

## Highlights

### **Low-level, prediction-based sensory and motor processes are unimpaired in Autism**

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- The emergence of the neurocognitive phenotype of autism is poorly understood
- Recent ideas frame psychiatric conditions as disorders of predictive inference
- Evidence for a deficit in predictive processing in autism is lacking
- Sensorimotor prediction in adults with and without autism was similar
- Our results do not support a general deficit in predictive processing in autism

# Low-level, prediction-based sensory and motor processes are unimpaired in Autism

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## ABSTRACT

A new promising account of human brain function suggests that sensory cortices try to optimise information processing via predictions that are based on prior experiences. The brain is thus likened to a probabilistic prediction machine. There has been a growing - though inconsistent - literature to suggest that features of autism spectrum conditions (ASCs) are associated with a deficit in modelling the world through such prediction-based inference. However empirical evidence for differences in low-level sensorimotor predictions in autism is still lacking. One approach to examining predictive processing in the sensorimotor domain is in the context of self-generated (predictable) as opposed to externally-generated (less predictable) effects. We employed two complementary tasks - forcematching and intentional binding - which examine self- versus externally-generated action effects in terms of sensory attenuation and intentional binding respectively in adults with and without autism. The results show that autism was associated with normal levels of sensory attenuation of internally-generated force and with unaltered temporal attraction of voluntary actions and their outcomes. Thus, our results do not support a general deficit in predictive processing in autism.

## 1. Introduction

The predictive processing framework accounts for how we deal optimally with ambiguous signals from our environment using prediction-based optimisation of inference (Teufel and Fletcher (2020); Friston and Kiebel (2009)). While initially developed as a framework to understand healthy brain function, this account also offers potential insights into the processes underlying psychiatric disorders (Moore (2015); Adams et al. (2016); Barrett et al. (2016); Sterzer et al. (2018); Gadsby and Hohwy (2019); Teufel and Fletcher (2016); Corlett and Fletcher (2014); Friston et al. (2014); Kube et al. (2020a,b); Fineberg et al. (2014)). There has been a growing interest in applying this framework to investigate differences in the cognitive, perceptual and neural processes in autism spectrum conditions (Qian and Lipkin (2011); Pellicano and Burr (2012); Sinha et al. (2014); Lawson et al. (2014); Van de Cruys et al. (2014); Rosenberg et al. (2015); van Boxtel and Lu (2013)). Much interest has been sparked by a proposal from Pellicano and Burr (Pellicano and Burr (2012)) suggesting that predictive deficits in individuals with autism are due to a diminished effect of prior expectations on the processing of ambiguous sensory information, leading to inferences that are more strongly based on sensory information. This atypicality in information processing, they speculate, could be a consequence of excessive endogenous neural noise although others have pointed out that reduced endogenous noise could yield comparable outcomes (Brock

(2012)). Alternative accounts suggest that the problem lies not in the prior expectations themselves but in altered precision of the prediction error - a key feedforward signal in the processing hierarchy (Van de Cruys et al. (2017); Lawson et al. (2014)).

Prima facie, the framework contributes a lot to understanding the characteristic clinical features of autism. For instance, it seems plausible to conjecture that deficits with the generation of predictions are at the core of difficulties with adapting to change, intolerance of uncertainty and certain sensory atypicalities in individuals with autism. Empirically, the evidence for these theories is still sparse and the idea of a global predictive impairment [...] shared across individuals (Sinha et al. (2014)) seems to be contradicted by an absence of apparent deficits in motion prediction of objects (Tewolde et al. (2018)), predictions about the weight of objects based on material cues (Arthur et al. (2019)) and other cognitive processes supposed to tap into predictive abilities (Croydon et al. (2017); Manning et al. (2017); Cruys et al. (2017); Maule et al. (2018)). Where group differences have been found, they mostly pertain to predictive deficits in the social domain (differences in neural substrates underlying prediction error coding for another's decisions (Balsters et al. (2017)), attenuated priors for inferring the intention and action of others (Chambon et al. (2017); Turi et al. (2017); Amoruso et al. (2019)) - especially when the use social context is required to disambiguate the action (von der Lühse et al. (2016))), but this is not universally true, as Pell and colleagues have found no deficits in prediction-based percep-

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tion of other people's gaze direction (Pell et al. (2016)). It is also unclear whether the observed deficits in prediction are due to low-level atypicalities in the predictive architecture or whether they might be the result of differences in other areas that prediction taps into such as the learning of action-outcome contingencies (Schuwerk et al. (2016)) and temporal processing (Brodeur et al. (2014); Szelag et al. (2004)).

In short, while a predictive processing deficit provides a credible explanatory model for features of autism, the experimental evidence is currently inconsistent and requires clarification. Moreover, many of the above tasks focus on higher level social or cognitive functions. In order to support the idea of a global prediction deficit in autism, however, a characterisation of basic mechanisms of sensory and motor prediction are mostly lacking (but see: Palmer et al. (2015)). In the current study we therefore used two complementary tasks known to index predictive processing in sensory and motor function: the forcematching task (Shergill et al. (2003)) and a modified version of the intentional binding paradigm (Moore and Haggard (2008)). We chose these tasks for two reasons: Firstly, in contrast to the higher-order cognitive paradigms mentioned above, the forcematching task focusses on basic mechanisms of sensory and motor prediction that laid the foundations for the predictive processing framework (Holst and Mittelstaedt (1950)). The IB task in turn bridges the gap between more basic predictive effects of sensory attenuation and prediction in the context of multisensory integration of volitional movements and their consequences. Secondly the tasks have robustly and reliably elicited responses in line with current views on prediction in healthy individuals and have, moreover, established the presence of altered responses in populations whose predictive architecture is conjectured to be compromised (Shergill et al. (2005); Voss et al. (2010); Synofzik et al. (2010)).

The forcematching task measures attenuation of the sensory consequences of self-generated actions. It is based on the principle of motor control theory which suggests that sensory consequences of predictable forces are anticipated and attenuated. Tasks exploring this phenomenon have reliably demonstrated that self-generated sensory consequences are perceived as weaker than externally-generated sensory consequences of the same intensity across a range of experimental paradigms, volunteers and laboratories (Wolpe et al. (2016, 2017); Shergill et al. (2003, 2005); Voss et al. (2007); Teufel et al. (2010); Walsh et al. (2011); Therrien et al. (2011); Pareés et al. (2014)).

The intentional binding (IB) effect refers to the finding that self-generated, voluntary actions and their sensory consequences are perceived to be closer together in time than movements externally forced upon the person and their sensory outcomes (Haggard et al. (2002); Prinz and Hommel (2002)). IB is thought to be an implicit measure of sense of agency (SoA) which in contrast to the sensory attenuation observed in the forcematching task, is speculated to rely both on predictive mechanisms as well as postdictive inferences. Predictive and postdictive contributions to agency have been investigated by varying the probability with which

the voluntary action produces the sensory outcome (Moore and Haggard (2008)). Moore and Haggard found that both processes operate, but that one dominates depending on the specific outcome probabilities: In blocks in which the action produced the sensory outcome with a high probability, healthy volunteers exhibited temporal binding even in the absence of the outcome, whereas temporal binding was only observed on those low outcome probability trials that did indeed produce the outcome. Based on this idea, a predictive and a postdictive component to IB have been identified in the literature (Voss et al. (2010); Moore and Haggard (2008); Moore et al. (2009)): When binding occurs in the absence of an outcome in high contingency environments, a predictive process is thought to drive the effect (that is, the tone has been predicted producing an effect that occurs even when the tone itself does not subsequently occur). Conversely if binding occurs in low contingency environments solely when an outcome (that has not been predicted) is present, a retroactive, postdictive binding to the action must be the main driving factor behind IB.

Thus, these two complementary tasks are well-suited to exploring different aspects of the predictive processing model of ASC: While the forcematching task is more likely to tap into basic predictive mechanisms of sensory gating (Chapman and Beauchamp (2006); Hughes et al. (2012)), intentional binding is thought to be largely attributable to temporal control and prediction (of the timing of the outcome). Therefore unimpaired performance on one, but not the other task would yield additional insight as to whether differences in predictive abilities in autism are more likely due to primary sensory deficits or more general issues with the timing and learning of action-outcome contingencies.

## 2. Experiment 1 - Forcematching in Autism

### 2.1. Method

#### 2.1.1. Participants

26 control participants (with no history of neurological or psychiatric illness) and 27 volunteers with a clinical diagnosis of an autism spectrum disorder took part in the study. Written informed consent was obtained from all participants. The ASC participants were recruited through an existing database, adverts with the University's Disability Resource Centre, local autism charities and the National Autistic Society.

Cognitive function for all study volunteers was assessed using the timed version of the Ravens Advanced Progressive Matrices (RAPM) (Raven et al. (1973)) and the Wechsler FSIQ in the case of one ASC volunteer. Furthermore all participants filled in the Edinburgh Handedness Inventory (Oldfield, 1971) as handedness can have an effect on force-perception and production (Park et al. (2008); Gertz et al. (2017)). On the inventory, a score of +40 reflects right-handedness and a score below -40 left-handedness.

Of the three participants with autism excluded, 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

**Table 1**  
Participant Demographics

Group	Age (SD, range)	Sex (m:f)	Handedness (SD)	IQ (SD, range)	AQ (SD, range)	PDI (SD, range)
ASC (N=24)	30.1 (9.2, 18-62)	11:13	53.8 (44.5)	105.2 (12.5, 84-131)	36.4 (7.7, 19-48)	64.9, (39.5 21-155)
Controls (N=26)	30.6 (6.0, 22-45)	9:17	75.3 (19.2)	106.8 (11.6, 82-129)	17.0 (6.6, 6-28)	41.8, (25.7 0-86)

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. (2006), Eaves and Ho (2008), White et al. (2009)). Co-morbid diagnoses of depression and/or anxiety were present in 10 of the participants with autism and 6 were currently taking selective serotonin reuptake inhibitors. 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 ( $\chi^2=0.654$ ,  $p=0.419$ ) 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 1).

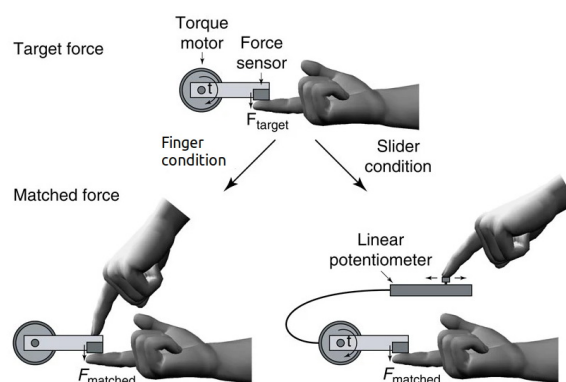
All but 3 of the ASC participants were assessed with module 4 of the Autism Diagnostic Observation Schedule (ADOS, (Lord et al., 2000)) and while the group was moderately symptomatic, only 9 participants met cut-off criteria for an autism spectrum condition and none met diagnostic criteria for autism. Low sensitivity of the ADOS module 4 has previously been reported and attributed to compensatory behaviour and milder ASDs (Bastiaansen et al. (2011)). Even among children, those with a diagnosis of an autism spectrum condition that is not childhood autism (ICD-10) often do not meet the diagnostic cut-off for the ADOS (Baird et al. (2006)).

Given previous reports of altered forcematching in individuals with high levels of schizotypy (Teufel et al. (2010)), we used the 21-item Peters Delusion Inventory (PDI, Peters and Garety (1996)) to quantify schizotypal traits in all participants. The Autism Spectrum Quotient (AQ, Baron-Cohen et al. (2001)), a 50-item self-administered questionnaire, was used as a measure of autistic traits. AQ and PDI scores were unavailable for one ASC participant. In contrast to the comparatively low ADOS scores, the mean of the AQ scores for both groups was in line with a previous meta-analysis reporting a mean score of 17 for non-autistic and 35 for autistic adults (Ruzich et al. (2015)).

### 2.1.2. Experimental Procedure

The experiment was modelled on the design by Shergill et al. (Shergill et al. (2003)) 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 equality (i.e. the point where the pressure felt the same) by either pressing directly on the lever with their right index

finger (finger condition) or by adjusting a slider which controlled the torque motor (slider condition), see Figure 1.



**Figure 1:** Illustration of the forcematching paradigm in which participants are asked to match a force applied to their left index finger via a lever, adapted from Wolpe et al. (2016). Participants had to reproduce the experienced force either by pushing down on the lever with their other index finger or by moving a slider.

As a result of forward prediction models for self-generated movements, participants routinely exceed the target force in the finger condition due to sensory attenuation, whereas the lack of a good forward model for the indirect control of the lever via the slider leads to a more accurate reproduction of the force.

The slider was a potentiometer which transduced a force gain at the ratio of 0.5 N/cm. 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. (2007)). Within each condition 10 different force magnitudes between 0.5N and 2.75N, differing in steps of 0.25N were applied in randomised order. Each force magnitude was presented for a total of 8 trials. Subjects first completed a 5-trial practice session for both conditions to ensure that they understood the task and were able to respond within the required time window. They then completed one finger and one slider 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.

### 2.1.3. Data Analysis

One ASC participant was excluded from further analysis as their performance in the finger condition was more than 9 standard deviations above the mean.

Basic force attenuation was indexed by calculating an overcompensation score based on the difference between the matched forces in the finger and slider condition (each normalised against the passively experienced force to account for small variations in the applied force) for each force level (see Humpston et al. (2017)). Individual regression lines of target force versus matched force for each subject were fitted for the finger and slider condition and then summarised as group regressions for both conditions. In addition to the basic overcompensation score, the slope and intercept of the regression lines can provide more detailed information about the matching performance of different groups (Wolpe et al. (2016)). Specifically the intercept in the finger condition is thought to represent the effect of the efference copy of the motor command (Wolpe et al. (2016)), whereas the slope in the slider condition more likely reflects perceptual sensitivity. Thus the finger intercept will be used as the main measure of predictive sensory attenuation.

Group differences were evaluated with Bayesian estimation using Markov Chain Monte Carlo methods to generate samples of the relevant posterior distributions. JAGS (Plummer (2003)) was implemented to build a Gibbs sampler and the default non-informative priors of the R package *BEST* (Kruschke (2013)) were implemented. The data is assumed to follow a t-distribution in *BEST* with  $\nu$  ( $1-\infty$ ) degrees of freedom controlling the width of the tails and thus acting as a measure of normality. The wide priors make the estimation of the posterior parameters (mean(s)  $\mu$ , standard deviation(s)  $\sigma$  and the shared normality parameter  $\nu$ ) very data driven. Convergence was assumed as long as the Brooks-Gelman-Rubin scale reduction factor (Gelman and Rubin (1992); Brooks and Gelman (1998)) was  $<1.1$ . The default values for number of chains (=100002) and burn-in trials (=1000) were retained. Bayesian correlations were calculated using the *BayesianFirstAid* package in R.

## 3. Results

Both groups showed the characteristic force attenuation with the posterior estimates of the mean overcompensation scores being 0.73 (credible interval/CI: [0.51, 1.00], estimated effect size: 1.58) and 0.80 (CI: [0.52, 1.10], estimated effect size: 1.33) for the control and autism group respectively. Handedness was unlikely to be associated with the magnitude of sensory attenuation (as measured by the overcompensation score) with an estimated correlation of  $r=-0.16$  and a 95% CI of [-0.45, 0.16] and was not included in the model.

Plotting the mean linear regressions for matched forces in the finger and slider conditions did not suggest any group differences (Figure 2a). Congruously, Bayesian estimation

yielded little evidence for a group difference on the means of overcompensation scores (estimated difference of means: -0.03, CI: [-0.37, 0.31], estimated effect size: -0.08, Figure 2b) or intercept (estimated difference of means: -0.04, CI: [-0.39, 0.32], estimated effect size: -0.09, Figure 2c) of the finger condition.

For a more in-depth view at these measures see Appendix A.

### 3.0.1. Questionnaire Measures

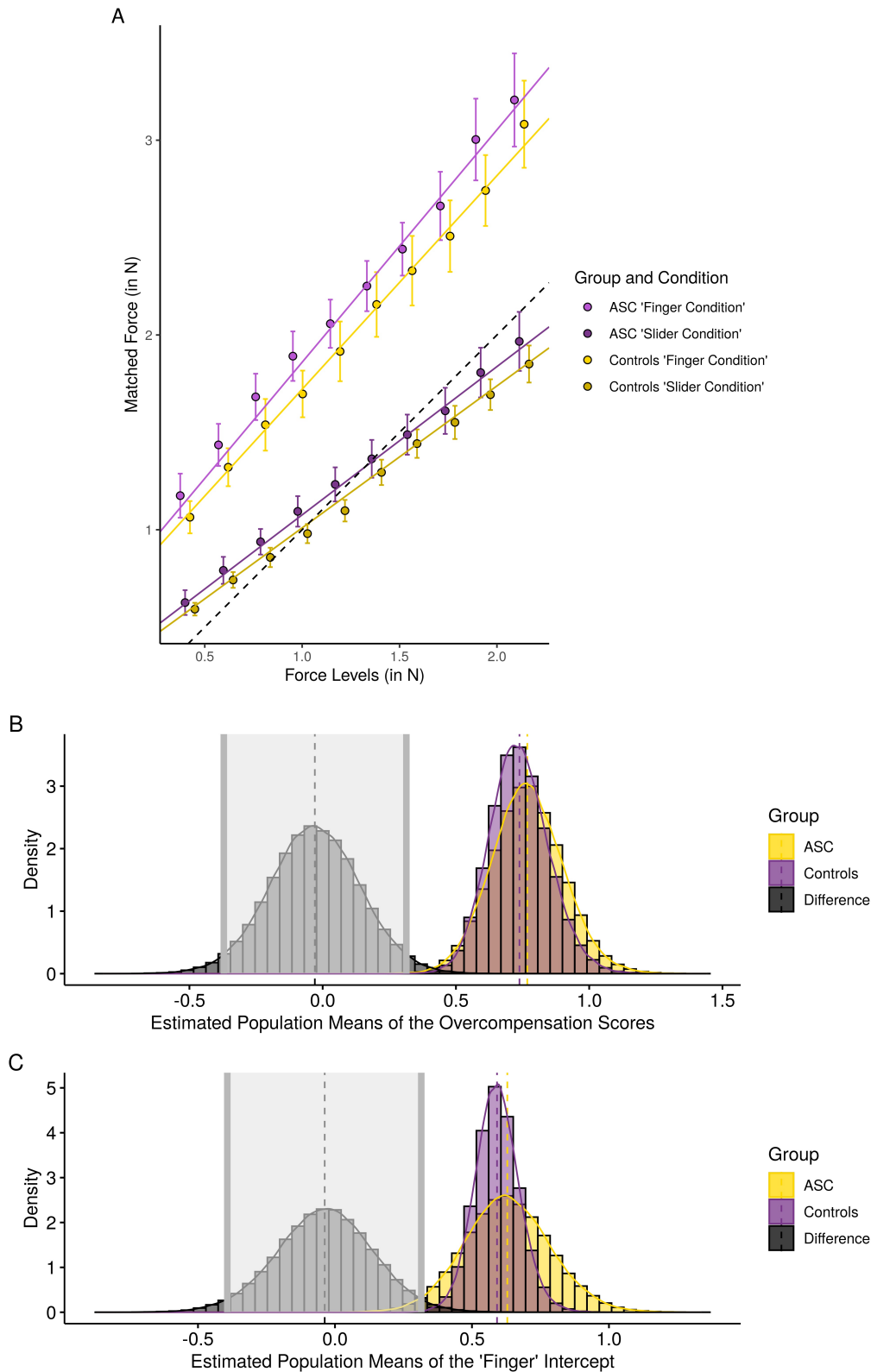
As expected, posterior estimates for group means on the AQ indicated a difference between the ASC and control group (estimated difference of means: -19.49, CI: [-24.03, -15.06], estimated effect size: -2.62) and perhaps more surprisingly there was also evidence in favour of the true group difference in means on the PDI being non-zero (estimated difference of means: -21.50, CI: [-42.22, -0.58], estimated effect size: -0.65) (Figure 3a).

In a separate analysis, using the intercept in the finger condition as the main measure of sensory attenuation (see: Wolpe et al. (2016)), we found - in line with previous observations (Teufel et al. (2010); but see: Humpston et al. (2017)) - that the probability that sensory attenuation has a negative relationship with schizotypy in the control group was 98% (estimated correlation: -0.41, CI: [-0.73, -0.07]), whereas evidence in the ASC group suggested no significant relationship (estimated correlation: 0.04, CI: [-0.40, 0.45]). This difference was statistically significant ( $Z = 1.783$ ,  $p = 0.037$ ). Conversely there did not seem to be an association between self-reported autistic traits on the AQ and sensory attenuation in the control group (estimated correlation: -0.01, CI: [0.42, 0.40]), but a trend for a positive relationship in the ASC group (estimated correlation: 0.36, CI: [-0.03, 0.70]), see Figure 3b and 3c.

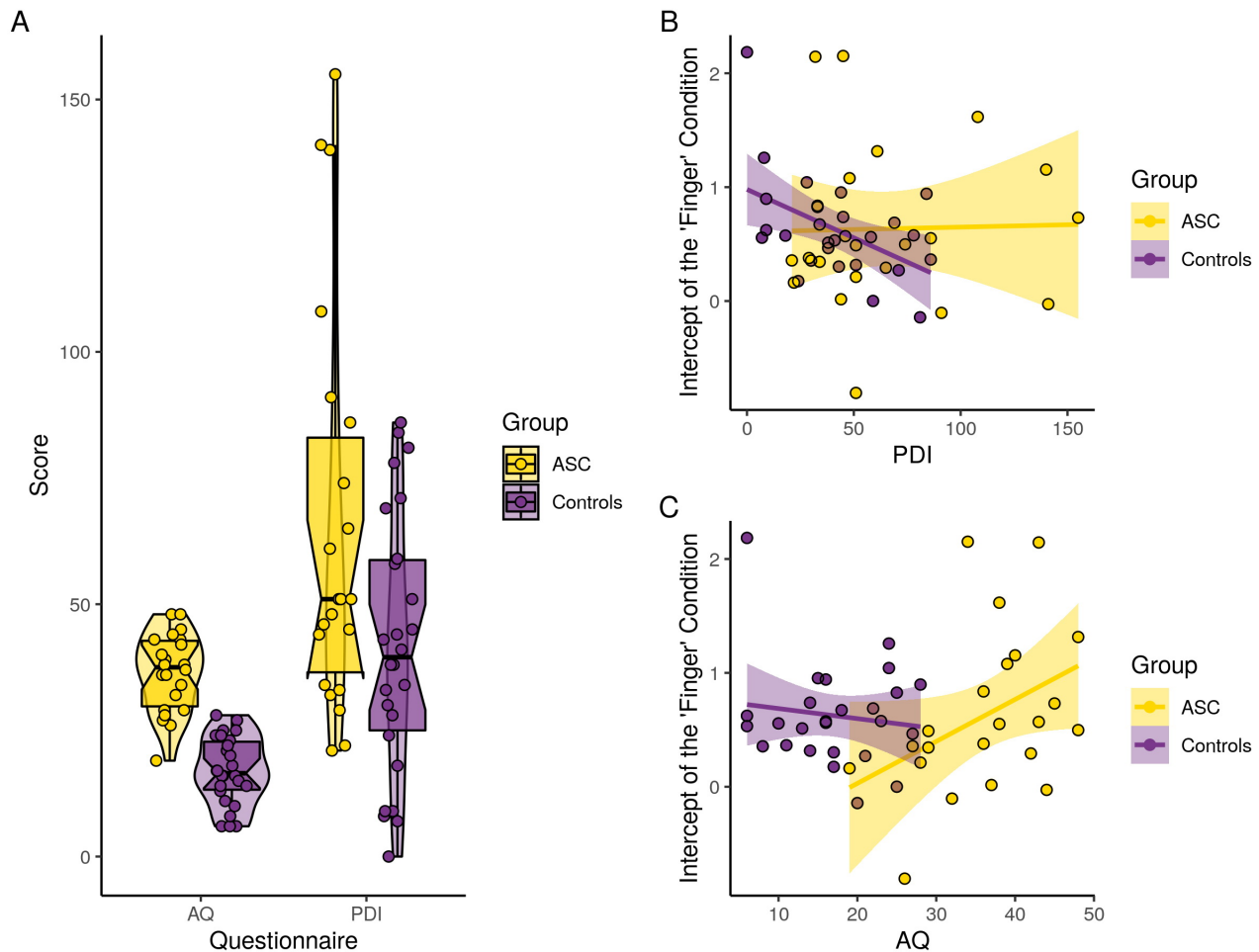
### 3.0.2. Summary

Overall, we found no evidence of a deficit in the attenuation of self-produced sensory consequences in autism, which is in contradiction of existing predictive processing models of the condition. A Bayesian analysis supported an absence of group differences in key measures of sensory attenuation. Interestingly, not only AQ (as predicted) but also a measure related to schizotypy (PDI) was higher in the ASC group. Moreover, in line with previous work, correlative analyses of sensory attenuation with schizotypy showed an expected negative relationship in control participants. No such correlation was found in ASC. However as the ASC group scored higher on the PDI, it is possible that the observed relationship for PDI scores in the control group simply does not hold for higher scores and thus the result does not necessarily indicate that sensory attenuation is influenced by different latent traits in the two groups. Conversely, AQ scores in the autism group seemed to have a positive association with sensory attenuation.

## Unimpaired Prediction in Autism



**Figure 2:** Main results for the forcematching task. **(A)** Mean linear regressions for the matched forces in the finger and slider conditions. Jitter was added to prevent overplotting. Error bars represent  $\pm 1$  standard error (SE) of the mean. Perfect matching performance is indicated by the dashed black line. **(B)** A plot of the posterior probability of the difference in means for the overcompensation score (black) with the estimated population means in yellow and purple respectively. The shaded area is the credible interval (CI), in this case the 95% Highest Density Interval (HDI) **(C)** Posterior probability of the difference in means for the intercept in the finger condition.



**Figure 3:** Results for the Questionnaire measures. **(A)** Plot showing the distribution of the questionnaire scores for both groups, including the median and interquartile ranges. **(B)** The correlation between sensory attenuation (as measured by the intercept in the finger condition) and the PDI. **(C)** The correlation between sensory attenuation (as measured by the intercept in the finger condition) and the AQ.

## 4. Experiment 2 - Intentional Binding in Autism

### 4.1. Method

#### 4.1.1. Participants

A total of 50 participants (25 per group) were recruited for the study. Written informed consent was obtained from all participants. Twenty-one of the control participants and all but one of the ASC volunteers also took part in experiment 1 and thus the same two volunteers with a history of psychosis were excluded.

Participants were matched for age, IQ (IQ information was unavailable for two control participants) and gender (see Table 2).

#### 4.1.2. Experimental Procedure

The basic structure of the task was similar to other intentional binding experiments (Haggard et al. (2002)): Participants were instructed to press a key with their right index finger at a time of their own choosing which could cause a

tone 250ms later. While they were engaged in this task, a Libet clock (Libet et al. (1993)) was visible in the middle of the screen with a clock-hand rotating at a rate of 2560ms per revolution.

For each block, participants were either instructed to recall the time at which they pressed the key (i.e. recall where the clock-hand was pointing to when they performed the key-press) or to enter the clock-hand's position when they heard the tone. The basic structure of the block remained the same and the instructions given at the start of each block were the only difference. Participants were informed of the response requirement (time estimation of the key press or tone occurrence) immediately prior to the blocks which otherwise did not differ visually from each other. After the keypress, the clock-hand continued to rotate for a random amount of time (which was sampled from a uniform distribution over all integer values between 1500 and 2500ms) in order to prevent participants from using the stopping point to infer the time of their keypress. Participants were told to avoid pressing at

**Table 2**  
Participant Demographics for the Intentional Binding Task

Group	Age ( <i>SD</i> , range)	Sex ( <i>m:f</i> )	IQ ( <i>SD</i> , range)	AQ ( <i>SD</i> , range)	PDI ( <i>SD</i> , range)
ASC ( <i>N</i> =23)	29.0 (6.1, 20-38)	11:12	105.2 (12.7, 84-131)	35.6 (7.6, 19-48)	62.6 (35.3, 21-155)
Controls ( <i>N</i> =25)	31.2 (5.7, 22-45)	10:15	104.6 (10.6, 82-122)	17.1 (7.0, 6-32)	45.0 (24.7, 7-86)

premeditated clock positions.

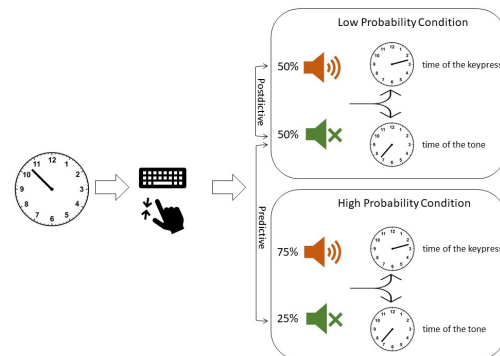
The difference between the true time of the key presses and the estimated time was used as the measure of intentional binding, hereafter called shift. By convention, binding for actions is indicated by a positive difference. The tone blocks were used so participants would still pay attention to the outcome of their actions and to adhere as closely as possible to the standard IB paradigm.

As in Moore's adapted version (Moore and Haggard (2008)), the probability of the tone occurring was manipulated: In half of the blocks the tone followed the key press 50% (low probability condition) of the time while in the other half it happened 75% (high probability condition) of the time (see Figure 4). When no tone occurred in the blocks where participants were asked to report the timing of the tone, a dummy value was reported instead. The order of blocks was randomised for each participant. In addition to 8 experimental blocks (4 per probability condition), the volunteers also completed a baseline task requiring them to judge the time of their key presses without any subsequent tone so that a baseline estimation error can be estimated for each individual. For the baseline condition participants were told that no tone would occur.

Based on Voss et al. (2010), the predictive component to the intentional binding effect was calculated as the difference in overall shift between no tone trials (i.e. the trials where no tone occurred) in the high probability blocks and no tone trials in the low probability blocks (no tone trials (75%) – no tone (50%)), see Figure 4. 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 75% probability blocks. Analogously the postdictive contribution was defined as the difference between no tone trials in the 50% blocks and the tone trials in the 50% blocks. The authors describe the 50% contingency as subjectively random, so participants should not be able to form helpful predictions about the occurrence of the tone on any given trial as it is equally likely to be present and absent. Therefore a larger binding effect for the trials where a tone occurs compared to no tone trials must be due to a postdictive component that acts on the temporal estimation process as a consequence of the outcome.

#### 4.1.3. Data Analysis

The analysis followed the typical protocol for IB studies. Initially, responses (with the exception of the baseline itself) were corrected against the mean shift 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



**Figure 4:** Illustration of the experimental procedure for IB with varying outcome contingencies. On some blocks the participants' button presses were followed by a tone 50% of the time (low probability condition), whereas for other blocks the tone followed the button press 75% (high probability condition). Participants were instructed for each block and had to estimate either the time of the key press or the time of the tone. The predictive component was calculated as the difference between the no tone trials (green) in the high and low probability conditions whereas the postdictive component was defined as the difference between the tone (orange) and no tone trials in the low probability condition.

to learn the contingencies.

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

## 5. Results

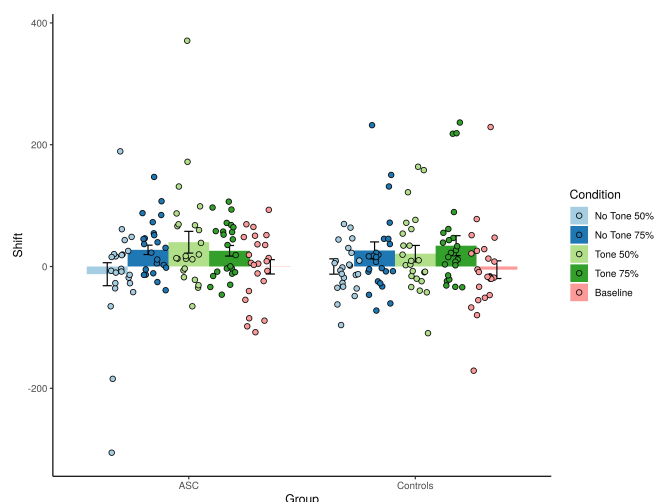
### 5.0.1. Basic Intentional Binding Effect

The resulting pattern resembled Moore and Haggard's (2008) results where intentional binding was observed in all conditions apart from the low-probability no-tone trials (see Figure 5).

### 5.0.2. Group Comparison on Predictive and Postdictive Components of Intentional Binding

Examining both predictive (estimated difference of means: -13.8, CI: [-65.4, 37.9], estimated effect size: -0.17, Figure 6a) and postdictive (estimated difference of means: -8.6, CI:





**Figure 5:** Baseline-corrected shift in the action estimates (ms) for each probability block in the no tone and tone conditions. Error bars represent  $\pm 1$  standard error (SE) of the mean.

[-58.8, 41.9], estimated effect size: -0.11, Figure 6b) conditions using Bayesian estimation showed that group differences in these parameters were unlikely.

### 5.0.3. Relationship between the Questionnaire Measures and Intentional Binding

There was little evidence that the AQ or PDI correlated with any of the measures: For the ASC group, the estimated correlation between the PDI and the predictive component was 0.07 (CI: [-0.37, 0.5]) and between the AQ and the predictive component -0.22 (CI: [-0.62, 0.23]). For the control group the analogous correlations were estimated to be 0.23 (CI: [-0.19, 0.62]) and 0.07 (CI: [-0.34, 0.48]) respectively. The postdictive component did not seem to be associated with the AQ (estimated correlation: -0.15, CI: [-0.57, 0.28]) or PDI (estimated correlation: 0.1, CI: [-0.35, 0.54]) in the ASC group. The same was also true for the control group: (AQ: estimated correlation: 0.04, CI: [-0.38, 0.46]; PDI: estimated correlation: 0.21, CI: [-0.21, 0.6]).

### 5.0.4. Relationship between the Forcematching and IB tasks

Due to the fact that several of the control and ASC participants took part in both experiments, 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. However comparing the predictive component of the IB task and the finger intercept of the forcematching task yielded no significant correlations ( $r=0.051$ ,  $p=0.750$  across groups and  $r=0.059$ ,  $p=0.806$  for the ASC and  $r=0.268$ ,  $p=0.240$  in the control group).

### 5.0.5. Summary

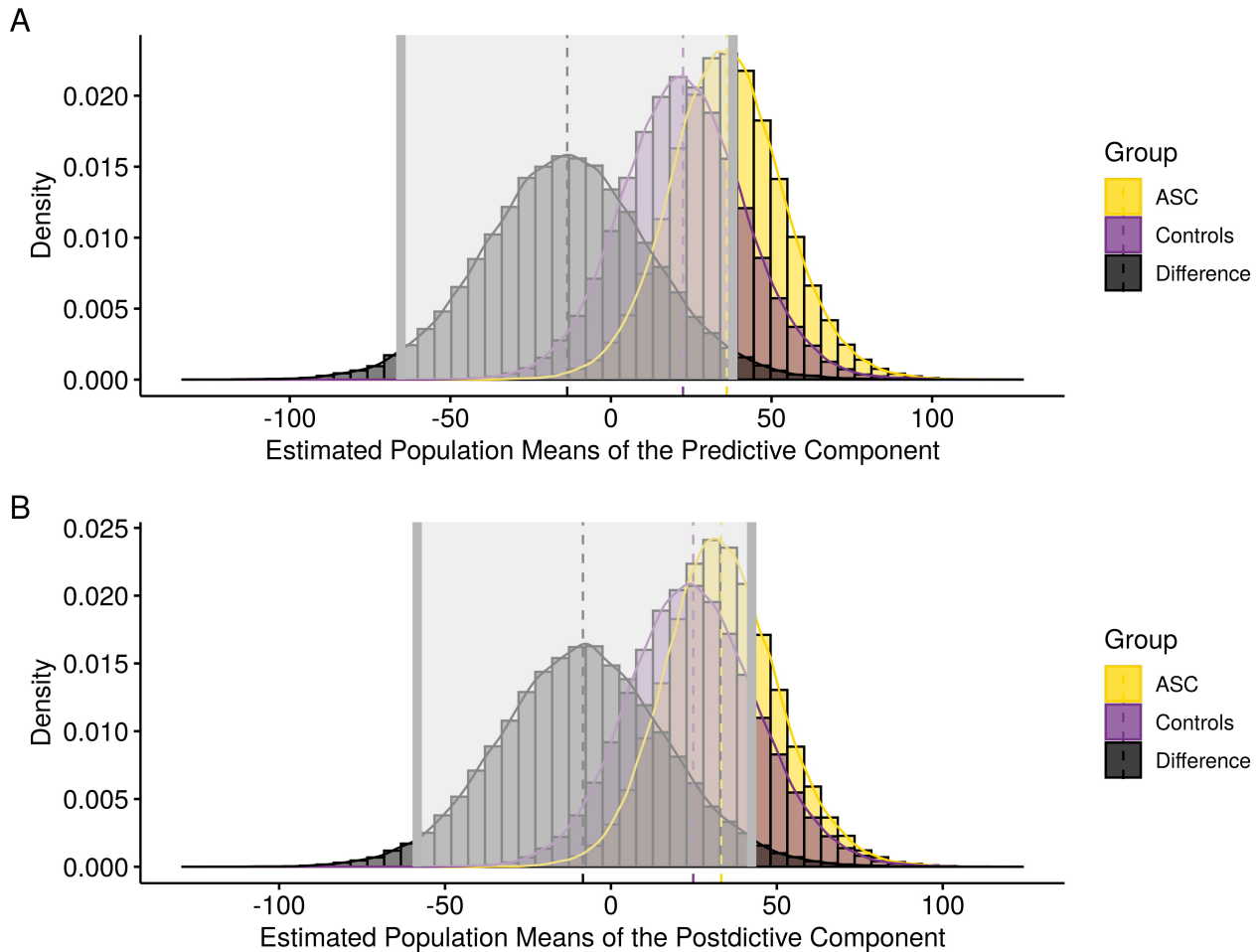
Overall, therefore, in keeping with the findings from the force-matching task in experiment 1, we found no group difference in intentional binding. Both groups showed expected reductions in the subjective experience of action-outcome

timing in both the predictive (tone expected, but absent) and postdictive (tone unexpected but present) conditions. Furthermore we found no association between performance on the predictive components of the forcematching and IB tasks confirming another recent finding by Tulver et al. (2019) who found no correlation between performance on several experimental paradigms 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. Discussion

In the past decade, a number of prominent hypotheses have suggested that autism is primarily a disorder of atypical predictive processes and that the range of alterations, particularly in perceptual experiences can be explained in terms of these atypicalities. However the empirical evidence supporting these hypotheses in the form of differences in low-level sensorimotor prediction has been lacking which led us to investigate sensory attenuation and agency-based temporal binding in adults with autism. In light of this theoretical work conceptualising autism as a disorder of prediction (Sinha et al. (2014)), one would expect to find reduced perceptual attenuation in the autistic group and a reduction of the predictive component to the intentional binding effect. Neither of these observations were made and our experiments do not support the idea of a deficit in predictive processing in autism. Despite relatively small group sizes, both ASC and control groups demonstrated sensory attenuation of self-generated stimuli with a magnitude consistent with previously reported results (Teufel et al. (2010); Shergill et al. (2003); Wolpe et al. (2016)) and both groups exhibited the basic pattern of inferential and predictive binding reported by Moore and Haggard (2008). Based on the average effect size ( $d = 0.93$ ) of available studies (Shergill et al. (2005); Pareés et al. (2014)) comparing clinical and non-clinical groups on the forcematching task, a power calculation with  $\alpha = 0.05$  and at a power of 0.9 yields a required sample size of  $N=26$  per group which is similar to our recruited sample. Individual variability is a known issue of IB paradigms, but the initial effect was demonstrated with just 10 participants (Moore and Haggard (2008)) and our trial numbers were significantly larger than those of previous papers (see Sperduti et al. (2014)). These findings indicate that global deficits in predictive processing cannot explain the observed cognitive, perceptual and motor differences in autism spectrum conditions.

However, one interesting group difference that emerged lay in the within-group relationship between odd or unusual beliefs, as measured by PDI and the magnitude of sensory attenuation. While we replicated the previous finding that an increase in the number of delusion-like beliefs was associated with more accurate force-matching (i.e. reduced sensory attenuation) in the control group, this relationship



**Figure 6:** Posterior distributions for the difference in estimated population means of the predictive (A) and postdictive (B) component of IB. The shaded area is the 95% Highest Density Interval (HDI).

was not seen in autism. However there was some preliminary evidence that higher autistic traits in autistic individuals could be related to an increase in sensorimotor prediction as indicated by increased sensory attenuation. The lack of correlation between attenuation and PDI in the autism group is intriguing. One possibility is that the PDI and AQ questionnaires do not measure the same underlying traits in autism as in controls (Murray et al. (2014)) which would caution against the use of the AQ to quantify and qualify intermediate endophenotypes or the use of "high AQ" scorers as a proxy for autism (Kitazoe et al. (2017); Gregory and Plaisted-Grant (2016)). An alternative explanation would be that sensory attenuation is indeed modulated by different latent traits in autistic and non-autistic individuals.

Compared to the schizophrenia literature, evidence for disruptions of sensory gating and agency processing in autism is scant: 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. (2006)) and adults with autism are just as good as their matched controls at judging agency based on whether visual feed-

back matched their own hand movements or not (David et al. (2008)). In contrast, Zalla et al. (2015) showed a decreased use of sensorimotor cues in making judgments of agency in adults with autism which was correlated with performance on a Theory of Mind 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. Preliminary studies on interoceptive deficits in autism seem to support this claim (Noel et al. (2018); Garfinkel et al. (2016)). Similarly, Zalla and Sperduti (2015) suggest that autism is characterised by an isolated impairment of predictive (but not postdictive) processes in the genesis of sense of agency. 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. (2014)). In light of our diverging results the differences between the two experiments 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. (2002); Maister and Plaisted-Grant (2011)), impeded performance for the ASC group. As Maister and Plaisted-Grant (2011) 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 and thus might elude being captured by the proportion error scores used in Sperduti et al. (2014). Other differences between the two studies include the smaller (N=15 for the autism group) 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 ~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.

The lack of phenotyping for sensory reactivity and abnormalities is certainly a caveat of the present study and could be addressed more thoroughly in future investigations. Detailed assessments 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. (2014)) and it is possible that differences in predictive abilities might be domain-specific. As predictive attenuation is not unique to the tactile domain (Benazet et al. (2016), Cardoso-Leite et al. (2010), Desantis et al. (2012), Hughes and Waszak (2011), Dinstein et al. (2010)), 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. (1981)), 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. (2016)).

A further limitation of the experiments presented here 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. Since structural priors are likely to either emerge from long-term aggregation of individual experiences or as embedded constraints acting on bottom-up processes (Teufel and Fletcher (2020)) - as opposed to the short-term learning of stochastic relationships for contextual priors - they supposedly are subject to developmental processes. As such the force-matching task would be the best candidate for a developmental approach to predictive coding

paradigms.

An obvious limitation of the study is the fact that a substantial amount of participants would not have qualified for a diagnosis based on behavioural observation tools alone given the low ADOS scores. 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. It is possible that predictive processing difficulties might be expressed only at higher levels. However, this idea seems implausible given that, if anything, the relationship between AQ scores and predictive processing pointed in the opposite direction: in the force-matching task, high autistic traits were related to performance that is indicative of enhanced, not reduced predictive processing. Second, the theoretical framework on which this study is based suggests that predictive processing difficulties are a core aspect of autism. The fact that we found no evidence to suggest such atypicalities in our sample challenges the idea that they are a core feature of autism, even if they would emerge at higher levels of symptoms.

Our study aimed to explore the predictive abilities of individuals with autism in two motor tasks that are thought to be subserved by partially overlapping, but different neural mechanisms. Previous efforts to investigate predictive processing in autism have yielded inconclusive results (mostly supporting aberrant prediction in the social domain), despite a comparatively large theoretical literature. Our present study militates against the idea of a general prediction deficit in autism as results indicate intact predictive and postdictive mechanisms of sensory attenuation and temporal attraction between actions and action outcomes. However results hinted at more subtle differences in the relationships between latent traits of schizotypy/autism and task performance in the two groups which illustrates the need to consider potential discrepancies in specific domains or subgroups.

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## A. Appendix - Additional Analyses for the Forcematching Task

### A.1. Slope and Intercept Measures

Bayesian estimation of the parameters for the slope of the regression for the finger condition did not indicate any

group differences (estimated difference of means:  $-0.04$ , CI:  $[-0.35, 0.27]$ , estimated effect size:  $-0.10$ ).

For the slider condition, Bayesian estimation equally did not suggest any group differences for the slope (estimated difference of means:  $-0.01$ , CI:  $[-0.18, 0.16]$ , estimated effect size:  $-0.04$ ), which has been interpreted as a measure of sensory sensitivity (Wolpe et al. (2016)).

## A.2. Noise and Precision

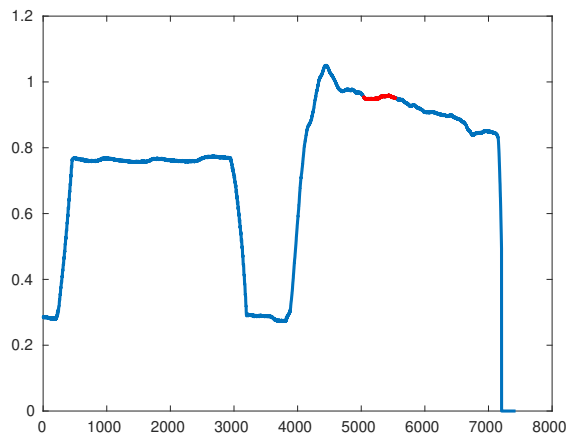
In line with previous research (Wang et al. (2017)), autistic participants demonstrated subtle motor deficits in force control as evidenced by noisier responses across both conditions: Mean-squared errors (MSE) for individual regression lines were computed (estimated difference of means:  $-0.18$ , CI:  $[-0.34, -0.02]$ , estimated effect size:  $-0.78$ , Figure 8a).

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.

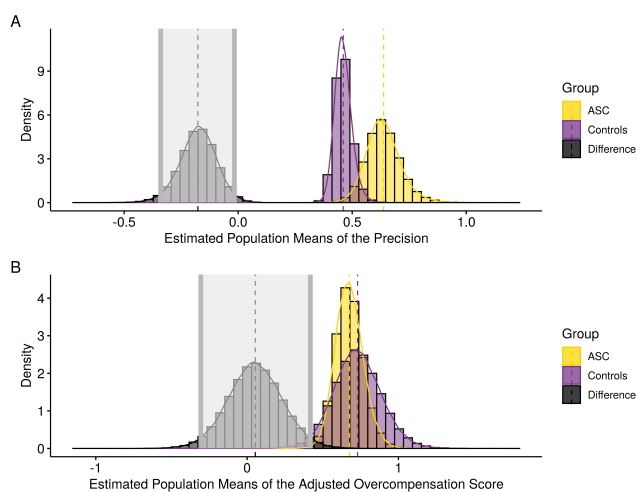
Thus, in an attempt to minimise the effect of the shape of individual force traces, an alternative measure was adapted from Wolpe et al. (2016) which searched for the 0.5 second time window with the least amount of variability on each trial (see figure 7). The time to stabilise, that is to say the time between the onset of movement and the calculated time window, did not differ between groups in either the finger (estimated difference of means:  $-32.2$ , CI:  $[-111, 44.2]$ , estimated effect size:  $-0.287$ ) or slider (estimated difference of means:  $-113$ , CI:  $[-245, 15.4]$ , estimated effect size:  $-0.53$ ) condition.

With this adjustment, no difference in the overcompensation scores could be detected (estimated difference of means:  $0.055$ , CI:  $[-0.31, 0.42]$ , estimated effect size:  $0.34$ ), see Figure 8b. We can thus conclude that the observed lack of a group difference is unlikely to be due to more noisy responses in the autism group.

The increased force variability seen in the autism group is still noteworthy as differences in motor control have been associated with other cognitive and perceptual differences in autism (Lindor et al. (2019); Nebel et al. (2016); Dziuk et al. (2007)). Furthermore 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. (2014); Collins et al. (2003); Magalhães and Kohn (2012); Kouzaki et al. (2012)). Support for the low endogenous neural noise hypothesis (Davis and Plaisted-Grant (2015)) 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 else-



**Figure 7:** Example of the new sampling method on a force trace with a downward drift (new time window in red)



**Figure 8:** (A) Posterior probability distribution of the difference of means for individual MSEs. (B) Posterior probability distribution of the difference of means for the adjusted Overcompensation Score.

where such as in the coloured noise associated with the neuromuscular periphery (Davids et al. (2006)). Additionally as the performance advantage of white Gaussian noise does not seem to be age-dependent (Deutsch and Newell (2001)), one can be sure that any group differences are not the result of a developmental delay in motor control.

## CRedit authorship contribution statement

**Johanna J. S. Finnemann:** Conceptualisation, Formal Analysis, Investigation, Methodology, Writing - Original Draft. **Kate Plaisted-Grant:** Conceptualisation, Supervision. **James Moore:** Methodology, Software. **Christoph Teufel:** Methodology, Supervision, Writing - Review Editing. **Paul C. Fletcher:** Methodology, Supervision, Writing - Review Editing.

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