries into sensory prediction in autism, the aim of this dissertation is to capitalise on the aforementioned link with psychotic disorders to fill in some of the experimental gaps regarding predictive processing in the former. Thus, experimental paradigms which have already shown their usefulness in assessing predictive processing differences in psychosis, will be implemented with autistic adults to probe different forms of perceptual inferences. In keeping with dimensional approaches to psychiatry, all participants also completed questionnaire measures of autistic and schizotypal traits so that their influence on prediction can be investigated. Such preliminary investigations cannot dissect or corroborate specific mechanistic accounts or neural circuitry underpinning predictive processing as outlined above, but rather they are intended to narrow down the range of plausible candidate theories. In doing so my ambition was to probe different kinds of sensory priors: Chapter 2 will explore how low-level predictions in the motor system can attenuate the subjective experience of sensory effects (in this case tactile stimulation) and

[24] Although some have hypothesised that the diverging evidence in schizophrenia could be better understood if the overreliance concerned higher-order priors only (Tulver et al. [562]).

whether this differs in adults with autism. In Chapter 3, higher-order contributions of volition and awareness to sensorimotor prediction are examined in autistic and non-autistic adults. In Chapter 4, a paradigm is presented which measures the contribution of prior knowledge to the disambiguation of complex visual scenes, thus bridging the gap to predictive processing in a non-motor modality. Again a group of autistic and non-autistic adults took part in this experiment. Finally, Chapter 5 attempts to shed some light on the psychometric properties of the autism and schizotypy questionnaires that were used in this dissertation and whether they are suitable for use in both autistic and non-autistic populations.

Chapter 2

Sensorimotor Attenuation

2.1 Introduction

> “ Τοῦ δὲ διεψεῦσθαι αἴτιον ὅτι οὐ μόνον τοῦ αἰσθητοῦ κινοῦντος φαίνεται ἀδήποτε ἀλλὰ καὶ τῆς αἰσθήσεως κινουμένης αὐτῆς, ἐὰν ὡσαύτως κινῆται ὥσπερ καὶ ὑπὸ τοῦ αἰσθητοῦ[1] ”

ARISTOTLE, PARVA NATURALIA: DE INSOMNIIS

The fact that the world appears stable despite our frequent saccadic eye movements has puzzled philosophers since Antiquity. How, if the same sensory input can be generated both by movement of the eye (reafference) and movement of the external world (exafference), can we distinguish the two? An initial hint as to the volitional aspect of this came from case studies involving patients with ocular paralysis who report an illusory shift of the environment in the direction of the intended (but not executed) saccade (Kornmüller [292], Perenin et al. [422]).

Mittelstaedt and Holst [237] suggested that this was due to an internal feedback process which they called ‘Reafferenzprinzip’ (‘principle of reafference’) wherein (re)afferent signals are attenuated by the extent to which they are congruent with the sensory signals predicted by an efference copy (‘Efferenzkopie’) of the outgoing motor command (see Figure 2.1).[2] More

[1] ‘The ground of such false judgements is that any appearances whatever present themselves, not only when its object stimulates a sense, but also when the sense by itself alone is stimulated, provided only it be stimulated in the manner as it is by the object.’ Translation taken from Aristotle et al. [12].

[2] Though it is important to note that the principle of reafference is probably not in itself sufficient to explain visual space constancy, see Bridgeman [63].

recent research has incorporated the idea of efference copies into a wider literature on the use of forward models in both engineering and biological systems (Wolpert and Ghahramani [611], Jordan and Rumelhart [257]) - thus called because of their objective to model causal relationships between actions and outcomes.[3] In motor control, forward models are thought to use the current state estimate of the body (eg. the degrees of freedom of the effector and its current configuration), the motor command and knowledge about the state of the external world to generate an estimate of the new state and the likely sensory consequences. They can thus sidestep the delays of the real sensory feedback, thereby promoting faster error correction when controlling vestibular functions (Cullen and Roy [111]), grip force (Flanagan and Wing [152]), motor coordination under altered but usually invariant physical principles (McIntyre et al. [361]) and the mental simulation of movements (Sirigu et al. [513]).

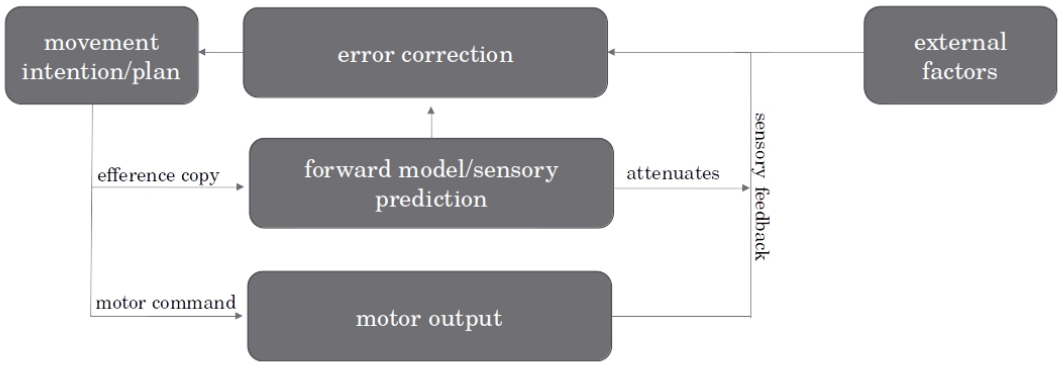


Figure 2.1 Illustration of sensory-motor transformations and sensory attenuation in motor control theory

2.1.1 Attenuation of Self-generated Sensory Input

An implementation of forward models in the motor-tactile domain would suggest that self-generated forces are perceived as weaker than externally-generated forces of the same magnitude as they will be associated with smaller prediction errors than externally generated sensory input (Blakemore et al. [51]). The overestimation of external forces when matched with a self-generated force is a surprisingly robust effect that has been replicated across a

[3] Alternative models build on inverse relationships between goals/target locations and the current state estimate to infer motor plans (Atkeson [19]). For a review of possible architectures of paired inverse and forward models see Lalazar and Vaadia [306]

range of experimental paradigms, volunteers and laboratories: Shergill et al. [505, 506], Voss et al. [589], Teufel et al. [552], Walsh et al. [593], Therrien et al. [556], Pareés et al. [413].

However voluntary action effects are not in themselves sufficient to generate precise predictions as has been shown by Blakemore et al. [52] who reported a lag between the anticipatory grip force when the load was produced indirectly (for example via a joystick) highlighting the importance of congruent sensory feedback to the acting organism.[4] This observation has given rise to a number of experimental paradigms where oblique or manipulated self-generated forces are used as a proxy for externally provoked sensory input. The task used in this chapter is one instantiation of this.

Sensorimotor Attenuation in Atypical Nervous Systems

One alleged 'use' of sensory attenuation is the labelling of actions as either self- or externally generated, thereby giving rise to a Sense of Agency (SoA). This has been a particularly attractive theoretical premise for investigating the mechanisms that lead to psychotic experiences such as hallucinations and delusions by way of misattribution of actions and their causes (Feinberg [149], Ford and Mathalon [154], Frith and Done [172], Ford et al. [155], Louzolo et al. [336]). Indeed such deficits in sensorimotor prediction have been demonstrated in patients with schizophrenia (SCZ) using the force matching task (Shergill et al. [506]) and saccadic suppression (Lindner et al. [327]). Furthermore self-reported delusion-proneness in subjects with no neurological or psychiatric history has been associated with a reduced effect of sensory attenuation (Teufel et al. [552]), although not all studies have been able to replicate this effect (Humpston et al. [244]).

However scaling of grip force in relation to the actual load is intact in SCZ (Delevoye-Turrell et al. [124]), but impaired in patients with reduced manual sensibility (Nowak and Hermsdörfer [402]) suggesting that the feedforward mechanism for generating predictions of sensory consequences of actions (impaired in SCZ) is distinguishable from predictions about state estimates and the ability to integrate sensory feedback during movement (intact) (Haggard et al. [207]).

Motivations to investigate sensory attenuation in autism are manifold: Similarities in the sensory processing differences between ASC and SCZ have been noted (Haque [216]). Furthermore in the same way that disruptions of SoA have been linked to positive symptoms

[4] In addition to this the magnitude of the attenuation effect (=prediction error amplitude?) is also affected by the timing (Bays [40], Bäss et al. [72]) and spatial co-alignment (Haggard et al. [207], Blakemore et al. [51], Kilteni and Ehrsson [279]) but not gain of the force production. No weighting of predictive attenuation according to the temporal-spatial congruence of the exerted forces has been reported under body-swap illusions (Van Doorn et al. [577]).

in psychosis, alterations of SoA in autism have been hypothesised to give rise to the social impairments (Lafleur et al. [301]). In fact disruptions of internal self-representations and self-reflections about one's intentions and mental states (Frith [173], Frith and Happé [175], Lombardo and Baron-Cohen [329]) have been noted in autism for years. Compared to controls, autistic individuals also tend to show greater force variability (Mosconi et al. [377], Wang et al. [594]) which has been associated with a higher reliance on forward models in older people (Wolpe et al. [610]). In line with this, children with autism also seem to build much stronger associations between motor commands and proprioceptive feedback than their non-autistic peers (Haswell et al. [220]). When interacting with novel objects, individuals with high autistic traits have less accurate postural anticipation (Schmitz et al. [485]) and sensorimotor predictions about the grip and load force needed (Buckingham et al. [70]).

Additionally, sensory attenuation of self-generated stimuli has anecdotally been implicated in the discrepant contrastive reactivity to sensory input by autistic children: Kanner noted about one of his patients that she 'was frightened by noises and anything moving toward her. She was so afraid of the vacuum cleaner that she would not even go near the closet where it was kept and when it was used, ran out into the garage, covering her ears with her hands. [...] Any noise, any interruption disturbed her.' (Kanner [261], p.230) 'Yet it is not the noise or motion itself that is dreaded. The disturbance comes from the noise or motion that intrudes itself, or threatens to intrude itself, upon the child's aloneness. The child himself can happily make as great a noise as any that he dreads and move objects about to his heart's desire.' (p. 245). This observation has also been echoed in my personal observations and communication with autistic people and their carers who have described a discrepancy in reactivity to sensory stimulation under the control of the person with autism versus sensory stimulation that is externally generated exceeding the expected differential response in non-autistic individuals. Thus while both autistic and non-autistic individuals will experience externally generated sensory input as more intense, this seems to be amplified in autism. Furthermore in autism, but not in typical development, sensory reactivity has been found to be related to intolerance of uncertainty (Neil et al. [393]).

However previous research on sensory attenuation in ASC has reported unimpaired cancellation of self-generated tactile stimulation in the form of self-tickling (Blakemore et al. [53]).[5] Examinations of ticklishness in response to self- and externally generated tactile input might be a special test case of sensory attenuation though as it 1) relies on self-report rather than more robust quantitative measures and 2) has a subjective experiential quality

[5]Individuals with high schizotypal traits are once again more likely to report self-generated tactile input as ticklish (Whitford et al. [601], Lemaitre et al. [320]) as are patients with auditory hallucinations and/or passivity experiences irrespective of the underlying psychiatric diagnosis (Blakemore et al. [54]).

that can be difficult to capture (Murray [385]). Furthermore the exact receptor mechanism of knismesis is not yet known (Selden [497]) and sensitivity to touch and ticklishness remain clearly distinguishable (Harris [219]) with Pacinian corpuscles being more likely involved in the sensation of low-threshold vibrations and Merkel's disks coding for (light) pressure (Purves et al. [442]). In line with predicted differences in sensory attenuation, other paradigms investigating tactile perception in autism found an increase of the dynamic detection threshold which led the authors to suggest an impairment in feed-forward inhibition which could then lead to the heightened sensory reactivity (Puts et al. [444]). Autistic individuals - in contrast to controls - also do not seem to exhibit an attenuation of the auditory N1 component when the tones are self-initiated versus externally generated (Laarhoven et al. [300]).

The following study therefore investigates sensory attenuation of tactile stimuli in individuals with and without ASC. Under the assumption of models that interpret autistic cognition as the result of a general disorder of predictive abilities such as Sinha et al. [512] and Pellicano and Burr [419], sensory attenuation should be reduced in autistic individuals due to a flat and/or noisy prior around the expected sensory consequences of self-generated actions. However as previously reported, structural priors (as opposed to contextual ones) seem to be largely intact in ASC (Pell et al. [417]) and evidence for a universal endophenotype of aberrant prediction in autism is still lacking. Furthermore as no deviant sensory reactivity to self-produced stimuli has been reported in autism, it seems unlikely that this is the root cause of tactile sensitivity. The latter could therefore equally be related to overly precise predictions for self-generated sensory input (and greater attenuation[6]) which in turn could make externally produced sensations comparatively more intense, although investigations into the subjective correlates of relative differences in intensity between self/other sensory processing have not yet been performed. This chapter aims to answer the following questions:

1. Do autistic individuals differ from non-autistic individuals in the extent to which they attenuate the sensory consequences of self-generated movements?

2. How do autistic and schizotypal traits - as measured by the AQ and PDI - relate to the magnitude of the sensory attenuation effect in autistic and nonautistic individuals?

[6]This is in line with reports of increased saccadic suppression in individuals with a high level of self-reported autistic traits (Crewther et al. [109]).

2.2 Methods

2.2.1 Participants

Twenty-seven volunteers with a clinical diagnosis of an autism spectrum disorder and 26 healthy control participants (with no history of neurological or psychiatric illness) took part in the study. The study protocol was approved by the Psychology Ethics Committee of the University of Cambridge and all participants gave written informed consent. Cognitive function for all study volunteers was assessed using the timed version of the Ravens Advanced Progressive Matrices (RAPM) (Raven et al. [450]) and the Wechsler FSIQ in the case of one ASC volunteer. Furthermore all participants filled in the Edinburgh Handedness Inventory [406] as handedness can have an effect on force-perception and production ([414, 184]). On the handedness inventory, a score above 40 reflects right-handedness and a score below -40 left-handedness.

Three ASC participants were excluded from the subsequent analysis as two had a diagnosis of schizophrenia or another psychotic disorder and one was unable to complete the experiment due to difficulties with maintaining the required arm posture. Aside from psychotic disorders no other psychiatric conditions served as exclusion criteria as anxiety, depression, OCD and other neurodevelopmental disorders such as ADHD and dyspraxia are thought to be extremely common/co-morbid in ASC (for prevalence estimates see Leyfer et al. [324], Eaves and Ho [137], White et al. [600]). So for pragmatic reasons with regard to recruitment and with the aim to test a representative sample of ASC individuals, reports of the aforementioned psychiatric diagnoses as well as current psychoactive drug use did not serve as exclusion criteria. Previous behavioural as well as neuroimaging studies have included high rates of autistic subjects currently on medication (50% in Dichter et al. [130], 36% in Koshino et al. [293], Monk et al. [368]) and found no effects of medication status on task performance. Ten of the participants with autism had co-morbid diagnoses of depression and/or anxiety and 6 were currently taking SSRIs. A further two people had a diagnosis of ADHD (one on medication) and one had unmedicated OCD.

Participants were well-matched for age, IQ (IQ information was unavailable for one control participant) and gender but the groups differed on the Edinburgh Handedness Inventory with three left-handed volunteers in the ASC group and none in the controls (see Table 2.1).

All but three of the ASC participants were assessed with module 4 of the Autism Diagnostic Observation Schedule (ADOS, [331])[7] and while the group was moderately

[7]Where ADOS scores were unavailable, the assessments were carried out by the author who has achieved research reliability (inter-rater reliability with a certified ADOS trainer) on all modules of the ADOS.

Table 2.1 Participant Demographics

Group	**Age** *(SD)*	**Sex** *(m:f)*	**Handedness** *(SD)*	**IQ** *(SD)*	**AQ** *(SD, range)*
ASC *(N=24)*	30.1 *(9.2)*	11:13	53.8 *(44.5)*	105.2 *(12.5)*	36.4 *(7.7, 19-48)*
Controls *(N=26)*	30.6 *(6.0)*	9:17	75.3 *(19.2)*	106.8 *(11.6)*	17.0 *(6.6, 6-28)*

symptomatic (mean score: 6.7), only nine participants met cut-off criteria for an autism spectrum condition and none met diagnostic criteria for autism. Low sensitivity of the ADOS module 4 has previously been reported and attributed to compensatory behaviour and 'milder ASDs' ([36]). Even among children, those with a diagnosis of an autism spectrum condition that is not 'childhood autism' (ICD-10) often to not meet the diagnostic cut-off for the ADOS (Baird et al. [23]). All of the participants included in this study had received a diagnosis of an autism spectrum disorder by a qualified clinician and scored above cut-off on the AQ (with the exception of one participant whose exclusion did not affect the interpretation of the final results).

2.2.2 Experimental Procedure

The experiment was modelled on the design by Shergill et al. [505] in which a lever – via a torque motor - exerts mild pressure onto the participants' left index finger. Depending on the condition, participants were asked to match the experienced pressure to the point of subjective inequality by either pressing directly on the lever with their right index finger ('internal condition') or by adjusting a slider which controlled the torque motor ('external condition'), see Figure 2.2. The slider was a potentiometer which transduced a force gain at the ratio of 0.5 N/cm.[8] The target force was presented for 2.5 seconds (ramped up and down linearly over 0.25 seconds) after which an auditory go-signal indicated that participants should make their response to ensure that the matching took place within 2 seconds of the target force being withdrawn. After 3 seconds a second auditory signal indicated the end of each trial and instructed participants to lift their right index finger from the lever or move the slider back to the starting position. Mean force production was measured between 2 and 2.5 seconds after the start of the matching period, as in previous studies (Voss et al. [589]). Within each condition 10 different force magnitudes (8 trials each) were applied in randomised order (0.5N, 0.75N, 1N, 1.25N, 1.5N, 1.75N, 2N, 2.25N, 2.5N, 2.75N). Subjects first completed a 5-trial practice session for both conditions to ensure that they understood

[8]The slider was not obscured during the task opening up the possibility that participants used visual feedback to guide their responses which might be different in ASC, see Mosconi et al. [377].

the task and were able to respond within the required time window. They then completed one 'internal' and one 'external' block with 80 trials (160 trials in total). Invalid trials due to too slow or fast responses were repeated until a total of 80 valid trials had been completed. Practice sessions and test blocks were counterbalanced across both experimental groups.

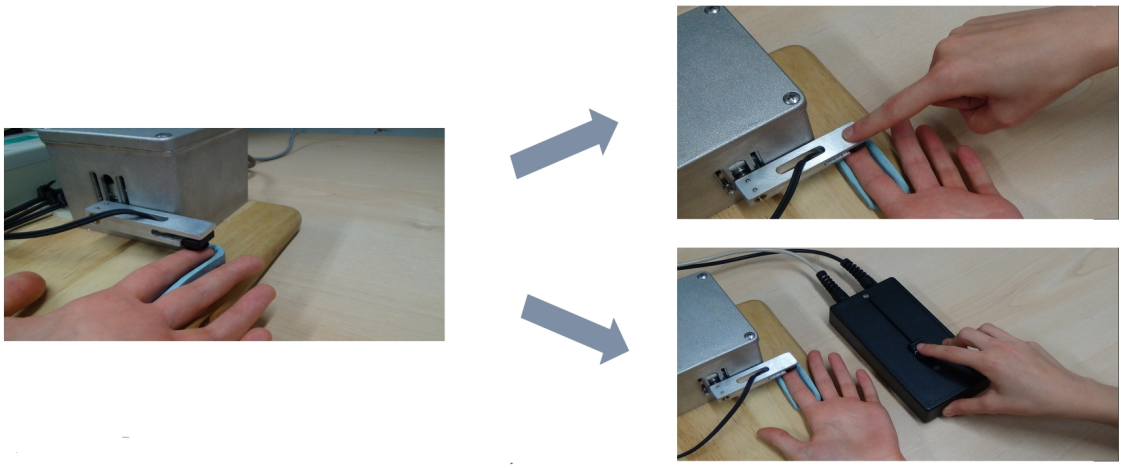


Figure 2.2 Illustration of the experimental setup

The 21-item Peters Delusion Inventory (PDI, Peters and Garety [425]) was used to quantify schizotypal traits in all participants and the Autism Spectrum Quotient (AQ, Baron-Cohen et al. [32]), a 50-item self-administered questionnaire, was used as a measure of autistic traits. AQ and PDI scores were unavailable for one ASC participant.

Data Analysis

Individual regression lines of target force versus matched force for each subject were fitted in MATLAB for the 'internal' and 'external' condition (as the target force magnitudes differed minimally from trial to trial) and then summarised as group regressions for both conditions. Basic force attenuation was measured by calculating an overcompensation score based on the difference between the matched forces in the 'internal' and 'external' condition for each force level. To explore potential differences in sensory attenuation, three separate mixed model analyses of variance (ANOVAs) with diagnostic status as the between-subject variable were run in SPSS to look at 1) the intercept of the regression lines in the 'internal' and 'external' conditions, 2) the slope of the regression lines in the 'internal' and 'external' conditions (which has been thought to be an indication of sensory sensitivity, see Wolpe

et al. [610]) and 3) the mean matching forces in the 'internal' and 'external' conditions normalised by the mean target forces. Visual inspection (histograms and Q-Q plots) of the data and Kolmogorov-Smirnov normality tests with Lilliefors correction suggested approximate normal distributions of the relevant measurements. All further analyses and graphs were done in R.

2.3 Results

2.3.1 Main Analysis

Matching forces correlated significantly with the target forces across all participants (at a level of $p<0.05$) with the exception of one ASC participant in the 'internal' condition ($r=0.617$, $p=0.058$) and one control volunteer in the 'external' condition ($r=0.517$, $p=0.126$). These subjects were still included in the analysis as they seemed to understand the task and performed as expected in the other condition. One ASC participant was excluded from further analysis as his matching values in the 'internal' condition were more than 9 standard deviations above the mean. There was no difference between the groups on the number of missed trials ($t_{47}=0.074$, $p=0.942$). Handedness was not associated with the magnitude of sensory attenuation[9] ($t_{47}=-0.075$, $p=0.610$).

Both groups showed the characteristic force attenuation with overcompensation scores being significantly different from 0 ($t_{22}=6.385$, $p<0.001$ and $t_{25}=6.647$, $p<0.001$ for the ASC and control group respectively), but there were no group differences on a mixed ANOVA (patients versus healthy subjects and 'internal' versus 'external') of the mean matching force normalised by the mean target force ($F_{(1,47)}=0.001$, $p=0.980$) or of the intercept ($F_{(1,47)}=0.087$, $p=0.769$) or slope ($F_{(1,47)}=0.038$, $p=0.847$) of the 'internal' and 'external' conditions (see Figure 2.3).

As expected groups differed on the AQ ($t_{46}=-9.367$, $p<0.001$) and more surprisingly also on the PDI ($t_{46}=-2.441$, $p=0.019$) (Figure 2.4). Distributions were non-normal for the PDI in the AQ group (Shapiro-Wilk statistic: 0.858, $p=0.005$), a Mann Whitney U test was still significant at $p=0.04$ ($U=385.000$).

Using the intercept in the internal condition as the main measure of sensory attenuation (Wolpe et al. [610]) the following correlations were observed: As observed previously (Teufel et al. [552]), sensory attenuation correlated negatively with schizotypy in the control ($r=-0.485$, $p=0.012$) but not ASC group ($r=0.000$, $p=1.000$) whereas there was a trend for a

[9] As measured by the overcompensation score.

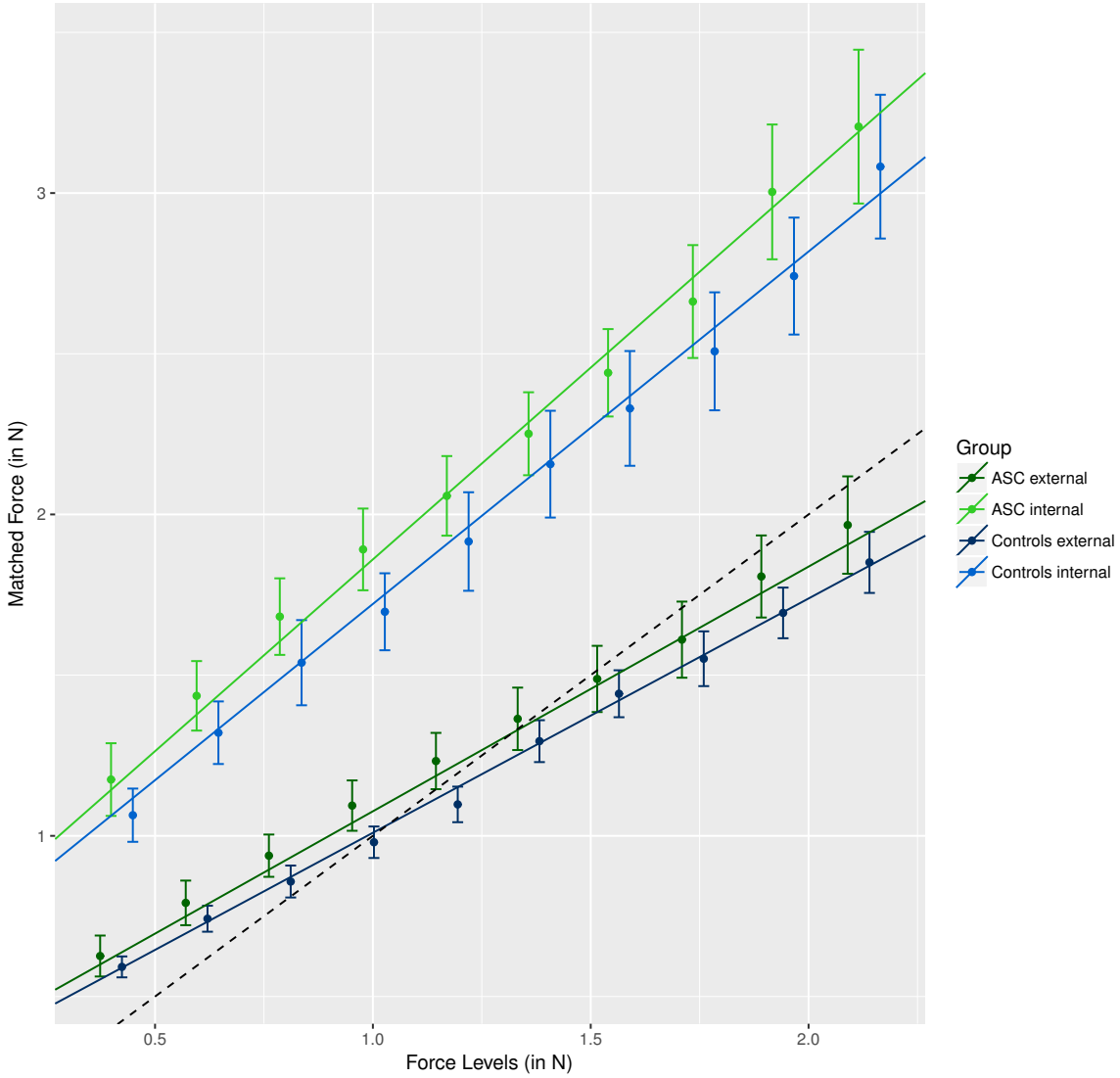


Figure 2.3 Mean linear regressions for the matched forces in the internal and external conditions. Jitter was added to prevent overplotting. Error bars represent ±1 standard error (SE) of the mean. Perfect matching performance is indicated by the dashed black line.

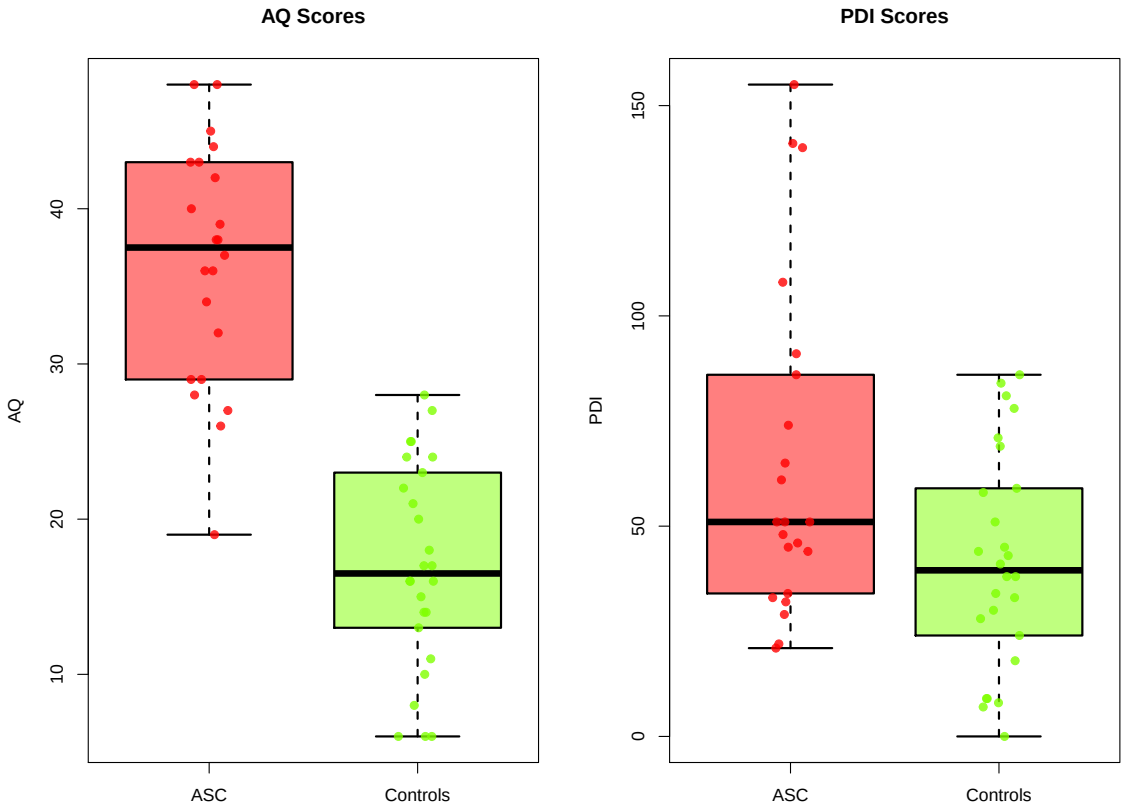


Figure 2.4 Boxplots with median and interquartile ranges for AQ and PDI scores.

positive relationship between self-reported autistic traits on the AQ and sensory attenuation in the ASC (r=0.408, p=0.059) but not control group (r=-0.131, p=0.525), see Figure 2.5. The correlation with AQ in the ASC group was mainly driven by the 'social' (r=0.500, p=0.021) and imagination (r=0.487, p=0.025) subscales of the AQ.

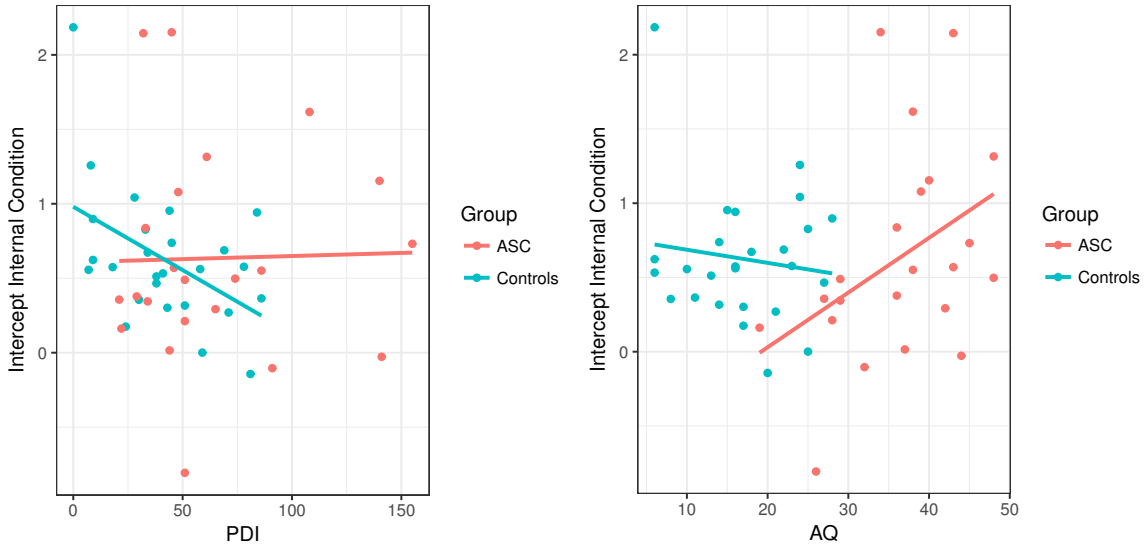


Figure 2.5 Correlations between sensory attenuation and questionnaire measures

Overall, ASC subjects were 'noisier' in their responses across both conditions when mean-squared errors (MSE) for individual regression lines were computed (Mann Whitney U=417.500, p=0.018.)[10], see Figure 2.6.

2.3.2 Exploratory Analysis

One possible explanation for a worse regression fit in the autistic group may lie in the sampling method. If the volunteers with autism differed on parameters such as the overall time to stabilise (eg. with longer initial overshoots), downward drift (fatigue) or other factors affecting the overall shape of the force traces, the pre-determined time window might not be sampling the intended matched force accurately on every trial. Thus, in an attempt to minimise the effect of the shape of individual force traces, an alternative measure was adapted from Wolpe et al. [610] which searched for the 0.5 second time window with the least amount of variability on each trial (for an example see Figure 2.7). The 'time to stabilise', that is to say the time between the onset of movement and the calculated time window, did not differ between groups in either the internal ($t_{(47)}$=-0.277, p=0.783) or external ($t_{(40.6)}$=-1.975,

[10]The distributions were non-normal in both groups with p<0.001.

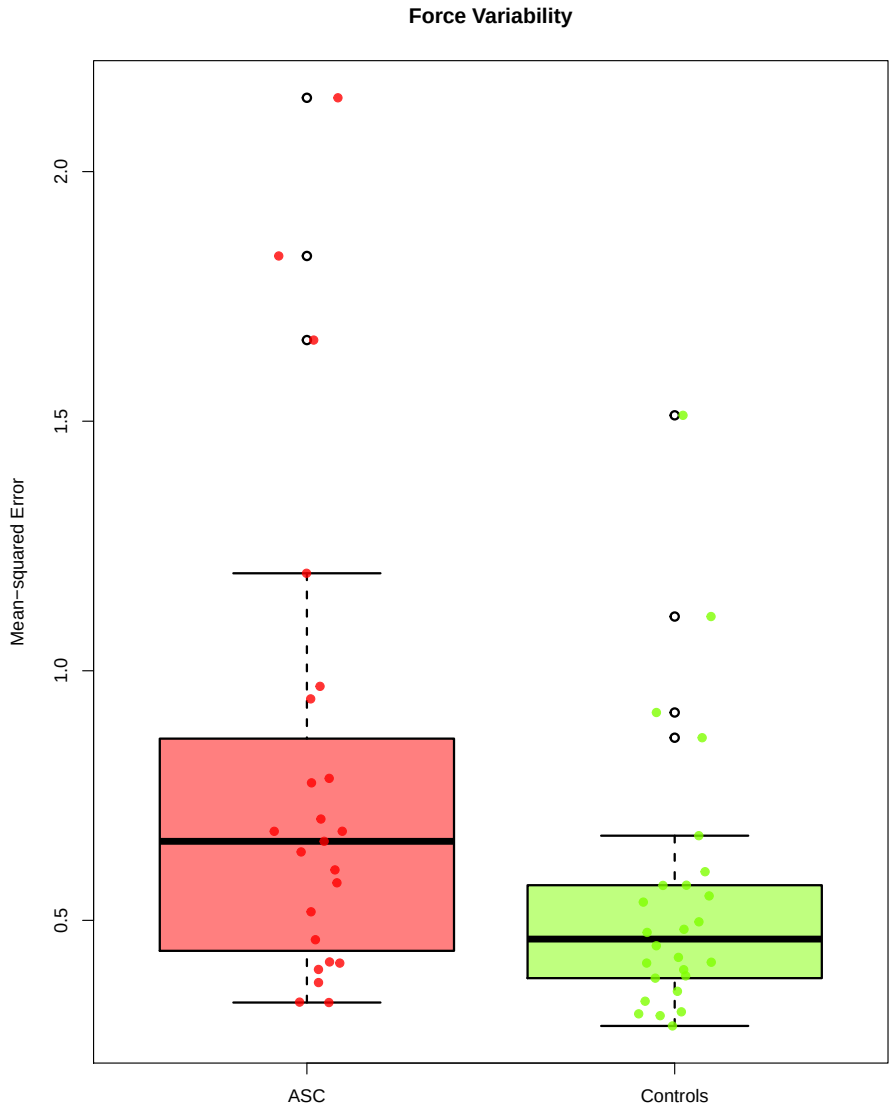


Figure 2.6 Boxplots with median and interquartile ranges for the MSEs of individual regression lines, both conditions combined

p=0.055, Welch's correction) condition though there was a trend for the ASC participants to take longer in the external condition.

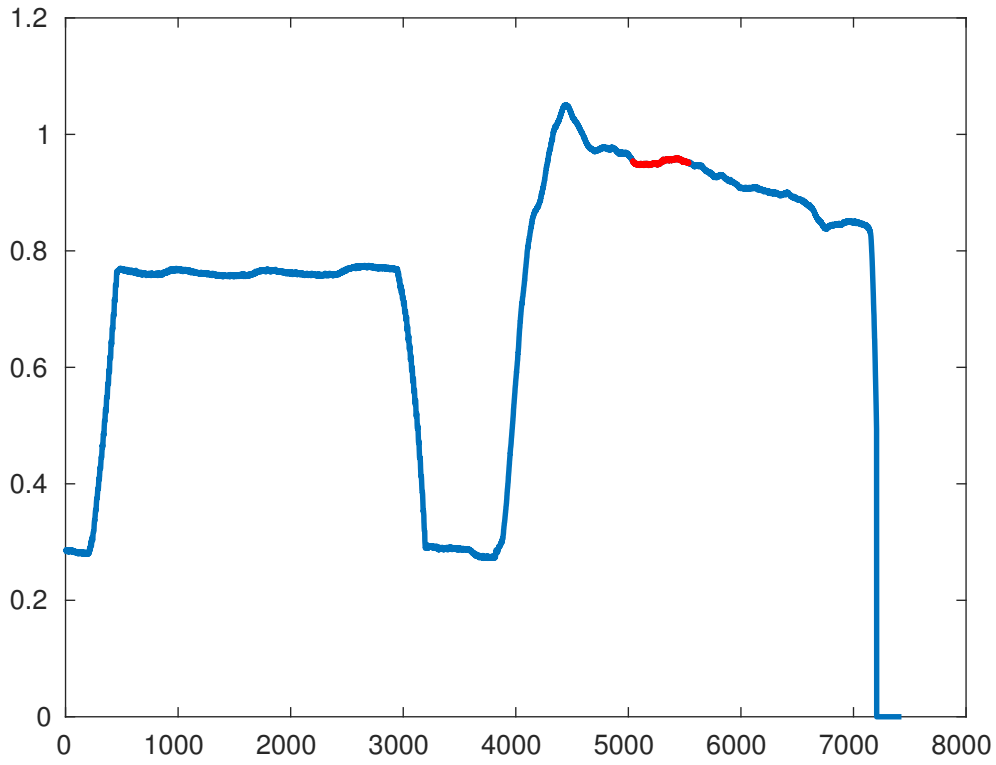


Figure 2.7 Example of the new sampling method on a force trace with a downward drift (new time window in red)

The results were similar to the main analysis: While the intercept for the 'internal' condition is higher for the ASC group in the adjusted analysis, the increased variance means that the results did not differ between standard time window and the adjusted sampling window on a mixed ANOVA (patients versus healthy subjects and 'internal' versus 'external') of the mean matching force normalised by the mean target force ($F_{(1,47)}$=0.645, p=0.426) or of the intercept ($F_{(1,47)}$=0.405, p=0.528) or slope ($F_{(1,47)}$=0.075, p=0.786) of the 'internal' and 'external' conditions (see Figure 2.8).

The correlations with questionnaire measures were largely the same: Sensory attenuation correlated negatively with schizotypy in the control (r=-0.462, p=0.018) but not ASC group (r=0.066, p=0.769) whereas there was a positive relationship between self-reported autistic traits on the AQ and sensory attenuation in the ASC (r=0.510, p=0.015) but not control group (r=-0.183, p=0.371), see Figure 2.9.

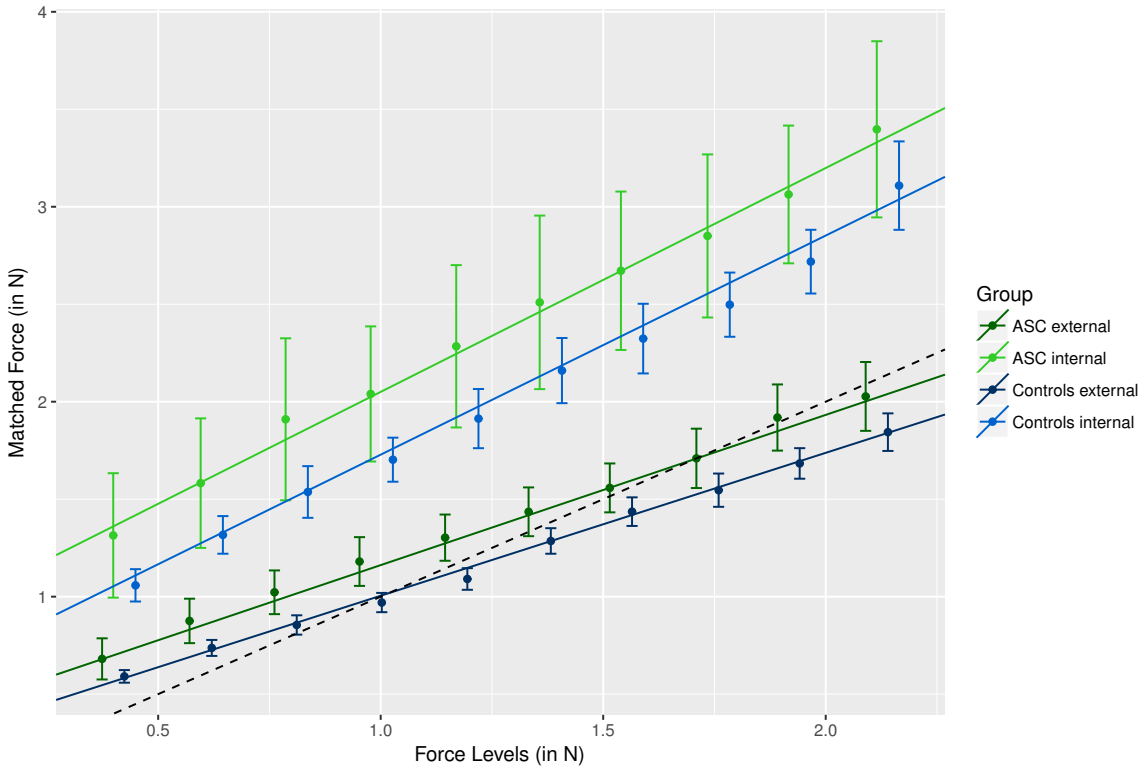


Figure 2.8 Exploratory analysis of the mean linear regressions for the matched forces in the internal and external conditions with an adjusted sampling window. Jitter was added to prevent overplotting. Error bars represent ±1 standard error (SE) of the mean. Perfect matching performance is indicated by the dashed black line.

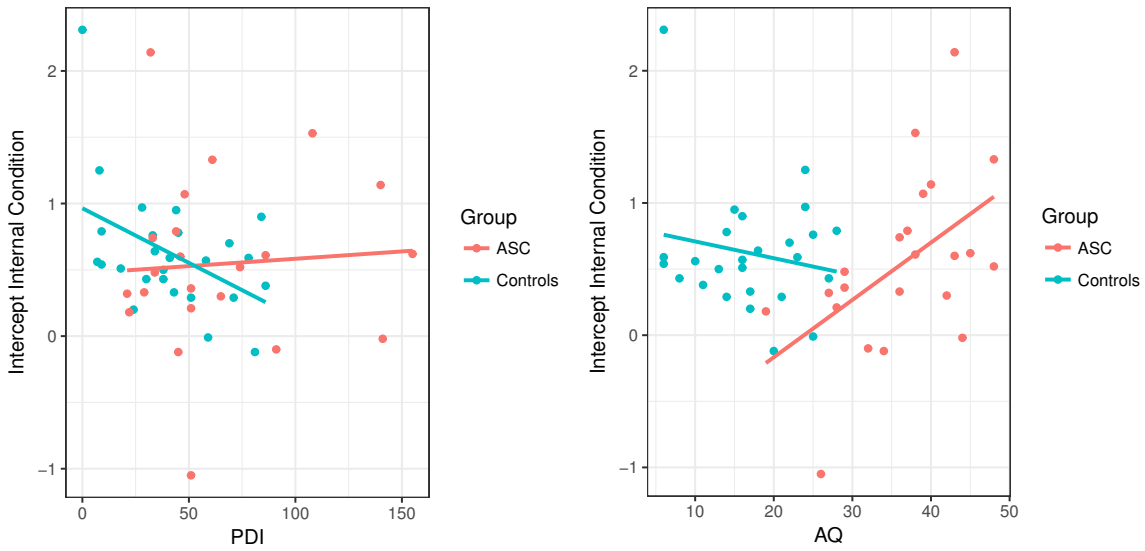


Figure 2.9 Correlations between sensory attenuation (adjusted analysis) and questionnaire measures

Visual inspection of the averaged force graphs across both conditions and groups suggested a downward drift in the finger condition in the ASC group (see Figure 2.10). Using the subtraction of the value at the end of the force delivery from the peak value as a proxy for the amount of drift, there was a trend for the ASC group to show a bigger difference ($t=_{(47)}$=-1.855, p=0.070).

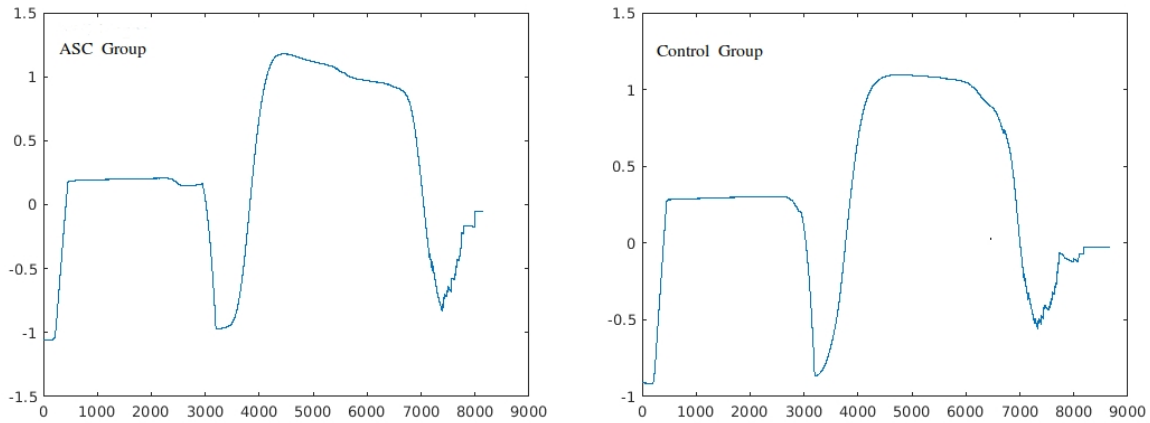


Figure 2.10 Averaged force traces in the 'finger' condition

2.4 Discussion

This study aimed to examine sensorimotor prediction in adults with autism by measuring the attenuation of self-generated tactile input that occurs as a result of a prediction generated by a forward model. In light of previous theoretical work that conceptualised autism as a 'disorder of prediction', one would expect to find reduced perceptual attenuation in the autistic group. My alternative hypothesis, namely that heightened sensory reactivity to externally produced stimuli in autism might be due to overly precise predictions of self-generated sensory input, would result in an increase of attenuation in the 'internal' condition.

The results do not support the idea of differences in the prediction of sensory consequences arising as a result of self-generated movement. Both groups demonstrated sensory attenuation of self-generated stimuli whose magnitude is in line with previously reported results (Teufel et al. [552], Shergill et al. [505], Wolpe et al. [610]). Some autistic participants demonstrated subtle motor deficits in force control replicating previous research (Wang et al. [594]).

Most strikingly the groups differed in the way that self-reported schizotypal and autistic traits related to task performance. This is especially noteworthy given the restricted range of

scores on the AQ for the ASC group (Bishop [48]). While the control group behaved in a way consistent with the hypothesis that abnormal agency experiences could be causally informed by a decrease in self-attenuation, the ASC participants did not follow this pattern. Instead higher autistic traits seem to be related to an increase in sensorimotor prediction. It is worth following up if sensory attenuation is indeed modulated by different latent traits in autistic and non-autistic individuals. An alternative explanation would be that the questionnaires used in this study do not measure the same latent traits in both groups for which there is some preliminary evidence (see Chapter 5).

It is also conceivable that rather than being a function of the level of autistic traits per se (as there was no correlation with autistic symptomatology on the ADOS), what is captured by the AQ are sensory processing differences; however the correlation did not seem to be driven by the 'attention to detail' subscale.

The lack of phenotyping for sensory reactivity and abnormalities is certainly a caveat of this study and could be addressed more thoroughly in future investigations.[11] More accurate assessment of sensory subtypes could also help to explain the commonly observed heterogeneity in task performance seen in the autistic group (Lane Alison E. et al. [307]). In the tactile domain, higher temporal resolution of tactile stimuli has been linked to hypersensitivity in individuals with autism (Ide et al. [247]) which was not assessed on this occasion. As predictive attenuation is not unique to the tactile domain (Benazet et al. [44], Cardoso-Leite et al. [74], Desantis et al. [127], Hughes and Waszak [242]), an investigation linking domain-specific sensory reactivity (like the frequently reported auditory defensiveness) to sensory attenuation might be better equipped to uncover potential differences. Furthermore although it is sometimes claimed that these sensorimotor processes are well understood given the extensive research into central and peripheral nervous system mechanisms supporting sensory gating (Rushton et al. [474]), their relationship with the perceptual attenuative processes seen in the force matching task is not entirely clear and there is some evidence that the two processes are functionally distinct (Palmer et al. [412]). While Holst and Mittelstaedt [237] conceived of the efference copy as an exact mirror of the motor command, Sperry's [523] contemporaneous work on optokinetic responses to eye rotation in fish investigated analogous sensorimotor transformations which he termed 'corollary discharge'. In contrast to the principle of reafference, Sperry's [523] corollary discharge does not have a specified

[11] Further limitations of the study include the high prevalence of psychiatric comorbidities (and associated medication) and the use of an adult population with a neurodevelopmental disorder who might have adopted some compensatory mechanisms for underlying atypicalities as indicated by the relatively low ADOS scores. Indeed some researchers have reported an improvement in the calibration of forward models with age in autistic individuals (Mosconi et al. [378]).

pathway of motor-sensory interaction; rather, the two streams can interact and depending on the tier of implementation its effect can be either facilitative or inhibitory (Crapse and Sommer [104]).

Thus tapping into predictive processes at other levels of the information processing hierarchy could still be useful in looking for possible shallow and deep endophenotypes of sensory processing differences. At the physiological level this could involve measuring the aforementioned amplitudes of sensory evoked potentials (SEPs) at the onset of self-paced movement and at the cognitive level one might probe higher level processes of sensory attenuation that are unrelated to efference copies such as priming effects (Sato [481], Sato [480]) and postdictive and expectation based attenuation (Voss et al. [590]). The issue of dissociating predictive and postdictive contributions to SoA will be the topic of the next chapter.

Chapter 3

Prediction and the Sense of Agency

3.1 Introduction

> *“What is an ‘I’, and why are such things found (at least so far) only in association with, as poet Russell Edson once wonderfully phrased it, ‘teetering bulbs of dread and dream’ – that is, only in association with certain kinds of gooey lumps encased in hard protective shells mounted atop mobile pedestals that roam the world on pairs of slightly fuzzy, jointed stilts?”*
>
> DOUGLAS R. HOFSTADTER, GÖDEL, ESCHER, BACH

The previous chapter explored the role of efference copies in the observed sensorimotor phenomena. But before Sperry, Holst and Mittelstaedt postulated their physiological theories, Helmholtz talked about the "Willensanstrengung" (‘effort of will’) that accompanies voluntary movements and can be used to reference certain spatial locations and thus support visual stability (Helmholtz [227]). Subsequent research on efference copies has confirmed the idea that sensory attenuation is at least partially driven by the volitional aspect of movement (Blakemore et al. [55])[1]: Disturbances of volition such as can be seen in patients with psychogenic movement disorders seem to reduce sensory attenuation (Pareés et al. [413]). Analogously, the impoverished sensory gating effects in schizophrenia are often seen as

[1] As mentioned before it is worth remembering that the reflex arc in vertebrates involving just spinal cord and primary and secondary sensory fibers is also capable of sensory gating mechanisms, i.e. generating and appraising proprioceptive prediction errors (Adams et al. [3], Crapse and Sommer [104]).

central to the higher-level disruptions of agency perception that manifest themselves in delusions of control (i.e. the decrease in sensory attenuation causes the agent to attribute his own movements to external forces). Paradoxically other symptoms cited in support of a breakdown of coherent agentic experiences in schizophrenia concern an *over-attribution* of causal efficacy: Individuals with schizophrenia occasionally report mistaking other people's actions for their own and experience augmented interference of external events on their own motor plans (Garbarini et al. [182]). They furthermore show strengthening of the 'causal self' under certain conditions (Haggard et al. [205], Maeda et al. [345]) and tolerate larger errors before the Sense of Agency (SoA) is stifled (Franck et al. [160], Knoblich et al. [288]). These phenomena cannot readily be explained by deviations in sensory gating mechanisms leading to the conclusion that SoA might not be a unitary process.

Indeed volitional movements involve numerous processes and distributed neural circuits responsible for the content and timing of actions (Zapparoli et al. [624], Mima et al. [366]). Voss and Synofzik [592] distinguish between the explicit judgement of agency and feelings of agency suggesting that the disparate findings in schizophrenia might be explained by a compensating mechanism whereby individuals respond to inadequate internal agency cues ('feeling') by increasing their reliance on external cues of agency ('judgement'), for example by widening the cue integration window during which sensory input is being interpreted as being caused by the self. Whereas the feelings of agency are almost entirely driven by predictive processes in the motor and visual systems, the judgment of agency (confusingly sometimes termed 'feelings of control' in the literature) leaves room for post-hoc inferences about external events. Thus, in the absence of cues from the motor system (such as during involuntary movement), external cues can modulate agentic experience instead and possibly even outweigh efferent signals (Moore et al. [375]).

3.1.1 SoA and Intentional Binding

One way to investigate the interplay between predictive mechanisms and SoA is the intentional binding (IB) paradigm. The IB effect refers to the fact that voluntary actions and their sensory consequences are perceived to be closer together in time than involuntary actions and their sensory outcomes (Haggard et al. [204], Prinz and Hommel [440]). The extent to which awareness of and belief about SoA facilitate this temporal contraction has been assessed with various paradigms including transcranial magnetic stimulation (Haggard et al. [204], Moore et al. [371]), manipulation of higher order causal beliefs (Desantis et al. [126], Haering and Kiesel [203]), passive movements (Nolden et al. [400], Engbert et al. [140]) and inhibited

actions with and without sensory consequences (Haggard et al. [206]). IB is relatively robust to manipulations of the sensory domains of the action effect (Haggard et al. [204], Ruess et al. [473], Engbert et al. [140]) and of the estimation method (Cornelio Martinez et al. [96], Nolden et al. [400], Cravo et al. [105], Humphreys and Buehner [243]), but the magnitude of the effect seems to rely on a complex interaction of voluntary intent, choice level, estimation method and predictability and temporal proximity of the stimulus. Contrasted with the forcematching task, higher order beliefs seem to play a larger role in SoA as measured by IB and are even present in cases where the outcome is not simply a sensory stimulus, but the action of another independent agent (Pfister et al. [429]). Furthermore IB is amenable to cognitive biases such as the self serving bias (Takahata et al. [545]) and emotional valence of the outcome stimuli can attenuate the effect (Christensen et al. [83], Yoshie and Haggard [618]). Depending on the experimental setup, subjectively shortened action-effect intervals have been reported to be steady for up to 4 seconds (Humphreys and Buehner [243]), to occur at 600ms but not 250ms (Nolden et al. [400]) or in contrary fashion to be more pronounced for intervals of 250ms compared to 650ms (Haggard et al. [204]). Thus the maximum measurable effect, and by inference SoA, may vary depending on the exact setup (Ruess et al. [472]).

The multitude of internal and external cues that play a part in giving rise to IB (and by extension SoA) also pose difficulties for converging on a unified theoretical framework: Suggestions that the central process of IB might be forward motor prediction (Wenke and Haggard [599]) are invalidated by binding effects in the absence of movement or presence of unpredictable outcome identity. Desantis et al. [125] aimed to separate the effects of both, motor identity prediction and temporal control on IB, and found that only temporal prediction drove the characteristic temporal compression of the interval between action and effect. A further popular account of IB conceptualises it as a consequence of ideomotor theory: Learned action-effect associations are stored as higher-order representations that can later be employed to select the appropriate action for the desired outcome (Nattkemper et al. [391]). Against this speaks the fact that intentional binding occurs even when non-actions or passive primes are associated with outcomes - the motor component thus does not seem obligatory (Kühn et al. [299], Moore et al. [375]).

The most compelling explanation simply frames it as another problem of ‘optimal cue integration’ that pits internal predictions and expectations against incoming sensory signals (Synofzik et al. [543], Moore and Fletcher [373]). The agency experience for predictable, simple and highly practised movements might be predominantly derived via efference copies whereas under more ambiguous authorship conditions more credence is given to prior beliefs

and representations about the current state of the world. Empirical evidence for shifts in the relative contribution for each system was provided by Wolpe et al. [609]. Synofzik and Voss [544] conclude that 'the sense of agency might not function as a unitary processing module (as it appears phenomenally), but in fact represent a complex supramodal phenomenon of largely heterogeneous functional and representational levels, with different agency cues receiving a different weighting on each level.'

Prediction or Postdiction?

The corollary of the optimal cue integration approach to IB is that the compression of time could be driven equally well by predictive or inferential processes. Lalazar and Vaadia [306] conclude that 'one of the fundamental problems in analyzing neural activity during learning is the inherent difficulty in breaking the closed sensory-motor loops. Since inverse and forward models are activated concurrently, they may each be represented simultaneously in neural activity within a behavioral trial. Therefore, it is often difficult to dissociate predictions of forward models from "predictions" which are none other than adapted inverse models. It remains a major challenge to devise experiments that can tease apart the neural activity underlying inverse models from those of forward models.'. Within the intentional binding paradigm, predictive and postdictive contributions have been investigated by varying the probability with which the voluntary action produces the sensory outcome (Moore and Haggard [370]). Moore and Haggard found that both processes operate, but that one dominates depending on the specific outcome probabilities: On high 'outcome probability' trials, healthy volunteers exhibited temporal binding even in the absence of the outcome, whereas subjective temporal compression was only observed on those low 'outcome probability' trials that did indeed produce the outcome.

The neural underpinnings of prediction and postdiction in sensory feedback loops vary depending on the exact task measures, but the sensory attenuation observed during the forcematching task in the previous chapter is thought to rely (in part) on the supplementary motor area (Haggard and Whitford [208]). Even in the absence of action, predictions about stimuli (such as their size or auditory features) can be tracked in the corresponding premotor areas (Schubotz and von Cramon [489]). Disrupting the anterior supplementary motor area (SMA) during execution of the IB task affects the subjective temporal shift of the outcome, but not the shift for the action itself (Moore et al. [371]) leading to the conclusion that only the binding of the action effect relies on predictive processes. Further evidence that prediction and postdiction are dissociable in IB are provided in Christensen et al.'s [83] emotional valence paradigm of intentional binding: Positive valences specifically increased

retrospective contributions to the binding effect when the valence and occurrence had low probability. High predictability of the valence of the outcome stimuli strengthened the predictive component, especially for neutral outcomes.

In line with the positive symptoms of aberrant SoA experiences in psychosis, patients with schizophrenia perform differently to controls on the basic IB task and exhibit an increase in the perceived temporal attraction between their action and the outcome (Haggard et al. [205]). When split into the prospective and retrospective components to IB, individuals with schizophrenia show an absence of predictive action binding, but an increase in the retrospective contribution to IB (Voss et al. [591]). In contrast, prodromal patients have a tendency towards stronger general binding facilitated by both an increase in predictive and postdictive processes (Hauser et al. [221]). Administration of ketamine, as a drug model of schizophrenia, boosts the IB effect in healthy volunteers (Moore et al. [374]) and specifically increases the predictive contribution to IB (Moore et al. [372]).

Autism and SoA

Agency monitoring (e.g. in the form of self-correcting errors before the outcome is visible) has been referred to as the 'cognitive version of efference copying' (Russell and Jarrold [475]) and differences in this kind of internal error correction are well-documented in schizophrenia. Evidence in autism is more sparse, but deficits in agency performance were theoretically derived based on links to self-awareness and mentalising: according to Searle [494], agency detection is the crucial link between self-awareness and ToM. Thus autism, historically conceptualised as a disorder with an 'impoverished sense of self' (Dawson and McKissick [120], Lyons and Fitzgerald [341]) whose core deficit might be a mentalising impairment, is a prime candidate to explore SoA. Pacherie [411] thought that basic impairments in SoA (possibly mediated by impaired mentalising) could also explain the executive function difficulties observed in autism. However on the surface of it, adults with autism are just as good as their matched controls at judging agency based on whether visual feedback matched their own hand movements or not (David et al. [115]). Zalla et al. [622] showed a decreased use of sensorimotor cues in making judgments of agency in adults with autism which was correlated with performance on a ToM task. They conclude that autistic individuals experience their internal signals as unreliable and might rely more on retrospective external cues (such as accuracy) to evaluate agency and some preliminary studies on interoceptive deficits in autism seem to support this claim (Noel et al. [399], Garfinkel et al. [183]). This stands in contrast to the present body of work which did not detect any differences in the use of efference copies as well as the previously mentioned finding that children with autism rely

more on proprioceptive feedback than their non-autistic peers (Haswell et al. [220]). Lafleur et al. [302] interprets Haswell et al. [220] as a consequence of *insufficient* prospective and retrospective visual indices for the development of implicit SoA and relates the poor use of external cues to the reported difficulties with imitation in autism and the theory of atypical connectivity in autism (Mostofsky and Ewen [379], Young et al. [619]). In a similar vein, Zalla and Sperduti [623] suggest that ASC is characterised by an isolated impairment of predictive (but not postdictive) processes in the genesis of SoA. A recent study has indeed found an attenuated intentional binding effect in adults with autism when tested with visual, auditory and audio-visual action outcomes (Sperduti et al. [522]). The study only employed the 'classic' IB paradigm and thus could not disentangle the respective contributions of predictive and inferential processes to IB.

This chapter therefore aims to answer the following questions:

1. Do individuals with ASC show a similar dynamic modulation of the influence of predictive and postdictive factors on IB effects as controls?

2. Do autistic or schizotypal traits play a mediating role in agency-based prediction and postdiction?

3.2 Methods

3.2.1 Participants

A total of 50 participants (25 per group) were recruited for the study. All but one of the ASC volunteers also took part in study 1 and thus the same two volunteers with a history of psychosis were excluded.

The study protocol was approved by the Psychology Ethics Committee of the University of Cambridge and all participants gave written informed consent. Cognitive function for all study volunteers was assessed using the timed version of the Ravens Advanced Progressive Matrices (RAPM) (Raven et al. [450]) and the Wechsler FSIQ in the case of one ASC volunteer. 11 of the participants with autism had co-morbid diagnoses of depression and/or anxiety and 6 were currently taking SSRIs. One participant had a history of OCD but was currently not taking medication. Reported comorbid neurodevelopmental disorders were ADHD (2 participants) and dyspraxia (1 participant).

Table 3.1 Participant Demographics for the Intentional Binding Task

Group	**Age** *(SD)*	**Sex** *(m:f)*	**IQ** *(SD)*	**AQ** *(SD, range)*
ASC *(N=23)*	29.0 *(6.1)*	11:12	105.2 *(12.7)*	35.6 *(7.5, 19-48)*
Controls *(N=25)*	31.2 *(5.7)*	10:15	104.6 *(10.6)*	17.1 *(7.0, 6-32)*

Participants were matched for age (t_{46}=1.309, p=0.197), IQ[2] (t_{44}=-0.163, p=0.871) and gender ($\chi^2_{(1)}$=0.298, p=0.771).

All but one of the ASC participants were assessed with module 4 of the Autism Diagnostic Observation Schedule (ADOS, [331]) and while the group was moderately symptomatic (mean score: 5.8), only six participants met cut-off criteria for an autism spectrum condition and none met diagnostic criteria for autism. All of the ASC participants in this study had received a diagnosis of an autism spectrum disorder by a qualified clinician and scored above cut-off on the AQ (except for one participant whose inclusion in the analysis did not affect the final interpretation of the results).

3.2.2 Experimental Procedure

The basic structure of the task was similar to other intentional binding experiments (Haggard et al. [204]): Participants were instructed to press a key with their right index finger at a time of their own choosing which caused a tone 250ms later. While they were engaged in this task, a Libet clock (Libet et al. [325]) was visible in the middle of the screen with a clock-hand rotating at a rate of 2560ms per revolution. After the keypress, the clock-hand continued to rotate for a random amount of time. Participants were told to avoid pressing at 'premeditated' clock positions.

In the 'action block' condition, participants had to recall the time at which they pressed the key (i.e. recall where the clock-hand was pointing to when they performed the keypress) while in 'tone blocks' participants were asked to enter the the clock-hand's position when they heard the tone.

In addition to 8 experimental blocks (4 per condition), the volunteers also completed a baseline task requiring them to judge the time of their key presses without any subsequent tone.

As in Moore's adapted version (Moore and Haggard [370]), the probability of the tone occurring was manipulated: In half of the blocks (2 per condition) the tone followed the key press 50% of the time while in the other half it happened 75% of the time (see Figure 3.1).

[2]IQ information was unavailable for two control participants.

When no tone occurred, participants were asked to report a dummy value. Participants were informed of the response requirement (time estimation of the key press or tone occurrence) immediately prior to the block which otherwise did not differ visually from each other. The order of blocks was randomised for each participant.

Blocks with the 50% probability for tone occurrence had 50 trials whereas blocks with tones occurring 75% of the time had 40 trials. Baseline blocks had 50 trials[3].

The data from one of the control participants was excluded from the analysis as it became clear in the debriefing that he had not been following the instructions.

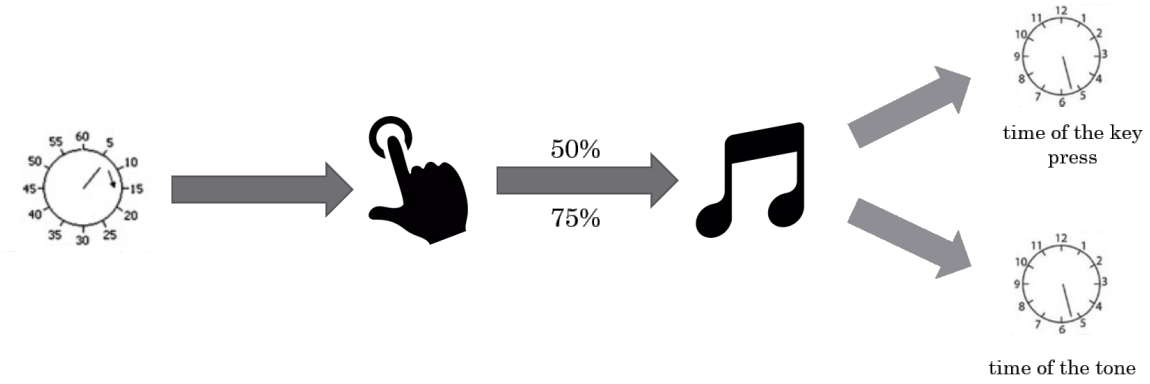


Figure 3.1 An illustration of the experimental procedure for IB with varying outcome contingencies

Data Analysis

Responses were corrected against the mean of all baseline trials for each participant. For the purposes of the analysis the first 10 trials of each block were not included as participants had to learn the contingencies. The reported shifts in the performed key presses were used as the measure of intentional binding rather than the perceived shifts in action outcomes (tones), as the latter were not present for each trial.

Based on Voss et al. [591], the predictive component to the intentional binding effect was calculated as the difference in overall shift between action only trials in the high probability blocks and action only trials in the low probability blocks ('action only' trials (75%) – 'action only' (50%)). Since the tone is observed in neither condition any difference in the strength of binding must be due to the higher predictive power of the 'action only' 75% probability blocks. Analogously the postdictive/inferential contribution was defined as

[3]Due to a technical error 2 control subjects had the trial numbers reversed and 3 controls and 7 ASC participants only completed 40 trials in the baseline task.

the average shift in 'tone only' trials in the 50% blocks. The authors describe the 50% contingency as subjectively 'random', so participants should not be able to form helpful predictions. Therefore any binding effect must be due to an inferential component that acts on the temporal estimation process after the tone occurs, see Figure 3.2.

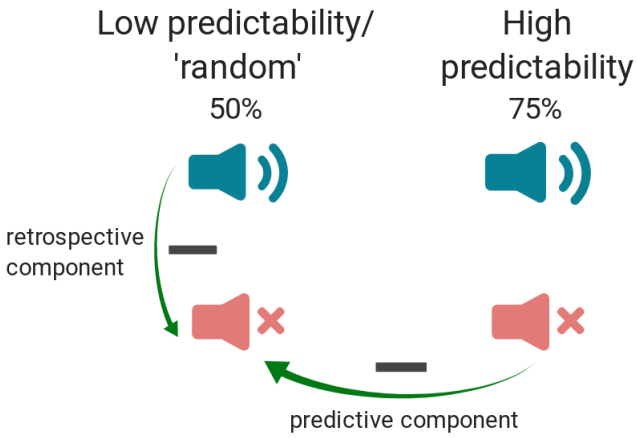


Figure 3.2 Illustration of calculations for the predictive and inferential components of IB

Visual inspection (histograms and Q-Q plots) of the data and Kolmogorov-Smirnov normality tests with Lilliefors correction suggested approximate normal distributions of the relevant measurements with the exception of the 50% probability 'action block' for the ASC group and the 75% probability 'tone only' blocks in the control group. Given that ANOVAs are robust in the face of small deviations from normality (Khan and Rayner [277], Schmider et al. [484]), it was chosen over non-parametric options. All further analyses and graphs were done in R.

3.3 Results

If prediction is indeed impaired in ASC, we would expect an interaction between group, condition and probability, reflecting an attenuated subjective compression of time in 'action only' trials in ASC participants relative to controls, and this difference in attenuation to be greatest on 75% trials.

Mean estimates (corrected against each participant's baseline) of tone judgements were calculated. A 4x2 ANOVA yielded a significant effect of condition ($F_{(3,135)}$=4.084, p=0.008), but not group ($F_{(1,45)}$=0.001, p=0.981) and no interaction between condition and group ($F_{(3,135)}$=0.630, p=0.597).

The resulting pattern resembled Moore and Haggard's [370] results where intentional binding was observed in all conditions apart from the low-probability no-tone trials (see Figure 3.3). Post-hoc analyses revealed that the low-probability no-tone block ('action50') was different from the other conditions, although the comparison with low-probability tone blocks was no longer significant at the adjusted α=0.017 (comparison of 'action 50' & 'tone 50': $t_{(46)}$=2.047, p=0.046; comparison of 'action 50' & 'action 75': $t_{(46)}$=-2.669, p=0.010; comparison of 'action 50' & 'tone 75': $t_{(46)}$=-2.750, p=0.008).

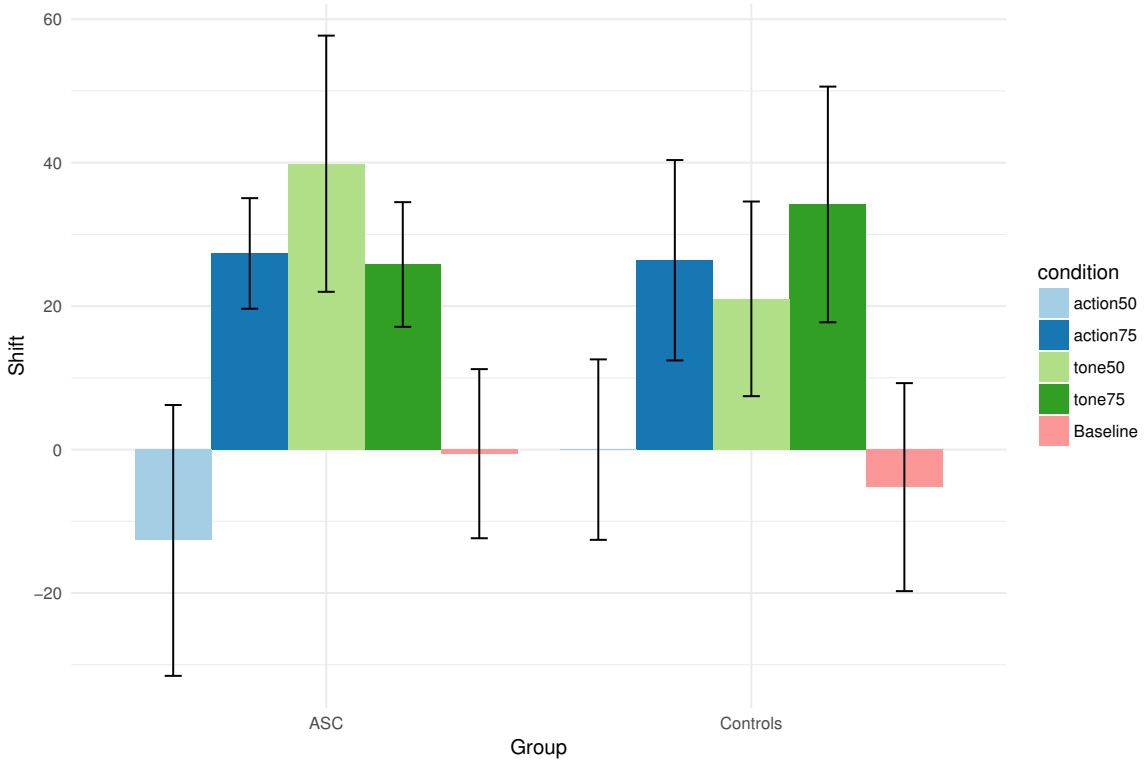


Figure 3.3 Baseline-corrected shift in the action estimates (ms) for each probability block in the 'action only' and 'tone only' conditions. Error bars represent ±1 standard error (SE) of the mean.

Predictive and inferential contributions to IB were highly correlated in both groups (ASC: r=0.974, p<0.001, Controls: r=0.973, p<0.001). There was a positive relationship between the 'Detail' subscale of the AQ and the magnitude of the retrospective and predictive components in the controls, but not the ASC group (r=0.559 p=0.005, r=0.433, p=0.34).

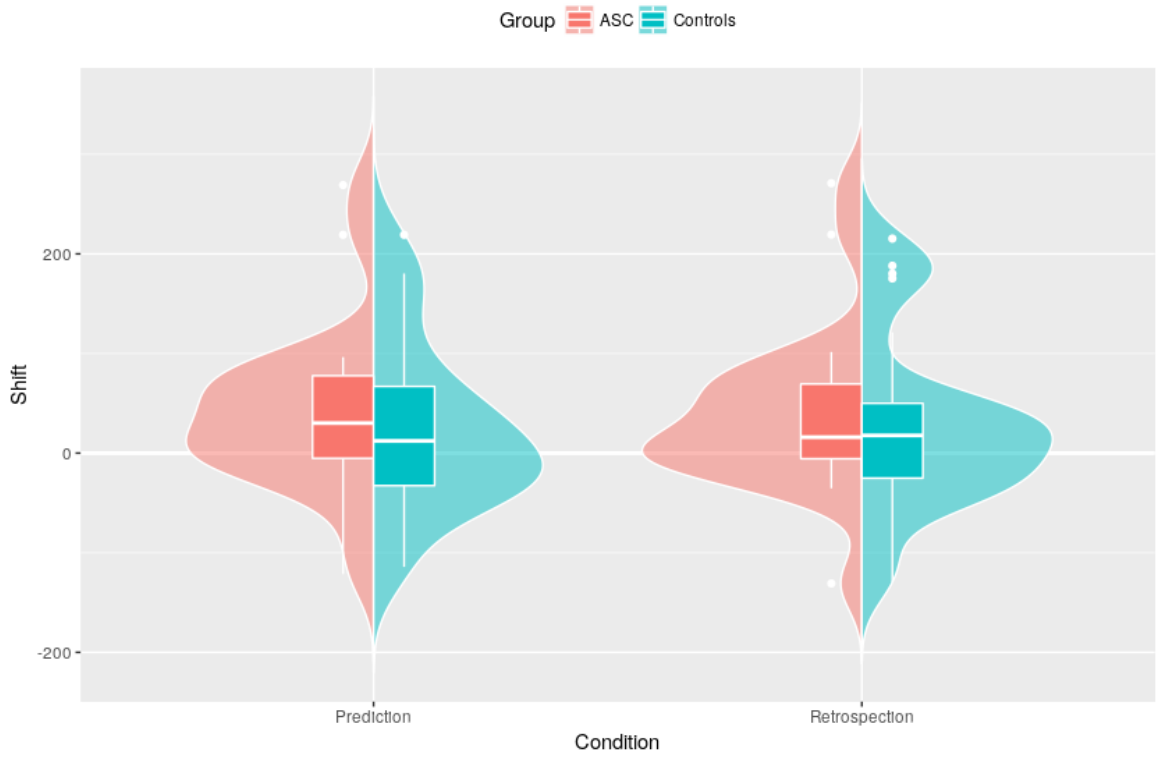


Figure 3.4 Violin Plots for the Retrospective and Predictive Components to IB

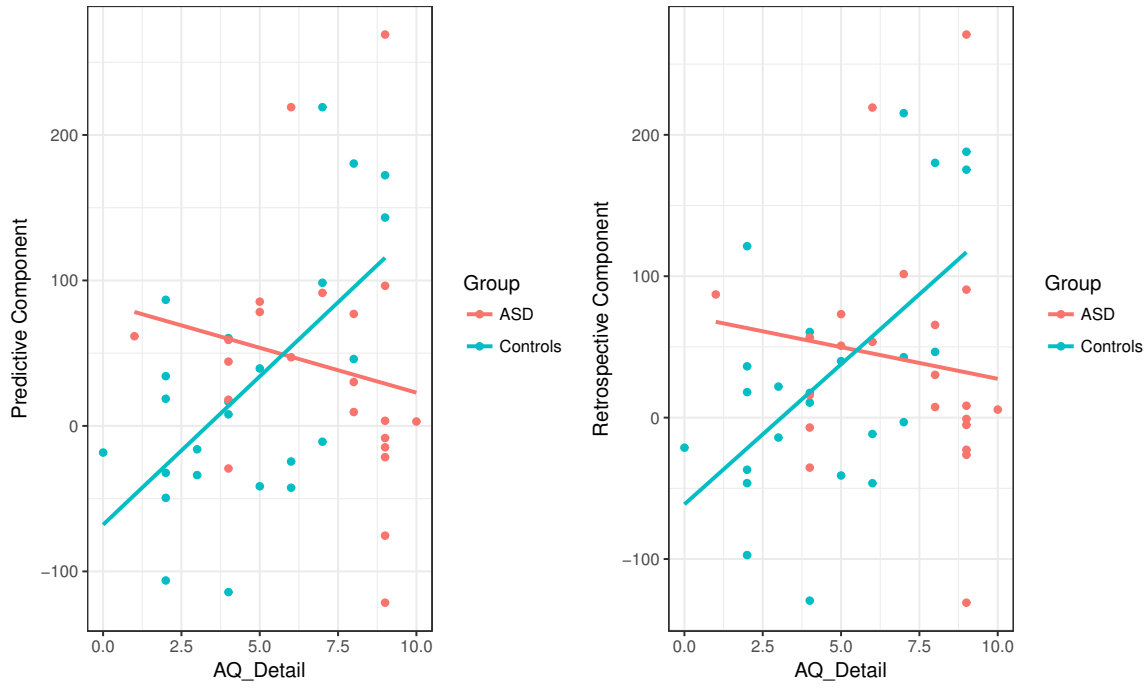


Figure 3.5 Correlations between IB and AQ subscales

3.3.1 Alternative Analysis of the Retrospective Component

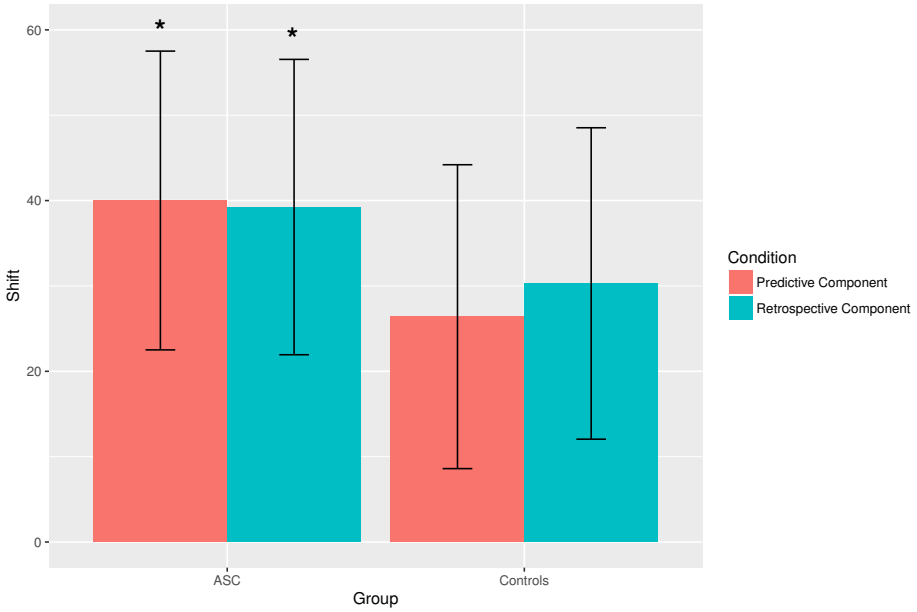
Another way to conceptualise postdiction in this IB task was to subtract the shift in the low probability action trials from the low probability tone trials. In this analysis, while the overall mean for the retrospective component increased in the ASC group, it was not significantly different from 0 (Figure 3.6).

3.4 Discussion

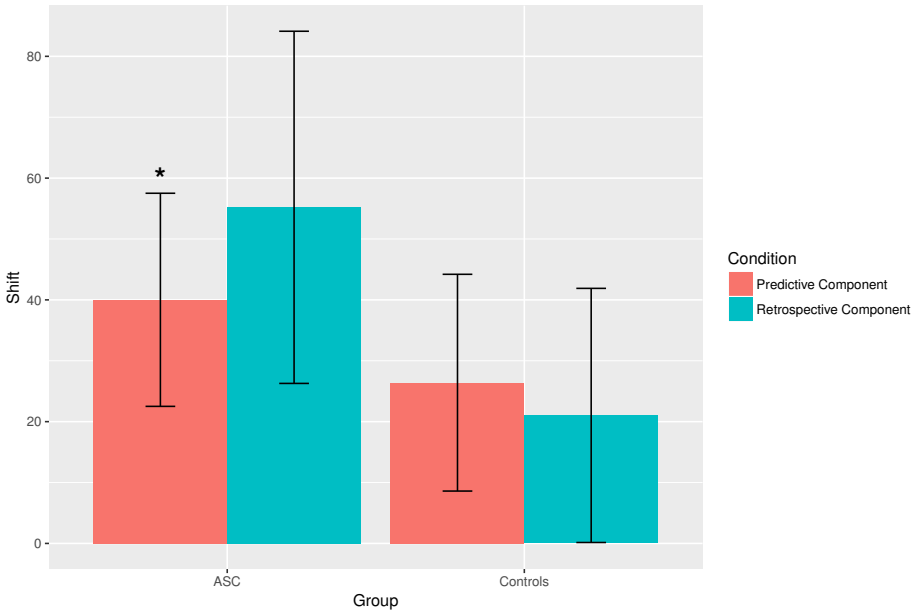
While both groups seemed to exhibit the basic pattern of inferential and predictive binding reported by Moore and Haggard [370], no group differences were found on any of the measures. If anything, the autistic group exhibited superior predictive and retrospective processing. Compared to the same task in the study by Voss et al. [591], both experimental groups seemed to have more consistent binding effects; Voss et al.'s control group seemed to almost exhibit no basic IB effect: Appendix A.1 shows comparative plots of the results by Voss et al. and the current study.

One caveat of this study is that the neural mechanism of intentional binding is still not well understood and that as Moore et al. [371] reported, only the action effect relies on predictive processes as mediated by the SMA. As the present experiment measured IB exclusively via the temporal shift of the action, rather than the action effect, the underlying mechanism of the 'predictive component' that was measured remains elusive. Certainly internal feedforward models are not the sole driving force behind the IB effect as initially assumed and thus any findings cannot be taken to necessarily reflect the strength or reliability of internal predictions (Synofzik and Voss [544]). The terminology used in the IB literature further obscures the theoretical foundations of predivtive and postdictive contributions to the emergence of SoA : While 'internal' and 'motoric' signals are often taken as equivalent (and distinct from 'external' reafferent sensory information), the efference copy approach which is often taken as an example for a predictive process, clearly makes use of both cues.

In light of the previously published contradicting finding of an IB deficit in autism (Sperduti et al. [522]), the difference between this and the study at hand need to be examined: The manipulation of the probability of the action effect occurring in the experiment that is presented here is unlikely to cause an enhancement in overall IB as it should introduce more uncertainty and more spurious binding effects. An obvious suggestion, given that Sperduti et al. employed three different delays between the action and action outcome, is that time estimation and temporal binding difficulties which are common in autism (Brock et al.



(a) Original analysis of the retrospective and predictive components of IB



(b) Adjusted analysis of the retrospective component

Figure 3.6 Error bars represent ±1 standard error (SE) of the mean. Asterisks indicate a significant difference from 0.

[67], Maister and Plaisted-Grant [348]), impeded performance for the ASC group. Sperduti et al. attempted to control for this by calculating and comparing proportion error scores for which actual interval durations were subtracted from the time estimations and dividing the difference by the actual interval duration. None of the main effects of group were significant for the proportion error scores comparisons, leading the authors to conclude that both groups exhibited similar timing accuracy. However as Maister and Plaisted-Grant [348] point out, impairments in estimating short time intervals between 0.5 and 2 seconds seem to be the result of deficits in attentional control in autistic individuals, rather than indicative of a more global temporal processing deficit. The main observation for such short intervals is an increase in intra-individual variability in the reproduction or estimation of the interval which might not be adequately captured by proportion error scores. Attentional control might be particularly challenged under conditions where the time elapsed between an action and a sensory outcome varies randomly from trial to trial. It is equally important to point out that the adequacy of proportion error scores has not been evaluated for paradigms such as IB where the time intervals are *expected* to be incorrectly estimated. Other differences between the two studies include the all-male participant panel in Sperduti et al.'s experiment, the different estimation methods (Libet clock vs. analogue scale) and the fact that each condition (interval and modality) was only presented 10 times with 180 trials in total by Sperduti et al. compared to $\sim$460 trials in the current study. If autistic individuals are indeed more variable in their responses due to attentional deficits, a higher number of trials would be needed to obtain the expected effect.

In conclusion the present study excludes the possibility of a general IB deficit in autism as results indicate intact (if not superior) predictive and postdictive mechanisms of temporal attraction between actions and action outcomes. To elucidate contradicting findings, further research could investigate effect sizes of particular contributions to IB in autistic and non-autistic individuals which tap into the various levels of agency processing that are thought to be involved. In contrast to the predictive processes explored with the forcematching task of the previous chapter, IB does not only rely on SoA cues that individuals acquire over the course of their lifetime, but also seems to be affected by what could be termed 'contextual' or 'situational' priors which only play out in isolated circumstances or are introduced as part of the experiment. The next chapter will employ an experimental paradigm which uses contextual priors to investigate prediction in the visual system.

Chapter 4

Perceptual Benefits of Prior Knowledge

4.1 Introduction

> *Peindre d'après nature, ce n'est pas copier l'objectif, c'est réaliser ses sensations.*[1]

PAUL CÉZANNE, PENSÉES SUR LA PEINTURE

In the same way that painting is an abstraction away from reality, (visual) perception is a result of hidden constructive processes that infer a three-dimensional world from a two-dimensional retinal image. Ambiguities are introduced by occlusion, lighting conditions, viewing angles, variable features between objects of the same category and other factors. And yet, our day-to-day visual experience of the world is surprisingly stable - such that the experience of visual illusions which exploit our highly-tuned probabilistic inference apparatus can be deeply unsettling. As outlined in the first chapter, we owe this disambiguation to the dynamic weighting of visual cues and prior constraints (Kersten et al. [275]). While the literature on sensory prediction is often perceived to be closely linked to ideas about efference copies, active inference and other motor components, some of the earliest empirical papers exploring predictive processes were published on visual perception (Rao and Ballard [448]). Prediction errors are present in and across other modalities (Lee and Noppeney [316], Stefanics et al. [528], Summerfield and Egner [538], Stekelenburg and Vroomen [530]) and visual prediction error signals may be detectable as early as V1 (Bannert and

[1] 'Painting from nature is not equivalent to copying the object; it is realising one's sensations.'

Bartels [27]) and occur as early as 80ms after stimulus onset (Aru et al. [14], Gamond et al. [181]).

One important aspect of perceptual (re)organisation as a result of prior information is figure-ground segregation: Which features form part of the figure and what part of the image is the background? On a lower level, grouping and edge assignment can provide clues, but under certain conditions rich and stable percepts can be formed with very little retinal information:

4.1.1 Two-tone Images

Two-tone, or 'Mooney' images are obtained by smoothing and thresholding greyscale images in such a way that automatic scene and image recognition are thwarted. They have now been widely used to investigate top-down influences on the processing of low-information stimuli (Dolan et al. [134], Hegdé et al. [224], Kemelmacher-Shlizerman et al. [269]). Initially developed to investigate the development of perceptual closure during face perception (Mooney [369]), the appeal of two-tone images to vision researchers rests on the fact that identical visuo-spatial features can give rise to very different percepts depending on the prior information observers have access to. Even after a single exposure to the greyscale template, the disambiguation effect can last a long time (Ludmer et al. [337]) and such rapid and robust perceptual reorganisation makes two-tone images the ideal candidates for inducing unique contextual priors. For an example of degraded two-tone images and their coloured originals, see Figure 4.1.

Several studies have demonstrated that unidentified two-tone images have similar neural representations, but once they are disambiguated the pattern of response in early retinotopic visual cortex is more similar to the response pattern of the greyscale template than the unrecognised two-tone image (Hsieh et al. [240], van Loon et al. [578], González-García et al. [191]). This also lends credibility to the argument that the prior knowledge conferred by two-tone images is at least partially distinct from basic priming effects: in functional imaging experiments, voxels involved in classic priming effects do not overlap with voxels that carry the crucial information about the content of the two-tone images (Gorlin et al. [192]).

In addition to an invocation of priming effects, another criticism of the Bayesian interpretation of two-tone image disambiguation are accounts that place attentional mechanisms at the heart of the apparent perceptual reorganisation of an impoverished stimulus: However the two theories need not be mutually exclusive (Hsieh et al. [240]), as orienting to particular

Figure 4.1 Example of two two-tone images depicting animals/people and their respective original photographs. These images were not part of the stimulus set used in this experiment but serve only as illustrations.

spatial locations could be one way in which top-down information influences bottom-up processing.

Significant developmental and culture-specific effects (Yoon et al. [617]), as well as general individual differences are found on two-tone image performance (with overall better performance in males, see Verhallen et al. [582] and Foreman [157]) and amygdala activation during the encoding of the greyscale templates can predict retention of disambiguation (Ludmer et al. [337]). According to Verhallen et al. [582], the disambiguation of two-tone images is 'all or none' whereas more recent research indicated that such perceptual insight might take place along a continuum of 'mental imagery resolution' (Davies et al. [118]). However this difference in resolution does not seem to be due to some local feature processing as the evidence that two-tone images are mostly being perceived holistically is quite strong even for non-face images (Steinberg Lowe et al. [529]).

Injection of ketamine[2] seems to disrupt top-down predictions to V1 and renders the pattern of response of disambiguated two-tone images less distinguishable, but does not

[2]Which, as pointed out in the introduction, is frequently used as a pharmacological model for schizophrenia (Frohlich and Van Horn [176]).

affect behavioural performance (van Loon et al. [578]). Generally speaking, the configural processing needed to disambiguate two-tone images seems to be poorer in individuals with high schizotypal traits (Batty et al. [38]) or psychosis (Rivolta et al. [463]), however when prior information is provided, these groups seem to derive more benefit from these top-down constraints than controls (Teufel et al. [553]). While these results might seem contradictory at first glance, they align with theories asserting that basic visual deficits in schizotypy and psychotic disorders lead to an increased reliance on prior information to form coherent mental pictures (Schmack et al. [483], Aleman et al. [6], Leitman et al. [319]). Some preliminary results furthermore suggest that deficits in perceptual closure tasks might be primarily related to what used to be known as the 'disorganised' subtype of schizophrenia (Uhlhaas et al. [565]).

4.1.2 Visual Processing in Autism

Most of the research on sensory processing in autism has been done in vision and various reviews have attempted to collate the emerging information: Bakroon and Lakshminarayanan [25], Griffiths and Milne [198] and Simmons et al. [509].

It has been claimed that whereas psychosis and psychosis-proneness are related to a stronger top-down bias on perception, individuals with high self-reported autistic traits are less likely to experience illusory perceptions due to modulated beliefs (Aru et al. [15]). In keeping with this, reports about a diminished influence of context on perception in autism have included reports of reduced shape constancy (Ropar and Mitchell [469]), diminished categorisation effects based on visual features (Soulières et al. [519]), a lack of susceptibility to certain visual illusions (Happé [214], Mitchell et al. [367])[3] and reduced change blindness (Loth et al. [332]). Atypical Gestalt processing as predicted by the WCC and its related theories has been found repeatedly in autistic individuals including reduced collinear facilitation and less benefit from closed contours in contour integration (Jachim et al. [251]). In one study by Bölte et al. [73], correlations between (generally poorer) performance on different tasks tapping into gestalt perception were found in the autistic groups, such that perceptual closure and similarity detection measures were linked to global processing of Navon stimuli, and the EFT and BD tests correlated negatively with susceptibility to five different visual illusions (Titchener, Ponzo, Muller-Lyer, Poggendorff, and Hering). Autistic individuals also exhibit atypical neurophysiological responses to contour integration tasks (Pei et al. [416]).

[3] But see Ropar and Mitchell [468] and Hoy et al. [239] for contradicting findings.

In the general population, autistic traits are related to weaker illusory percepts in one specific illusion paradigm, but this effect does not extend to other visual illusions (Aru et al. [15]).

However there is a comparable body of literature that has not been able to replicate findings of reduced gestalt processing and susceptibility to visual illusions in autism: Autistic children perceive illusory contours, such as might be evoked by the Kanizsa Triangle or two-tone images, in the same way than their peers (Milne and Scope [365]) and exhibit the neural activity alterations in V1 in response to Kanizsa illusions (Utzerath et al. [567]). According to Blake et al. [50], the grouping of lines into global figures is unimpaired in children with autism and Del Viva et al. [123] and Kemner et al. [270] report similar detection thresholds for a closed chain of Gabor patches in autistic and non-autistic children. Spanò et al. [521] finds intact figure-ground segregation in autistic individuals and comparable influences of low-level (convexity and surface integration) and high-level (memory) contributions to the former. The detection of symmetry might even be enhanced in autistic adults (Perreault et al. [423]) and recent doctoral work by Subri [535] concluded that the mixed results are best interpreted within the EPF framework which posits intact global and superior local processing.

Processing of Two-tone Images and Autism

As with the original task, the majority of autism research using two-tone images has used them as a way to investigate possible atypicalities in face perception rather than as a more general task assessing the influence of prior knowledge on visual disambiguation. When the task is employed in this manner, performance on two-tone images of faces (but not objects) seems to be impaired in autism (Loth et al. [333]), hinting at a more domain specific-deficit. Yet, this finding could not be replicated in a larger cohort and with a bigger stimulus set (Naumann et al. [392]). In the general population no correlations between the autistic traits (AQ) and a Mooney face detection task were detected by either Verhallen et al. [583] or Tulver et al. [562].

A recent experiment most closely resembling the paradigm in this chapter was performed by Van de Cruys et al. [574] who reported no differences on a pre-post two-tone disambiguation task (including non-face stimuli) in adolescents with autism and no correlation with AQ. Given the developmental advantage observed in the disambiguation of two-tone stimuli, the adults taking part in this study should perform at least equally well on the task.

I therefore wanted to answer the following questions:

1. Can the result of Van de Cruys et al. [574] be replicated in autistic adults?

2. Is there a difference in the effect of template exposure on disambiguation for social vs. non-social stimuli in autism?

4.2 Methods

4.2.1 Participants

Twenty-six volunteers with a clinical diagnosis of an autism spectrum disorder and 26 healthy control participants (with no history of neurological or psychiatric illness) took part in the study. The study protocol was approved by the Psychology Ethics Committee of the University of Cambridge and all participants gave written informed consent. Cognitive function for all study volunteers was assessed using the timed version of the RAPM (Raven et al. [450]) and the Wechsler FSIQ in the case of one ASC volunteer. One ASC participants was excluded from the subsequent analysis as she had a diagnosis of schizophrenia.

Ten of the participants with autism had co-morbid diagnoses of depression and/or anxiety and three were currently taking SSRIs and one was taking a typical antipsychotic drug with a further participant taking an antipsychotic drug and benzodiazepine on demand. Two participants disclosed an ADHD diagnosis, but neither was taking medication.

Participants were well-matched for age (t_{48}=0.344, p=0.733), IQ (t_{49}=-0.577, p=0.567) and gender ($\chi^2{}_{(1)}$=0.481, p=0.488), see Table 4.1.

All but two of the ASC participants were assessed with module 4 of the Autism Diagnostic Observation Schedule (ADOS, [331])[4] and while the group was moderately symptomatic (mean score: 7.5), only four participants met cut-off criteria for autism and a further six participants met the cut-off for an autism spectrum condition. All ASC participants with the exception of two scored above cut-off on the AQ and the two individuals who scored below scored above cut-off for autism on the ADOS and were therefore still included in the analysis. Four participants from the control group scored above the cut-off on the AQ, but excluding them from the main analysis did not affect the results and they were therefore retained in the analysis.

4.2.2 Stimuli

The stimuli have been used for two previously reported studies (Teufel et al. [551, 553]). Colour photos from the Corel Photo Library were used to generate both greyscale versions

[4]Only sum scores of the ADOS were available for a third participant.

Table 4.1 Participant Demographics

Group	**Age** *(SD)*	**Sex** *(m:f)*	**IQ** *(SD)*	**AQ** *(SD, range)*
ASC *(N=25)*	29.2 *(8.4)*	13:12	103.4 *(16.1)*	33.8 *(9.7, 9-48)*
Controls *(N=26)*	28.4 *(7.1)*	11:15	105.8 *(14.7)*	19.8 *(9.3, 4-36)*

of the images as well as two-tone images. The set of two-tone images used were carefully generated and selected in the following way: About 1000 initial greyscale images (500 images of people and 500 images of different animals)[5] were convolved with different Gaussian filters and thresholded at different cut-off values. For each image, I selected the two-tone image from the set of possible filter and threshold permutations that was deemed to be both 1) difficult to disambiguate without exposure to the original greyscale template as well as 2) amenable to effortless perceptual closure with knowledge of the templates. A second experimenter (Christoph Teufel) then reviewed the choices before all selected two-tone images were piloted in 27 naive observers for the two properties described above. The 30 images that were rated best on clarity after template exposure while still being difficult to decode without prior information were selected for this experiment. This final set included 13 images depicting people or faces and 17 images depicting animals.

4.2.3 Experimental Procedure

Participants were seated in front of a laptop at a viewing distance of 16” and instructed that they would see images composed of initially meaningless black and white blobs but that each image did in fact contain a person/people or an animal/animals. A red dot appeared somewhere on the image and participants were asked to indicate whether they thought the red dot was on the figure (person/animal) or the background. Responses were made by using the left (‘figure’) and right (‘background’) arrow keys. Each block contained three different two-tone images and each image was presented in four different conditions: 1) with the dot on a white patch and on the figure 2) with the dot on a white patch and on the background 3) with the dot on a black patch and on the figure and finally 4) with the dot on a black patch and on the background, leading to a total of 12 stimuli. The stimulus duration was 1.5 seconds (including ramping up and ramping down) and the red dot appeared 0.2 seconds after stimulus onset. Participants were then reminded of the arrow key mapping and if they didn’t respond within 3 seconds the message ‘Please respond.’ appeared on the screen. After

[5] Images depicting still lifes tend to have more uniform background surfaces and are thus on average easier to disambiguate.

selecting responses for the 12 presented stimuli, participants were told to move their hand to the mouse and to look carefully at the greyscale templates that were presented successively. After a delay of 3 seconds, the cursor appeared on a grey patch either left, right, below or above the template and participants had to move the cursor onto the image and click to proceed to the next template. Each greyscale template was shown three times. Next, the same 12 two-tone images were presented again in a randomised order and participants made the same choices about the location of the red dot. Participants were then allowed to take a break before proceeding to the next block. The experiment was comprised of eight blocks with a total of 24 different images that were chosen at random from the original set of 30. The location of the dot was chosen to be equidistant from centre of image for the two conditions ('on' and 'off' figure).

Before completing the task, each participant was shown an example of a two-tone image and the corresponding template until they were able to disambiguate the two-tone image based on the template. Everyone also completed a short practice session with just two images (shown twice) that mirrored the structure of the task.

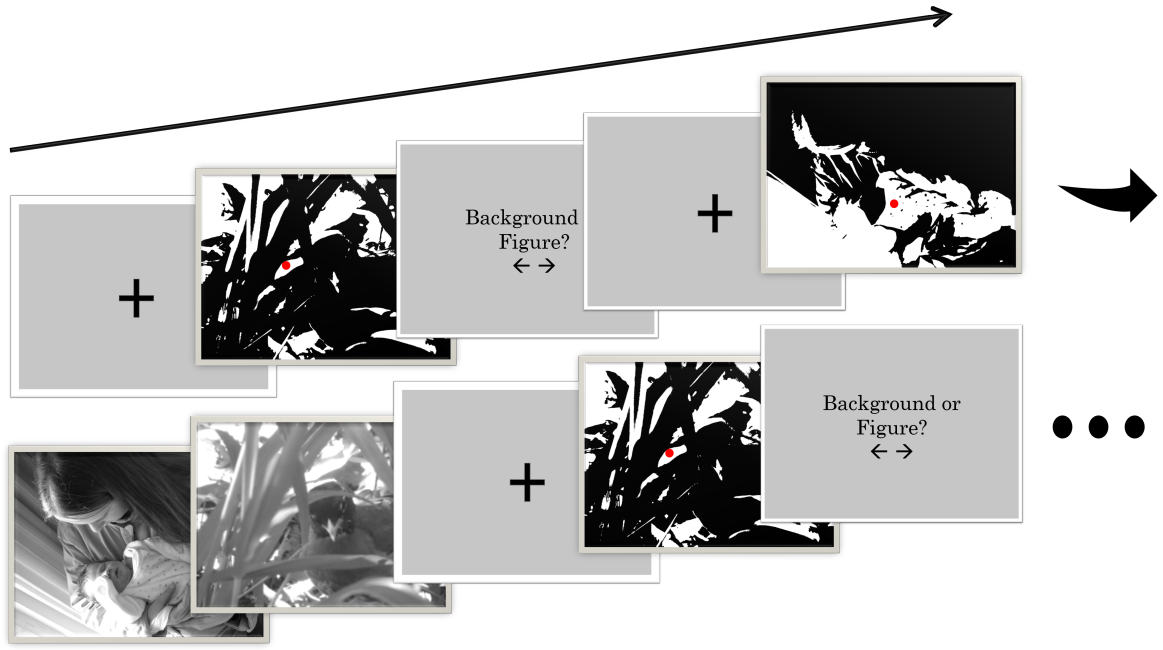


Figure 4.2 Illustration of the experimental procedure

4.2.4 Analysis

Analyses were conducted in R. The responses were analysed within a Signal Detection Theory framework, such that *hits* were correct identifications of the red dot being on the figure, *correct rejections* were correct responses about the dot being located on the background, *false alarms* were responses indication that the dot was on the figure when it was in fact on the background and *misses* were trials during which participants incorrectly responded that the dot was on the background. The R package *psycho* was used to estimate (perceptual) discriminability d' and response bias β for the 'Before' and 'After' blocks separately. The respective functions in R are:

```
dprime <- function(hit,fa)
{qnorm(hit)- qnorm(fa)}

beta <- function(hit,fa)
{zhr <- qnorm(hit)
zfar <- qnorm(fa)
exp(-zhr*zhr/2+zfar*zfar/2)}
```

Visual inspection (histograms and Q-Q plots) of the data and Kolmogorov-Smirnov normality tests with Lilliefors correction suggested approximate normal distributions of the relevant measurements.

Both d' and β were analysed as two independent samples t-tests with diagnostic status as the between-subject variable.

4.3 Results

4.3.1 Main Analysis

There was no difference between the groups on the difference between d' (t_{49}=1.363, p=0.179) or the β (t_{49}=-0.231, p=0.818) in the 'Before' and 'After' blocks.

Both groups showed the expected advantage after exposure to the greyscale templates (t_{24}=11.289, p<0.001 and t_{25}=10.755, p<0.001 for the ASC and control group respectively), see Figure 4.3.

Both groups also exhibited a tendency of a shift of the response bias towards reporting more dots on the figure after having seen the templates ($t_{(50)}$=1.873, p=0.067). There was no difference between groups ($t_{(49)}$=-0.231, p=0.818).

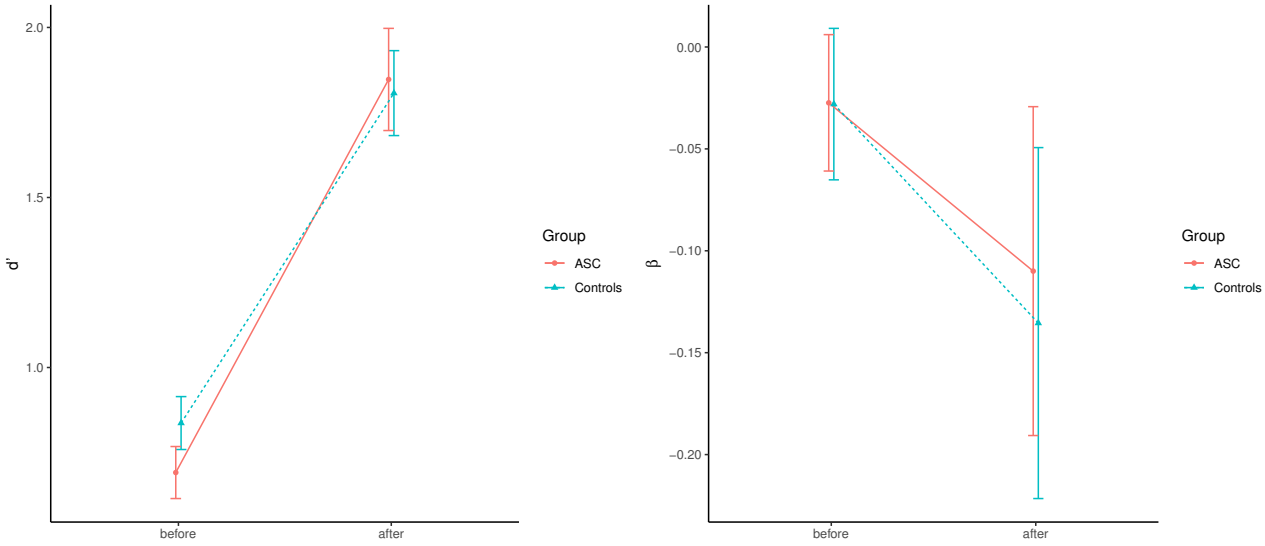


Figure 4.3 Sensitivity scores (d') and response bias (β) before and after exposure to the greyscale templates for both groups. Jitter was added to prevent overplotting. Error bars represent ±1 SEM.

Contrary to previous findings (Teufel et al. [553]), schizotypy was not related to task performance. Furthermore neither AQ (and any of its subscales), nor ADOS scores correlated with the improvement in disambiguation as a result of template exposure. The PDI score of the autistic group exhibited a positive relationship with the AQ 'Communication' subscale ($r = 0.626, p = 0.002$, uncorrected).

4.3.2 Further Analyses: Reaction Times and Social Stimuli

Reaction and Viewing Times

There was no group difference in reaction times for the 'Before' ($t_{(49)}$=-0.062, p=0.951) or 'After' block ($t_{(49)}$=-0.586, p=0.561).

However control participants used the free viewing time of the greyscale templates to examine them for longer than the ASC participants (Welch's t=-2.769, p=0.009), see Figure 4.4.

Images of People vs. Images of Animals

There is a wide literature on differences in the processing of social stimuli (especially face processing) in autism (Crawford et al. [106], Hadjikhani et al. [202]). Autistic children

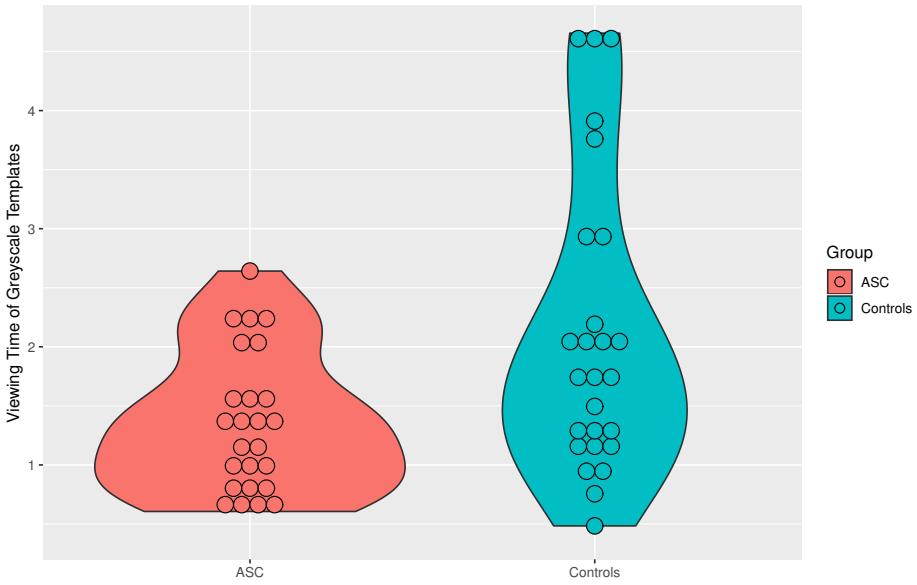


Figure 4.4 Length of viewing time for the greyscale templates. The mean (SD) is 2.116 (1.250) and 1.363 (0.589) for the control and ASC group respectively.

are reported to look less at people compared to objects (Swettenham et al. [542], Riby and Hancock [457, 458]) and have an increased rate of prosopagnosia (Weigelt et al. [597], Shah et al. [502], Tanaka and Sung [547]).

Specific difficulties in disambiguating two-tone images of faces have been reported in autism (Loth et al. [333]) and thus it was important to make sure that the social content of our chosen two-tone images did not affect performance in the ASC group. When analysing only the trials which contained an image of a face or person, there was no difference for d' across the two blocks ($t_{(49)}$=-0.502, p=0.618), nor for β ($t_{(49)}$=1.044, p=0.302), see Figure 4.5.

4.3.3 Exploratory Analysis: Relationship Between the Three Tasks

The experiments in this dissertation were not planned as a within-participant multi-paradigm approach to predictive processing, but due to recruitment limitations with the clinical group a few of the participants took part in two or even all three experiments. Therefore it was possible to look at correlations across tasks in order to see if there is a common factor underpinning performance on 'predictive processing' tasks.

None of the tasks correlated with each other, not in the group as a whole nor within groups:

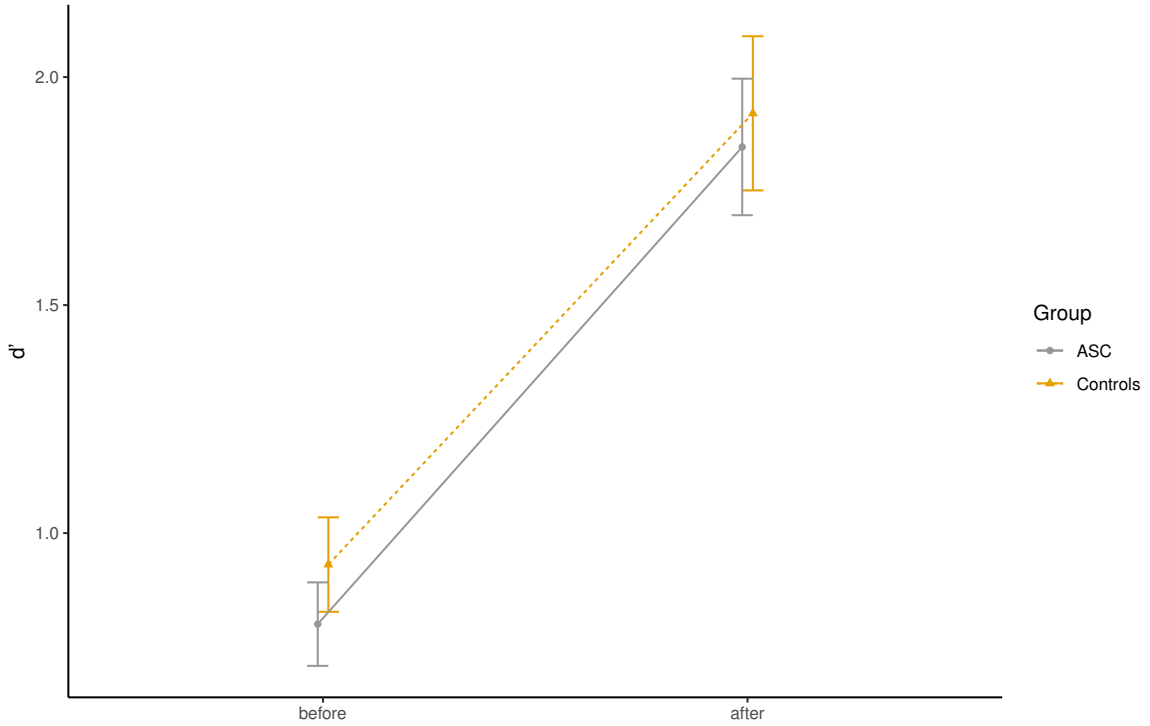


Figure 4.5 Sensitivity scores (d') before and after exposure to the greyscale templates for images displaying people only. Jitter was added to prevent overplotting. Error bars represent ±1 SEM.

1. Comparing the Force Matching and Two-tone Task (7 controls and 14 ASC):
   Using the intercept of the regression line in the 'internal' condition and d' from the two-tone disambiguation task there was a correlation of $r = -0.286, p = 0.209$ for both groups combined. In the ASC group only the correlation was $r = -0.261, p = 0.367$ and for the controls $r = 0.003, p = 0.994$.

2. Comparing the Force Matching and the IB task (21 controls, 20 ASC):
   The intercept from the force matching task and the 'prediction' measure from the IB task yielded a correlation of $r = 0.051, p- = 0.750$ across groups and $r = -0.059, p = 0.806$ for the ASC and $r = 0.268, p = 0.240$ in the control group.

3. Comparing the IB and Two-tone Task (9 controls and 14 ASC):
   The 'prediction' measure of the IB task and d' from the two-tone task had a Pearson correlation of $r = -0.204, p = 0.350$ in both groups combined. In the ASC group the correlation was $r = -0.197, p = 0.500$ and it was $r = -0.163, p = 0.675$ in the controls.

4.4 Discussion

This is the first study looking at the impact of template exposure on two-tone image disambiguation in adults with ASC. Behaviourally, both groups benefitted from the information provided by the templates to a similar extent. Both groups also slightly lowered their response bias (towards reporting more dots on the figure than off) as a result of seeing the templates which is puzzling given that the instructions were not to report the presence or absence of a signal but rather to choose between two possible locations of the stimulus. Possibly it feels more natural to think of a target being located on the figure rather than the background such that being more confident in identifying the figure increases the likelihood of reporting this expected outcome.

One confounding factor in the results is that the control participants used the self-timed exposure to the greyscale templates more extensively than the ASC group which is something that should be controlled in future experiments. Recent research has demonstrated however that at least in the area of contour integration, autistic individuals do not seem to benefit from longer stimulus durations (Subri [535]). Furthermore, if the viewing time had any bearing on performance in the control group, it would strengthen the argument that performance in the autism group was unimpaired.

Despite this, several individuals with ASC commented in the debriefing on their subjectively experienced difficulty with completing the task. Discrepancies between subjective and objective performance are not uncommon in cognitive neuroscience (Lau and Passingham [311], Melloni et al. [363]) and thus it might be interesting to investigate empirically if the subjective experience of perceptual inference differs in subjects with autism. The neural circuitry supporting subjective conscious experience of top-down processing also seems to differ from the pathways supporting objective task performance (Melloni et al. [363]) which might explain why no global or pervasive differences in sensory processing have been found despite repeated qualitative descriptions of sensory perturbations.

The positive relationship between the AQ 'Imagination' subscale and disambiguation performance (both before template exposure and as a difference score) that Van de Cruys et al. [574] reported could not be replicated in this study. The PDI also didn't show any association with any of the task measures, but was related positively to the AQ 'Communication' score: This might hint at the fact that autistic individuals might struggle to understand the pragmatic content of some of the questions on the PDI. This hypothesis will be further explored in the next chapter.

The results from the exploratory analysis of possible associations between the Forcematching, IB and two-tone tasks also confirmed another recent finding by Tulver et al. [562] who found no correlation between performance on several experimental paradigms[6] which supposedly assess 'predictive processing'. This calls into question any theories asserting that the inference of distal causes of sensory input is underpinned by a discrete, unitary 'prediction' system with fixed (hypo-) parameters that percolate down to the sub-units responsible for the processing of a given stimulus.

[6]Namely: The use of priors in Mooney face recognition, illusory contours, blur detection and representational momentum.

Chapter 5

Psychometric Measurements

5.1 Introduction

> *Es sind Züge verarbeitet, die wir von allen den autistischen Kindern her zusammengetragen haben. Nicht jedes von ihnen hat jeden Zug – derartiges kann man ja von keiner typologischen Betrachtungsweise verlangen. Aber für den, der solche Kinder kennt, ist es immer wieder erstaunlich, in wie vielen, in wie scheinbar anfälligen Einzelheiten sie übereinstimmen, wie einheitlich also der Typus ist. Dabei sind aber doch wieder die individuellen Unterschiede innerhalb des Typus groß. Wir würden selber unsere Betrachtungsweise für falsch halten, wenn uns solche Unterschiede verschwänden, wenn uns die unwiederholbare Persönlichkeit hinter dem Typus zurückträte.*[1]
>
> HANS ASPERGER, DIE AUTISTISCHEN PSYCHOPATHEN IM KINDESALTER

Heterogeneity of behavioural presentation has been a challenge for the investigation of autism from when it was first described, and the changes in and widening of diagnostic criteria have only accentuated this issue to the extent that it is now often seen as a hallmark

[1] 'The information we draw on comes from all our cases, but, as expected with any typological approach, not every case has every feature. Nevertheless, those who know such children never cease to be surprised at the striking coincidences of detail. The autistic personality is highly distinctive despite wide individual differences. Our method would have failed if it ignored the differences and if it let each child's unique personality vanish behind the type.' Translation taken from Frith [174].

of the condition (Masi et al. [355], Abrahams and Geschwind [1], Amaral et al. [8], Chang et al. [78]). Furthermore autism itself is sometimes believed by some to be the extreme end of a continuum that extends into the typically developing population, rather than a category in its own right (Baron-Cohen et al. [32], Posserud et al. [438], Lai et al. [303], Skuse et al. [517], Geschwind [185], James et al. [252]) - mirroring other thought processes in the field of dimensional psychiatry (Widiger and Samuel [603]). It is for these reasons that measuring group differences (even when the groups are matched on common factors such as chronological age and cognitive functioning) has come under criticism: Mean measures of performance on various tasks might obscure individual differences within the group which in turn might hold clues about causal mechanisms of the cognitive system in question (Thomas et al. [557]). However since group comparisons are more convenient both in terms of recruitment and analysis (Connections [90]), they remain the predominant approach to experimental design in autism research and are justified by assuming that 'it is the average (and not the variation around the average) that matters; that it captures some essential property of autism.' (Brock [66]). To supplement this approach, researchers resort to questionnaires as quasi-dimensional or subtype-clustering approaches. Yet all too often questionnaire scores are derived and correlated with task outcomes without any thought being given to their psychometric properties and whether it is appropriate to use them in the context of wanting to quantify and qualify intermediate endophenotypes and relate them to behavioural performance.

As the experimental paradigms employed for this thesis are based on similar investigations in the psychosis literature (Shergill et al. [505], Voss et al. [591], Teufel et al. [553]), I am using two widely used questionnaire measures throughout to appraise autistic and schizotypal traits. Although both questionnaires (the AQ and the PDI) have enjoyed popularity, more detailed studies on how these measures capture proposed latent traits in clinical and non-clinical populations are still lacking. This chapter therefore aims to answer the following questions:

1. What are the basic psychometric properties and factorial structures of the PDI and AQ in autistic and non-autistic populations?

2. Do the AQ and PDI measure the purported latent traits equivalently in individuals with and without an autism spectrum condition?

5.1.1 Short Detour: Schizotypy

Schizotypy has been explored as both a risk factor for developing psychosis as well as a personality trait with schizophreniform disorders marking the extreme end of the continuum. Multiple research groups are now arguing for a fully dimensional interpretation of schizotypy and psychosis (Nelson et al. [394], Johns and van Os [254]), although psychotic-like experiences, prodromal phases of the disorder and trait schizoptypy are not always clearly delineated (Mason and Claridge [357], Barrantes-Vidal et al. [34]). Furthermore the continuous distribution of schizotypal traits in the general population does not rule out the existence of more than one continuum nor the possibility of a qualitative difference between psychosis and normal experiences as some have argued (David [114], Lawrie et al. [312]). However there is evidence for a specific continuity from psychotic symptoms in childhood to a diagnosis of schizophreniform disorders in adulthood (Poulton et al. [439], Wolff et al. [608]. In the same way that the relationship between psychotic disorders and autism is still under debate (as outlined in Chapter 1), the question about the dimensionality and predictive value of schizotypy in non-psychotic individuals has not been settled yet.

5.1.2 Measures: AQ and PDI

AQ

The Autism Spectrum Quotient (AQ) is a 50-item self-report measure which was initially designed to measure the level of autistic traits in cognitively able adults (Baron-Cohen et al. [32]) and has since also been validated as a possible screening tool in a clinical context (Woodbury-Smith et al. [612]).

More recent studies have questioned its usefulness and specificity as a screening tool for referrals (Ashwood et al. [17]) with concerns over the AQ picking up other psychopathologies such as anxiety (Ashwood et al. [17], Reed et al. [452]), disorders on the psychotic spectrum (Lugnegård et al. [338], Naito et al. [389]) and other mental health conditions (Sizoo et al. [515]). For individuals who are already referred for an autism assessment the AQ provides no further discriminatory power (Conner et al. [91]).

In the research setting, the AQ is frequently employed to control for autistic traits in the control group or alternatively a group of individuals scoring high on the AQ is used as a proxy for an ASC group. The AQ seems to capture heterogeneous traits in non-clinical populations (Kitazoe et al. [283]), calling into question whether it is picking up symptoms pathognomic for autism - a skepticism which seems to be justifiable given a lack of congruence in perceptual mechanisms between healthy high-AQ and ASC individuals

(Gregory and Plaisted-Grant [197]). In a small pilot study with 148 autistic participants and 166 controls, Murray et al. [384] demonstrated configural and metric, but not scalar invariance of the AQ. The authors conclude that this might be attributable to different reporting between diagnosed and undiagnosed autistic individuals. Alternatively a lack of insight and deficits in self-perception could cause under-reporting in the autistic group compared to the controls.

A recent meta-analysis of the AQ (Ruzich et al. [479]) reported an average score of 16.94 for control subjects and 35.19 for individuals with ASC. Furthermore the previously reported sex difference in the nonclinical population with males scoring higher than females (Baron-Cohen et al. [32]) was replicated. In clinical populations, the sex difference is occasionally reported in the opposite direction with females on the spectrum endorsing higher autistic traits (Lai et al. [304]), notwithstanding the fact that the AQ is though to be biased towards the male phenotype of autism (Murray et al. [382]).

Different factor structures have been reported for the AQ from the originally proposed 5-factor model ('social skill', 'attention switching', 'attention to detail', 'communication', 'imagination', Baron-Cohen et al. [32]; 'social skills', 'communication/mindreading', 'restricted/repetitive behaviour', 'imagination', 'attention to detail', Kloosterman et al. [285]), to 4 factors ('socialness', 'pattern', 'understanding others/communication', 'imagination', Stewart and Austin [533]), 3 factors ('social skill', 'details/pattern', 'communication/mindreading', Austin [21] and Hurst et al. [246]) and 2 factor models ('social interaction', 'attention to detail', Hoekstra et al. [232] and Valla et al. [568]). Model fit (where reported) for the factor analyses of the AQ can be found in Table 5.1. None of the studies investigating the AQs factor structure had a big enough sample to run a factor analysis on a patient group. The 5 conceptually-derived subscales proposed in the original publication also do not seem to hold up to internal consistency and test–retest reliability standards (Stevenson and Hart [532], Freeth et al. [161], Ingersoll et al. [248]) and are much more variable for subscale scores.

Table 5.1 Model Fit for Reported Factor Structures of the AQ

Model (Number of Factors)	χ^2	df	p	RMSEA[1]	CFI[2]	TLI/CAIC[3]	SRMR[4]
Hurst et al., 2007 (2)	4947.967	1176	<0.001	0.080	0.500	0.479/—	0.167
Hoekstra et al., 2008 (2)	—	—	—	0.05	0.96	—	—
Austin et al. 2009 (4)	—	—	—	0.055	—	—/-5492.05	0.072
Kloosterman, 2011 (5)	542.03	340	<0.01	0.052	0.827	—/-137.97	0.074
Baron-Cohen, 2001 (5)	4673.203	1170	<0.001	0.078	0.535	0.514/—	0.313

For the purposes of this study the 4-point Likert scale of the AQ was retained in favour of dichotomising items for most of the following analyses as it has been found to have superior psychometric properties (Murray et al. [383], Murray et al. [384], Stevenson and Hart [532]). Items 3, 8, 10, 11, 14, 15, 17, 24, 25, 27, 28, 29, 30, 31, 32, 34, 36, 37, 38, 40, 44, 47, 48, 49 and 50 are reverse-scored such that higher scores are indicative of a higher endorsement of autistic traits.

PDI

The PDI is a 40-item questionnaire designed to measure sub-clinical delusional thinking and schizotypy (Peters et al. [426], Peters et al. [427]). Item selection for the shorter, 21-item version (PDI-21) (Peters and Garety [425], Peters et al. [424]) was based on the assumption of delusional ideation(s) as a unidimensional construct (Peters et al. [424]) rather than on multifactorial solutions provided by Principal Component Analyses (PCA). All versions of the PDI have a binary response structure which incorporates 5-point Likert scale ratings of distress, preoccupation and conviction for every endorsed item. Although there is a relationship with age (with lower scores in older people), no gender differences have been found and the mean score for healthy controls on the dichotomous scale was 6.7. Mean scores for the distress, preoccupation and conviction subscales were 15.5, 15.4 and 20.4 respectively. The mean scores of a deluded comparison group were 11.9 (dichotomous scale), 36.7 (distress), 36.1 (preoccupation) and 44.5 (conviction).

Subsequent efforts found 7-factor PCA solutions (‘paranoid’, ‘experiences of influence’, ‘grandiosity’, ‘religiousness’, ‘magical thinking’, ‘referential’, ‘depressive’ in López-Ilundain et al. [343] and Verdoux et al. [581] with ‘suspiciousness’ and ‘persecutory ideas’, ‘thought disturbances and jealousy’, ‘grandiose ideas’, ‘religious ideation’, ‘paranormal beliefs’, ‘ideas of reference and guilt’, ‘apocalyptic ideations’). Using more robust parallel analysis (Hayton et al. [223]), a 3-factor solution was deemed most appropriate (McCarthy-Jones and Fernyhough [359]), but only a 2-item factor corresponding to religiosity had satisfactory internal reliability (Cronbach’s α >0.7). Taking the lack of a reliable factor structure into account, a unidimensional scoring system as originally proposed might indeed be most appropriate.

[1]Root Mean Square Error of Approximation
[2]Confirmatory Fit Index
[3]Tucker Lewis Index and Consistent Akaike’s Information Criterion
[4]Standardised Root Mean Squared Residual

5.1.3 Introduction to Psychometrics: Confirmatory Factor Analysis and Item Response Theory

Underlying the theoretical model of most psychometric theories is the idea that responses or indicators (for example responses to items of a questionnaire) can be related to the individual's position on an unobserved latent factor or factors (Borsboom et al. [59]).

In both exploratory and confirmatory factor analysis the following sources of information are entered into the model: the association between indicators and the factors (= factors loading matrix), the variance and co-variance between factors themselves (= factor correlation matrix) and unexplained information in the indicators (= unique factor matrix) (Xing and Hall [613]). Estimation of parameters is commonly based on maximum likelihood (ML) calculations (Xing and Hall [613]). Given the assumptions of multivariate normality and linearity that classic CFA has, categorical SEM is to be preferred for questionnaires with less than 5 ordinal categories (Rhemtulla et al. [456]). In categorical approaches, a polychoric correlation matrix is generated instead of using Pearson's correlations to assess the relationship among a set of observed indicators. The alternative to ML is mean- and variance-adjusted weighted least squares (WLSMV) as ML can become computationally laborious with multiple factors.

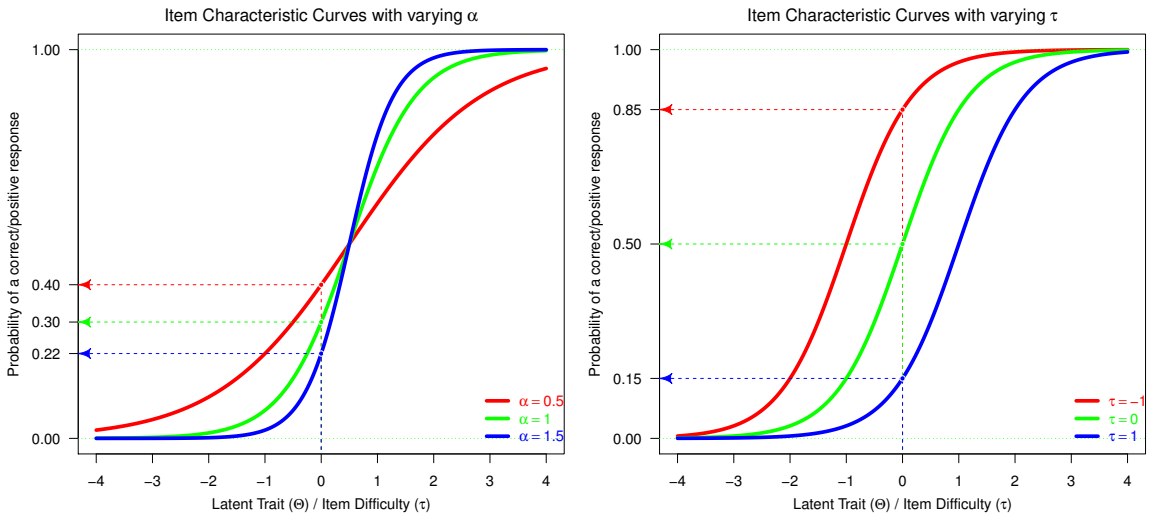
Item response theory (IRT) also uses generalised linear models to relate item responses to the unknown latent trait they are supposed to measure. It traditionally assumes unidimensionality, but more recent modifications have led to the development of multidimensional and polytomous IRT, including approaches that are equivalent to factor analyses (Kamata and Bauer [260]).

Within IRT, the item characteristic curve (ICC) is a sigmoid function that models a monotonic relationship between the response to an item and the latent trait of the respondent. In its general form, the model can be conceptualised as:

$$P_{ij}(\theta) = \frac{e^{\alpha_i(\theta_j+\tau_i)}}{1+e^{\alpha_i(\theta_j+\tau_i)}} \tag{5.1}$$

where $P_{ij}(\theta)$ is the probability that person j with latent trait level θ endorses item i. α is the discriminatory power of the item, or the slope at the inflection point. And τ is the threshold of the item or difficulty parameter. For an illustration of the different parameters see Figure 5.1.

Models are denoted as 1PL (one-parameter logistic), 2PL, 3PL and so forth based on the number of fixed parameters. In one parameter logistic models (1PL), only the item difficulty τ is freely varying, in 2PL models item difficulty τ and item discrimination α are free. 3PL



(a) ICCs with different α values

(b) ICCs with different τ levels

Figure 5.1 Examples of different item characteristic curves, based on code from Castro [75]

and 4PL models add guessing and carelessness parameters which serve as lower and upper bounds respectively.

The equivalent of α in classical FA would be the factor loadings and the intercept in categorical CFA is equivalent to τ. The equivalent CFA model therefore is:

$$x = \tau_x + \alpha_x \theta + \delta \tag{5.2}$$

with τ_x being the vector of intercepts for the observed variables x, α_x as the factor loading matrix, θ the latent variable(s) and δ the measurement error of x.

Both IRT and CFAs are used to assess measurement invariance, i.e. the extent to which different populations (such as a clinical and a control group) will respond similarly to items given the same value on the latent variable (Davidov et al. [116]). A lack of measurement invariance (i.e. non-equivalent measurement model parameters across groups) in turn might hint at the fact that certain items are interpreted differently by each group or differ in terms of their relevance for each group.

Measurement invariance is usually assessed in the following logically structured way:

1. Configural/Pattern variance: Is the factor structure the same across groups (no equality constraints)

2. Metric invariance: Are the factor loadings the same across groups? (α_x does not vary across groups/is constrained to be equal across groups)

3. Scalar invariance: Are the means the same across groups? (Both α_x and τ_x are invariant across groups, thus any differences in means on the item responses are attributable to differences in means on the latent variables.)

4. Strict invariance: Are the loadings, intercepts/thresholds and residuals the same across groups?

Within the IRT framework, measurement invariance is called Differential Item Functioning (DIF) and describes a difference in the probability of an endorsing an item by individuals with the same latent trait level θ.

5.1.4 Dimensional Structure of the Questionnaires: Exploratory Graph Analysis (EGA)

Exploratory Graph Analysis (EGA) is a recent approach within the wider field of network psychometrics (Marsman et al. [354]) which models the relationship between (questionnaire) items as an undirected network: Each item is a vertex (or node) and the pairwise relationship between vertices is indicated by (undirected) edges. Undirected graphical models and their data generating structures are also called Markov Random Fields (MRF), because the vertices satisfy the local Markov property: Given the Markov blanket of a vertex (which consists of all of its neighbouring nodes), the vertex is independent of all other vertices outside it.

EGA, due to its data-driven and undirected nature, is especially useful in situations where the theoretical underpinnings are still vague and of limited use in specifying relations and constructs. This makes network analyses especially attractive to psychiatric research where symptom structures and interactions both across and within diagnostic categories are still poorly understood. Graph theory applications of mental disorders promise to identify important symptoms or symptom clusters (Fried et al. [163]) and have been applied to a variety of psycho(patho)logy: depression (Boschloo et al. [60]), psychosis (Isvoranu et al. [250]), PTSD (Armour et al. [13], Knefel et al. [286]) and eating disorders (Olatunji et al. [404]). Outside of psychiatry, personality traits are also assumed to surface as clusters in EGAs (Cramer et al. [103], Mõttus and Allerhand [387]). However as with many other exploratory techniques, inferences drawn based on EGA should only be seen as providing interesting starting points for future investigations.

5.2 Methods

The data analysis plan was uploaded prior to analysis to the Open Science Framework and can be accessed here (Finnemann [151]).

5.2.1 Participants

Cohorts

For the first cohort, 496 subjects were recruited between October 2012 and April 2013 via adverts to college mailing lists of the University of Cambridge as part of a bigger participant panel. The vast majority of respondents were students (79.44%). No diagnostic information was obtained other than screening for depression and dyslexia.

806 students completed a second recruitment push (cohort 2) which included a question about the pre-existence of an autism diagnosis.

For the comparison study of clinical and non-clinical populations, a total of 696 participants were recruited via social media (157), online survey platforms (76), university mailing lists (57) and through an existing departmental database for autism research (406). The survey data was checked for duplicates (by comparing email addresses from those who supplied them) and one duplicate was detected and excluded from analysis. Subjects with <10% completion rate were also excluded.

Further information on the demographics of the three cohorts and exclusions can be found in Table B.1 in Appendix 1. Table B.2 provides further information on disclosed psychiatric conditions for cohort 3.

171 of ASC participants indicated that they were diagnosed by a psychologist and 110 received their diagnosis from a psychiatrist. A further 12 were diagnosed by a paediatrician and 8 by a neurologist. For the remaining participants, information abut the diagnostician was missing or unspecific (e.g. ‘doctor’).

Basic Demographics

The ASC group was older than the control group with a mean age of 38.42. The control group’s mean age was 25.77 ($t_{410.3}$=-17.91, p<0.001). There were no sex difference between the two groups (χ^2=15.714, p> 0.999).

The mean AQ score for the ASC group was 36.27 (SD: 10.85, N=264) and the mean PDI score was 59.92 (SD: 40.49, N=287). The control group obtained an average AQ score of 19.86 (SD: 7.87, N=1302) and a total PDI score of 41.67 (SD: 31.39, N=1026). While

still scoring significantly lower than the ASC group, the control group had higher AQ scores than the healthy controls in the meta-analysis by Ruzich [479] (p<0.001) and lower PDI scores than the healthy controls in the original paper of the short version (Peters et al. [424]) (p<0.001). For an illustration of the distributions see Figure 5.2.

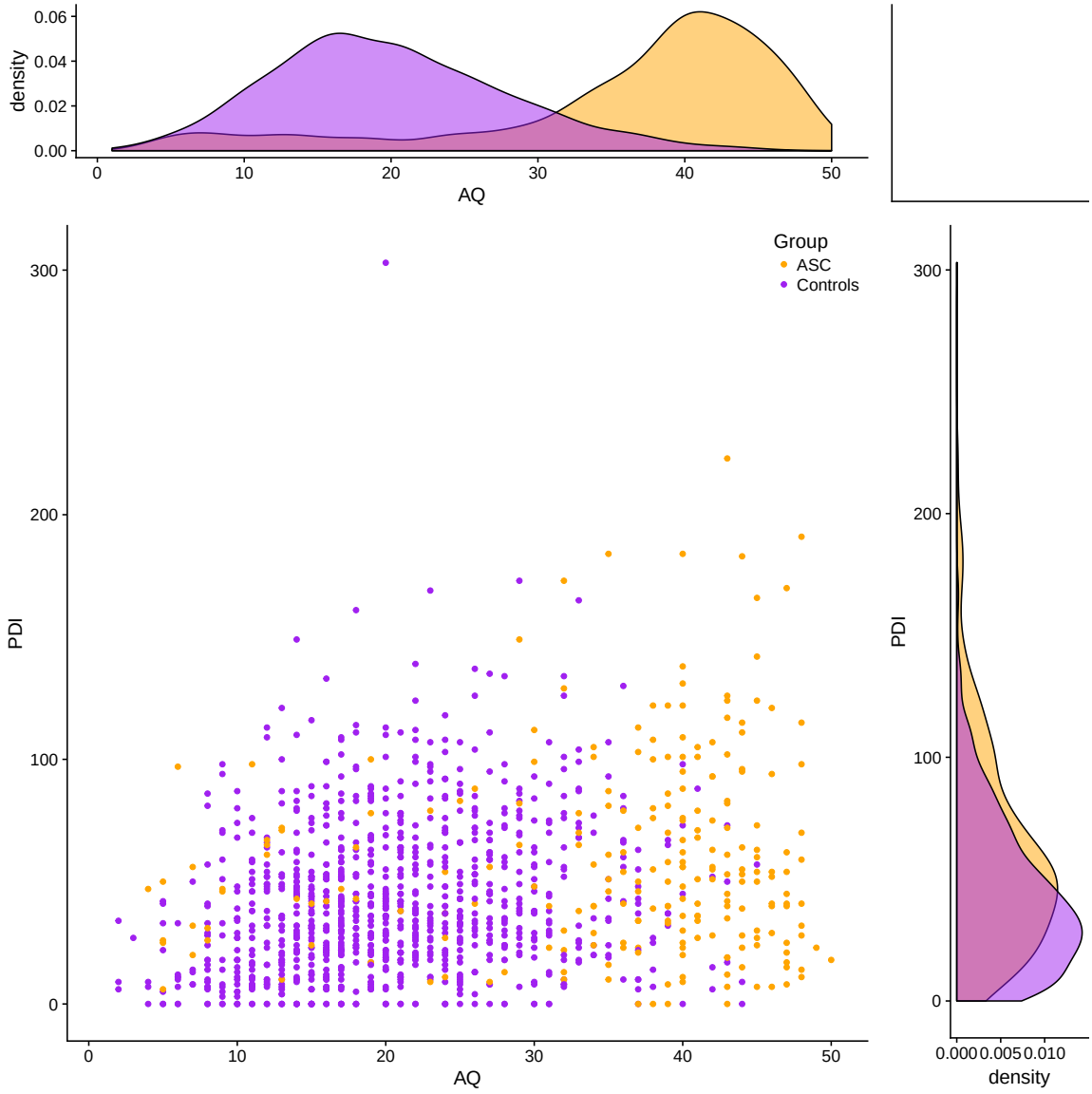


Figure 5.2 Densities of PDI and AQ

In line with previous research, typically developing males had higher scores than typically developing females on the AQ (t=2.960, p=0.003), but there were no differences in the ASC group. Neither group exhibited sex differences on the PDI, see Figure 5.3.

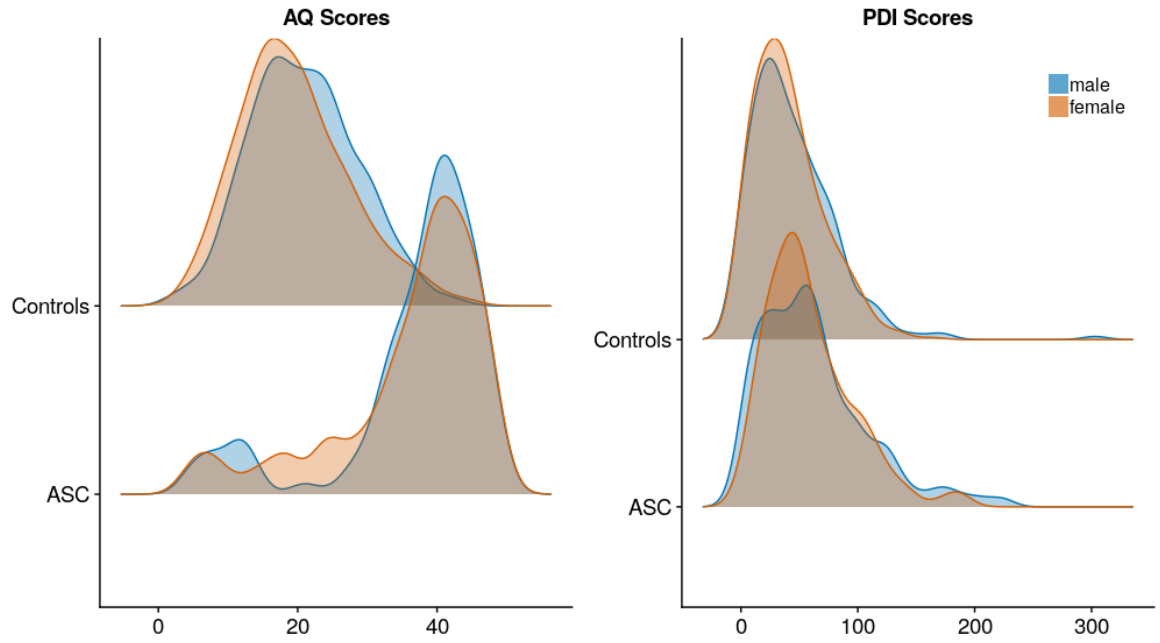


Figure 5.3 Gender Distributions for the AQ and PDI

5.2.2 EGA

EGA estimates the interaction between items (i.e. the correlation matrix of the variables) with a penalised maximum likelihood approach. For this dataset the basic partial correlation network was estimated using Pearson's pairwise correlations. As correlations between nodes are rarely exactly zero and therefore may be spurious or due to 'ambient noise' (Lykken [340]), the visualisation of an unweighted correlation matrix would yield uninterpretable results. Thus a sparsity penalty needs to be introduced to prevent overfitting and identify edges that are absent at the population level. Finding this sparse matrix however is not computationally trivial. Taking into account the high number of edges (and necessary corrections for multiple comparisons within the classic significance testing framework), most partial networks are estimated using the 'least absolute shrinkage and selection operator' (LASSO) approximation algorithm (Friedman et al. [164]). LASSO can simultaneously perform parameter estimation and model selection by penalising the maximum likelihood for the sum of parameter estimates by a predetermined parameter. The *EBICglasso* function of the R library *qgraph* (Epskamp et al. [143]) which was used for the analysis of the AQ, can run the analysis for 100 different penalisation parameters and return the network that minimises the Extended Bayesian Information Criterion (EBIC) (Chen and Chen [80], Foygel and Drton [159]).

Discrete MRFs, i.e. those generated by binary data such as the PDI, are known to be difficult to compute and it is only recently that robust estimation methods have been published: Using the Ising model in physics which describes the mutual influence of discrete atomic spins of neighbouring magnetic dipoles and LASSO estimation methods using EBIC, nodewise logistic regressions can yield stable regularised networks (van Borkulo et al. [569]). The R *IsingFit* package was used for all dichotomous network estimations.

The position of nodes in any EGA was determined by the Fruchterman-Reingold algorithm ([177]).

In the present dataset, EGA was used to explore the dimensionality of the AQ and the PDI in the ASC and control cohorts. This visualisation of conditional dependencies between items can provide a first indication of the underlying latent variables as observed clusters (or 'communities') of nodes. In certain cases these are statistically equivalent to the factor structure (Golino and Epskamp [189])[5].

Centrality plots, which were originally developed for the analysis of social networks, can offer information about important nodes. Three common measures to evaluate the centrality of nodes are their strength, closeness and betweenness. Strength is related to the most basic measure of centrality, called 'degree', which counts the number of edges a node is connected to, but sums the weight of the edges. Closeness is defined as the (average) length of the shortest path between the node and all other nodes. Finally, betweenness measures the number of times the node has to be passed along the shortest path between two other nodes. As all of these measures were primarily developed for network analysis of social connections or message passing systems (where edges are known rather than correlational), it is important to delineate a possible interpretation of centrality in the context of psychometric networks. In statistical terms, higher centrality of nodes indicates increased shared variance with other nodes. However, returning to the notion of questionnaires as measurement devices for unobserved latent factors, centrality of single items could quantify the degree to which items capture one or more of the latent traits. In the psychometrics literature this is sometimes likened to the 'importance' of the variable or item while others (Bringmann et al. [64]) also emphasise that psychological networks might not satisfy all of the assumptions generally underlying network analyses with known edges. Furthermore centrality measures are relative metrics, as each node is compared to the other nodes in a network regardless of overall connective strength.

[5]For example if clusters of items are unconnected/insular, the underlying latent factors are close to orthogonal.

Inferences based on measures such as centrality indices and edge strength are only as good as their associated accuracy and stability. Therefore it is crucial to assess the influence of sampling variation and number of observations on the network structure (Epskamp et al. [142]). Using the R package *bootnet* (Epskamp and Fried [144]) the differences between parameter estimates in the present dataset was assessed using bootstrap samples. A non-parametric boostrap was used as the edge weights are not expected to be normally distributed as a result of the glasso regularisation procedure. To estimate the stability of the centrality indices, a case-dropping bootstrap is most appropriate. The results from the latter can also inform more general appraisals of network similarity: The R library *NetworkComparisonTest* assesses network structure invariance, global strength invariance[6] (the overall level of connectivity in the networks) and edge strength invariance of two networks.

Since we cannot be certain about the unidimensionality of both questionnaires, several algorithms will be employed to identify potentially stable 'communities' of nodes across both groups. Four different approaches will be used based on options available in the R *igraph* package: The *walktrap* algorithm, *spinglass* algorithm, the *leading eigenvector* algorithm and the *fast greedy modularity optimisation* algorithm. The *walktrap* algorithm is based on random walks, i.e. a 'walk' which starts in a randomly selected node and continues on a randomly selected path (via edges). After several iterations, 'communities' are identified as clusters of nodes with short random walks between them. This method of identifying dense subgraphs within sparse networks was first suggested by Pons and Latapy [436] and is deemed appropriate for the sample sizes in this analysis (Yang et al. [615]). The *spinglass* algorithm has been adapted from stochastic mathematical models of magnetism: Modelling (via simulation) which particles prefer to be in the x of n possible spin states is equivalent to modelling which pairs of nodes belong to the same community. There is an upper (but not lower) bound on the number of possible spin states (or communities). Developed by Newman [397], the *leading eigenvector* algorithm computes the leading (=largest positive eigenvalue) non-negative eigenvector of the modularity matrix of the network. This optimisation is achieved by iteratively splitting the network(s) into two sections for as long as the split increases the modularity. Ultimately, communities are differentiated by their corresponding elements in the eigenvector. This method is particularly suitable for highly connected networks. The *fast greedy modularity optimisation* algorithm also aims to optimise the modularity function, but works in the opposite direction: At the start each node is assumed

[6]The global strength invariance test uses permutations to estimate the distributions of the absolute sum of all edges of the two networks under H0.

to belong to its own unique community and nodes are merged over time for as long as the overall modularity is increased by the largest possible amount. If no stable communities of nodes can be identified, the DIF analysis will be completed on all items of each questionnaire.

For this EGA the short 28-item version of the AQ was used (Hoekstra et al. [233]) since the rule of thumb is that one would like to have >500 participants per group for graphs with more than 30 nodes. Given the small number of autistic respondents, the network graph for the ASC group might be less stable than for the controls. Correspondingly, in case of the PDI, only the dichotomous scale was used.

For the analysis, complete data was available for 332 ASC participants and 1384 controls.

5.2.3 Item Response Theory: Differential Item Functioning (DIF)

Depending on the communities identified with the EGA, differential item functioning (DIF) should either be assessed separately for each dimension of a multidimensional questionnaire or for the questionnaire as a whole if it is unidimensional. Although one could employ a rating scale model for the polytomous AQ measures, both DIF analyses were undertaken with N parameter logistic models (using the $difNLR$ and $difR$ packages in R) such that comparable ICCs as outlined in the introduction can be drawn. The N will be determined based on best model fit.

5.3 Results

5.3.1 Dimensional Structure of the Questionnaires: EGA

AQ

Figure 5.4 illustrates the estimated network of the 28-item AQ with the factors reported by Hoekstra et al. [233]. The network graphs indicate that the least stable cluster for the ASC group seems to be the 'routine' subscale and the 'numbers' subscale for the controls. Item 4 ('I frequently get so absorbed in one thing that I lose sight of other things.') looks to be more closely linked to the 'numbers' cluster in the autism group than its 'switching' factor. Item 22 ('I find it hard to make new friends.') is also disconnected from its suggested 'social' subscale.

Overall the EGA for the control group has more negative weights.

Visual inspection yields a few common, strong connections in both networks: Questions 3 ('If I try to imagine something I find it very easy to create a picture in my mind.') and

8 (‘When I’m reading a story, I can easily imagine what the characters might look like.’) appear to have strong positive relationships. Given the wording of these items, they are potentially measuring the same thing. Items 32 (‘I find it easy to do more than one thing at once.’) and 37 (‘If there is an interruption, I can switch back to what I was doing very quickly.’) appear to be tapping into executive function ability and are strongly connected on both graphs. Sociability and extraversion are potentially assessed by items 44 (‘I enjoy social occasions.’) and 47 (‘I enjoy meeting new people.’) which also exhibit a strong positive edge weight in both groups.

In the ASC group only, items 6 (‘I usually notice car number plates or similar strings of information.’) and 23 (‘I notice patterns in things all the time.’) have a strong common edge.

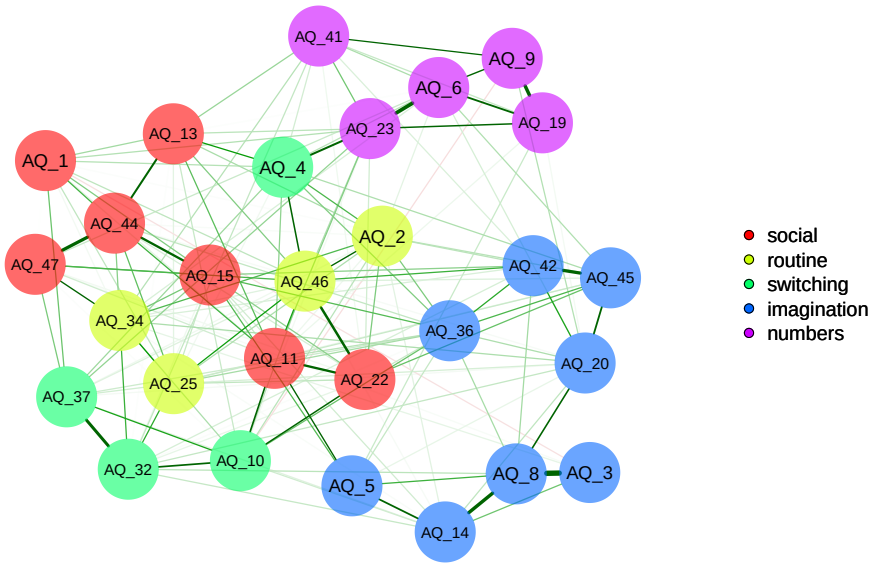
For the control group items 11 (‘I find social situations easy.’) and 46 (‘New situations make me anxious.’) are strongly connected across clusters, potentially capturing a social anxiety trait.

The centrality plots (Figure 5.5) indicate a few differences (but also similarities) across groups on a number of items. Item 11 on the AQ (‘I find social situations easy.’) seems to be a central node for both groups and one can speculate that it is closest to capturing the essence of what the AQ measures. Another contender might be node 46 (‘New situations make me anxious.’). Items with the apparently biggest discrepancy in centrality between the two groups include item 22 (‘I find it hard to make new friends.’) and item 23 (‘I notice patterns in things all the time.’). Notably both nodes belong to the subset of items which did not seem to cluster well in the control group and in line with this exhibit lower centrality indices than might be expected based on the results in the ASC group.

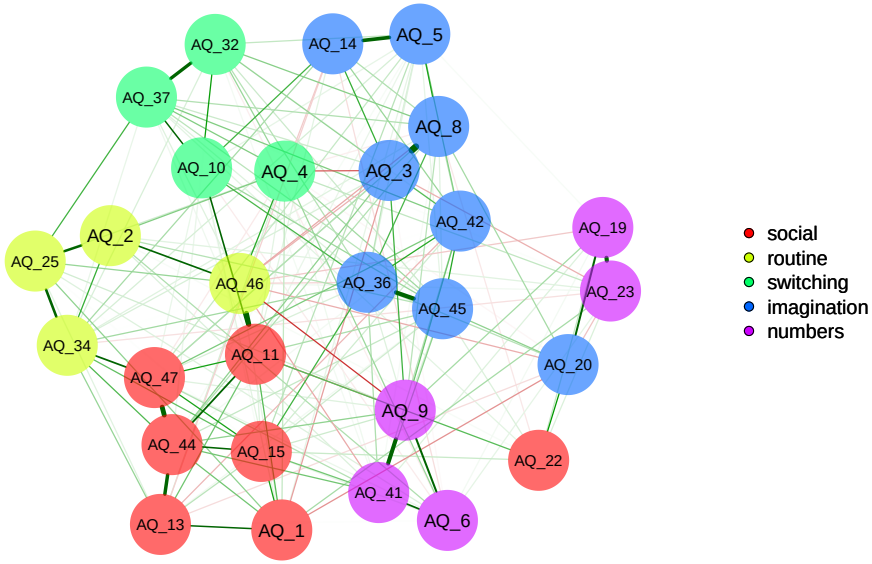
PDI

On visual inspection the basic connections between items look similar in both groups, however the ASC network notably exhibits more isolated and weakly connected nodes (see Figure 5.6). This could be either due to a lack of power or due to genuine orthogonal/unrelated factors. A few clusters do not seem to extend beyond two nodes which may hint towards weak factorial structure.

The emerging doublets in both groups include items 8 (‘Do you ever feel that you are especially close to God?’) and 11 (‘Do you ever feel as if you have been chosen by God in some way?’), items 4 (‘Do you ever feel as if you are being persecuted in some way?’) and 5 (‘Do you ever feel as if there is a conspiracy against you?’) and items 6 (‘Do you ever feel as if you are, or destined to be someone very important?’) and 7 (‘Do you ever feel that you are a very special or unusual person?’). These small content clusters could be



(a) ASC respondents



(b) Control respondents

Figure 5.4 EGAs for the AQ in the ASC and Control group. Green edges signify positive relationships, whereas red edges are negative. Connection strength is indicated by the thickness of the line. The displayed clusters are taken from Hoekstra et al. [233] and based on the original factors determined by Baron-Cohen et al. [32]. The minimum, maximum and cutoff scores are set at 0, 1 and 0.15 respectively to make edge thickness comparable between graphs.

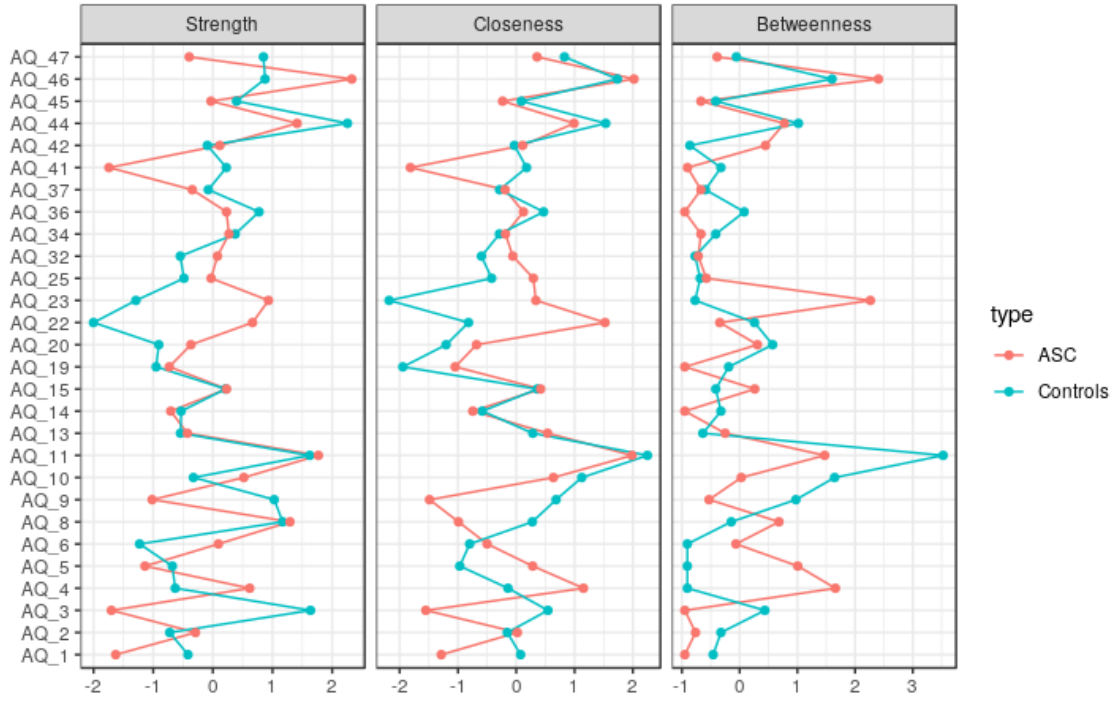


Figure 5.5 Centrality Plots for the AQ on measures of strength, closeness and betweenness

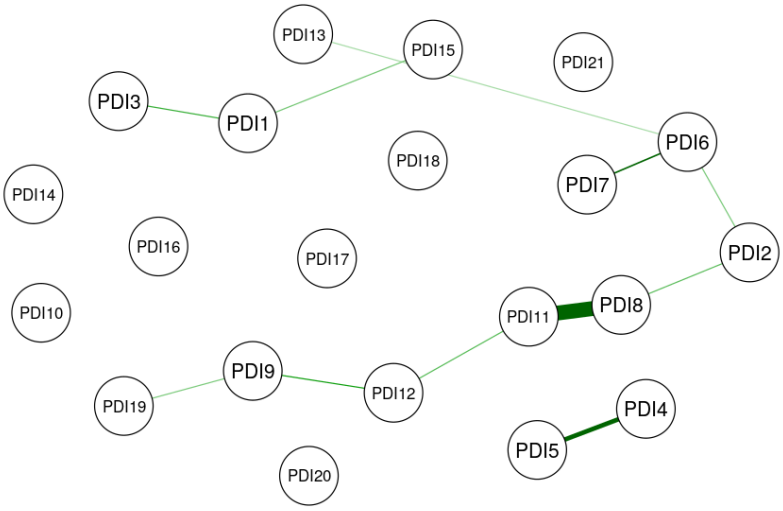
labelled as measuring religious ideas, paranoid ideas and delusions of grandeur respectively. In the control group only, item 18 ('Do your thoughts ever feel alien to you in some way?') and 20 ('Do you ever feel as if your own thoughts were being echoed back to you?') are also strongly linked and seem to reflect uncertainty about the agency of mental actions. The last obvious cluster covers beliefs in the paranormal: item 9 ('Do you ever think people can communicate telepathically?') and item 12 ('Do you believe in the power of witchcraft, voodoo or the occult?').

The centrality plots for the PDI (Figure 5.7) look a bit more disparate between the groups, but certain items still seem to have higher psychometric importance such as item 12 ('Do you believe in the power of witchcraft, voodoo or the occult?'). However, given the low overall connectivity of the network (particularly in the ASC group), not much can be gleaned from these indices and it is to be expected that their accuracy and stability is rendering them uninformative - which will be explored in the next section.

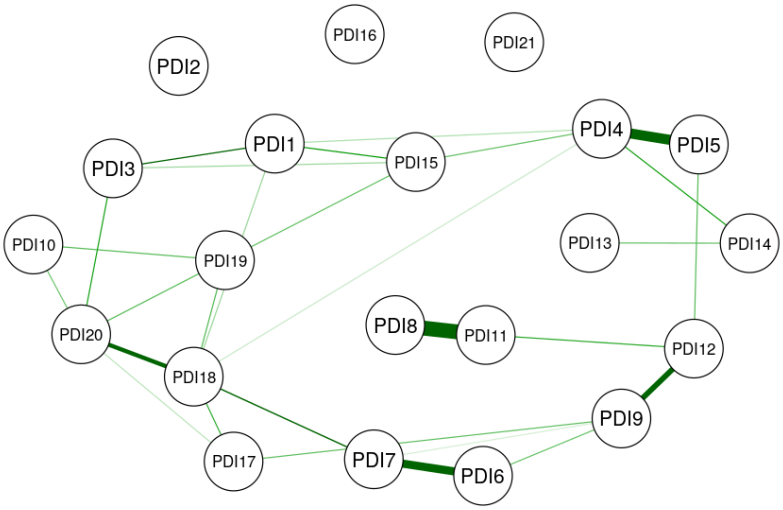
Accuracy and Stability of the Networks

Centrality Indices

The accuracy of these measures is particularly important when centrality indices of individual



(a) ASC respondents



(b) Control respondents

Figure 5.6 EGAs for the PDI in the ASC and Control group. Green edges signify positive relationships, whereas red edges are negative. Connection strength is indicated by the thickness of the line.

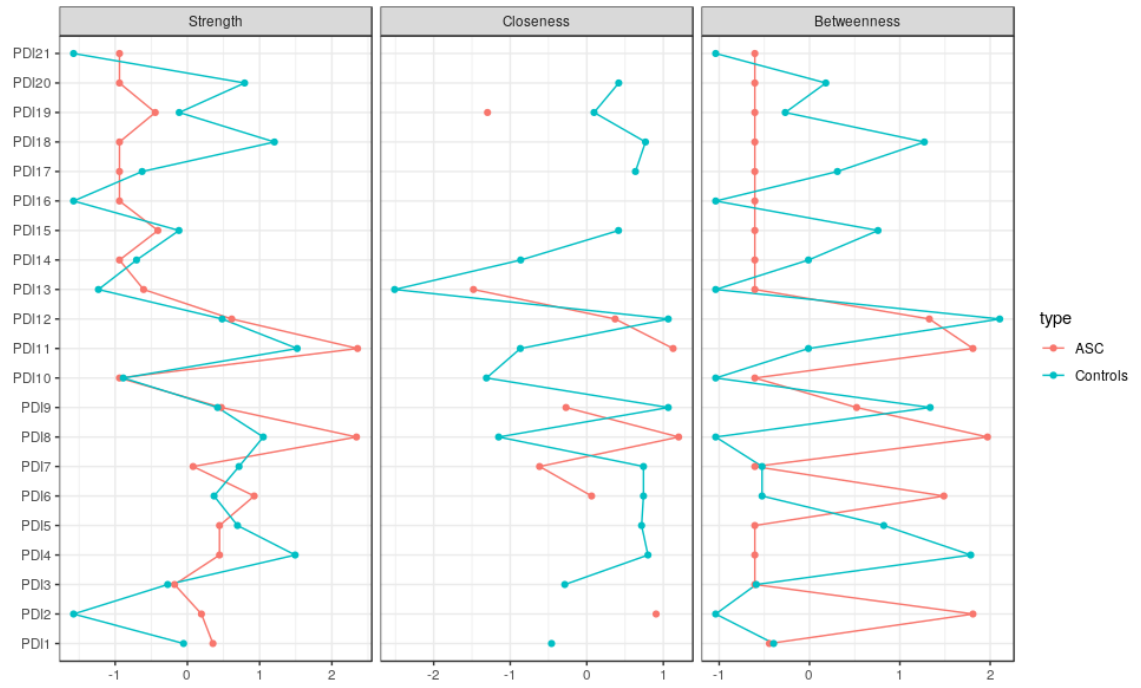
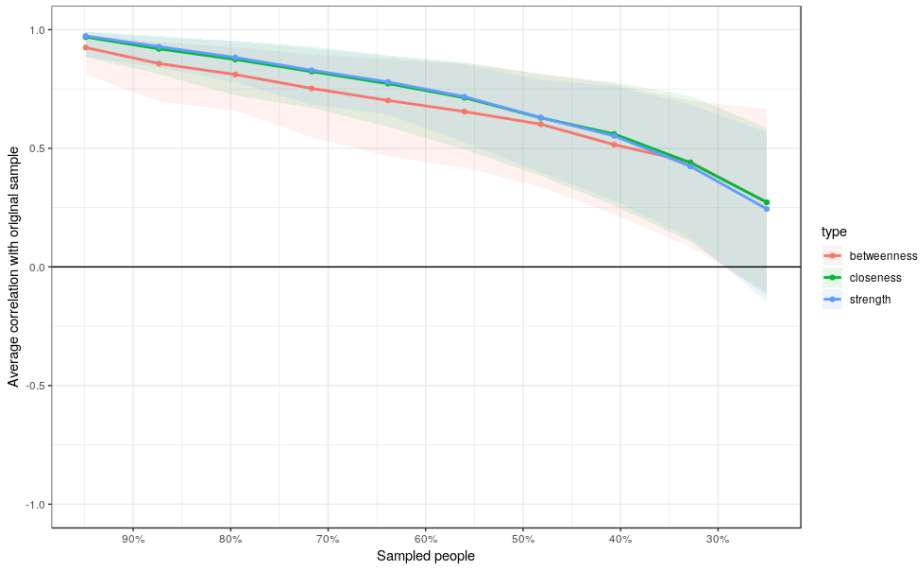


Figure 5.7 Centrality Plots for the PDI on measures of strength, closeness and betweenness

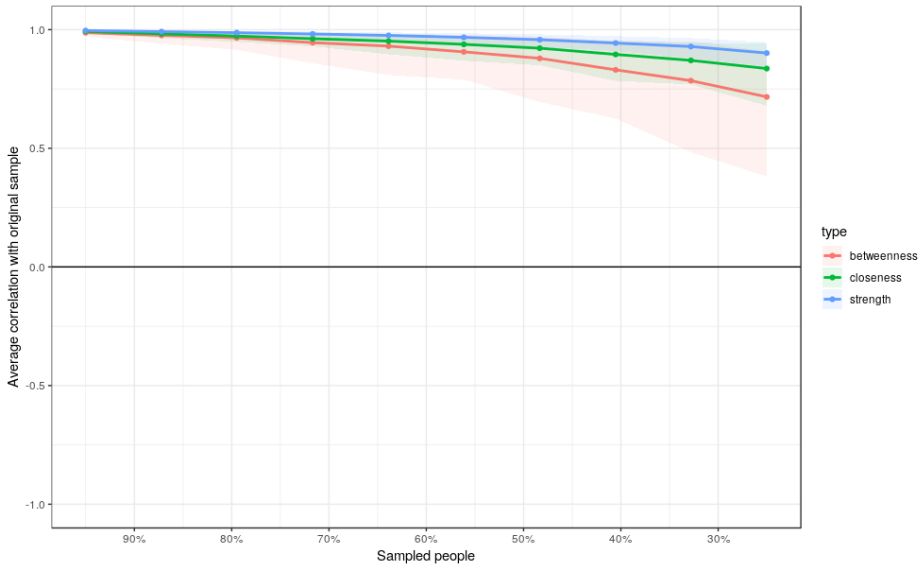
nodes are meant to be interpreted clinically (i.e. 'strong' nodes are a target for intervention). One way to assess the stability of centrality measures is to calculate the correlation stability (CS) coefficient, which is the maximum proportion of cases that can be dropped from the dataset while the correlation between original centrality indices and centrality of networks based on subsets remains at 0.7 (this is the default) or higher with a probability of 95%. According to Epskamp et al. [142], the CS-coefficient should ideally be above 0.5 and not below 0.25 although these are based on a single simulation study only.

Using 2500 bootstrapped samples, the maximum drop proportions for the AQ network structure in the control group to retain a correlation of 0.7 are at the highest level tested (>0.75) for both the strength as well as closeness. The CS coefficient for betweenness was 0.594 which puts all three measures into a comfortable range for estimated accuracy (see Figure 5.8b).

In the ASC group the maximum drop proportions to retain correlation of 0.7 in at least 95% of the bootstrapped samples are 0.127 for 'betweenness', 0.205 for 'closeness' and 0.283 for 'strength'. Thus only 'strength' is interpretable with some caution, see Figure 5.8a. It is a common effect that betweenness and closeness measures are only stable in very large samples, whereas strength is a comparatively robust metric (Jr et al. [258]).



(a) ASC respondents



(b) Control respondents

Figure 5.8 Average correlations between centrality indices of the original sample and networks with dropped cases for the AQ data. Shaded areas depict the range from the 2.5th quantile to the 97.5th quantile.

The stability estimates for the PDI are not as good as for the AQ: All three centrality indices were below the acceptable threshold for the PDI in the ASC group (see Figure 5.9) and none exhibited sufficient stability in the control group either (the only computable one being strength at 0.205). Because of the high number of missing nodes (i.e. nodes isolated form the main network), 'closeness' did not contain any variance in either group and is therefore not shown.

Edges

The non-parametric bootstrap (2500 samples) was used to estimate the confidence intervals around the regularised edge weights. Figure 5.10 displays the edge weights for the AQ from the original sample and their bootstrapped means with 95% confidence intervals. Given the larger sample size in the control data set, it is not surprising that the confidence intervals around the edge weights are smaller in this group. In the ASC group, very few edge weights are significantly different from each other. Several confidence intervals overlap with zero in both bootstrap graphs, however as these tests are performed after the regularisation, a procedure that is meant to only leave edges that are likely to be different from zero, this cannot be interpreted in the familiar way. Even when the confidence interval overlaps with zero, the confidence of the true, unbiased parameter might not. Contrarily no overlap with zero on the bootstrapping confidence interval always means that the confidence interval of the true parameter also does not include zero (Epskamp et al. [142]).

Another way to visualise statistically significant differences is to display them in a matrix displaying difference testing between individual edges: Figure 5.11.

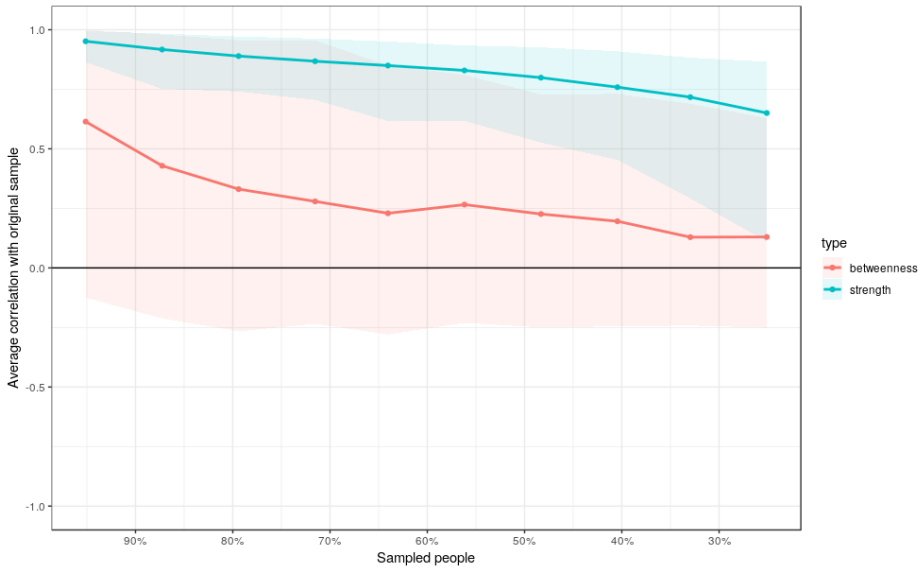
For a full list of all edge characteristics and bootstrapped CI values see Appendix B.3 for the autism group and B.4 for the control group.

For the PDI network, the bootstrapped edge weight estimates look fairly similar in both groups (Figure 5.12), but confidence intervals on single edges are larger than in the AQ. The contrast matrix is displayed in Figure 5.13 and confirms that only a small number of edges are significantly different from each other.

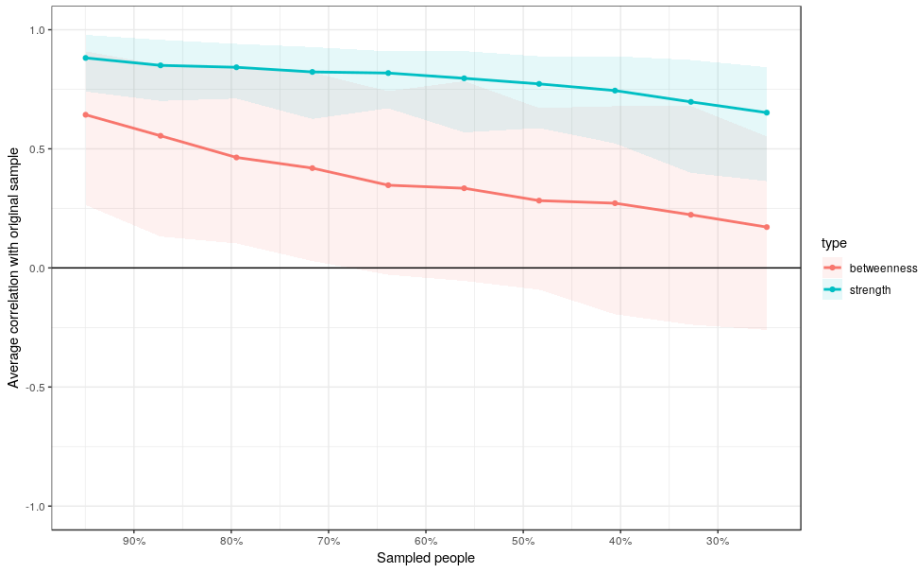
Network Comparison

AQ

Since I already examined the differences between individual edges, only global strength invariance was calculated (Figure 5.14). The global strength values of the individual networks for the AQ were 11.906 and 12.707 for the ASC and control groups respectively ($p = 0.746$).

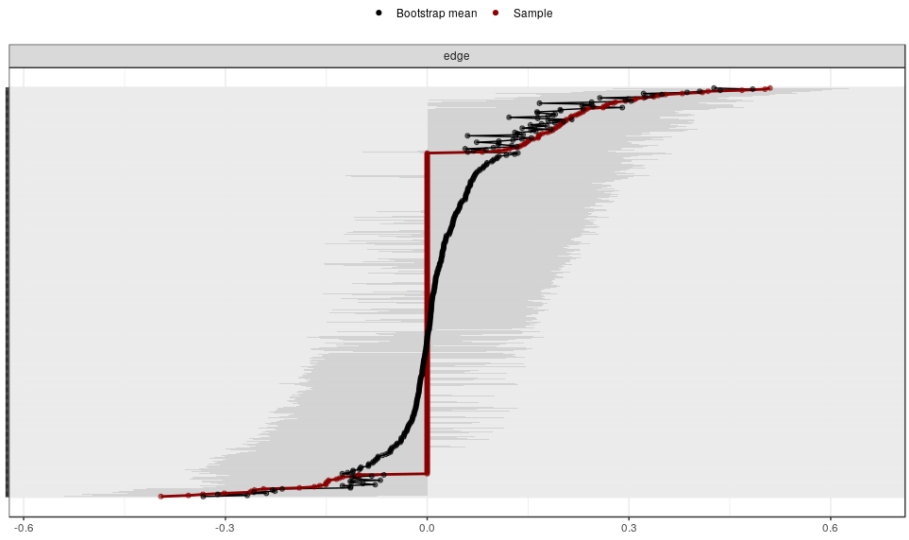


(a) ASC respondents

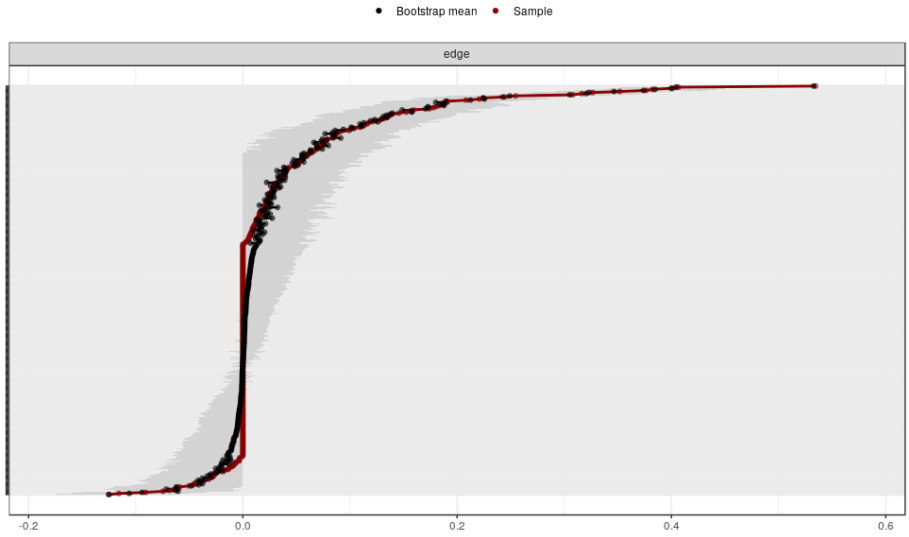


(b) Control respondents

Figure 5.9 Average correlations between centrality indices of the original sample and networks with dropped cases for the PDI data. Shaded areas depict the range from the 2.5th quantile to the 97.5th quantile.

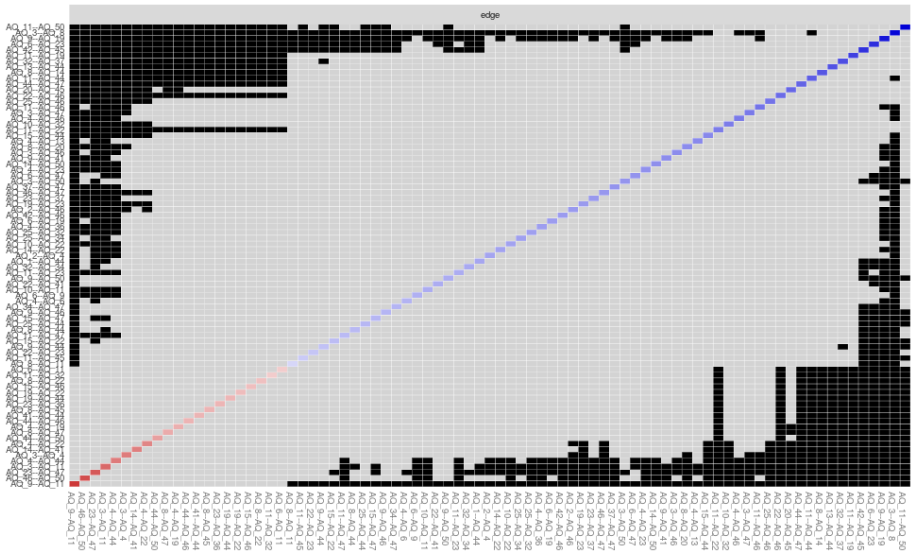


(a) ASC respondents

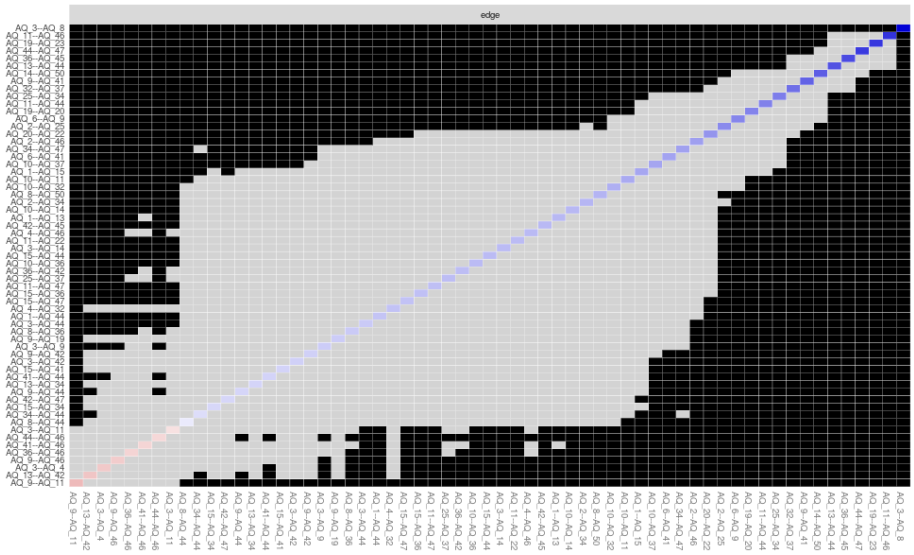


(b) Control respondents

Figure 5.10 Original edge weights and bootstrapped means and their 95% confidence intervals for the AQ network. (Possible) pairwise edge weights on the y-axis are ordered from the highest (top) to lowest (bottom).

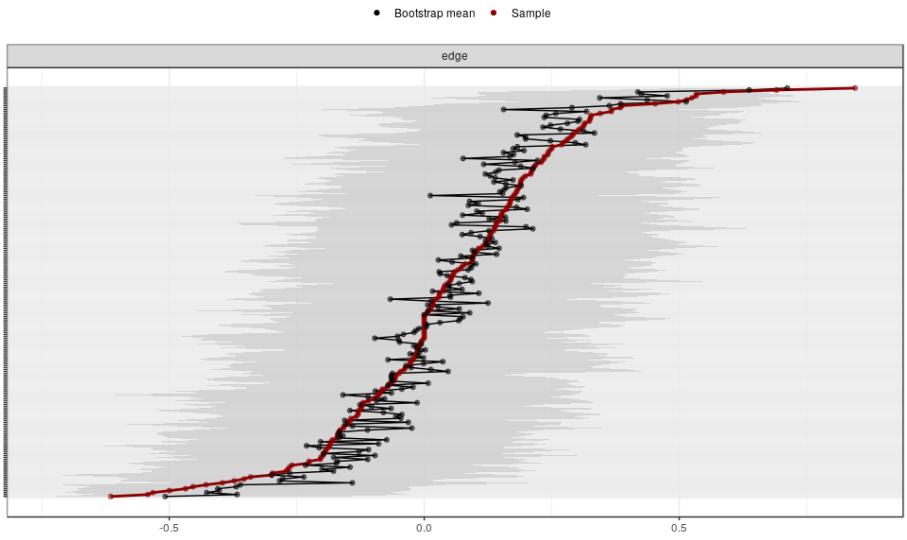


(a) ASC respondents

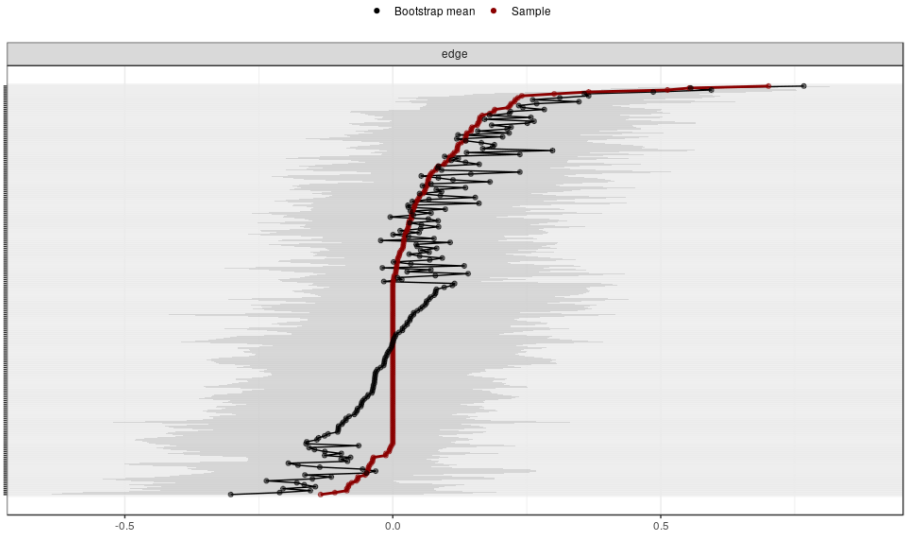


(b) Control respondents

Figure 5.11 Matrix showing significant differences ($\alpha = 0.05$, uncorrected) of edge strength for the AQ networks. Black boxes indicate a significant difference between two edge weights, grey squares indicate non-significance.

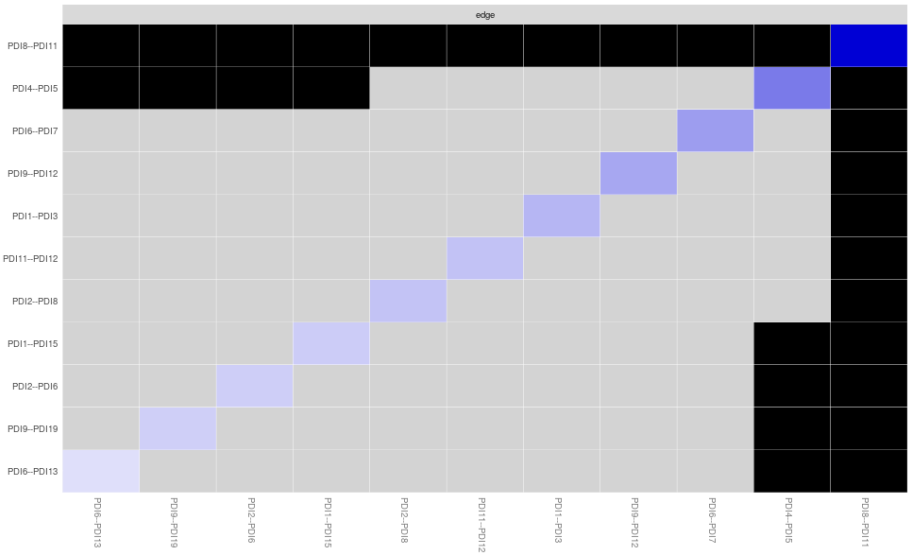


(a) ASC respondents

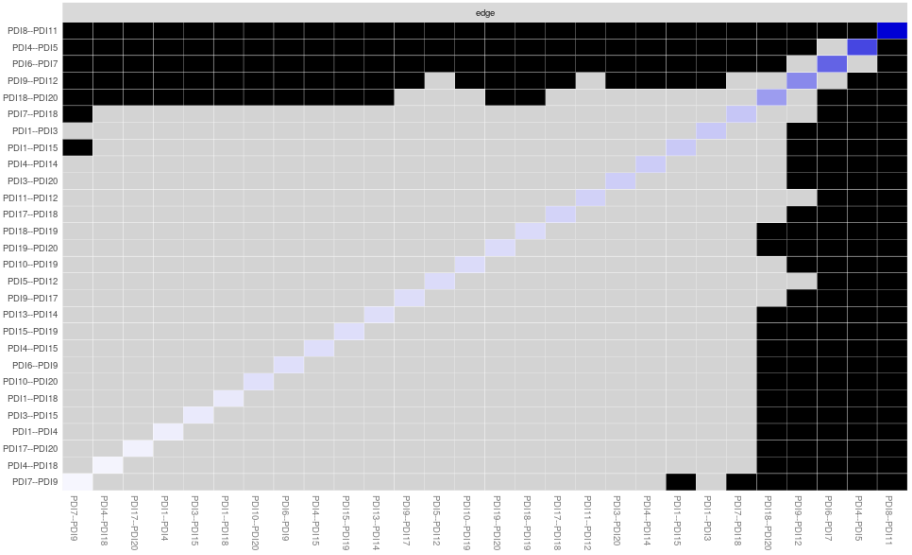


(b) Control respondents

Figure 5.12 Original edge weights and bootstrapped means and their 95% confidence intervals for the PDI network. (Possible) pairwise edge weights on the y-axis are ordered from the highest (top) to lowest (bottom).

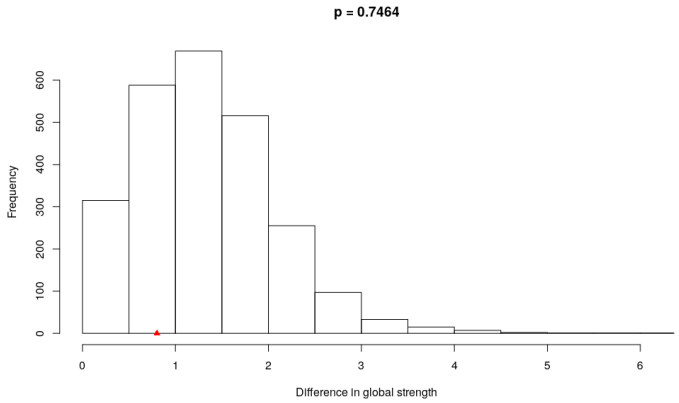


(a) ASC respondents

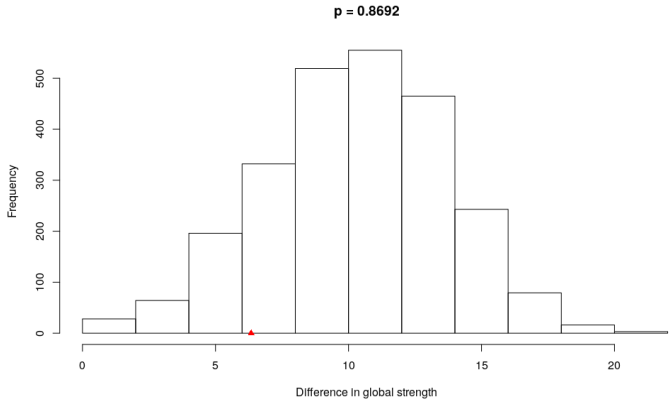


(b) Control respondents

Figure 5.13 Matrix showing significant differences ($\alpha = 0.05$, uncorrected) of edge strength for the PDI networks. Black boxes indicate a significant difference between two edge weights, grey squares indicate non-significance.



(a) Global Strength Differences for the AQ



(b) Global Strength Differences for the PDI

Figure 5.14 Reference distributions of the difference in global strength for the AQ (a) and PDI (b) networks. The red triangle shows the test statistic based on the observed (real) data.

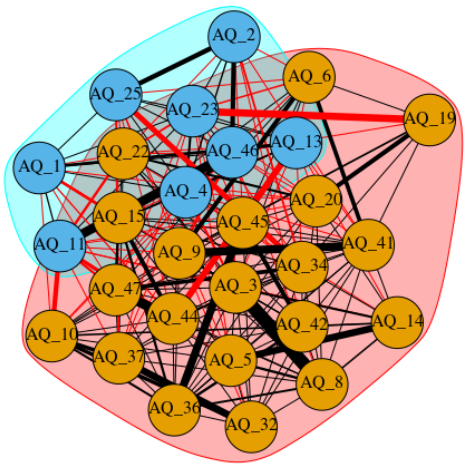
The PDI also did not exhibit any group differences in overall network strength with values of 10.745 (ASC) and 17.088 (Controls) ($p = 0.869$).

Cluster Detection in Both Groups Combined

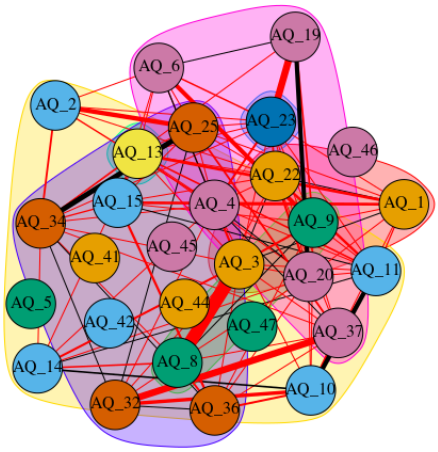
In order to perform an adequate DIF analysis, communities across both groups are estimated with the help of the *walktrap* algorithm, *spinglass* algorithm, the *leading eigenvector* algorithm and the *fast greedy modularity optimisation* algorithm. Community detection algorithms did not yield consistent clusters of nodes for neither the AQ (Figure 5.15 - c) and d) share some similarities though) nor the PDI (Figure 5.16).

Table 5.2 Communities for the AQ

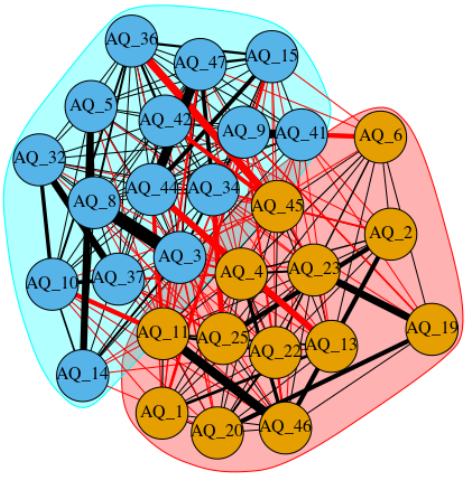
Cluster	Walktrap	Spinglass	Leading Eigenvector	Fast Greedy
1	1,2,4,11,13,23,25,46	6,9,41	1,2,4,6,11,13,19,20,22,23,25,45,46	1,2,4,6,9,11,13,15,19,20,22,23,25,41,45,46
2	3,6,8,9,10,11,14,15,19,20,22,32,36,37,41,42,44,45,47,50	36,42,45	3,8,9,10,14,15,32,34,36,37,41,42,44,47,50	3,8,10,14,32,34,36,37,42,44,47,50
3		1,11,13,15,44,47		
4		3,8,14,50		
5		2,4,25,34,46		
6		19,20,22,23		
7		10,32,37		



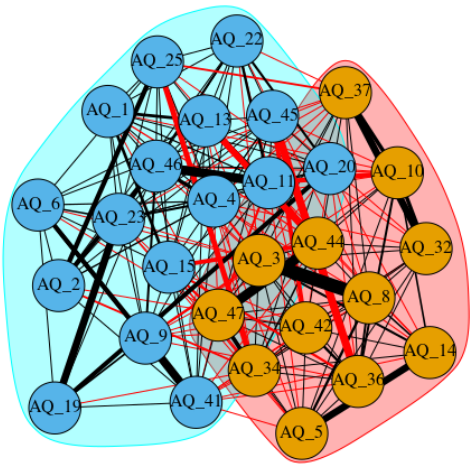
(a) Walktrap Algorithm



(b) Spinglass Algorithm



(c) Leading Eigenvector Algorithm

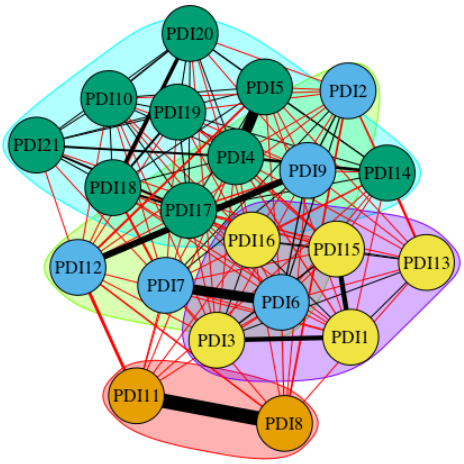


(d) Fast Greedy Modularity Optimisation

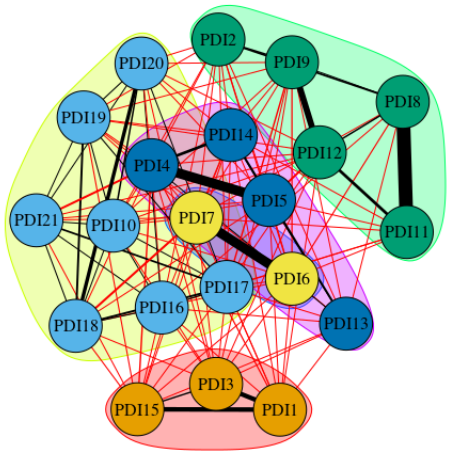
Figure 5.15 Clusters/Communities of nodes in the AQ network for both groups combined based on four different algorithms

Table 5.3 Communities for the PDI

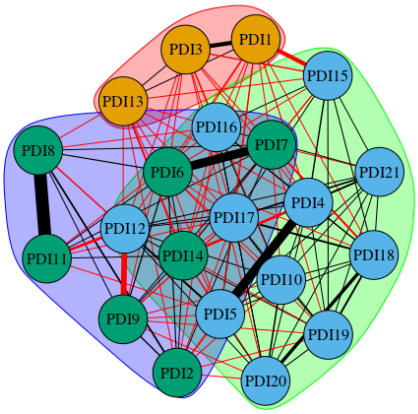
Cluster	Walktrap	Spinglass	Leading Eigenvector	Fast Greedy
1	8,11	6,7	1,3,13	1,2,5,6,8,9,11,12,13,14,17
2	2,6,7,9,12	4,5,13,14	4,5,10,12,15,16,17,18,19,20,21	3,4,7,10,15,16,18,19,20,21
3	4,5,10,14,17,18,19,20,21	10,16,17,18,19,20,21	2,6,7,8,9,11,14	
4	1,3,13,15,16	1,3,15		
5		2,8,9,11,12		



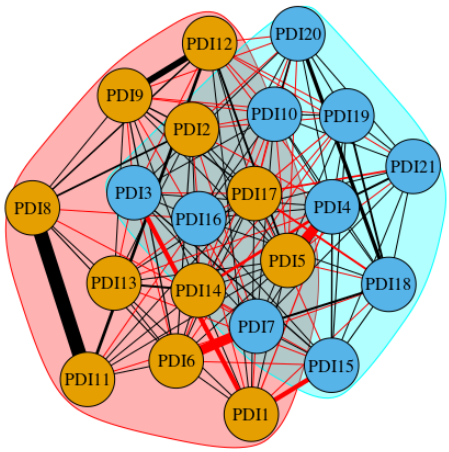
(a) Walktrap Algorithm



(b) Spinglass Algorithm



(c) Leading Eigenvector Algorithm



(d) Fast Greedy Modularity Optimisation

Figure 5.16 Clusters/Communities of nodes in the PDI network for both groups combined based on four different algorithms

5.3.2 IRT/DIF

No common, robust clusters could be identified by comparing results for *spinglass*, *walktrap*, *leading eigenvector* and *fast-greedy* for either the AQ or PDI. Therefore both IRT analyses were conducted on the questionnaire(s) as a whole.

AQ

Contrary to the analysis plan (Finnemann [151]), the dichotomous AQ scores were analysed using a 2PL model rather than a rating scale model to allow for plotting of ICCs. The 2PL model was selected as it had the best model fit (see Table 5.4). 3Pl and 4PL were not assessed as they should only be considered when there is a strong prior hypothesis that they model the data.

Table 5.4 IRT Model Comparison Between 1PL and 2PL for the AQ-28, the better model fit is marked in bold. AIC is the Akaike information criterion ([5]), AICc has a correction for finite sample size, BIC is the Bayesian information criterion ([493]) and SABIC is the Sample-sized adjusted BIC criterion.

Model	AIC	AICc	BIC	SABIC	χ^2	df	p
1PL	53821.846	53822.976	53977.233	53885.107	NaN	NaN	NaN
2PL	**52427.751**	**52431.973**	**52727.81**	**52549.91**	1448.095	27	0

Using the *difNLR* and *difR* packages, a generalised logistic regression likelihood ratio χ^2 statistic based on 2PL was fitted to the AQ-28. Parameters were estimated with non-linear least squares. Item purification was not applied and multiple comparisons were adjusted for with the Benjamini-Hochberg correction.

The majority of AQ-28 items exhibited differential functioning (Table 5.5): AQ2 ('I prefer to do things the same way over and over again.'), AQ3 ('If I try to imagine something I find it very easy to create a picture in my mind.'), AQ4 ('I frequently get so absorbed in one thing that I lose sight of other things.'), AQ6 ('I usually notice car number plates or similar strings of information.'), AQ8 ('When I'm reading a story, I can easily imagine what the characters might look like.'), AQ9 ('I am fascinated by dates.'), AQ10 ('In a social group, I can easily keep track of several different people's conversations.'), AQ11 ('I find social situations easy.'), AQ13 ('I would rather go to a library than a party.'), AQ14 ('I find making up stories easy.'), AQ19 ('I am fascinated by numbers.'), AQ20 ('When I'm reading a story, I find it difficult to work out the characters' intentions.'), AQ22 ('I find it hard to make new

Table 5.5 Differential Item Functioning for the AQ-28, the detection threshold is set at 5.992 (α=0.05).

Item	χ^2	p	adj. p	
$AQ1$	2.134	0.344	0.385	
$AQ2$	46.088	<0.001	<0.001	***
$AQ3$	160.175	<0.001	<0.001	***
$AQ4$	94.405	<0.001	<0.001	***
$AQ6$	19.771	<0.001	<0.001	***
$AQ8$	84.733	<0.001	<0.001	***
$AQ9$	44.480	<0.001	<0.001	***
$AQ10$	7.876	0.020	0.027	*
$AQ11$	35.825	<0.001	<0.001	***
$AQ13$	8.873	0.012	0.017	*
$AQ14$	23.807	<0.001	<0.001	***
$AQ15$	0.712	0.700	0.726	
$AQ19$	22.979	<0.001	<0.001	***
$AQ20$	124.001	<0.001	<0.001	***
$AQ22$	47.547	<0.001	<0.001	***
$AQ23$	143.377	<0.001	<0.001	***
$AQ25$	21.238	<0.001	<0.001	***
$AQ32$	1.476	0.478	0.515	
$AQ34$	12.723	0.002	0.003	**
$AQ36$	0.154	0.926	0.926	
$AQ37$	13.341	0.001	0.002	**
$AQ41$	5.350	0.070	0.088	
$AQ42$	5.443	0.066	0.088	
$AQ44$	25.768	<0.001	<0.001	***
$AQ45$	4.722	0.094	0.114	
$AQ46$	125.250	<0.001	<0.001	***
$AQ47$	38.778	<0.001	<0.001	***
$AQ50$	3.944	0.139	0.162	

friends.'), AQ23 ('I notice patterns in things all the time.'), AQ25 ('It does not upset me if my daily routine is disturbed.'), AQ34 ('I enjoy doing things spontaneously.'), AQ37 ('If there is an interruption, I can switch back to what I was doing very quickly.'), AQ44 ('I enjoy social occasions.'), AQ46 ('New situations make me anxious.') and AQ47 ('I enjoy meeting new people.').

Figure 5.17 displays the item characteristic curves for these items. Items 3, 8, 9, 13, 34 and 37 display uniform DIF (where one group is always at a disadvantage when responding to the question) whereas the remainder display non-uniform DIF (where the lines intersect and the same group will have an advantage or a disadvantage at varying proficiency levels).

Notably, the control groups seems to have a low α for a fair number of times (AQ2, AQ4, AQ6, AQ19, AQ20, AQ22, AQ23 and AQ46) suggesting that these items do not adequately differentiate individuals with low latent trait levels of θ.

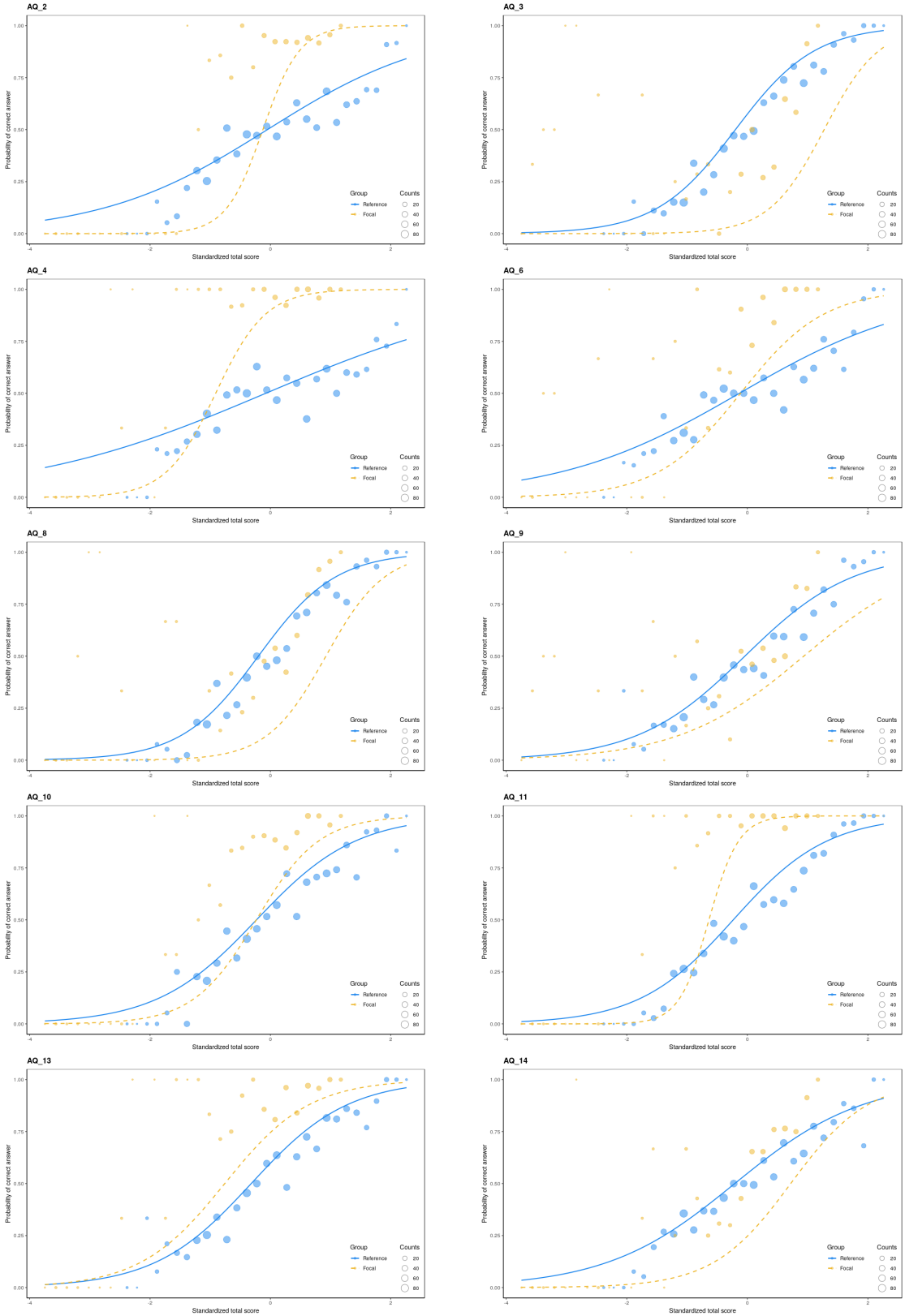


Figure 5.17 ICCs for the differentially functioning items of the AQ, the focal group (yellow) are the responses from the ASC group, the reference group (blue) are the controls. Continued on next page.

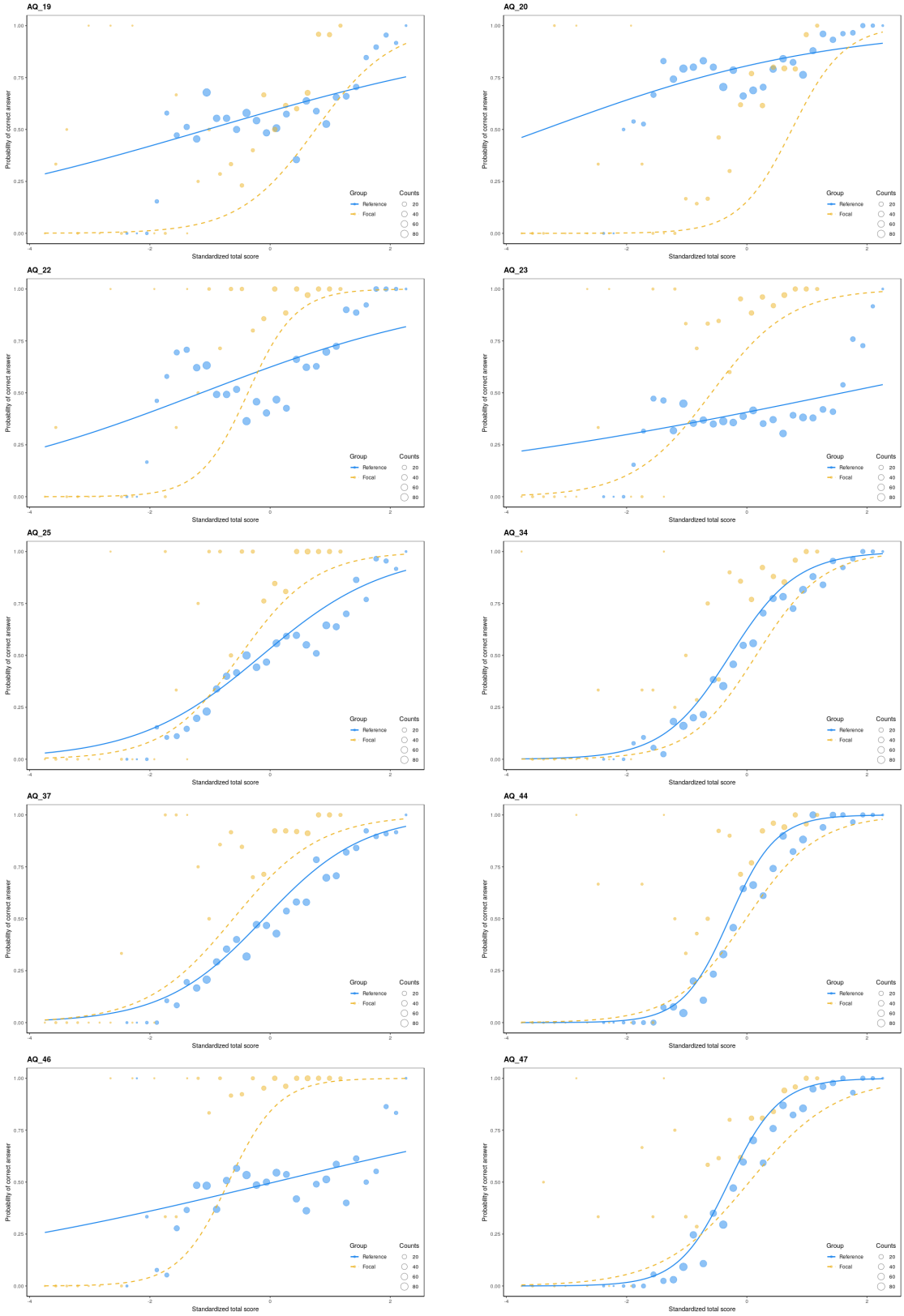


Figure 5.17 ICCs for the differentially functioning items of the AQ, the focal group (yellow) are the responses from the ASC group, the reference group (blue) are the controls.

PDI

Using a marginal maximum-likelihood method (and assuming θ is normally distributed), a model comparison between 1PL and 2PL resulted in overall more support for the 2PL model (see Table 5.6).

Table 5.6 IRT Model Comparison Between 1PL and 2PL for the PDI, the better model fit is marked in bold. AIC is the Akaike information criterion ([5]), AICc has a correction for finite sample size, BIC is the Bayesian information criterion ([493]) and SABIC is the Sample-sized adjusted BIC criterion.

Model	AIC	AICc	BIC	SABIC	χ^2	df	p
1PL	51254.425	51254.892	**51372.043**	51305.325	NaN	NaN	NaN
2PL	**51192.115**	**51193.789**	51416.151	**51289.069**	100.31	19	0

Differentially functioning items are: PDI1 ('Do you ever feel as if people seem to drop hints about you or say things with a double meaning?'), PDI4 ('Do you ever feel as if you are being persecuted in some way?'), PDI5 ('Do you ever feel as if there is a conspiracy against you?'), PDI6 ('Do you ever feel as if you are, or destined to be someone very important?'), PDI8 ('Do you ever feel that you are especially close to God?'), PDI10 ('Do you ever feel as if electrical devices such as computers can influence the way you think?'), PDI13 ('Are you often worried that your partner may be unfaithful?'), PDI15 ('Do you ever feel that people look at you oddly because of your appearance?') and PDI21 ('Do you ever feel as if you are a robot or zombie without a will of your own?'). Figure 5.18 displays the item characteristic curves for these items. PDI items 4, 5, 6, 8 and 10 display uniform DIF (where one group is always at a disadvantage when responding to the question) whereas items 1, 13, 15 and 21 display non-uniform DIF (where the lines intersect and the same group will have an advantage or a disadvantage at varying proficiency levels).

On DIF items of social suspiciousness and paranoia (1,4,5), ASC participants consistently display lower item difficulty (smaller τ) - that is to say given the same estimated level of the underlying latent trait θ, they are more likely to respond positively to questions about feelings of persecution or paranoia. An exception to this is item 13 which assesses suspicion about the faithfulness of one's partner. As autistic individuals are less likely to be in a romantic relationship both compared to their non-autistic peers as well as compared to other psychiatric groups (Hofvander et al. [235], Barneveld et al. [30]), this question might simply not have applied to the majority of respondents. The pattern of lower item difficulty for the ASC group is reversed for questions 6, 8 and 10 which cover religious beliefs and thought insertion. For item 12 on paranormal beliefs and item 21 on lack of sense of agency, ASC

Table 5.7 Differential Item Functioning for the PDI, the detection threshold is set at 5.992 (α=0.05).

Item	χ^2	p	adj. p	
PDI1	16.634	<0.000	0.001	**
PDI2	4.897	0.086	0.151	
PDI3	1.596	0.450	0.556	
PDI4	32.967	<0.001	<0.001	***
PDI5	36.739	<0.001	0.001	***
PDI6	31.796	<0.001	<0.001	***
PDI7	2.054	0.358	0.470	
PDI8	8.517	0.014	0.033	*
PDI9	3.296	0.193	0.311	
PDI10	23.557	<0.001	<0.001	***
PDI11	2.524	0.283	0.425	
PDI12	7.266	0.026	0.056	
PDI13	9.354	0.009	0.024	*
PDI14	0.623	0.732	0.786	
PDI15	14.631	<0.001	0.002	**
PDI16	0.288	0.866	0.866	
PDI17	1.110	0.574	0.670	
PDI18	2.172	0.338	0.470	
PDI19	0.580	0.748	0.786	
PDI20	6.208	0.045	0.086	
PDI21	12.570	0.002	0.006	**

participants exhibit a lower item difficulty for low levels of θ, but lower α means that at higher θ the control group shows a lower item difficulty. The reverse is true of item 15.

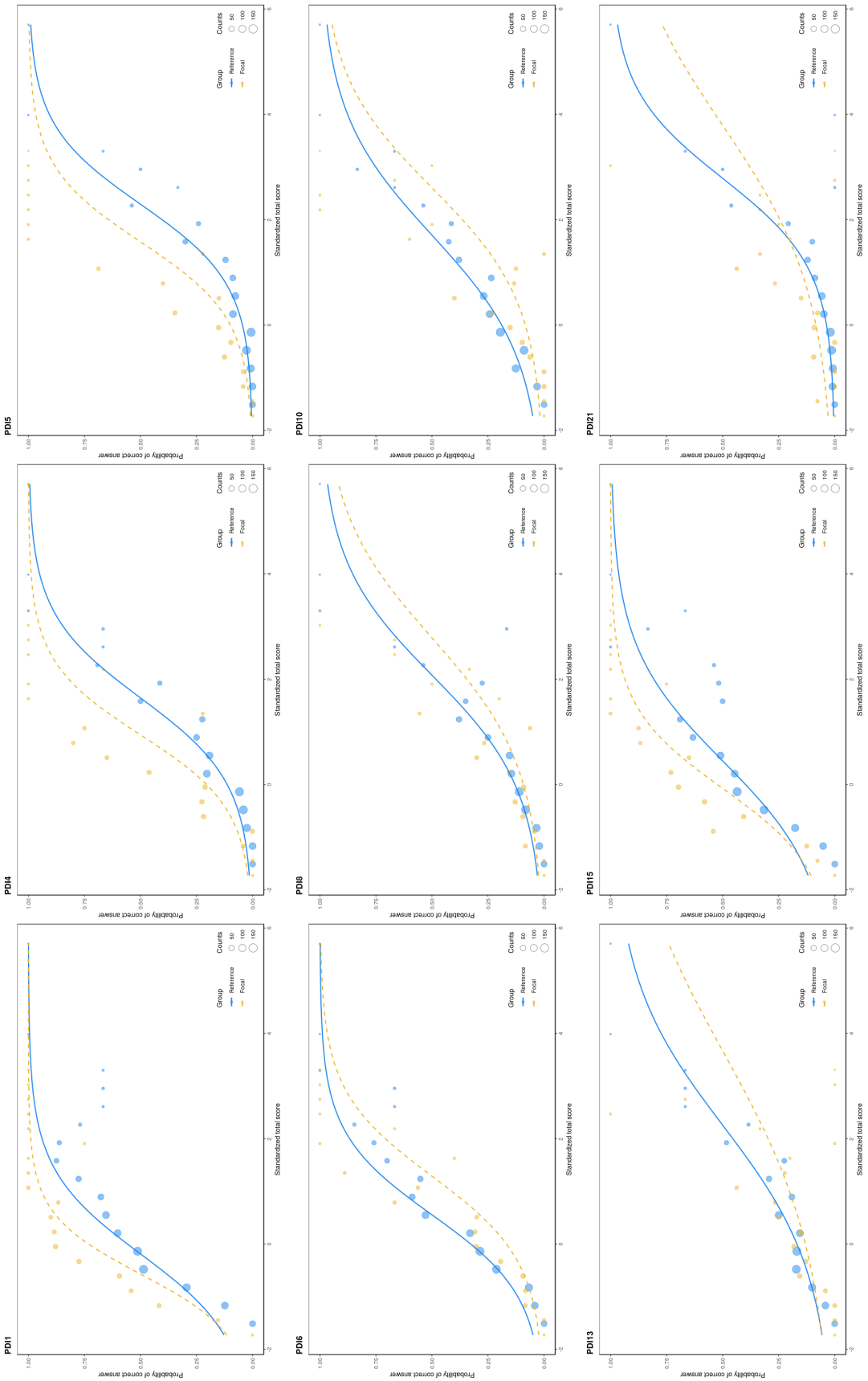


Figure 5.18 ICCs for the differentially functioning items of the PDI, the focal group (yellow) are the responses from the ASC group, the reference group (blue) are the controls.

5.4 Discussion

In view of the fact that the AQ and PDI are both routinely used (including by this dissertation!) to quantify levels of the character traits in question as well as screen for patients which might meet diagnostic criteria, a cursory evaluation of their construct validity has been long overdue. The present study was designed to assess the aspect of generalisability across groups, or 'measurement invariance', in different populations. While initial differences were already apparent at the level of configural invariance (or item clusters), a definite group difference in global connectivity strength could not be determined and the edge and centrality measures were not robust enough to test for statistical differences. More pronounced differences were evident in the DIF analysis for several items in both the AQ and the PDI. Neither group had a systematic advantage, rather respondents from the two groups might not attribute the same meaning to the items.

For instance the AQ item clusters 11 & 46 in the EGA might measure social anxiety in the control group, but tap into social difficulties in the ASC group, thus leading to the observed DIF of both items. This would support the previously mentioned claims that the AQ is detecting a broader liability for psychopathology in non-autistic individuals (Ashwood et al. [17], Reed et al. [452], Lugnegård et al. [338], Naito et al. [389], Sizoo et al. [515]). In line with this, previous large-scale studies of non-clinical populations report a unique association of high AQ scores with reports of social anxiety (Russell-Smith et al. [478]).

Of particular interest is also the low discriminatory value of several AQ items for the control group indicating that only a small amount of variability in the responses to those items is attributable to real differences of the underlying latent trait θ. These items therefore do not seem appropriate for measuring autistic traits in non-autistic individuals.

In the PDI, autistic respondents displayed lower item difficulty for questions addressing paranoia and suspiciousness. Paranoid thoughts and behaviours have previously been reported in autism and appear to be related to an increased frequency of negative social experiences (such as being bullied) in the autistic population as well as difficulty with Theory of Mind and as such have different causal pathways to paranoia in the context of schizotypy in the general population (Spain et al. [520], Pinkham et al. [431]). Conversely, the ASC group were less likely to endorse items about religious beliefs at the same estimated latent trait level. This fits with data suggesting that - perhaps due to mentalising deficits - autistic individuals are less likely to express belief in a deity (Norenzayan et al. [401])[7]. Based on the correlation between the PDI and communication difficulties reported in Chapter 4 and

[7] However see Reddish et al. [451] for a different opinion.

personal anecdotal evidence, individual items might also be misinterpreted by some people on the spectrum: One of my participants responded affirmatively to the item 'Do you ever feel as if electrical devices such as computers can influence the way you think?', because he thought that acquiring knowledge by way of reading about various topics on the internet does of course have an effect on his views and opinions.

Taken together these substantial differences indicate that one should exercise caution when using the AQ or PDI to compare latent traits across autistic and non-autistic populations. The present study only investigated differential item functioning across groups, so no inferences can be made about the validity of the questionnaires within groups or across different groups such as gender. The network graph looked more plausible in the ASC group, both in terms of clustering as well as general (positive) connectivity which is perhaps not surprising as it was designed with this group in mind. The discriminatory power of the AQ items was also much better for the autistic group, so we might have more confidence in using the AQ to quantify autistic traits in individuals with an existing diagnosis of autism than in its applicability and appropriateness for healthy individuals. Other measures such as the Broad Autism Phenotype Questionnaire (Hurley et al. [245]) have been developed to capture autistic traits in the general population, but so far very little research has been dedicated to validate them in different clinical and non-clinical samples. In the same way that the meaningfulness of the AQ is compromised for non-autistic individuals, the PDI's validity for the ASC group could not be established. Overall, item associations were weaker than in the control group and it is unclear to what extent the PDI might be measuring traits associated with autism, such as social difficulties. Based on the results of this analysis, higher total scores of schizotypy measures in autistic participants should not automatically be interpreted as an association between schizotypy and autism.

The development of instruments that can measure autistic traits is a pressing research need as can be seen by the wide use of the AQ[8]. Future work should focus on developing well-validated tools to assess constructs such as autistic traits and schizotypy in both clinical and non-clinical populations instead of applying instruments which were developed for use in a specific sub-group to the population at large or vice versa. The improvement of existing instruments and potential development of novel measurements is also an opportunity to enhance and refine our understanding of schizotypy and autistic traits and the suggested continuum of trait distributions within or across groups.

[8] A Pubmed search for the terms 'Autism Spectrum Quotient', 'autism quotient', 'autism spectrum quotient' or 'Autism Quotient' resulted in more than 800 papers.

At the same time it is also important to acknowledge that the vacillating ideas about the construct of autism and schizophrenia create a moving target for psychometricians. Caveats of the present research effort include the reasonably small ASC sample, lack of matching for age and presence of psychopathology in the control group. Even though autism is becoming an increasingly common diagnosis, clinical samples are still difficult to recruit and thus the present sample size of the clinical data might be regarded as small for psychometric investigations that usually employ datasets with >1000 respondents. That being said, Kim and Yoon [280] reported true positive rates of 100% for DIF analyses with polytomous data with a sample size of 200 and acceptable detection for dichotomous data at 500 observations. Type 1 error rates are also relatively robust in logistic regression DIF models under unequal sample sizes (Herrera and Gómez [228]). Similar recommendations for sample size (500 for binary data of 10-30 nodes and 250 for polytomous data) have been put forward by Dalege et al. [113] for the EGA. The control group exceeds both of those cutoffs whereas the ASC group has under 500 respondents but more than 100 from which we can infer at least reasonable power. Other DIF studies with similar item counts have employed even smaller sample sizes (Baer et al. [22]).

Chapter 6

General Discussion: Nothing New under the Sun?

> *There is, it seems to us,*
> *At best, only a limited value*
> *In the knowledge derived from experience.*
> *The knowledge imposes a pattern, and falsifies,*
> *For the pattern is new in every moment*
> *And every moment is a new and shocking*
> *Valuation of all we have been.*
>
> T.S. ELIOT, FOUR QUARTETS

6.1 Limitations and Future Research

In view of the increasing number of papers using the predictive coding framework to explain a range of behavioural phenomena, I felt it was still worthwhile to gather some empirical data with paradigms that have invoked predictive processing. This thesis provides converging evidence that basic sensory predictive mechanisms are intact in ASC and extends the list of investigations with comparable results: Tewolde et al. [555], Van de Cruys et al. [574], Manning et al. [350]. As the aim of this work was to investigate a broad range of potential

differences in sensory prediction, none of the paradigms was investigated in enough detail (via tweaks and amendments) to distinguish between the proposed theories of sensory processing in autism presented in Chapter 1. Another area that was deliberately left out in the current investigations were predictive processes dealing with higher-order (i.e. social) priors. Notably these have been found to be impaired (Balsters et al. [26], Chambon et al. [76]), but most theories on prediction place the hypothesised atypicalities in autism at a more global processing stage.

The results presented here also continue a general trend in autism research where previously reported deficits are not corroborated in more recent publications. One explanation could be that the aforementioned changes in diagnostic criteria have changed the demographics of the test cohorts used in research (and the higher rate of female participants is one obvious difference) such that more recent experiments are conducted with overall less impaired individuals. This is certainly a limitation of the current work as a substantial amount of participants would not have qualified for a diagnosis based on behavioural observation tools. From an ethical and practical point of view it is clear why 1) participants with better adaptive skills volunteer more readily for research projects and 2) researchers refrain from recruiting individuals where concerns over the distress caused by the participation or consent could be raised. Nevertheless it is important to be mindful of the fact that findings from one cohort might only have limited generalisability to groups with a very different phenotypic expression. However from a quantitative standpoint, stratification by the ADOS, AQ (and PDI) - the weaknesses of which were discussed in Chapter 5 - did not support a general hypothesis that those with more pronounced (autistic) traits exhibited more atypical responses on the tasks employed in this piece of work.

A further limitation of this thesis was the exclusion of younger populations for the experiments. As autism is a neurodevelopmental disorder, it would be worth exploring if the trajectories for acquiring and refining internal models of the external world are different in autistic individuals even if performance is indistinguishable at a later developmental stage. Out of the studies presented here, the force-matching and two-tone image tasks would lend themselves to be employed with children or teenagers. Since structural priors emerge from long-term aggregation of individual experiences (as opposed to the short-term learning of stochastic relationships for contextual priors), they supposedly are subject to developmental processes and as such the force-matching task would be the best candidate for a developmental approach to predictive coding paradigms. In addition, the task offers an opportunity to shed light on the previously discussed hypotheses concerning low (Davis and Plaisted-Grant [119]) or high (Simmons et al. [508]) endogenous neural noise in autism.

In order to uncover the origin of the increased force variability seen in the autism group, a new condition could be added to the experiment: Mechanical white input noise improves performance on a range of human motor functions including static isometric force production (Trenado et al. [561], Collins et al. [87], Magalhães and Kohn [346], Kouzaki et al. [294]). Support for the low endogenous neural noise hypothesis would imply that autistic individuals improve the steadiness of their force output with comparatively higher amplitudes of the input noise. If however the autism group does not benefit from the added white noise, the source of the force variability may have to be sought elsewhere such as in the coloured noise associated with the neuromuscular periphery (Davids et al. [117]).

Another suggestion would have been to tease apart potential differences by conducting thorough sensory phenotyping in order to relate domain-specific symptoms to domain-specific tasks. Regrettably, despite consistent group differences on self-report of sensory reactivity, the majority of questionnaires such as the Short Sensory Profile have been deemed to be psychometrically unreliable in differentiating between different sensory phenotypes (Williams et al. [605]) and a two-factor solution including 'uniformly elevated scores' and 'avoidance' seems to be the best fit (Simpson et al. [511]). Both hypo- and hyperreactivity commonly co-occur in individuals with autism (Baranek et al. [29], Greenspan and Wieder [196]) and this is more frequently seen in individuals with overall more pronounced sensory atypicalities (Elwin et al. [139]). Thus, unless better measures become available it is unlikely that questionnaire measures alone will help to unpack performance variability on these tasks within the group. More sensitive and novel approaches that do not exclusively rely on self-report (such as The Sensory Processing Scale Assessment, Schoen et al. [486]) in combination with other measures would be an interesting supplement to this line of research. Existing scales also primarily assess sensory reactivity as a stable trait, rather than a context-dependent state even though it has been widely acknowledged that precipitating and additive events contribute to sensory overload (Brown and Dunn [69], Belmonte and Yurgelun-Todd [43]). Sensory avoidance is potentially a good measure of state anxiety (Engel-Yeger and Dunn [141]), but so far no validated measures have been published that could be used as assessments of acute sensory processing difficulties per se.

Aside from refuting the hypothesis that autism is characterised by a 'predictive impairment [...] shared across individuals' (Sinha et al. [512]), the findings presented here also do not support the notion of 'predictive ability' as a unitary process across tasks and individuals. Others have equally pointed out that 'the heterogeneity of experimental manipulations that are thought to reflect predictive processes are likely to measure different processing steps, making their direct comparison difficult.'(Grotheer and Kovács [199]) and the experiments

in this thesis are no different. The magnitude of 'prediction' as measured on one task was not related to the task execution of other paradigms suggesting that the mechanisms at work during sensorimotor attenuation differ - if not at the computational or algorithmic level - at least at the level of implementation. If computational theories are going to advance our understanding of psychiatric conditions, the insights yielded by their proposed experimental underpinnings should initially be well described and explored in non-clinical populations. The lack of cross-paradigm correspondences should thus be addressed by - for example - building up evidence in favour of multi-domain equivalency of effects.

6.2 Conclusions

> *We demand rigidly defined areas of doubt and uncertainty!*
>
> DOUGLAS ADAMS, THE HITCHHIKER'S GUIDE TO THE GALAXY

While I have outlined a few ideas and specific areas into which current autism research could expand its investigations, it might also be time to start resisting the lure of sweeping claims and unifying theories to focus instead on steady progress in creating and testing more circumscribed models of sensory processing. This would mirror efforts in other related fields which have called for concentrated efforts in preference to broad-brush conclusions about nebulous and largely descriptive outcomes such as 'optimal' versus 'non-optimal' behaviour (Rahnev and Denison [447]).

The danger of such abstract models lies in the fact that incompatible results may be explained as still adhering to the overarching theoretical framework - either due to local, task-dependent differences or a lack of exactitude in the specification of the model parameters. As a recent discussion pointed out, the question about how high precision and information gain are optimised in predictive processing has still not been answered (Kwisthout et al. [297]) and as such leaves ample room for the causal interpretation of atypical behavioural or neurophysiological responses. If, on the other hand we stick to a more general model, the knowledge gleaned from framing empirical results within its specifications is limited and remains largely descriptive. As researchers in psychiatry, we ultimately aim to uncover neurobiological mechanisms for symptoms, but above all improve care and diagnostic validity

and timeliness. If framing clinically and phenomenologically established differences within a particular theoretical framework does neither add to the mechanistic understanding or treatment potential, nor provide pointers for future investigations, it might be time to retire such ambitions to the realm of philosophical discourse.

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Appendix A

Appendix to Chapter 3

A.0.1 Comparison with Voss et al. data

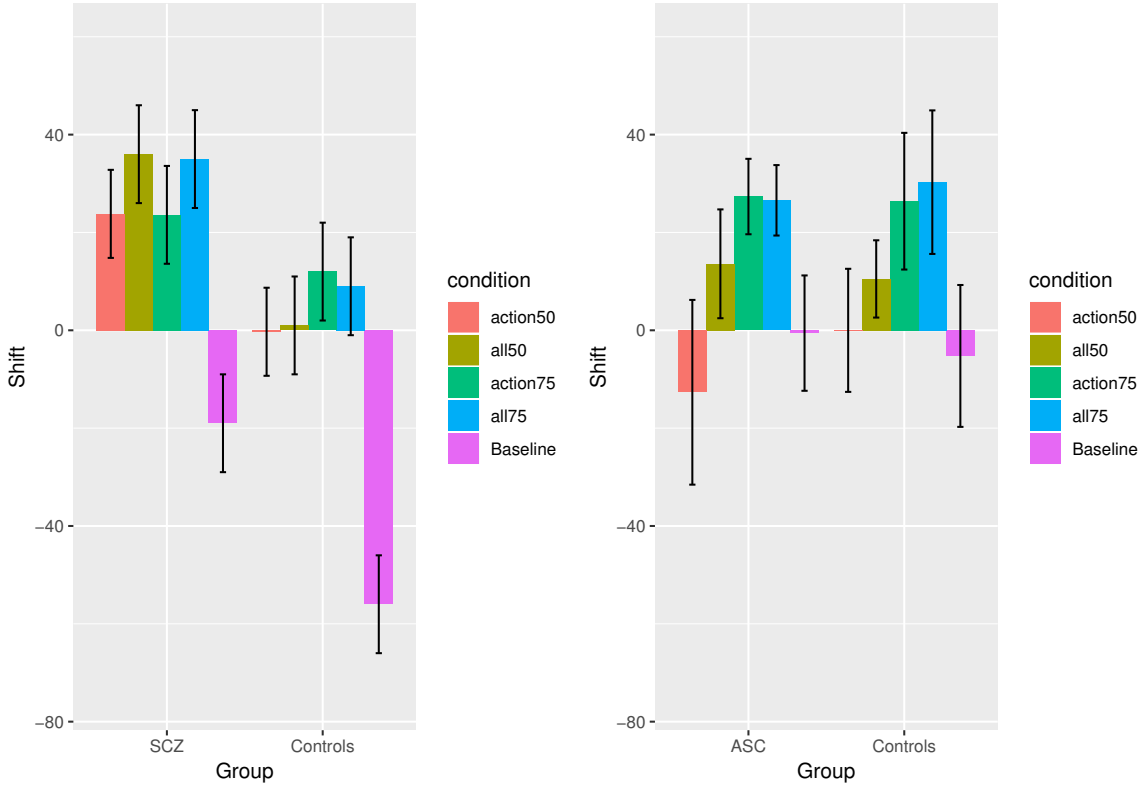

Figure A.1 Comparison of the IB data acquired in SCZ by Voss et al. [591] and the present study in autism. The control group in the data by Voss et al. do not seem to exhibit a significant binding effect and general IB was only demonstrated when all groups were collapsed. The SCZ group seems to differ from controls and ASC participants mainly in the increased binding for low probability action only trials.

Appendix B

Appendix Chapter 5

B.1 Survey Demographics

Table B.1 Survey Demographics

	N[1]	Gender			Complete	Age	Language		Education[2]					
		M	F	Other		mean/range	English	Other	Doctoral	Master	Bachelor	A-levels	GCSE	Other
Cohort 1 *(N=496)*	496	124	372	0	100%	23.34 (18-63)	365	131	2.4%	22.8%	24.6%	47.4%	0.4%	1%
Cohort 2 *(N=806)*	702	214	483	5	50%	24.31 (18-60)	536	256	-	-	-	-	-	-
ASC	15	6	8	1	80%	23.07 (19-27)	15	0	-	-	-	-	-	-
Controls *(suspected ASC: 5)*	644	193	449	2	52.5%	24.36 (18-60)	436	210	-	-	-	-	-	-
Missing	43	15	26	2	2.3%	22.67 (21-25)	85	46	-	-	-	-	-	-
Cohort 3 *(N=696)*	555	162	373	19	75.5%	34.97 (18-74)	496	59	6.80%	2.3%	12.8%	6.6%	71.6%	-
ASC	316	112	191	13	78.5%	39.22 (18-66)	283	33	13.3%	1.6%	14.2%	6.6%	64.2%	-
Controls *(suspected ASC: 52)*	239	50	182	6	71.6%	34.43 (18-74)	213	26	8.4%	4.2%	12.1%	9.6%	65.3%	-

[1] Number of surveys after excluding responses with <10% completion rate and duplicates

[2] For Cohort 3 this is based on binning years of education into 0-11 yrs, 11-13 yrs, 13-16 yrs, 16-17 yrs, 17-max yrs)

Table B.2 Reported Medical Conditions

	Anorexia Nervosa	ADHD	Anxiety	Bipolar Disorder	Bulimia	Depression	Dyslexia	Dyspraxia	Epilepsy	Language Delay	Learning Difficulties	OCD	Personality Disorders	Prospagnosia	Psychotic Disorders	PTSD	Tourette's
Cohort 1 *(N=496)*	-	-	-	-	-	54[1]	13	-	-	-	-	-	-	-	-	-	-
Cohort 3 *(N=767)*	27	74	67	24	15	288	37	40	20	18	24	57	38	2	13	9	4
ASC	20	55	46	16	11	202	30	34	11	16	21	44	28	2	11	7	4
Controls	8	19	21	8	4	86	7	6	9	2	3	13	10	0	2	2	0

[1] Based on asking about recent depressive symptoms rather than diagnosis.

B.2 Bootstrapped Edge Weight Estimates

Table B.3 ASC Group - AQ

Edge	Sample	Mean	SD	CI (lower)	CI (upper)	q2.5	q97.5A
AQ_1–AQ_10	0.00	0.00	0.01	-0.02	0.02	0.00	0.05
AQ_1–AQ_11	0.02	0.02	0.03	-0.04	0.07	0.00	0.09
AQ_1–AQ_13	0.05	0.05	0.04	-0.04	0.14	0.00	0.15
AQ_1–AQ_14	0.00	0.00	0.01	-0.01	0.01	0.00	0.01
AQ_1–AQ_15	0.07	0.06	0.05	-0.02	0.17	0.00	0.17
AQ_1–AQ_19	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_1–AQ_2	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_1–AQ_20	0.00	-0.00	0.00	-0.01	0.01	0.00	0.00
AQ_1–AQ_22	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_1–AQ_23	0.04	0.04	0.04	-0.03	0.12	0.00	0.13
AQ_1–AQ_25	0.04	0.03	0.04	-0.04	0.12	0.00	0.13
AQ_1–AQ_3	-0.02	-0.03	0.04	-0.10	0.06	-0.13	0.00
AQ_1–AQ_32	0.00	0.00	0.01	-0.03	0.03	0.00	0.05
AQ_1–AQ_34	0.01	0.02	0.03	-0.05	0.06	0.00	0.09
AQ_1–AQ_36	0.00	0.00	0.01	-0.01	0.01	0.00	0.01
AQ_1–AQ_37	0.09	0.07	0.05	-0.02	0.19	0.00	0.18
AQ_1–AQ_4	0.04	0.04	0.04	-0.05	0.13	0.00	0.15
AQ_1–AQ_41	0.00	-0.00	0.00	-0.01	0.01	0.00	0.00
AQ_1–AQ_42	0.00	0.02	0.03	-0.06	0.06	0.00	0.10
AQ_1–AQ_44	0.11	0.10	0.05	0.01	0.21	0.01	0.20
AQ_1–AQ_45	0.00	0.00	0.01	-0.01	0.01	0.00	0.01
AQ_1–AQ_46	0.04	0.03	0.03	-0.03	0.10	0.00	0.11
AQ_1–AQ_47	0.00	0.02	0.03	-0.05	0.05	0.00	0.09
AQ_1–AQ_50	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_1–AQ_6	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_1–AQ_8	0.00	-0.00	0.01	-0.02	0.02	-0.04	0.00
AQ_1–AQ_9	0.00	-0.02	0.03	-0.06	0.06	-0.10	0.00
AQ_10–AQ_11	0.17	0.15	0.05	0.07	0.27	0.06	0.26
AQ_10–AQ_13	0.03	0.03	0.04	-0.04	0.11	0.00	0.12
AQ_10–AQ_14	0.03	0.02	0.03	-0.03	0.08	0.00	0.09

Table B.3 Continued: ASC Group - AQ

AQ_10–AQ_15	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_10–AQ_19	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_10–AQ_20	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_10–AQ_22	0.16	0.15	0.05	0.05	0.27	0.04	0.26
AQ_10–AQ_23	0.01	0.02	0.03	-0.05	0.06	0.00	0.09
AQ_10–AQ_25	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_10–AQ_32	0.18	0.16	0.06	0.05	0.30	0.04	0.29
AQ_10–AQ_34	0.05	0.04	0.04	-0.03	0.13	0.00	0.13
AQ_10–AQ_36	0.09	0.07	0.05	-0.01	0.19	0.00	0.18
AQ_10–AQ_37	0.14	0.12	0.06	0.02	0.26	0.00	0.24
AQ_10–AQ_41	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_10–AQ_42	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_10–AQ_44	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_10–AQ_45	0.00	0.01	0.02	-0.04	0.04	0.00	0.08
AQ_10–AQ_46	0.04	0.04	0.04	-0.04	0.12	0.00	0.13
AQ_10–AQ_47	0.00	0.01	0.02	-0.04	0.05	0.00	0.08
AQ_10–AQ_50	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_11–AQ_13	0.10	0.09	0.05	-0.00	0.20	0.00	0.20
AQ_11–AQ_14	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_11–AQ_15	0.03	0.03	0.04	-0.04	0.10	0.00	0.12
AQ_11–AQ_19	0.01	0.02	0.03	-0.04	0.06	0.00	0.09
AQ_11–AQ_20	0.00	0.01	0.03	-0.06	0.06	0.00	0.10
AQ_11–AQ_22	0.21	0.21	0.07	0.08	0.34	0.08	0.33
AQ_11–AQ_23	0.07	0.06	0.04	-0.01	0.16	0.00	0.15
AQ_11–AQ_25	0.02	0.03	0.03	-0.04	0.09	0.00	0.11
AQ_11–AQ_32	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_11–AQ_34	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_11–AQ_36	0.04	0.04	0.04	-0.04	0.12	0.00	0.13
AQ_11–AQ_37	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_11–AQ_41	0.00	0.00	0.01	-0.01	0.01	0.00	0.01
AQ_11–AQ_42	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_11–AQ_44	0.11	0.11	0.05	0.02	0.21	0.02	0.20
AQ_11–AQ_45	0.01	0.02	0.03	-0.04	0.07	0.00	0.09

Table B.3 Continued: ASC Group - AQ

AQ_11–AQ_46	0.14	0.14	0.05	0.03	0.24	0.04	0.25
AQ_11–AQ_47	0.03	0.02	0.03	-0.03	0.09	0.00	0.10
AQ_11–AQ_50	0.15	0.14	0.06	0.04	0.27	0.03	0.25
AQ_13–AQ_14	0.00	0.01	0.01	-0.03	0.03	0.00	0.05
AQ_13–AQ_15	0.00	0.01	0.02	-0.04	0.04	0.00	0.08
AQ_13–AQ_19	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_13–AQ_20	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_13–AQ_22	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_13–AQ_23	0.04	0.03	0.04	-0.04	0.12	0.00	0.13
AQ_13–AQ_25	0.02	0.02	0.03	-0.05	0.09	0.00	0.11
AQ_13–AQ_32	0.00	0.01	0.02	-0.04	0.05	0.00	0.08
AQ_13–AQ_34	0.00	0.00	0.01	-0.02	0.02	0.00	0.05
AQ_13–AQ_36	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_13–AQ_37	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_13–AQ_41	0.06	0.05	0.04	-0.03	0.15	0.00	0.15
AQ_13–AQ_42	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_13–AQ_44	0.19	0.18	0.06	0.08	0.31	0.07	0.29
AQ_13–AQ_45	0.00	0.01	0.01	-0.03	0.03	0.00	0.05
AQ_13–AQ_46	0.07	0.07	0.05	-0.03	0.17	0.00	0.17
AQ_13–AQ_47	0.00	0.01	0.02	-0.04	0.04	0.00	0.08
AQ_13–AQ_50	0.00	0.00	0.01	-0.03	0.03	0.00	0.05
AQ_14–AQ_15	0.03	0.03	0.03	-0.03	0.09	0.00	0.10
AQ_14–AQ_19	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_14–AQ_20	0.04	0.03	0.03	-0.03	0.11	0.00	0.11
AQ_14–AQ_22	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_14–AQ_23	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_14–AQ_25	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_14–AQ_32	0.03	0.03	0.03	-0.03	0.10	0.00	0.11
AQ_14–AQ_34	0.00	0.01	0.02	-0.04	0.05	0.00	0.09
AQ_14–AQ_36	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_14–AQ_37	0.02	0.02	0.03	-0.04	0.08	0.00	0.11
AQ_14–AQ_41	0.00	-0.02	0.04	-0.07	0.07	-0.12	0.00
AQ_14–AQ_42	0.00	0.01	0.02	-0.03	0.03	0.00	0.06

Table B.3 Continued: ASC Group - AQ

AQ_14–AQ_44	0.00	0.00	0.01	-0.02	0.02	0.00	0.02
AQ_14–AQ_45	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_14–AQ_46	0.01	0.01	0.02	-0.03	0.04	0.00	0.07
AQ_14–AQ_47	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_14–AQ_50	0.18	0.17	0.05	0.07	0.29	0.06	0.28
AQ_15–AQ_19	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_15–AQ_20	0.00	0.00	0.01	-0.02	0.02	0.00	0.05
AQ_15–AQ_22	0.07	0.05	0.04	-0.02	0.15	0.00	0.15
AQ_15–AQ_23	0.00	0.01	0.02	-0.05	0.05	0.00	0.08
AQ_15–AQ_25	0.03	0.04	0.04	-0.05	0.12	0.00	0.14
AQ_15–AQ_32	0.01	0.02	0.03	-0.05	0.07	0.00	0.10
AQ_15–AQ_34	0.08	0.07	0.05	-0.02	0.18	0.00	0.17
AQ_15–AQ_36	0.10	0.09	0.05	-0.01	0.21	0.00	0.20
AQ_15–AQ_37	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_15–AQ_41	0.00	0.00	0.01	-0.02	0.02	0.00	0.05
AQ_15–AQ_42	0.07	0.06	0.05	-0.03	0.17	0.00	0.17
AQ_15–AQ_44	0.21	0.20	0.05	0.10	0.32	0.10	0.31
AQ_15–AQ_45	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_15–AQ_46	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_15–AQ_47	0.09	0.09	0.05	-0.01	0.20	0.00	0.19
AQ_15–AQ_50	0.10	0.09	0.05	-0.00	0.21	0.00	0.20
AQ_19–AQ_20	0.01	0.01	0.02	-0.04	0.05	0.00	0.08
AQ_19–AQ_22	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_19–AQ_23	0.16	0.15	0.05	0.07	0.26	0.06	0.25
AQ_19–AQ_25	0.00	0.00	0.01	-0.01	0.01	0.00	0.01
AQ_19–AQ_32	0.00	-0.01	0.02	-0.04	0.04	-0.07	0.00
AQ_19–AQ_34	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_19–AQ_36	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_19–AQ_37	0.00	-0.00	0.01	-0.01	0.01	0.00	0.00
AQ_19–AQ_41	0.03	0.04	0.04	-0.05	0.12	0.00	0.14
AQ_19–AQ_42	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_19–AQ_44	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_19–AQ_45	0.00	-0.00	0.01	-0.01	0.01	-0.02	0.00

Table B.3 Continued: ASC Group - AQ

AQ_19–AQ_46	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_19–AQ_47	0.00	0.00	0.01	-0.01	0.01	0.00	0.03
AQ_19–AQ_50	0.03	0.03	0.04	-0.04	0.10	0.00	0.11
AQ_2–AQ_10	0.04	0.04	0.04	-0.04	0.12	0.00	0.14
AQ_2–AQ_11	0.01	0.02	0.03	-0.05	0.07	0.00	0.10
AQ_2–AQ_13	0.05	0.04	0.04	-0.03	0.13	0.00	0.14
AQ_2–AQ_14	0.02	0.02	0.03	-0.04	0.07	0.00	0.10
AQ_2–AQ_15	0.00	0.01	0.02	-0.05	0.05	0.00	0.08
AQ_2–AQ_19	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_2–AQ_20	0.00	0.01	0.02	-0.04	0.04	0.00	0.08
AQ_2–AQ_22	0.08	0.07	0.06	-0.03	0.19	0.00	0.19
AQ_2–AQ_23	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_2–AQ_25	0.05	0.04	0.04	-0.04	0.13	0.00	0.14
AQ_2–AQ_3	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_2–AQ_32	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_2–AQ_34	0.10	0.08	0.05	0.00	0.20	0.00	0.19
AQ_2–AQ_36	0.08	0.07	0.06	-0.04	0.19	0.00	0.19
AQ_2–AQ_37	0.01	0.02	0.03	-0.05	0.07	0.00	0.10
AQ_2–AQ_4	0.11	0.09	0.05	0.01	0.20	0.00	0.19
AQ_2–AQ_41	0.00	0.01	0.02	-0.05	0.05	0.00	0.08
AQ_2–AQ_42	0.04	0.04	0.04	-0.04	0.12	0.00	0.13
AQ_2–AQ_44	0.00	0.02	0.03	-0.05	0.06	0.00	0.09
AQ_2–AQ_45	0.04	0.03	0.04	-0.03	0.12	0.00	0.12
AQ_2–AQ_46	0.16	0.15	0.05	0.05	0.26	0.05	0.25
AQ_2–AQ_47	0.00	0.01	0.02	-0.04	0.04	0.00	0.06
AQ_2–AQ_50	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_2–AQ_6	0.02	0.02	0.03	-0.04	0.07	0.00	0.09
AQ_2–AQ_8	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_2–AQ_9	0.00	-0.00	0.01	-0.01	0.01	0.00	0.00
AQ_20–AQ_22	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_20–AQ_23	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_20–AQ_25	0.02	0.02	0.03	-0.04	0.09	0.00	0.10
AQ_20–AQ_32	0.03	0.03	0.04	-0.04	0.11	0.00	0.12

Table B.3 Continued: ASC Group - AQ

AQ_20–AQ_34	0.05	0.03	0.04	-0.02	0.12	0.00	0.12
AQ_20–AQ_36	0.05	0.04	0.04	-0.03	0.12	0.00	0.13
AQ_20–AQ_37	0.01	0.01	0.02	-0.04	0.05	0.00	0.08
AQ_20–AQ_41	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_20–AQ_42	0.13	0.12	0.05	0.03	0.23	0.02	0.22
AQ_20–AQ_44	0.01	0.01	0.02	-0.03	0.04	0.00	0.07
AQ_20–AQ_45 5	0.18	0.17	0.06	0.07	0.30	0.06	0.28
AQ_20–AQ_46	0.00	0.00	0.01	-0.03	0.03	0.00	0.05
AQ_20–AQ_47	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_20–AQ_50	0.01	0.02	0.03	-0.05	0.08	0.00	0.11
AQ_22–AQ_23	0.03	0.03	0.03	-0.04	0.09	0.00	0.11
AQ_22–AQ_25	0.04	0.04	0.05	-0.06	0.13	0.00	0.16
AQ_22–AQ_32	0.01	0.02	0.03	-0.05	0.08	0.00	0.11
AQ_22–AQ_34	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_22–AQ_36	0.03	0.04	0.04	-0.05	0.11	0.00	0.14
AQ_22–AQ_37	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_22–AQ_41	0.01	0.02	0.03	-0.04	0.06	0.00	0.09
AQ_22–AQ_42	0.00	0.02	0.03	-0.06	0.06	0.00	0.10
AQ_22–AQ_44	0.02	0.03	0.03	-0.04	0.09	0.00	0.11
AQ_22–AQ_45	0.09	0.07	0.04	0.00	0.18	0.00	0.16
AQ_22–AQ_46	0.22	0.21	0.07	0.09	0.36	0.08	0.34
AQ_22–AQ_47	0.01	0.02	0.03	-0.04	0.06	0.00	0.09
AQ_22–AQ_50	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_23–AQ_25	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_23–AQ_32	0.00	0.00	0.01	-0.03	0.03	0.00	0.05
AQ_23–AQ_34	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_23–AQ_36	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_23–AQ_37	0.06	0.06	0.05	-0.03	0.15	0.00	0.16
AQ_23–AQ_41	0.08	0.07	0.05	-0.02	0.18	0.00	0.18
AQ_23–AQ_42	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_23–AQ_44	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_23–AQ_45	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_23–AQ_46	0.05	0.03	0.04	-0.03	0.12	0.00	0.12

Table B.3 Continued: ASC Group - AQ

AQ_23–AQ_47	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_23–AQ_50	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_25–AQ_32	0.12	0.11	0.06	-0.00	0.25	0.00	0.24
AQ_25–AQ_34	0.15	0.14	0.05	0.04	0.26	0.03	0.25
AQ_25–AQ_36	0.05	0.05	0.04	-0.04	0.14	0.00	0.15
AQ_25–AQ_37	0.03	0.03	0.04	-0.05	0.10	0.00	0.12
AQ_25–AQ_41	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_25–AQ_42	0.00	0.00	0.01	-0.02	0.02	0.00	0.02
AQ_25–AQ_44	0.09	0.08	0.05	-0.01	0.20	0.00	0.19
AQ_25–AQ_45	0.03	0.03	0.04	-0.04	0.10	0.00	0.12
AQ_25–AQ_46	0.14	0.13	0.05	0.04	0.25	0.03	0.24
AQ_25–AQ_47	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_25–AQ_50	0.00	0.00	0.01	-0.03	0.03	0.00	0.05
AQ_3–AQ_10	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_3–AQ_11	0.00	-0.02	0.03	-0.05	0.05	-0.08	0.00
AQ_3–AQ_13	0.00	-0.01	0.02	-0.04	0.04	-0.07	0.00
AQ_3–AQ_14	0.08	0.08	0.05	-0.02	0.19	0.00	0.19
AQ_3–AQ_15	0.00	-0.00	0.00	-0.01	0.01	0.00	0.00
AQ_3–AQ_19	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_3–AQ_20	0.00	0.01	0.02	-0.05	0.05	0.00	0.08
AQ_3–AQ_22	0.00	-0.00	0.00	-0.01	0.01	0.00	0.00
AQ_3–AQ_23	0.00	-0.00	0.00	-0.01	0.01	-0.01	0.00
AQ_3–AQ_25	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_3–AQ_32	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_3–AQ_34	0.01	0.03	0.03	-0.05	0.08	0.00	0.10
AQ_3–AQ_36	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_3–AQ_37	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_3–AQ_4	0.00	-0.03	0.03	-0.07	0.07	-0.11	0.00
AQ_3–AQ_41	0.00	0.00	0.01	-0.03	0.03	0.00	0.05
AQ_3–AQ_42	0.00	0.01	0.02	-0.05	0.05	0.00	0.08
AQ_3–AQ_44	0.00	-0.00	0.01	-0.02	0.02	-0.04	0.00
AQ_3–AQ_45	0.00	-0.00	0.01	-0.02	0.02	-0.04	0.00
AQ_3–AQ_46	0.00	0.00	0.01	-0.01	0.01	0.00	0.01

Table B.3 Continued: ASC Group - AQ

AQ_3–AQ_47	0.00	0.02	0.03	-0.05	0.06	0.00	0.09
AQ_3–AQ_50	0.00	0.02	0.03	-0.05	0.05	0.00	0.10
AQ_3–AQ_6	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_3–AQ_8	0.39	0.39	0.05	0.30	0.49	0.29	0.48
AQ_3–AQ_9	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_32–AQ_34	0.12	0.11	0.05	0.02	0.22	0.00	0.21
AQ_32–AQ_36	0.00	0.02	0.03	-0.05	0.06	0.00	0.09
AQ_32–AQ_37	0.25	0.24	0.06	0.13	0.38	0.12	0.36
AQ_32–AQ_41	0.03	0.04	0.04	-0.05	0.12	0.00	0.13
AQ_32–AQ_42	0.02	0.02	0.03	-0.03	0.08	0.00	0.10
AQ_32–AQ_44	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_32–AQ_45	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_32–AQ_46	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_32–AQ_47	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_32–AQ_50	0.00	0.01	0.03	-0.05	0.05	0.00	0.09
AQ_34–AQ_36	0.00	0.01	0.02	-0.04	0.04	0.00	0.06
AQ_34–AQ_37	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_34–AQ_41	0.04	0.04	0.04	-0.04	0.13	0.00	0.14
AQ_34–AQ_42	0.02	0.02	0.03	-0.04	0.09	0.00	0.11
AQ_34–AQ_44	0.07	0.06	0.04	-0.02	0.16	0.00	0.16
AQ_34–AQ_45	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_34–AQ_46	0.02	0.03	0.04	-0.05	0.09	0.00	0.12
AQ_34–AQ_47	0.15	0.14	0.06	0.04	0.27	0.02	0.25
AQ_34–AQ_50	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_36–AQ_37	0.03	0.03	0.04	-0.04	0.10	0.00	0.11
AQ_36–AQ_41	0.00	0.01	0.02	-0.05	0.05	0.00	0.08
AQ_36–AQ_42	0.13	0.12	0.06	0.01	0.24	0.01	0.23
AQ_36–AQ_44	0.04	0.03	0.04	-0.04	0.11	0.00	0.12
AQ_36–AQ_45	0.11	0.10	0.05	0.01	0.21	0.00	0.21
AQ_36–AQ_46	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_36–AQ_47	0.00	0.00	0.01	-0.01	0.01	0.00	0.01
AQ_36–AQ_50	0.03	0.03	0.04	-0.04	0.11	0.00	0.13
AQ_37–AQ_41	0.00	0.00	0.01	-0.01	0.01	0.00	0.02

Table B.3 Continued: ASC Group - AQ

AQ_37–AQ_42	0.00	0.00	0.01	-0.03	0.03	0.00	0.05
AQ_37–AQ_44	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_37–AQ_45	0.00	0.00	0.01	-0.03	0.03	0.00	0.05
AQ_37–AQ_46	0.02	0.02	0.03	-0.04	0.08	0.00	0.10
AQ_37–AQ_47	0.08	0.07	0.05	-0.02	0.17	0.00	0.16
AQ_37–AQ_50	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_4–AQ_10	0.00	0.02	0.03	-0.06	0.06	0.00	0.10
AQ_4–AQ_11	0.07	0.06	0.05	-0.03	0.16	0.00	0.17
AQ_4–AQ_13	0.15	0.13	0.06	0.03	0.27	0.01	0.25
AQ_4–AQ_14	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_4–AQ_15	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_4–AQ_19	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_4–AQ_20	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_4–AQ_22	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_4–AQ_23	0.19	0.18	0.05	0.09	0.30	0.08	0.28
AQ_4–AQ_25	0.01	0.02	0.03	-0.06	0.08	0.00	0.12
AQ_4–AQ_32	0.00	0.00	0.01	-0.02	0.02	0.00	0.05
AQ_4–AQ_34	0.00	-0.00	0.00	-0.01	0.01	0.00	0.00
AQ_4–AQ_36	0.07	0.07	0.04	-0.02	0.16	0.00	0.16
AQ_4–AQ_37	0.06	0.05	0.04	-0.03	0.14	0.00	0.14
AQ_4–AQ_41	0.03	0.03	0.04	-0.04	0.10	0.00	0.12
AQ_4–AQ_42	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_4–AQ_44	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_4–AQ_45	0.05	0.04	0.04	-0.03	0.12	0.00	0.13
AQ_4–AQ_46	0.16	0.16	0.06	0.04	0.28	0.04	0.27
AQ_4–AQ_47	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_4–AQ_50	0.00	0.01	0.02	-0.04	0.04	0.00	0.08
AQ_4–AQ_6	0.04	0.04	0.04	-0.03	0.11	0.00	0.12
AQ_4–AQ_8	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_4–AQ_9	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_41–AQ_42	0.02	0.02	0.03	-0.04	0.08	0.00	0.10
AQ_41–AQ_44	0.00	-0.00	0.01	-0.02	0.02	-0.02	0.00
AQ_41–AQ_45	0.00	0.00	0.01	-0.02	0.02	0.00	0.03

Table B.3 Continued: ASC Group - AQ

AQ_41–AQ_46	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_41–AQ_47	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_41–AQ_50	0.00	0.01	0.03	-0.05	0.05	0.00	0.09
AQ_42–AQ_44	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_42–AQ_45	0.27	0.26	0.06	0.14	0.39	0.14	0.38
AQ_42–AQ_46	0.12	0.09	0.05	0.02	0.21	0.00	0.18
AQ_42–AQ_47	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_42–AQ_50	0.00	0.00	0.01	-0.03	0.03	0.00	0.05
AQ_44–AQ_45	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_44–AQ_46	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_44–AQ_47	0.27	0.26	0.05	0.16	0.38	0.16	0.37
AQ_44–AQ_50	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_45–AQ_46	0.02	0.02	0.03	-0.04	0.08	0.00	0.10
AQ_45–AQ_47	0.00	0.01	0.03	-0.05	0.05	0.00	0.09
AQ_45–AQ_50	0.00	-0.00	0.01	-0.01	0.01	-0.01	0.00
AQ_46–AQ_47	0.11	0.09	0.05	0.01	0.20	0.00	0.19
AQ_46–AQ_50	0.00	-0.00	0.01	-0.01	0.01	-0.02	0.00
AQ_47–AQ_50	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_6–AQ_10	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_6–AQ_11	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_6–AQ_13	0.00	0.01	0.02	-0.05	0.05	0.00	0.09
AQ_6–AQ_14	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_6–AQ_15	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_6–AQ_19	0.18	0.18	0.05	0.08	0.29	0.08	0.28
AQ_6–AQ_20	0.02	0.02	0.03	-0.04	0.08	0.00	0.10
AQ_6–AQ_22	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_6–AQ_23	0.30	0.29	0.06	0.18	0.41	0.17	0.40
AQ_6–AQ_25	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_6–AQ_32	0.00	-0.00	0.01	-0.01	0.01	-0.02	0.00
AQ_6–AQ_34	0.02	0.02	0.03	-0.04	0.09	0.00	0.11
AQ_6–AQ_36	0.00	-0.00	0.00	-0.01	0.01	0.00	0.00
AQ_6–AQ_37	0.00	-0.00	0.01	-0.01	0.01	0.00	0.00
AQ_6–AQ_41	0.05	0.05	0.04	-0.04	0.14	0.00	0.15

Table B.3 Continued: ASC Group - AQ

AQ_6–AQ_42	0.00	0.00	0.01	-0.03	0.03	0.00	0.05
AQ_6–AQ_44	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_6–AQ_45	0.05	0.04	0.04	-0.03	0.12	0.00	0.13
AQ_6–AQ_46	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_6–AQ_47	0.03	0.03	0.04	-0.04	0.10	0.00	0.12
AQ_6–AQ_50	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_6–AQ_8	0.00	-0.00	0.00	-0.01	0.01	-0.00	0.00
AQ_6–AQ_9	0.16	0.16	0.05	0.06	0.26	0.06	0.26
AQ_8–AQ_10	0.00	0.01	0.02	-0.04	0.04	0.00	0.07
AQ_8–AQ_11	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_8–AQ_13	0.00	-0.00	0.01	-0.01	0.01	-0.01	0.00
AQ_8–AQ_14	0.28	0.27	0.05	0.17	0.38	0.17	0.38
AQ_8–AQ_15	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_8–AQ_19	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_8–AQ_20	0.17	0.16	0.05	0.08	0.26	0.06	0.25
AQ_8–AQ_22	0.00	-0.00	0.01	-0.01	0.01	0.00	0.00
AQ_8–AQ_23	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_8–AQ_25	0.00	-0.00	0.00	-0.01	0.01	0.00	0.00
AQ_8–AQ_32	0.04	0.04	0.04	-0.03	0.11	0.00	0.12
AQ_8–AQ_34	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_8–AQ_36	0.05	0.05	0.04	-0.03	0.13	0.00	0.14
AQ_8–AQ_37	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_8–AQ_41	0.00	0.01	0.02	-0.03	0.03	0.00	0.06
AQ_8–AQ_42	0.05	0.05	0.04	-0.02	0.13	0.00	0.13
AQ_8–AQ_44	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_8–AQ_45	0.00	-0.00	0.01	-0.02	0.02	-0.05	0.00
AQ_8–AQ_46	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_8–AQ_47	0.00	-0.00	0.01	-0.01	0.01	0.00	0.00
AQ_8–AQ_50	0.11	0.10	0.05	0.01	0.21	0.00	0.20
AQ_8–AQ_9	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_9–AQ_10	-0.02	-0.03	0.04	-0.09	0.05	-0.12	0.00
AQ_9–AQ_11	0.00	-0.01	0.02	-0.05	0.05	-0.08	0.00
AQ_9–AQ_13	0.00	-0.00	0.00	-0.01	0.01	0.00	0.00

Table B.3 Continued: ASC Group - AQ

AQ_9–AQ_14	0.00	-0.00	0.01	-0.02	0.02	-0.05	0.00
AQ_9–AQ_15	0.00	0.00	0.01	-0.02	0.02	0.00	0.02
AQ_9–AQ_19	0.28	0.28	0.06	0.17	0.39	0.17	0.38
AQ_9–AQ_20	0.04	0.05	0.04	-0.04	0.13	0.00	0.14
AQ_9–AQ_22	0.00	-0.00	0.01	-0.01	0.01	-0.01	0.00
AQ_9–AQ_23	0.00	0.00	0.01	-0.03	0.03	0.00	0.05
AQ_9–AQ_25	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_9–AQ_32	0.00	-0.00	0.01	-0.02	0.02	-0.04	0.00
AQ_9–AQ_34	0.00	-0.00	0.00	-0.01	0.01	0.00	0.00
AQ_9–AQ_36	0.00	-0.00	0.00	-0.01	0.01	-0.00	0.00
AQ_9–AQ_37	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_9–AQ_41	0.15	0.15	0.05	0.04	0.26	0.04	0.25
AQ_9–AQ_42	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_9–AQ_44	0.00	-0.00	0.01	-0.01	0.01	-0.02	0.00
AQ_9–AQ_45	0.00	-0.00	0.00	-0.01	0.01	0.00	0.00
AQ_9–AQ_46	0.00	-0.00	0.00	-0.01	0.01	0.00	0.00
AQ_9–AQ_47	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_9–AQ_50	0.00	0.00	0.01	-0.02	0.02	0.00	0.03

Table B.4 Control Group - AQ

Edge	Sample	Mean	SD	CI (lower)	CI (upper)	q2.5	q97.5A
AQ_1–AQ_10	0.02	0.02	0.02	-0.02	0.05	0.00	0.06
AQ_1–AQ_11	0.10	0.10	0.02	0.05	0.15	0.05	0.14
AQ_1–AQ_13	0.16	0.16	0.03	0.11	0.21	0.11	0.21
AQ_1–AQ_14	0.00	-0.01	0.01	-0.02	0.02	-0.04	0.00
AQ_1–AQ_15	0.14	0.13	0.03	0.09	0.19	0.08	0.18
AQ_1–AQ_19	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_1–AQ_2	0.00	0.01	0.01	-0.02	0.02	0.00	0.04
AQ_1–AQ_20	-0.06	-0.05	0.03	-0.12	-0.01	-0.11	0.00
AQ_1–AQ_22	0.00	0.01	0.01	-0.02	0.02	0.00	0.04
AQ_1–AQ_23	0.05	0.04	0.02	0.01	0.10	0.00	0.09
AQ_1–AQ_25	0.07	0.06	0.03	0.01	0.12	0.00	0.11

Table B.4 Continued: Control Group - AQ

AQ_1–AQ_3	-0.06	-0.04	0.02	-0.10	-0.01	-0.08	0.00
AQ_1–AQ_32	0.00	-0.00	0.00	-0.00	0.00	-0.00	0.00
AQ_1–AQ_34	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_1–AQ_36	0.00	-0.00	0.00	-0.01	0.01	-0.01	0.00
AQ_1–AQ_37	0.00	-0.00	0.01	-0.01	0.01	-0.02	0.00
AQ_1–AQ_4	0.03	0.03	0.02	-0.01	0.08	0.00	0.08
AQ_1–AQ_41	0.00	-0.00	0.00	-0.01	0.01	-0.01	0.00
AQ_1–AQ_42	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_1–AQ_44	0.08	0.08	0.02	0.04	0.13	0.03	0.12
AQ_1–AQ_45	0.00	-0.00	0.00	-0.01	0.01	-0.01	0.00
AQ_1–AQ_46	0.01	0.02	0.02	-0.02	0.05	0.00	0.06
AQ_1–AQ_47	0.00	0.01	0.01	-0.03	0.03	0.00	0.04
AQ_1–AQ_50	0.01	0.01	0.02	-0.02	0.05	0.00	0.05
AQ_1–AQ_6	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_1–AQ_8	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_1–AQ_9	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_10–AQ_11	0.17	0.16	0.02	0.12	0.22	0.12	0.21
AQ_10–AQ_13	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_10–AQ_14	0.11	0.10	0.02	0.06	0.16	0.06	0.15
AQ_10–AQ_15	0.00	0.01	0.02	-0.03	0.03	0.00	0.05
AQ_10–AQ_19	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_10–AQ_20	0.00	-0.00	0.00	-0.01	0.01	-0.02	0.00
AQ_10–AQ_22	0.00	-0.00	0.00	-0.01	0.01	-0.01	0.00
AQ_10–AQ_23	0.00	-0.00	0.01	-0.01	0.01	-0.02	0.00
AQ_10–AQ_25	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_10–AQ_32	0.14	0.14	0.03	0.09	0.20	0.09	0.19
AQ_10–AQ_34	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_10–AQ_36	0.11	0.10	0.02	0.06	0.15	0.05	0.15
AQ_10–AQ_37	0.16	0.16	0.03	0.11	0.21	0.11	0.21
AQ_10–AQ_41	0.00	-0.00	0.01	-0.01	0.01	-0.02	0.00
AQ_10–AQ_42	0.00	0.00	0.00	-0.00	0.00	0.00	0.01
AQ_10–AQ_44	0.01	0.01	0.02	-0.02	0.04	0.00	0.05
AQ_10–AQ_45	0.06	0.06	0.03	0.01	0.11	0.01	0.11

Table B.4 Continued: Control Group - AQ

AQ_10–AQ_46	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_10–AQ_47	0.01	0.01	0.02	-0.02	0.04	0.00	0.05
AQ_10–AQ_50	0.01	0.02	0.02	-0.02	0.05	0.00	0.06
AQ_11–AQ_13	0.08	0.08	0.02	0.04	0.13	0.03	0.13
AQ_11–AQ_14	0.02	0.01	0.02	-0.02	0.05	0.00	0.05
AQ_11–AQ_15	0.02	0.02	0.02	-0.02	0.06	0.00	0.06
AQ_11–AQ_19	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_11–AQ_20	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_11–AQ_22	0.10	0.09	0.02	0.05	0.15	0.04	0.14
AQ_11–AQ_23	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_11–AQ_25	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_11–AQ_32	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_11–AQ_34	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_11–AQ_36	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_11–AQ_37	0.01	0.01	0.01	-0.02	0.04	0.00	0.05
AQ_11–AQ_41	0.00	-0.00	0.00	-0.01	0.01	-0.00	0.00
AQ_11–AQ_42	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_11–AQ_44	0.18	0.18	0.02	0.14	0.23	0.13	0.22
AQ_11–AQ_45	0.05	0.04	0.02	0.01	0.09	0.00	0.09
AQ_11–AQ_46	0.36	0.35	0.02	0.32	0.41	0.31	0.40
AQ_11–AQ_47	0.14	0.13	0.02	0.09	0.18	0.09	0.18
AQ_11–AQ_50	0.01	0.01	0.01	-0.02	0.04	0.00	0.05
AQ_13–AQ_14	-0.03	-0.02	0.02	-0.06	0.01	-0.06	0.00
AQ_13–AQ_15	0.04	0.04	0.02	-0.01	0.09	0.00	0.09
AQ_13–AQ_19	0.00	-0.00	0.01	-0.01	0.01	-0.02	0.00
AQ_13–AQ_20	-0.02	-0.02	0.02	-0.06	0.02	-0.06	0.00
AQ_13–AQ_22	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_13–AQ_23	0.00	0.00	0.00	-0.01	0.01	0.00	0.02
AQ_13–AQ_25	0.02	0.02	0.02	-0.02	0.05	0.00	0.06
AQ_13–AQ_32	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_13–AQ_34	0.06	0.06	0.02	0.01	0.11	0.01	0.11
AQ_13–AQ_36	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_13–AQ_37	0.00	-0.00	0.01	-0.01	0.01	-0.02	0.00

Table B.4 Continued: Control Group - AQ

AQ_13–AQ_41	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_13–AQ_42	-0.04	-0.03	0.02	-0.09	0.00	-0.07	0.00
AQ_13–AQ_44	0.28	0.28	0.02	0.24	0.32	0.23	0.32
AQ_13–AQ_45	0.01	0.01	0.01	-0.02	0.04	0.00	0.05
AQ_13–AQ_46	0.00	0.00	0.01	-0.01	0.01	0.00	0.03
AQ_13–AQ_47	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_13–AQ_50	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_14–AQ_15	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_14–AQ_19	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_14–AQ_20	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_14–AQ_22	0.00	-0.00	0.00	-0.01	0.01	-0.01	0.00
AQ_14–AQ_23	0.00	-0.00	0.01	-0.01	0.01	-0.03	0.00
AQ_14–AQ_25	0.00	0.00	0.01	-0.01	0.01	0.00	0.03
AQ_14–AQ_32	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_14–AQ_34	0.05	0.04	0.02	0.01	0.10	0.00	0.09
AQ_14–AQ_36	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_14–AQ_37	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_14–AQ_41	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_14–AQ_42	0.06	0.06	0.02	0.01	0.11	0.01	0.10
AQ_14–AQ_44	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_14–AQ_45	0.00	0.00	0.01	-0.01	0.01	0.00	0.03
AQ_14–AQ_46	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_14–AQ_47	0.00	0.01	0.01	-0.02	0.02	0.00	0.04
AQ_14–AQ_50	0.29	0.28	0.03	0.24	0.34	0.23	0.33
AQ_15–AQ_19	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_15–AQ_20	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_15–AQ_22	0.03	0.02	0.02	-0.01	0.07	0.00	0.07
AQ_15–AQ_23	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_15–AQ_25	0.00	0.01	0.01	-0.02	0.03	0.00	0.04
AQ_15–AQ_32	0.00	0.00	0.00	-0.01	0.01	0.00	0.02
AQ_15–AQ_34	0.09	0.09	0.03	0.04	0.14	0.04	0.14
AQ_15–AQ_36	0.11	0.11	0.02	0.06	0.16	0.06	0.15
AQ_15–AQ_37	0.00	0.00	0.00	-0.01	0.01	0.00	0.02

Table B.4 Continued: Control Group - AQ

AQ_15–AQ_41	0.08	0.08	0.02	0.03	0.13	0.03	0.13
AQ_15–AQ_42	0.03	0.02	0.02	-0.02	0.07	0.00	0.07
AQ_15–AQ_44	0.17	0.17	0.02	0.12	0.21	0.12	0.21
AQ_15–AQ_45	0.03	0.03	0.02	-0.02	0.07	0.00	0.08
AQ_15–AQ_46	0.00	-0.00	0.00	-0.01	0.01	-0.01	0.00
AQ_15–AQ_47	0.14	0.14	0.03	0.08	0.19	0.09	0.19
AQ_15–AQ_50	0.04	0.03	0.02	-0.01	0.09	0.00	0.08
AQ_19–AQ_20	0.19	0.18	0.03	0.14	0.24	0.13	0.23
AQ_19–AQ_22	0.03	0.03	0.02	-0.01	0.08	0.00	0.08
AQ_19–AQ_23	0.32	0.31	0.03	0.27	0.38	0.25	0.36
AQ_19–AQ_25	0.00	-0.00	0.01	-0.01	0.01	-0.02	0.00
AQ_19–AQ_32	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_19–AQ_34	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_19–AQ_36	0.00	0.00	0.00	-0.01	0.01	0.00	0.02
AQ_19–AQ_37	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_19–AQ_41	0.02	0.02	0.02	-0.02	0.06	0.00	0.06
AQ_19–AQ_42	0.00	-0.00	0.00	-0.01	0.01	-0.01	0.00
AQ_19–AQ_44	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_19–AQ_45	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_19–AQ_46	-0.03	-0.03	0.02	-0.07	0.01	-0.07	0.00
AQ_19–AQ_47	0.00	-0.00	0.00	-0.01	0.01	-0.01	0.00
AQ_19–AQ_50	0.00	0.01	0.01	-0.02	0.03	0.00	0.04
AQ_2–AQ_10	0.00	-0.00	0.00	-0.01	0.01	-0.02	0.00
AQ_2–AQ_11	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_2–AQ_13	0.00	-0.00	0.00	-0.01	0.01	-0.00	0.00
AQ_2–AQ_14	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_2–AQ_15	0.00	0.01	0.01	-0.02	0.02	0.00	0.04
AQ_2–AQ_19	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_2–AQ_20	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_2–AQ_22	0.00	0.01	0.01	-0.02	0.03	0.00	0.05
AQ_2–AQ_23	0.02	0.01	0.02	-0.02	0.05	0.00	0.06
AQ_2–AQ_25	0.22	0.22	0.03	0.17	0.27	0.16	0.27
AQ_2–AQ_3	0.00	-0.00	0.00	-0.01	0.01	-0.02	0.00

Table B.4 Continued: Control Group - AQ

AQ_2–AQ_32	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_2–AQ_34	0.12	0.11	0.02	0.07	0.17	0.07	0.16
AQ_2–AQ_36	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_2–AQ_37	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_2–AQ_4	0.07	0.07	0.03	0.02	0.13	0.01	0.12
AQ_2–AQ_41	0.02	0.01	0.02	-0.01	0.05	0.00	0.05
AQ_2–AQ_42	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_2–AQ_44	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_2–AQ_45	0.04	0.04	0.02	-0.00	0.09	0.00	0.08
AQ_2–AQ_46	0.17	0.16	0.02	0.13	0.22	0.11	0.21
AQ_2–AQ_47	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_2–AQ_50	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_2–AQ_6	0.05	0.04	0.02	-0.00	0.10	0.00	0.09
AQ_2–AQ_8	0.00	-0.00	0.00	-0.00	0.00	-0.00	0.00
AQ_2–AQ_9	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_20–AQ_22	0.15	0.14	0.03	0.09	0.20	0.08	0.19
AQ_20–AQ_23	-0.03	-0.02	0.02	-0.08	0.01	-0.08	0.00
AQ_20–AQ_25	0.00	-0.00	0.01	-0.01	0.01	-0.02	0.00
AQ_20–AQ_32	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_20–AQ_34	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_20–AQ_36	0.04	0.04	0.02	-0.00	0.09	0.00	0.08
AQ_20–AQ_37	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_20–AQ_41	0.00	0.01	0.01	-0.02	0.02	0.00	0.04
AQ_20–AQ_42	0.00	0.01	0.01	-0.03	0.03	0.00	0.04
AQ_20–AQ_44	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_20–AQ_45	0.07	0.06	0.03	0.01	0.12	0.00	0.11
AQ_20–AQ_46	-0.05	-0.04	0.02	-0.09	-0.00	-0.09	0.00
AQ_20–AQ_47	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_20–AQ_50	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_22–AQ_23	0.02	0.02	0.02	-0.02	0.06	0.00	0.06
AQ_22–AQ_25	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_22–AQ_32	0.00	-0.00	0.00	-0.01	0.01	-0.00	0.00
AQ_22–AQ_34	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00

Table B.4 Continued: Control Group - AQ

AQ_22–AQ_36	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_22–AQ_37	0.02	0.01	0.02	-0.02	0.05	0.00	0.06
AQ_22–AQ_41	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_22–AQ_42	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_22–AQ_44	0.00	0.00	0.00	-0.01	0.01	0.00	0.02
AQ_22–AQ_45	0.02	0.02	0.02	-0.02	0.06	0.00	0.06
AQ_22–AQ_46	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_22–AQ_47	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_22–AQ_50	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_23–AQ_25	0.00	0.01	0.01	-0.02	0.02	0.00	0.04
AQ_23–AQ_32	0.00	-0.00	0.00	-0.01	0.01	-0.01	0.00
AQ_23–AQ_34	-0.02	-0.01	0.02	-0.05	0.02	-0.06	0.00
AQ_23–AQ_36	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_23–AQ_37	0.00	0.01	0.01	-0.02	0.03	0.00	0.04
AQ_23–AQ_41	0.00	-0.00	0.00	-0.01	0.01	-0.01	0.00
AQ_23–AQ_42	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_23–AQ_44	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_23–AQ_45	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_23–AQ_46	0.01	0.01	0.01	-0.02	0.03	0.00	0.05
AQ_23–AQ_47	0.00	-0.00	0.00	-0.00	0.00	-0.00	0.00
AQ_23–AQ_50	0.00	0.00	0.01	-0.01	0.01	0.00	0.03
AQ_25–AQ_32	0.00	0.01	0.01	-0.02	0.02	0.00	0.04
AQ_25–AQ_34	0.23	0.22	0.02	0.18	0.28	0.17	0.27
AQ_25–AQ_36	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_25–AQ_37	0.11	0.10	0.02	0.06	0.16	0.05	0.15
AQ_25–AQ_41	0.00	0.01	0.01	-0.02	0.02	0.00	0.03
AQ_25–AQ_42	0.00	0.00	0.00	-0.01	0.01	0.00	0.02
AQ_25–AQ_44	0.00	0.01	0.01	-0.02	0.02	0.00	0.04
AQ_25–AQ_45	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_25–AQ_46	0.06	0.05	0.02	0.01	0.11	0.01	0.10
AQ_25–AQ_47	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_25–AQ_50	0.00	0.01	0.01	-0.02	0.02	0.00	0.04
AQ_3–AQ_10	0.00	0.00	0.00	-0.00	0.00	0.00	0.00

Table B.4 Continued: Control Group - AQ

AQ_3–AQ_11	-0.00	-0.00	0.01	-0.02	0.01	-0.03	0.00
AQ_3–AQ_13	0.00	-0.00	0.01	-0.01	0.01	-0.03	0.00
AQ_3–AQ_14	0.12	0.12	0.02	0.07	0.17	0.07	0.16
AQ_3–AQ_15	0.00	0.00	0.01	-0.01	0.01	0.00	0.03
AQ_3–AQ_19	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_3–AQ_20	0.00	0.01	0.01	-0.02	0.02	0.00	0.04
AQ_3–AQ_22	0.00	-0.00	0.00	-0.01	0.01	-0.02	0.00
AQ_3–AQ_23	-0.05	-0.04	0.02	-0.09	-0.01	-0.08	0.00
AQ_3–AQ_25	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_3–AQ_32	0.06	0.06	0.02	0.01	0.11	0.01	0.10
AQ_3–AQ_34	0.02	0.02	0.02	-0.01	0.06	0.00	0.06
AQ_3–AQ_36	0.04	0.04	0.02	-0.00	0.08	0.00	0.08
AQ_3–AQ_37	0.03	0.03	0.02	-0.01	0.07	0.00	0.07
AQ_3–AQ_4	-0.09	-0.08	0.02	-0.14	-0.05	-0.12	-0.04
AQ_3–AQ_41	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_3–AQ_42	0.10	0.10	0.03	0.05	0.15	0.05	0.15
AQ_3–AQ_44	0.07	0.06	0.02	0.02	0.11	0.02	0.10
AQ_3–AQ_45	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_3–AQ_46	-0.05	-0.04	0.02	-0.09	-0.00	-0.09	0.00
AQ_3–AQ_47	0.04	0.04	0.02	-0.00	0.08	0.00	0.08
AQ_3–AQ_50	0.02	0.02	0.02	-0.02	0.06	0.00	0.07
AQ_3–AQ_6	-0.00	-0.00	0.01	-0.02	0.02	-0.04	0.00
AQ_3–AQ_8	0.48	0.47	0.02	0.43	0.52	0.43	0.51
AQ_3–AQ_9	0.11	0.11	0.02	0.07	0.16	0.07	0.15
AQ_32–AQ_34	0.02	0.02	0.02	-0.02	0.06	0.00	0.06
AQ_32–AQ_36	0.04	0.04	0.02	-0.01	0.09	0.00	0.09
AQ_32–AQ_37	0.27	0.27	0.03	0.22	0.32	0.22	0.32
AQ_32–AQ_41	0.01	0.01	0.01	-0.02	0.03	0.00	0.04
AQ_32–AQ_42	0.01	0.01	0.02	-0.03	0.05	0.00	0.06
AQ_32–AQ_44	0.00	0.01	0.01	-0.02	0.02	0.00	0.03
AQ_32–AQ_45	0.03	0.03	0.02	-0.02	0.08	0.00	0.08
AQ_32–AQ_46	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_32–AQ_47	0.00	0.00	0.01	-0.01	0.01	0.00	0.03

Table B.4 Continued: Control Group - AQ

AQ_32–AQ_50	0.04	0.03	0.02	-0.01	0.08	0.00	0.08
AQ_34–AQ_36	0.05	0.05	0.02	0.01	0.10	0.00	0.09
AQ_34–AQ_37	0.01	0.02	0.02	-0.02	0.05	0.00	0.06
AQ_34–AQ_41	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_34–AQ_42	0.00	0.01	0.01	-0.03	0.03	0.00	0.04
AQ_34–AQ_44	0.10	0.10	0.02	0.06	0.15	0.06	0.15
AQ_34–AQ_45	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_34–AQ_46	0.00	0.01	0.01	-0.02	0.02	0.00	0.04
AQ_34–AQ_47	0.18	0.17	0.03	0.13	0.23	0.13	0.23
AQ_34–AQ_50	0.02	0.02	0.02	-0.02	0.06	0.00	0.07
AQ_36–AQ_37	0.02	0.02	0.02	-0.02	0.06	0.00	0.07
AQ_36–AQ_41	0.05	0.04	0.02	-0.00	0.09	0.00	0.09
AQ_36–AQ_42	0.13	0.13	0.03	0.08	0.18	0.07	0.18
AQ_36–AQ_44	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_36–AQ_45	0.33	0.32	0.03	0.27	0.38	0.27	0.37
AQ_36–AQ_46	-0.02	-0.02	0.02	-0.06	0.01	-0.06	0.00
AQ_36–AQ_47	0.02	0.02	0.02	-0.02	0.06	0.00	0.07
AQ_36–AQ_50	0.02	0.02	0.02	-0.02	0.06	0.00	0.06
AQ_37–AQ_41	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_37–AQ_42	0.07	0.07	0.02	0.03	0.12	0.02	0.11
AQ_37–AQ_44	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_37–AQ_45	0.04	0.04	0.02	-0.01	0.09	0.00	0.09
AQ_37–AQ_46	0.02	0.01	0.02	-0.02	0.05	0.00	0.06
AQ_37–AQ_47	0.00	0.01	0.01	-0.02	0.02	0.00	0.03
AQ_37–AQ_50	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_4–AQ_10	0.00	0.00	0.01	-0.02	0.02	0.00	0.04
AQ_4–AQ_11	0.02	0.02	0.02	-0.02	0.05	0.00	0.06
AQ_4–AQ_13	0.02	0.02	0.02	-0.02	0.06	0.00	0.07
AQ_4–AQ_14	-0.03	-0.02	0.02	-0.07	0.01	-0.07	0.00
AQ_4–AQ_15	0.00	-0.00	0.01	-0.01	0.01	-0.02	0.00
AQ_4–AQ_19	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_4–AQ_20	-0.01	-0.01	0.01	-0.03	0.02	-0.05	0.00
AQ_4–AQ_22	0.00	0.00	0.01	-0.01	0.01	0.00	0.02

Table B.4 Continued: Control Group - AQ

AQ_4–AQ_23	0.04	0.03	0.02	-0.01	0.08	0.00	0.08
AQ_4–AQ_25	0.03	0.03	0.02	-0.02	0.07	0.00	0.08
AQ_4–AQ_32	0.06	0.05	0.03	0.01	0.11	0.00	0.10
AQ_4–AQ_34	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_4–AQ_36	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_4–AQ_37	0.07	0.07	0.03	0.02	0.13	0.02	0.12
AQ_4–AQ_41	0.01	0.01	0.01	-0.02	0.04	0.00	0.05
AQ_4–AQ_42	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_4–AQ_44	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_4–AQ_45	0.06	0.05	0.02	0.01	0.10	0.00	0.09
AQ_4–AQ_46	0.13	0.12	0.03	0.08	0.18	0.07	0.17
AQ_4–AQ_47	-0.01	-0.01	0.01	-0.04	0.01	-0.04	0.00
AQ_4–AQ_50	0.00	-0.00	0.01	-0.01	0.01	-0.02	0.00
AQ_4–AQ_6	0.06	0.05	0.03	0.01	0.11	0.00	0.10
AQ_4–AQ_8	-0.01	-0.01	0.01	-0.04	0.02	-0.05	0.00
AQ_4–AQ_9	0.00	-0.00	0.00	-0.01	0.01	-0.00	0.00
AQ_41–AQ_42	0.04	0.04	0.02	-0.01	0.09	0.00	0.09
AQ_41–AQ_44	0.08	0.08	0.02	0.04	0.13	0.04	0.13
AQ_41–AQ_45	0.07	0.06	0.02	0.02	0.11	0.02	0.11
AQ_41–AQ_46	-0.05	-0.04	0.02	-0.09	-0.01	-0.09	0.00
AQ_41–AQ_47	0.04	0.04	0.02	-0.00	0.09	0.00	0.09
AQ_41–AQ_50	0.00	0.00	0.01	-0.02	0.02	0.00	0.03
AQ_42–AQ_44	0.01	0.01	0.01	-0.02	0.03	0.00	0.04
AQ_42–AQ_45	0.13	0.13	0.03	0.08	0.18	0.08	0.17
AQ_42–AQ_46	0.00	-0.00	0.01	-0.01	0.01	-0.02	0.00
AQ_42–AQ_47	0.08	0.07	0.02	0.03	0.12	0.02	0.11
AQ_42–AQ_50	0.07	0.07	0.03	0.02	0.12	0.02	0.12
AQ_44–AQ_45	0.03	0.03	0.02	-0.00	0.07	0.00	0.07
AQ_44–AQ_46	0.00	-0.00	0.00	-0.01	0.01	-0.01	0.00
AQ_44–AQ_47	0.36	0.36	0.02	0.32	0.41	0.31	0.40
AQ_44–AQ_50	0.01	0.01	0.01	-0.02	0.03	0.00	0.05
AQ_45–AQ_46	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_45–AQ_47	0.00	0.00	0.01	-0.01	0.01	0.00	0.02

Table B.4 Continued: Control Group - AQ

AQ_45–AQ_50	0.01	0.02	0.02	-0.02	0.05	0.00	0.06
AQ_46–AQ_47	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_46–AQ_50	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_47–AQ_50	0.03	0.03	0.02	-0.01	0.08	0.00	0.07
AQ_6–AQ_10	-0.01	-0.01	0.01	-0.04	0.02	-0.05	0.00
AQ_6–AQ_11	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_6–AQ_13	0.00	0.01	0.01	-0.02	0.02	0.00	0.04
AQ_6–AQ_14	-0.02	-0.01	0.02	-0.05	0.01	-0.05	0.00
AQ_6–AQ_15	0.02	0.01	0.02	-0.02	0.05	0.00	0.05
AQ_6–AQ_19	0.01	0.01	0.02	-0.03	0.04	0.00	0.06
AQ_6–AQ_20	0.00	-0.00	0.01	-0.01	0.01	-0.03	0.00
AQ_6–AQ_22	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_6–AQ_23	0.01	0.01	0.02	-0.02	0.05	0.00	0.06
AQ_6–AQ_25	0.02	0.02	0.02	-0.02	0.06	0.00	0.06
AQ_6–AQ_32	-0.01	-0.01	0.01	-0.04	0.02	-0.05	0.00
AQ_6–AQ_34	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_6–AQ_36	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_6–AQ_37	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_6–AQ_41	0.18	0.17	0.03	0.12	0.23	0.12	0.22
AQ_6–AQ_42	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_6–AQ_44	0.00	0.00	0.01	-0.01	0.01	0.00	0.02
AQ_6–AQ_45	0.00	0.00	0.00	-0.01	0.01	0.00	0.02
AQ_6–AQ_46	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_6–AQ_47	0.01	0.01	0.01	-0.02	0.04	0.00	0.05
AQ_6–AQ_50	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
AQ_6–AQ_8	0.00	-0.00	0.00	-0.01	0.01	-0.02	0.00
AQ_6–AQ_9	0.19	0.19	0.03	0.14	0.25	0.14	0.24
AQ_8–AQ_10	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
AQ_8–AQ_11	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_8–AQ_13	0.00	-0.00	0.00	-0.01	0.01	-0.01	0.00
AQ_8–AQ_14	0.05	0.05	0.02	-0.00	0.09	0.00	0.09
AQ_8–AQ_15	0.00	0.00	0.00	-0.01	0.01	0.00	0.02
AQ_8–AQ_19	0.00	0.00	0.00	-0.00	0.00	0.00	0.00

Table B.4 Continued: Control Group - AQ

AQ_8–AQ_20	0.05	0.04	0.02	0.01	0.10	0.00	0.08
AQ_8–AQ_22	-0.01	-0.01	0.01	-0.04	0.01	-0.04	0.00
AQ_8–AQ_23	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_8–AQ_25	0.00	-0.00	0.00	-0.00	0.00	0.00	0.00
AQ_8–AQ_32	0.07	0.07	0.02	0.02	0.12	0.02	0.11
AQ_8–AQ_34	0.02	0.02	0.02	-0.02	0.05	0.00	0.06
AQ_8–AQ_36	0.11	0.11	0.02	0.06	0.15	0.06	0.15
AQ_8–AQ_37	0.05	0.04	0.02	0.00	0.09	0.00	0.09
AQ_8–AQ_41	0.01	0.01	0.01	-0.02	0.03	0.00	0.05
AQ_8–AQ_42	0.04	0.04	0.02	-0.01	0.08	0.00	0.09
AQ_8–AQ_44	0.04	0.04	0.02	0.01	0.08	0.00	0.08
AQ_8–AQ_45	0.02	0.02	0.02	-0.02	0.05	0.00	0.06
AQ_8–AQ_46	-0.05	-0.05	0.02	-0.09	-0.01	-0.09	0.00
AQ_8–AQ_47	0.04	0.04	0.02	-0.00	0.09	0.00	0.09
AQ_8–AQ_50	0.13	0.13	0.02	0.09	0.18	0.08	0.18
AQ_8–AQ_9	0.04	0.04	0.02	-0.01	0.09	0.00	0.09
AQ_9–AQ_10	0.00	-0.00	0.00	-0.01	0.01	-0.02	0.00
AQ_9–AQ_11	-0.07	-0.05	0.02	-0.12	-0.03	-0.10	-0.00
AQ_9–AQ_13	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_9–AQ_14	0.00	-0.00	0.00	-0.00	0.00	-0.00	0.00
AQ_9–AQ_15	0.03	0.03	0.02	-0.01	0.07	0.00	0.07
AQ_9–AQ_19	0.05	0.05	0.02	0.01	0.10	0.00	0.09
AQ_9–AQ_20	0.00	0.00	0.00	-0.01	0.01	0.00	0.02
AQ_9–AQ_22	0.00	-0.00	0.00	-0.01	0.01	0.00	0.00
AQ_9–AQ_23	0.00	-0.00	0.01	-0.02	0.02	-0.03	0.00
AQ_9–AQ_25	0.03	0.02	0.02	-0.01	0.06	0.00	0.06
AQ_9–AQ_32	0.00	0.00	0.00	-0.00	0.00	0.00	0.01
AQ_9–AQ_34	0.00	0.01	0.01	-0.02	0.02	0.00	0.04
AQ_9–AQ_36	0.02	0.02	0.02	-0.02	0.06	0.00	0.07
AQ_9–AQ_37	0.00	0.00	0.00	-0.01	0.01	0.00	0.01
AQ_9–AQ_41	0.31	0.31	0.02	0.27	0.36	0.27	0.36
AQ_9–AQ_42	0.09	0.08	0.02	0.04	0.13	0.03	0.13
AQ_9–AQ_44	0.07	0.06	0.02	0.02	0.11	0.01	0.10

Table B.4 Continued: Control Group - AQ

AQ_9–AQ_45	0.02	0.02	0.02	-0.02	0.07	0.00	0.07
AQ_9–AQ_46	-0.12	-0.12	0.02	-0.17	-0.08	-0.17	-0.08
AQ_9–AQ_47	0.02	0.02	0.02	-0.02	0.06	0.00	0.06
AQ_9–AQ_50	0.00	0.00	0.00	-0.01	0.01	0.00	0.02

Table B.5 ASC Group - PDI

Edge	Sample	Mean	SD	CI (lower)	CI (upper)	q2.5	q97.5A
PDI1–PDI10	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PDI1–PDI11	0.00	-0.00	0.02	-0.04	0.04	0.00	0.00
PDI1–PDI12	0.00	0.00	0.02	-0.03	0.03	0.00	0.00
PDI1–PDI13	0.00	0.00	0.05	-0.10	0.10	0.00	0.00
PDI1–PDI14	0.00	0.00	0.05	-0.10	0.10	0.00	0.00
PDI1–PDI15	0.58	0.55	0.36	-0.14	1.29	0.00	1.22
PDI1–PDI16	0.00	0.02	0.12	-0.24	0.24	0.00	0.50
PDI1–PDI17	0.00	0.03	0.16	-0.31	0.31	0.00	0.56
PDI1–PDI18	0.00	0.03	0.13	-0.26	0.26	0.00	0.51
PDI1–PDI19	0.00	0.02	0.11	-0.22	0.22	0.00	0.43
PDI1–PDI2	0.00	0.02	0.11	-0.22	0.22	0.00	0.30
PDI1–PDI20	0.00	0.00	0.01	-0.01	0.01	0.00	0.00
PDI1–PDI21	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PDI1–PDI3	0.83	0.77	0.41	0.01	1.65	0.00	1.51
PDI1–PDI4	0.00	0.10	0.21	-0.43	0.43	0.00	0.71
PDI1–PDI5	0.00	0.02	0.11	-0.21	0.21	0.00	0.36
PDI1–PDI6	0.00	0.01	0.07	-0.13	0.13	0.00	0.22
PDI1–PDI7	0.00	0.01	0.07	-0.14	0.14	0.00	0.30
PDI1–PDI8	0.00	-0.00	0.03	-0.06	0.06	0.00	0.00
PDI1–PDI9	0.00	0.00	0.04	-0.08	0.08	0.00	0.00
PDI10–PDI11	0.00	0.01	0.07	-0.15	0.15	0.00	0.00
PDI10–PDI12	0.00	0.04	0.16	-0.33	0.33	0.00	0.62
PDI10–PDI13	0.00	0.00	0.01	-0.02	0.02	0.00	0.00
PDI10–PDI14	0.00	0.00	0.03	-0.07	0.07	0.00	0.00
PDI10–PDI15	0.00	0.01	0.08	-0.16	0.16	0.00	0.00

Table B.5 Continued: ASC Group - PDI

PDI10–PDI16	0.00	0.00	0.04	-0.09	0.09	0.00	0.00
PDI10–PDI17	0.00	0.08	0.22	-0.44	0.44	0.00	0.80
PDI10–PDI18	0.00	0.04	0.15	-0.30	0.30	0.00	0.61
PDI10–PDI19	0.00	0.00	0.03	-0.07	0.07	0.00	0.00
PDI10–PDI20	0.00	0.04	0.16	-0.33	0.33	0.00	0.67
PDI10–PDI21	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PDI11–PDI12	0.70	0.62	2.76	-4.83	6.22	0.00	1.58
PDI11–PDI13	0.00	-0.00	0.08	-0.15	0.15	0.00	0.00
PDI11–PDI14	0.00	0.05	0.41	-0.83	0.83	0.00	0.74
PDI11–PDI15	0.00	0.04	1.48	-2.96	2.96	0.00	0.00
PDI11–PDI16	0.00	-0.07	1.97	-3.95	3.95	-0.64	0.00
PDI11–PDI17	0.00	0.02	0.34	-0.67	0.67	0.00	0.00
PDI11–PDI18	0.00	-0.00	0.01	-0.02	0.02	0.00	0.00
PDI11–PDI19	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PDI11–PDI20	0.00	-0.00	0.01	-0.03	0.03	0.00	0.00
PDI11–PDI21	0.00	0.04	1.15	-2.31	2.31	0.00	0.00
PDI12–PDI13	0.00	0.00	0.05	-0.10	0.10	0.00	0.00
PDI12–PDI14	0.00	0.00	0.05	-0.10	0.10	0.00	0.00
PDI12–PDI15	0.00	0.00	0.03	-0.07	0.07	0.00	0.00
PDI12–PDI16	0.00	-0.00	0.01	-0.02	0.02	0.00	0.00
PDI12–PDI17	0.00	0.00	0.03	-0.05	0.05	0.00	0.00
PDI12–PDI18	0.00	0.00	0.02	-0.04	0.04	0.00	0.00
PDI12–PDI19	0.00	0.04	0.14	-0.28	0.28	0.00	0.57
PDI12–PDI20	0.00	0.01	0.09	-0.18	0.18	0.00	0.00
PDI12–PDI21	0.00	0.01	0.09	-0.19	0.19	0.00	0.00
PDI13–PDI14	0.00	0.05	0.18	-0.37	0.37	0.00	0.75
PDI13–PDI15	0.00	0.07	0.21	-0.42	0.42	0.00	0.80
PDI13–PDI16	0.00	0.00	0.01	-0.03	0.03	0.00	0.00
PDI13–PDI17	0.00	-0.00	0.05	-0.10	0.10	0.00	0.00
PDI13–PDI18	0.00	-0.00	0.04	-0.07	0.07	0.00	0.00
PDI13–PDI19	0.00	-0.00	0.01	-0.03	0.03	0.00	0.00
PDI13–PDI20	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PDI13–PDI21	0.00	-0.00	0.02	-0.04	0.04	0.00	0.00

Table B.5 Continued: ASC Group - PDI

PDI14–PDI15	0.00	0.02	0.12	-0.24	0.24	0.00	0.48
PDI14–PDI16	0.00	0.01	0.09	-0.17	0.17	0.00	0.00
PDI14–PDI17	0.00	0.02	0.11	-0.21	0.21	0.00	0.38
PDI14–PDI18	0.00	-0.00	0.02	-0.03	0.03	0.00	0.00
PDI14–PDI19	0.00	0.09	0.24	-0.48	0.48	0.00	0.83
PDI14–PDI20	0.00	0.00	0.03	-0.06	0.06	0.00	0.00
PDI14–PDI21	0.00	0.00	0.04	-0.08	0.08	0.00	0.00
PDI15–PDI16	0.00	0.04	0.15	-0.31	0.31	0.00	0.63
PDI15–PDI17	0.00	0.03	0.12	-0.25	0.25	0.00	0.46
PDI15–PDI18	0.00	0.42	0.37	-0.74	0.74	0.00	1.15
PDI15–PDI19	0.00	0.00	0.03	-0.06	0.06	0.00	0.00
PDI15–PDI20	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PDI15–PDI21	0.00	0.16	0.34	-0.67	0.67	0.00	1.15
PDI16–PDI17	0.00	0.13	0.29	-0.59	0.59	0.00	0.99
PDI16–PDI18	0.00	0.06	0.18	-0.37	0.37	0.00	0.67
PDI16–PDI19	0.00	0.00	0.02	-0.04	0.04	0.00	0.00
PDI16–PDI20	0.00	0.10	0.27	-0.53	0.53	0.00	0.95
PDI16–PDI21	0.00	0.00	0.05	-0.09	0.09	0.00	0.00
PDI17–PDI18	0.00	0.35	0.39	-0.79	0.79	0.00	1.17
PDI17–PDI19	0.00	0.26	0.39	-0.77	0.77	0.00	1.17
PDI17–PDI20	0.00	0.13	0.29	-0.57	0.57	0.00	0.94
PDI17–PDI21	0.00	0.01	0.07	-0.15	0.15	0.00	0.00
PDI18–PDI19	0.00	0.12	0.26	-0.51	0.51	0.00	0.84
PDI18–PDI20	0.00	0.01	0.07	-0.14	0.14	0.00	0.00
PDI18–PDI21	0.00	0.16	0.32	-0.65	0.65	0.00	1.02
PDI19–PDI20	0.00	0.04	0.16	-0.31	0.31	0.00	0.64
PDI19–PDI21	0.00	0.00	0.06	-0.11	0.11	0.00	0.00
PDI2–PDI10	0.00	0.00	0.05	-0.10	0.10	0.00	0.00
PDI2–PDI11	0.00	0.00	0.06	-0.13	0.13	0.00	0.00
PDI2–PDI12	0.00	0.05	0.18	-0.36	0.36	0.00	0.70
PDI2–PDI13	0.00	0.01	0.10	-0.19	0.19	0.00	0.00
PDI2–PDI14	0.00	0.01	0.10	-0.20	0.20	0.00	0.00
PDI2–PDI15	0.00	0.00	0.03	-0.07	0.07	0.00	0.00

Table B.5 Continued: ASC Group - PDI

PDI2–PDI16	0.00	0.01	0.07	-0.14	0.14	0.00	0.00
PDI2–PDI17	0.00	0.45	0.51	-1.01	1.01	0.00	1.49
PDI2–PDI18	0.00	0.00	0.02	-0.04	0.04	0.00	0.00
PDI2–PDI19	0.00	0.01	0.06	-0.13	0.13	0.00	0.00
PDI2–PDI20	0.00	0.03	0.15	-0.30	0.30	0.00	0.62
PDI2–PDI21	0.00	-0.00	0.03	-0.07	0.07	0.00	0.00
PDI2–PDI3	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PDI2–PDI4	0.00	0.05	0.17	-0.34	0.34	0.00	0.64
PDI2–PDI5	0.00	0.12	0.26	-0.52	0.52	0.00	0.88
PDI2–PDI6	0.56	0.47	0.44	-0.32	1.43	0.00	1.33
PDI2–PDI7	0.00	0.00	0.03	-0.06	0.06	0.00	0.00
PDI2–PDI8	0.68	0.43	0.52	-0.35	1.71	0.00	1.52
PDI2–PDI9	0.00	0.03	0.13	-0.26	0.26	0.00	0.52
PDI20–PDI21	0.00	0.02	0.12	-0.24	0.24	0.00	0.45
PDI3–PDI10	0.00	0.04	0.18	-0.36	0.36	0.00	0.76
PDI3–PDI11	0.00	-0.01	0.51	-1.02	1.02	0.00	0.00
PDI3–PDI12	0.00	-0.00	0.02	-0.05	0.05	0.00	0.00
PDI3–PDI13	0.00	0.00	0.01	-0.01	0.01	0.00	0.00
PDI3–PDI14	0.00	-0.01	0.07	-0.14	0.14	0.00	0.00
PDI3–PDI15	0.00	0.13	0.25	-0.50	0.50	0.00	0.82
PDI3–PDI16	0.00	0.00	0.02	-0.05	0.05	0.00	0.00
PDI3–PDI17	0.00	0.00	0.03	-0.06	0.06	0.00	0.00
PDI3–PDI18	0.00	-0.01	0.07	-0.14	0.14	0.00	0.00
PDI3–PDI19	0.00	0.00	0.02	-0.04	0.04	0.00	0.00
PDI3–PDI20	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PDI3–PDI21	0.00	0.00	0.06	-0.12	0.12	0.00	0.00
PDI3–PDI4	0.00	0.27	0.36	-0.73	0.73	0.00	1.11
PDI3–PDI5	0.00	0.01	0.09	-0.18	0.18	0.00	0.00
PDI3–PDI6	0.00	0.00	0.02	-0.04	0.04	0.00	0.00
PDI3–PDI7	0.00	0.02	0.09	-0.18	0.18	0.00	0.34
PDI3–PDI8	0.00	-0.00	0.03	-0.06	0.06	0.00	0.00
PDI3–PDI9	0.00	-0.00	0.02	-0.04	0.04	0.00	0.00
PDI4–PDI10	0.00	0.12	0.27	-0.53	0.53	0.00	0.90

Table B.5 Continued: ASC Group - PDI

PDI4–PDI11	0.00	0.00	0.02	-0.03	0.03	0.00	0.00
PDI4–PDI12	0.00	-0.00	0.02	-0.04	0.04	0.00	0.00
PDI4–PDI13	0.00	0.02	0.11	-0.22	0.22	0.00	0.44
PDI4–PDI14	0.00	0.29	0.38	-0.77	0.77	0.00	1.15
PDI4–PDI15	0.00	0.00	0.02	-0.04	0.04	0.00	0.00
PDI4–PDI16	0.00	0.00	0.02	-0.04	0.04	0.00	0.00
PDI4–PDI17	0.00	0.01	0.07	-0.15	0.15	0.00	0.00
PDI4–PDI18	0.00	0.03	0.11	-0.21	0.21	0.00	0.43
PDI4–PDI19	0.00	0.06	0.17	-0.35	0.35	0.00	0.64
PDI4–PDI20	0.00	0.05	0.18	-0.36	0.36	0.00	0.68
PDI4–PDI21	0.00	0.10	0.26	-0.52	0.52	0.00	0.94
PDI4–PDI5	1.51	1.68	0.34	0.83	2.18	1.06	2.35
PDI4–PDI6	0.00	0.01	0.06	-0.11	0.11	0.00	0.10
PDI4–PDI7	0.00	0.03	0.11	-0.23	0.23	0.00	0.43
PDI4–PDI8	0.00	0.01	0.05	-0.11	0.11	0.00	0.00
PDI4–PDI9	0.00	0.01	0.06	-0.11	0.11	0.00	0.00
PDI5–PDI10	0.00	0.05	0.17	-0.34	0.34	0.00	0.64
PDI5–PDI11	0.00	0.01	0.11	-0.21	0.21	0.00	0.00
PDI5–PDI12	0.00	0.06	0.18	-0.36	0.36	0.00	0.66
PDI5–PDI13	0.00	0.02	0.10	-0.20	0.20	0.00	0.39
PDI5–PDI14	0.00	0.06	0.18	-0.37	0.37	0.00	0.66
PDI5–PDI15	0.00	0.00	0.01	-0.03	0.03	0.00	0.00
PDI5–PDI16	0.00	0.00	0.01	-0.01	0.01	0.00	0.00
PDI5–PDI17	0.00	0.07	0.20	-0.40	0.40	0.00	0.71
PDI5–PDI18	0.00	0.17	0.28	-0.56	0.56	0.00	0.88
PDI5–PDI19	0.00	0.02	0.10	-0.19	0.19	0.00	0.38
PDI5–PDI20	0.00	0.01	0.08	-0.16	0.16	0.00	0.21
PDI5–PDI21	0.00	0.00	0.05	-0.10	0.10	0.00	0.00
PDI5–PDI6	0.00	0.25	0.31	-0.63	0.63	0.00	0.94
PDI5–PDI7	0.00	-0.00	0.03	-0.05	0.05	0.00	0.00
PDI5–PDI8	0.00	0.00	0.07	-0.13	0.13	0.00	0.00
PDI5–PDI9	0.00	0.07	0.18	-0.36	0.36	0.00	0.62
PDI6–PDI10	0.00	0.02	0.09	-0.18	0.18	0.00	0.36

Table B.5 Continued: ASC Group - PDI

PDI6–PDI11	0.00	0.01	0.20	-0.40	0.40	0.00	0.00
PDI6–PDI12	0.00	-0.00	0.05	-0.10	0.10	0.00	0.00
PDI6–PDI13	0.36	0.39	0.42	-0.48	1.21	0.00	1.23
PDI6–PDI14	0.00	0.02	0.09	-0.19	0.19	0.00	0.36
PDI6–PDI15	0.00	0.11	0.20	-0.40	0.40	0.00	0.68
PDI6–PDI16	0.00	0.01	0.05	-0.11	0.11	0.00	0.00
PDI6–PDI17	0.00	0.03	0.14	-0.28	0.28	0.00	0.58
PDI6–PDI18	0.00	0.13	0.23	-0.47	0.47	0.00	0.75
PDI6–PDI19	0.00	0.00	0.03	-0.05	0.05	0.00	0.00
PDI6–PDI20	0.00	0.02	0.11	-0.23	0.23	0.00	0.41
PDI6–PDI21	0.00	0.00	0.03	-0.05	0.05	0.00	0.00
PDI6–PDI7	1.11	1.29	0.36	0.39	1.83	0.63	2.04
PDI6–PDI8	0.00	0.09	0.23	-0.46	0.46	0.00	0.80
PDI6–PDI9	0.00	0.06	0.16	-0.33	0.33	0.00	0.56
PDI7–PDI10	0.00	0.01	0.07	-0.14	0.14	0.00	0.00
PDI7–PDI11	0.00	0.00	0.05	-0.10	0.10	0.00	0.00
PDI7–PDI12	0.00	0.00	0.03	-0.07	0.07	0.00	0.00
PDI7–PDI13	0.00	0.00	0.02	-0.04	0.04	0.00	0.00
PDI7–PDI14	0.00	0.07	0.20	-0.41	0.41	0.00	0.77
PDI7–PDI15	0.00	0.09	0.18	-0.36	0.36	0.00	0.58
PDI7–PDI16	0.00	0.00	0.04	-0.08	0.08	0.00	0.00
PDI7–PDI17	0.00	-0.01	0.06	-0.13	0.13	0.00	0.00
PDI7–PDI18	0.00	0.02	0.08	-0.17	0.17	0.00	0.32
PDI7–PDI19	0.00	0.00	0.03	-0.06	0.06	0.00	0.00
PDI7–PDI20	0.00	-0.00	0.05	-0.10	0.10	0.00	0.00
PDI7–PDI21	0.00	0.01	0.07	-0.14	0.14	0.00	0.00
PDI7–PDI8	0.00	0.01	0.06	-0.12	0.12	0.00	0.00
PDI7–PDI9	0.00	0.05	0.17	-0.34	0.34	0.00	0.61
PDI8–PDI10	0.00	0.01	0.10	-0.19	0.19	0.00	0.00
PDI8–PDI11	2.89	3.11	4.45	-6.01	11.80	1.98	3.92
PDI8–PDI12	0.00	0.02	0.12	-0.24	0.24	0.00	0.47
PDI8–PDI13	0.00	0.00	0.01	-0.02	0.02	0.00	0.00
PDI8–PDI14	0.00	0.00	0.01	-0.02	0.02	0.00	0.00

Table B.5 Continued: ASC Group - PDI

PDI8–PDI15	0.00	0.00	0.02	-0.03	0.03	0.00	0.00
PDI8–PDI16	0.00	0.00	0.02	-0.04	0.04	0.00	0.00
PDI8–PDI17	0.00	0.00	0.04	-0.08	0.08	0.00	0.00
PDI8–PDI18	0.00	-0.00	0.01	-0.03	0.03	0.00	0.00
PDI8–PDI19	0.00	-0.00	0.01	-0.02	0.02	0.00	0.00
PDI8–PDI20	0.00	0.00	0.03	-0.05	0.05	0.00	0.00
PDI8–PDI21	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PDI8–PDI9	0.00	0.13	0.30	-0.60	0.60	0.00	0.99
PDI9–PDI10	0.00	0.06	0.20	-0.40	0.40	0.00	0.76
PDI9–PDI11	0.00	-0.01	0.13	-0.25	0.25	0.00	0.00
PDI9–PDI12	1.00	0.84	0.46	0.08	1.91	0.00	1.60
PDI9–PDI13	0.00	0.01	0.06	-0.11	0.11	0.00	0.00
PDI9–PDI14	0.00	0.00	0.04	-0.09	0.09	0.00	0.00
PDI9–PDI15	0.00	-0.00	0.02	-0.04	0.04	0.00	0.00
PDI9–PDI16	0.00	0.00	0.03	-0.07	0.07	0.00	0.00
PDI9–PDI17	0.00	0.04	0.14	-0.28	0.28	0.00	0.55
PDI9–PDI18	0.00	0.00	0.04	-0.09	0.09	0.00	0.00
PDI9–PDI19	0.54	0.52	0.45	-0.37	1.45	0.00	1.38
PDI9–PDI20	0.00	0.01	0.09	-0.17	0.17	0.00	0.24
PDI9–PDI21	0.00	0.00	0.01	-0.02	0.02	0.00	0.00

Table B.6 Control Group - PDI

Edge	Sample	Mean	SD	CI (lower)	CI (upper)	q2.5	q97.5A
PDI1–PDI10	0.00	0.00	0.01	-0.03	0.03	0.00	0.00
PDI1–PDI11	0.00	0.01	0.05	-0.09	0.09	0.00	0.00
PDI1–PDI12	0.00	0.02	0.08	-0.16	0.16	0.00	0.31
PDI1–PDI13	0.00	0.04	0.10	-0.20	0.20	0.00	0.35
PDI1–PDI14	0.00	0.01	0.06	-0.12	0.12	0.00	0.25
PDI1–PDI15	0.57	0.61	0.14	0.29	0.85	0.34	0.88
PDI1–PDI16	0.00	0.04	0.11	-0.21	0.21	0.00	0.39
PDI1–PDI17	0.00	0.01	0.07	-0.13	0.13	0.00	0.27
PDI1–PDI18	0.23	0.29	0.20	-0.16	0.62	0.00	0.67

Table B.6 Continued: Control Group - PDI

PDI1–PDI19	0.00	0.00	0.03	-0.05	0.05	0.00	0.00
PDI1–PDI2	0.00	0.00	0.02	-0.03	0.03	0.00	0.00
PDI1–PDI20	0.00	0.00	0.03	-0.06	0.06	0.00	0.00
PDI1–PDI21	0.00	-0.00	0.04	-0.08	0.08	0.00	0.00
PDI1–PDI3	0.59	0.61	0.16	0.28	0.90	0.31	0.91
PDI1–PDI4	0.18	0.23	0.21	-0.24	0.61	0.00	0.66
PDI1–PDI5	0.00	0.18	0.28	-0.56	0.56	0.00	0.86
PDI1–PDI6	0.00	0.01	0.04	-0.08	0.08	0.00	0.15
PDI1–PDI7	0.00	0.04	0.08	-0.17	0.17	0.00	0.28
PDI1–PDI8	0.00	-0.00	0.02	-0.03	0.03	0.00	0.00
PDI1–PDI9	0.00	0.00	0.03	-0.06	0.06	0.00	0.00
PDI10–PDI11	0.00	-0.00	0.01	-0.01	0.01	0.00	0.00
PDI10–PDI12	0.00	0.01	0.06	-0.11	0.11	0.00	0.20
PDI10–PDI13	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PDI10–PDI14	0.00	0.00	0.01	-0.02	0.02	0.00	0.00
PDI10–PDI15	0.00	-0.00	0.01	-0.03	0.03	0.00	0.00
PDI10–PDI16	0.00	0.08	0.18	-0.35	0.35	0.00	0.57
PDI10–PDI17	0.00	0.01	0.07	-0.13	0.13	0.00	0.21
PDI10–PDI18	0.00	0.01	0.06	-0.11	0.11	0.00	0.21
PDI10–PDI19	0.38	0.33	0.29	-0.20	0.97	0.00	0.86
PDI10–PDI20	0.33	0.30	0.28	-0.24	0.90	0.00	0.85
PDI10–PDI21	0.00	0.06	0.17	-0.34	0.34	0.00	0.61
PDI11–PDI12	0.48	0.48	0.38	-0.28	1.25	0.00	1.20
PDI11–PDI13	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PDI11–PDI14	0.00	0.02	0.10	-0.20	0.20	0.00	0.43
PDI11–PDI15	0.00	-0.01	0.06	-0.12	0.12	0.00	0.00
PDI11–PDI16	0.00	-0.00	0.02	-0.04	0.04	0.00	0.00
PDI11–PDI17	0.00	0.00	0.03	-0.07	0.07	0.00	0.00
PDI11–PDI18	0.00	-0.00	0.01	-0.02	0.02	0.00	0.00
PDI11–PDI19	0.00	-0.00	0.05	-0.10	0.10	0.00	0.00
PDI11–PDI20	0.00	0.05	0.15	-0.30	0.30	0.00	0.59
PDI11–PDI21	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PDI12–PDI13	0.00	0.00	0.00	-0.00	0.00	0.00	0.00

Table B.6 Continued: Control Group - PDI

PDI12–PDI14	0.00	0.00	0.01	-0.02	0.02	0.00	0.00
PDI12–PDI15	0.00	0.00	0.03	-0.05	0.05	0.00	0.00
PDI12–PDI16	0.00	0.00	0.02	-0.03	0.03	0.00	0.00
PDI12–PDI17	0.00	0.02	0.09	-0.19	0.19	0.00	0.36
PDI12–PDI18	0.00	0.02	0.08	-0.17	0.17	0.00	0.32
PDI12–PDI19	0.00	0.01	0.05	-0.10	0.10	0.00	0.00
PDI12–PDI20	0.00	0.15	0.23	-0.46	0.46	0.00	0.71
PDI12–PDI21	0.00	0.44	0.44	-0.88	0.88	0.00	1.30
PDI13–PDI14	0.36	0.29	0.30	-0.24	0.95	0.00	0.85
PDI13–PDI15	0.00	0.05	0.11	-0.22	0.22	0.00	0.38
PDI13–PDI16	0.00	0.01	0.07	-0.14	0.14	0.00	0.27
PDI13–PDI17	0.00	0.05	0.14	-0.28	0.28	0.00	0.51
PDI13–PDI18	0.00	0.00	0.03	-0.05	0.05	0.00	0.00
PDI13–PDI19	0.00	0.00	0.04	-0.08	0.08	0.00	0.00
PDI13–PDI20	0.00	-0.00	0.02	-0.03	0.03	0.00	0.00
PDI13–PDI21	0.00	0.00	0.03	-0.06	0.06	0.00	0.00
PDI14–PDI15	0.00	0.01	0.05	-0.10	0.10	0.00	0.19
PDI14–PDI16	0.00	0.01	0.07	-0.13	0.13	0.00	0.26
PDI14–PDI17	0.00	0.03	0.12	-0.24	0.24	0.00	0.49
PDI14–PDI18	0.00	0.03	0.11	-0.21	0.21	0.00	0.41
PDI14–PDI19	0.00	0.00	0.02	-0.04	0.04	0.00	0.00
PDI14–PDI20	0.00	0.03	0.11	-0.22	0.22	0.00	0.42
PDI14–PDI21	0.00	0.00	0.04	-0.08	0.08	0.00	0.00
PDI15–PDI16	0.00	0.00	0.03	-0.06	0.06	0.00	0.00
PDI15–PDI17	0.00	0.03	0.10	-0.19	0.19	0.00	0.37
PDI15–PDI18	0.00	0.08	0.13	-0.26	0.26	0.00	0.41
PDI15–PDI19	0.36	0.29	0.24	-0.13	0.84	0.00	0.74
PDI15–PDI20	0.00	0.00	0.03	-0.06	0.06	0.00	0.00
PDI15–PDI21	0.00	0.02	0.09	-0.18	0.18	0.00	0.37
PDI16–PDI17	0.00	0.02	0.10	-0.21	0.21	0.00	0.42
PDI16–PDI18	0.00	0.05	0.13	-0.26	0.26	0.00	0.47
PDI16–PDI19	0.00	0.00	0.03	-0.06	0.06	0.00	0.00
PDI16–PDI20	0.00	0.00	0.03	-0.06	0.06	0.00	0.00

Table B.6 Continued: Control Group - PDI

PDI16–PDI21	0.00	0.23	0.35	-0.70	0.70	0.00	1.03
PDI17–PDI18	0.47	0.43	0.30	-0.14	1.08	0.00	1.00
PDI17–PDI19	0.00	0.13	0.23	-0.46	0.46	0.00	0.74
PDI17–PDI20	0.15	0.16	0.24	-0.33	0.63	0.00	0.74
PDI17–PDI21	0.00	0.58	0.48	-0.96	0.96	0.00	1.45
PDI18–PDI19	0.39	0.36	0.27	-0.14	0.92	0.00	0.86
PDI18–PDI20	1.05	1.04	0.24	0.58	1.53	0.57	1.51
PDI18–PDI21	0.00	0.27	0.34	-0.67	0.67	0.00	1.01
PDI19–PDI20	0.38	0.26	0.28	-0.17	0.94	0.00	0.83
PDI19–PDI21	0.00	0.18	0.30	-0.60	0.60	0.00	0.95
PDI2–PDI10	0.00	0.08	0.17	-0.34	0.34	0.00	0.56
PDI2–PDI11	0.00	0.01	0.06	-0.11	0.11	0.00	0.00
PDI2–PDI12	0.00	0.00	0.02	-0.05	0.05	0.00	0.00
PDI2–PDI13	0.00	0.00	0.02	-0.05	0.05	0.00	0.00
PDI2–PDI14	0.00	0.03	0.11	-0.22	0.22	0.00	0.45
PDI2–PDI15	0.00	0.00	0.02	-0.05	0.05	0.00	0.00
PDI2–PDI16	0.00	0.04	0.14	-0.28	0.28	0.00	0.55
PDI2–PDI17	0.00	0.04	0.13	-0.26	0.26	0.00	0.51
PDI2–PDI18	0.00	0.04	0.12	-0.25	0.25	0.00	0.45
PDI2–PDI19	0.00	0.24	0.31	-0.61	0.61	0.00	0.89
PDI2–PDI20	0.00	0.01	0.06	-0.12	0.12	0.00	0.15
PDI2–PDI21	0.00	0.02	0.11	-0.22	0.22	0.00	0.41
PDI2–PDI3	0.00	0.01	0.04	-0.09	0.09	0.00	0.00
PDI2–PDI4	0.00	0.08	0.19	-0.38	0.38	0.00	0.66
PDI2–PDI5	0.00	0.01	0.07	-0.14	0.14	0.00	0.00
PDI2–PDI6	0.00	0.08	0.16	-0.32	0.32	0.00	0.51
PDI2–PDI7	0.00	0.05	0.13	-0.25	0.25	0.00	0.47
PDI2–PDI8	0.00	0.14	0.25	-0.49	0.49	0.00	0.74
PDI2–PDI9	0.00	0.28	0.31	-0.62	0.62	0.00	0.89
PDI20–PDI21	0.00	0.01	0.06	-0.11	0.11	0.00	0.00
PDI3–PDI10	0.00	0.02	0.08	-0.15	0.15	0.00	0.29
PDI3–PDI11	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PDI3–PDI12	0.00	0.12	0.21	-0.42	0.42	0.00	0.66

Table B.6 Continued: Control Group - PDI

PDI3–PDI13	0.00	0.03	0.10	-0.20	0.20	0.00	0.39
PDI3–PDI14	0.00	0.00	0.02	-0.05	0.05	0.00	0.00
PDI3–PDI15	0.22	0.25	0.18	-0.13	0.58	0.00	0.59
PDI3–PDI16	0.00	0.01	0.06	-0.12	0.12	0.00	0.26
PDI3–PDI17	0.00	0.03	0.11	-0.21	0.21	0.00	0.41
PDI3–PDI18	0.00	0.04	0.10	-0.21	0.21	0.00	0.36
PDI3–PDI19	0.00	-0.00	0.03	-0.06	0.06	0.00	0.00
PDI3–PDI20	0.53	0.45	0.31	-0.09	1.16	0.00	1.04
PDI3–PDI21	0.00	-0.00	0.02	-0.03	0.03	0.00	0.00
PDI3–PDI4	0.00	0.00	0.04	-0.08	0.08	0.00	0.00
PDI3–PDI5	0.00	0.00	0.01	-0.01	0.01	0.00	0.00
PDI3–PDI6	0.00	0.01	0.06	-0.12	0.12	0.00	0.23
PDI3–PDI7	0.00	0.01	0.03	-0.06	0.06	0.00	0.10
PDI3–PDI8	0.00	0.01	0.05	-0.11	0.11	0.00	0.15
PDI3–PDI9	0.00	0.03	0.09	-0.18	0.18	0.00	0.35
PDI4–PDI10	0.00	0.10	0.18	-0.36	0.36	0.00	0.55
PDI4–PDI11	0.00	0.00	0.01	-0.01	0.01	0.00	0.00
PDI4–PDI12	0.00	0.11	0.20	-0.39	0.39	0.00	0.63
PDI4–PDI13	0.00	0.06	0.15	-0.29	0.29	0.00	0.50
PDI4–PDI14	0.54	0.47	0.32	-0.09	1.18	0.00	1.02
PDI4–PDI15	0.35	0.38	0.24	-0.12	0.83	0.00	0.82
PDI4–PDI16	0.00	0.01	0.05	-0.10	0.10	0.00	0.00
PDI4–PDI17	0.00	0.01	0.07	-0.14	0.14	0.00	0.27
PDI4–PDI18	0.12	0.17	0.21	-0.29	0.53	0.00	0.61
PDI4–PDI19	0.00	0.01	0.05	-0.11	0.11	0.00	0.18
PDI4–PDI20	0.00	0.02	0.08	-0.16	0.16	0.00	0.30
PDI4–PDI21	0.00	0.39	0.40	-0.80	0.80	0.00	1.19
PDI4–PDI5	1.96	1.97	0.28	1.41	2.52	1.44	2.52
PDI4–PDI6	0.00	0.00	0.02	-0.04	0.04	0.00	0.00
PDI4–PDI7	0.00	0.05	0.11	-0.23	0.23	0.00	0.39
PDI4–PDI8	0.00	0.00	0.03	-0.06	0.06	0.00	0.00
PDI4–PDI9	0.00	0.00	0.04	-0.08	0.08	0.00	0.00
PDI5–PDI10	0.00	0.01	0.05	-0.11	0.11	0.00	0.00

Table B.6 Continued: Control Group - PDI

PDI5–PDI11	0.00	0.18	0.31	-0.62	0.62	0.00	0.97
PDI5–PDI12	0.38	0.39	0.38	-0.39	1.15	0.00	1.15
PDI5–PDI13	0.00	0.04	0.15	-0.30	0.30	0.00	0.57
PDI5–PDI14	0.00	0.00	0.04	-0.08	0.08	0.00	0.00
PDI5–PDI15	0.00	0.00	0.04	-0.08	0.08	0.00	0.00
PDI5–PDI16	0.00	0.00	0.01	-0.02	0.02	0.00	0.00
PDI5–PDI17	0.00	0.03	0.13	-0.25	0.25	0.00	0.53
PDI5–PDI18	0.00	0.08	0.20	-0.39	0.39	0.00	0.67
PDI5–PDI19	0.00	-0.00	0.03	-0.05	0.05	0.00	0.00
PDI5–PDI20	0.00	0.03	0.12	-0.24	0.24	0.00	0.49
PDI5–PDI21	0.00	0.02	0.10	-0.21	0.21	0.00	0.34
PDI5–PDI6	0.00	0.03	0.10	-0.21	0.21	0.00	0.42
PDI5–PDI7	0.00	0.00	0.03	-0.06	0.06	0.00	0.00
PDI5–PDI8	0.00	0.00	0.01	-0.02	0.02	0.00	0.00
PDI5–PDI9	0.00	0.00	0.04	-0.07	0.07	0.00	0.00
PDI6–PDI10	0.00	0.03	0.08	-0.17	0.17	0.00	0.34
PDI6–PDI11	0.00	0.12	0.21	-0.42	0.42	0.00	0.65
PDI6–PDI12	0.00	0.06	0.13	-0.26	0.26	0.00	0.45
PDI6–PDI13	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
PDI6–PDI14	0.00	0.02	0.07	-0.14	0.14	0.00	0.28
PDI6–PDI15	0.00	0.01	0.04	-0.07	0.07	0.00	0.12
PDI6–PDI16	0.00	-0.00	0.02	-0.03	0.03	0.00	0.00
PDI6–PDI17	0.00	0.08	0.15	-0.31	0.31	0.00	0.50
PDI6–PDI18	0.00	0.05	0.10	-0.21	0.21	0.00	0.35
PDI6–PDI19	0.00	0.02	0.08	-0.16	0.16	0.00	0.30
PDI6–PDI20	0.00	-0.00	0.01	-0.02	0.02	0.00	0.00
PDI6–PDI21	0.00	0.06	0.17	-0.33	0.33	0.00	0.62
PDI6–PDI7	1.66	1.67	0.17	1.33	2.00	1.35	2.01
PDI6–PDI8	0.00	0.26	0.24	-0.47	0.47	0.00	0.72
PDI6–PDI9	0.35	0.31	0.22	-0.09	0.78	0.00	0.71
PDI7–PDI10	0.00	0.01	0.05	-0.11	0.11	0.00	0.22
PDI7–PDI11	0.00	0.00	0.04	-0.08	0.08	0.00	0.00
PDI7–PDI12	0.00	0.00	0.03	-0.07	0.07	0.00	0.00

Table B.6 Continued: Control Group - PDI

PDI7–PDI13	0.00	-0.00	0.02	-0.03	0.03	0.00	0.00
PDI7–PDI14	0.00	0.02	0.07	-0.13	0.13	0.00	0.26
PDI7–PDI15	0.00	0.02	0.06	-0.12	0.12	0.00	0.23
PDI7–PDI16	0.00	0.00	0.01	-0.02	0.02	0.00	0.00
PDI7–PDI17	0.00	0.01	0.04	-0.09	0.09	0.00	0.00
PDI7–PDI18	0.61	0.63	0.19	0.23	0.99	0.27	1.01
PDI7–PDI19	0.00	0.03	0.09	-0.19	0.19	0.00	0.33
PDI7–PDI20	0.00	0.00	0.04	-0.07	0.07	0.00	0.00
PDI7–PDI21	0.00	0.00	0.02	-0.05	0.05	0.00	0.00
PDI7–PDI8	0.00	0.13	0.19	-0.38	0.38	0.00	0.57
PDI7–PDI9	0.09	0.11	0.16	-0.24	0.42	0.00	0.52
PDI8–PDI10	0.00	0.00	0.00	-0.00	0.00	0.00	0.00
PDI8–PDI11	2.71	2.83	0.26	2.19	3.23	2.32	3.34
PDI8–PDI12	0.00	0.49	0.38	-0.76	0.76	0.00	1.15
PDI8–PDI13	0.00	-0.00	0.04	-0.09	0.09	0.00	0.00
PDI8–PDI14	0.00	0.00	0.01	-0.01	0.01	0.00	0.00
PDI8–PDI15	0.00	0.00	0.01	-0.01	0.01	0.00	0.00
PDI8–PDI16	0.00	-0.00	0.02	-0.04	0.04	0.00	0.00
PDI8–PDI17	0.00	0.00	0.02	-0.05	0.05	0.00	0.00
PDI8–PDI18	0.00	-0.00	0.02	-0.04	0.04	0.00	0.00
PDI8–PDI19	0.00	0.03	0.11	-0.22	0.22	0.00	0.43
PDI8–PDI20	0.00	0.00	0.02	-0.04	0.04	0.00	0.00
PDI8–PDI21	0.00	0.00	0.00	0.00	0.00	0.00	0.00
PDI8–PDI9	0.00	0.00	0.02	-0.04	0.04	0.00	0.00
PDI9–PDI10	0.00	0.05	0.12	-0.25	0.25	0.00	0.43
PDI9–PDI11	0.00	0.05	0.15	-0.29	0.29	0.00	0.55
PDI9–PDI12	1.26	1.26	0.25	0.76	1.76	0.76	1.75
PDI9–PDI13	0.00	0.01	0.05	-0.11	0.11	0.00	0.15
PDI9–PDI14	0.00	0.02	0.08	-0.16	0.16	0.00	0.33
PDI9–PDI15	0.00	-0.00	0.03	-0.07	0.07	0.00	0.00
PDI9–PDI16	0.00	0.00	0.00	-0.01	0.01	0.00	0.00
PDI9–PDI17	0.36	0.30	0.30	-0.25	0.97	0.00	0.89
PDI9–PDI18	0.00	-0.00	0.04	-0.08	0.08	0.00	0.00

Table B.6 Continued: Control Group - PDI

PDI9–PDI19	0.00	0.01	0.04	-0.08	0.08	0.00	0.00
PDI9–PDI20	0.00	0.09	0.17	-0.35	0.35	0.00	0.58
PDI9–PDI21	0.00	0.09	0.21	-0.42	0.42	0.00	0.73