

# 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 - force-matching and intentional binding - which examine self- versus externally-generated action effects in terms of sensory attenuation and attentional 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.

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## 1. Introduction

1     The predictive processing framework accounts for how we deal optimally  
2     with ambiguous signals from our environment using prediction-based opti-  
3     misation of inference (Teufel and Fletcher [1], Friston and Kiebel [2]). While  
4     initially developed as a framework to understand healthy brain function,  
5     this account also offers potential insights into the processes underlying psy-  
6     chiatric disorders (Moore [3], Adams et al. [4], Barrett et al. [5], Sterzer et al.  
7     [6], Gadsby and Hohwy [7], Teufel and Fletcher [8], Corlett and Fletcher

8 [9], Friston et al. [10], Kube et al. [11, 12], Fineberg et al. [13]). There has  
9 been a growing interest in applying this framework to investigate differences  
10 in the cognitive, perceptual and neural processes in autism spectrum condi-  
11 tions (Qian and Lipkin [14], Pellicano and Burr [15], Sinha et al. [16], Lawson  
12 et al. [17], Van de Cruys et al. [18], Rosenberg et al. [19], van Boxtel and  
13 Lu [20]). Much interest has been sparked by a proposal from Pellicano and  
14 Burr [15] suggesting that predictive deficits in individuals with autism are  
15 due to a diminished effect of prior expectations on the processing of am-  
16 biguous sensory information, leading to inferences that are more strongly  
17 based on sensory information. This atypicality in information processing,  
18 they speculate, could be a consequence of excessive endogenous neural noise  
19 although others have pointed out that reduced endogenous noise could yield  
20 comparable outcomes (Brock [21]). Alternative accounts suggest that the  
21 problem lies not in the prior expectations themselves but in altered precision  
22 of the prediction error - a key feedforward signal in the processing hierarchy  
23 (Van de Cruys et al. [22], Lawson et al. [17]).

24 Prima facie, the framework contributes a lot to understanding the char-  
25 aracteristic clinical features of autism. For instance, it seems plausible to con-  
26 jecture that deficits with the generation of predictions are at the core of  
27 difficulties with adapting to change, intolerance of uncertainty and certain  
28 sensory atypicalities in individuals with autism. Empirically, the evidence for  
29 these theories is still sparse and the idea of a global “predictive impairment  
30 [...] shared across individuals” (Sinha et al. [16]) seems to be contradicted  
31 by an absence of apparent deficits in motion prediction of objects (Tewolde  
32 et al. [23]), predictions about the weight of objects based on material cues  
33 (Arthur et al. [24]) and other cognitive processes supposed to tap into predic-  
34 tive abilities (Croydon et al. [25], Manning et al. [26], Cruys et al. [27], Maule  
35 et al. [28]). Where group differences have been found, they mostly pertain  
36 to predictive deficits in the social domain: Balsters et al. [29], Chambon  
37 et al. [30], Turi et al. [31], Amoruso et al. [32], von der Lühne et al. [33], but  
38 this is not universally true, as Pell and colleagues have found no deficits in  
39 prediction-based perception of other people’s gaze direction (Pell et al. [34]).  
40 It is also unclear whether the observed deficits in prediction are due to low-  
41 level atypicalities in the predictive architecture or whether they might be  
42 the result of differences in other areas that prediction taps into such as the  
43 learning of action-outcome contingencies (Schuwerk et al. [35]) and temporal  
44 processing (Brodeur et al. [36], Szelag et al. [37]).

45 In short, while a predictive processing deficit provides a credible explana-

46 tory model for features of autism, the experimental evidence is currently  
47 inconsistent and requires clarification. Moreover, all of the paradigms men-  
48 tioned above tap into higher-order perceptual and cognitive functions. In  
49 order to support the idea of a global prediction deficit in autism, how-  
50 ever, a characterisation of basic mechanisms of sensory and motor prediction  
51 are currently lacking. These basic predictive mechanisms initially laid the  
52 foundations for the predictive processing framework (Holst and Mittelstaedt  
53 [38], Helmholtz [39]) but, surprisingly, have not been studied in ASD. In the  
54 current study we therefore used two complementary tasks known to index  
55 predictive processing in basic sensory and motor function: the forcematch-  
56 ing task (Shergill et al. [40]) and a modified version of the intentional binding  
57 paradigm (Moore and Haggard [41]). We chose these tasks for two reasons:  
58 Firstly, in contrast to the higher-order cognitive paradigms mentioned above,  
59 both experiments focus on basic mechanisms of sensory and motor predic-  
60 tion that laid the foundations for the predictive processing framework ([38]).  
61 Secondly the tasks have robustly and reliably elicited responses in line with  
62 current views on prediction in healthy individuals and have, moreover, es-  
63 tablished the presence of altered responses in populations whose predictive  
64 architecture is conjectured to be compromised (Shergill et al. [42], Voss et al.  
65 [43], Synofzik et al. [44]).

66 The forcematching task measures attenuation of the sensory consequences  
67 of self-generated actions. It is based on the principle of motor control theory  
68 which suggests that sensory consequences of predictable forces are anticipated  
69 and attenuated. Tasks exploring this phenomenon have reliably demon-  
70 strated that self-generated sensory consequences are perceived as weaker  
71 than externally-generated sensory consequences of the same intensity across  
72 a range of experimental paradigms, volunteers and laboratories (Wolpe et al.  
73 [45, 46], Shergill et al. [40, 42], Voss et al. [47], Teufel et al. [48], Walsh et al.  
74 [49], Therrien et al. [50], Pareés et al. [51]).

75 The intentional binding (IB) effect refers to the finding that self-generated,  
76 voluntary actions and their sensory consequences are perceived to be closer  
77 together in time than movements externally forced upon the person and their  
78 sensory outcomes (Haggard et al. [52], Prinz and Hommel [53]). IB is thought  
79 to be an implicit measure of sense of agency (SoA) which in contrast to the  
80 sensory attenuation observed in the forcematching task, is speculated to rely  
81 both on predictive mechanisms as well as post-hoc inferences. Predictive  
82 and postdictive contributions to agency have been investigated by varying  
83 the probability with which the voluntary action produces the sensory out-

84 come (Moore and Haggard [41]). Moore and Haggard found that both pro-  
85 cesses operate, but that one dominates depending on the specific outcome  
86 probabilities: On trials, on which the action produced an outcome with a  
87 high probability, healthy volunteers exhibited temporal binding even in the  
88 absence of the outcome, whereas subjective temporal compression was only  
89 observed on those low “outcome probability” trials that did indeed produce  
90 the outcome.

91 Thus, these two complementary tasks are well-suited to exploring different  
92 aspects of the predictive processing model of ASC: While the forcematching  
93 task is more likely to tap into basic predictive mechanisms of sensory gat-  
94 ing (Chapman and Beauchamp [54], Hughes et al. [55]), intentional binding  
95 is thought to be largely attributable to temporal control and prediction (of  
96 the timing of the outcome). Therefore unimpaired performance on one, but  
97 not the other task would yield additional insight as to whether differences in  
98 predictive abilities in autism are more likely due to primary sensory deficits  
99 or more general issues with the timing and learning of action-outcome con-  
100 tingencies.

## 101 **2. Experiment 1 - Forcematching in Autism**

### 102 *2.1. Method*

#### 103 *2.1.1. Participants*

104 27 volunteers with a clinical diagnosis of an autism spectrum disorder and  
105 26 healthy control participants (with no history of neurological or psychiatric  
106 illness) took part in the study. Written informed consent was obtained from  
107 all participants. Cognitive function for all study volunteers was assessed using  
108 the timed version of the Ravens Advanced Progressive Matrices (RAPM)  
109 (Raven et al. [56]) and the Wechsler FSIQ in the case of one ASC volunteer.  
110 Furthermore all participants filled in the Edinburgh Handedness Inventory  
111 [57] as handedness can have an effect on force-perception and production  
112 (Park et al. [58], Gertz et al. [59]). On the inventory, a score of +40 reflects  
113 right-handedness and a score below -40 left-handedness.

114 3 ASC participants were excluded from the subsequent analysis as two  
115 had a diagnosis of schizophrenia or another psychotic disorder and one was  
116 unable to complete the experiment due to difficulties with maintaining the  
117 required arm posture. Aside from psychotic disorders no other psychiatric  
118 conditions served as exclusion criteria as anxiety, depression, OCD and other  
119 neurodevelopmental disorders such as ADHD and dyspraxia are thought to

**Table 1:** Participant Demographics

<b>Group</b>	<b>Age (<i>SD</i>)</b>	<b>Sex (<i>m:f</i>)</b>	<b>Handedness (<i>SD</i>)</b>	<b>IQ (<i>SD</i>)</b>
ASC ( <i>N=24</i> )	30.1 ( <i>9.2</i> )	11:13	53.8 ( <i>44.5</i> )	105.2 ( <i>12.5</i> )
Controls ( <i>N=26</i> )	30.6 ( <i>6.0</i> )	9:17	75.3 ( <i>19.2</i> )	106.8 ( <i>11.6</i> )

120 be extremely common/co-morbid in ASC (for prevalence estimates see Leyfer  
121 et al. [60], Eaves and Ho [61], White et al. [62]). 10 of the participants with  
122 autism had co-morbid diagnoses of depression and/or anxiety and 6 were  
123 currently taking SSRIs. A further two people had a diagnosis of ADHD (one  
124 on medication) and one had unmedicated OCD.

125 Participants were well-matched for age, IQ (IQ information was unavail-  
126 able for one control participant) and gender but the groups differed on the  
127 Edinburgh Handedness Inventory with three left-handed volunteers in the  
128 ASC group and none in the controls (see Table 1).

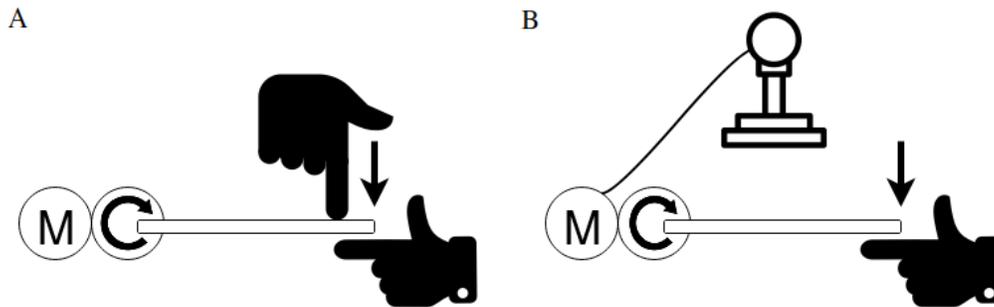
129 All but 3 of the ASC participants were assessed with module 4 of the  
130 Autism Diagnostic Observation Schedule (ADOS, [63]) and while the group  
131 was moderately symptomatic (mean score: 6.7), only 9 participants met  
132 cut-off criteria for an autism spectrum condition and none met diagnostic  
133 criteria for autism. Low sensitivity of the ADOS module 4 has previously  
134 been reported and attributed to compensatory behaviour and “milder ASDs”  
135 ([64]). Even among children, those with a diagnosis of an autism spectrum  
136 condition that is not “childhood autism” (ICD-10) often do not meet the  
137 diagnostic cut-off for the ADOS (Baird et al. [65]).

138 Given previous reports of altered forcematching in individuals with high  
139 levels of schizotypy (Teufel et al. [48]), we used the 21-item Peters Delusion  
140 Inventory (PDI, Peters and Garety [66]) to quantify schizotypal traits in all  
141 participants. The Autism Spectrum Quotient (AQ, Baron-Cohen et al. [67]),  
142 a 50-item self-administered questionnaire, was used as a measure of autistic  
143 traits. AQ and PDI scores were unavailable for one ASC participant.

#### 144 2.1.2. *Experimental Procedure*

145 The experiment was modelled on the design by Shergill et al. [40] in which  
146 a lever – via a torque motor - exerts mild pressure onto the participants’ left  
147 index finger. Depending on the condition, participants were asked to match  
148 the experienced pressure to the point of subjective equality (i.e. the point  
149 where the pressure felt the same) by either pressing directly on the lever

150 with their right index finger (“finger condition”) or by adjusting a slider  
151 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. Participants had to reproduce the experienced force either by pushing down on the lever with their other index finger (A) or by moving a slider (B).

152 As a result of forward prediction models for self-generated movements,  
153 participants routinely exceed the target force in the “finger” condition due to  
154 sensory attenuation, whereas predictions for the indirect control of the lever  
155 via the slider are less precise and participants thus tend to be more accurate  
156 in their reproduction of the force.

157 The slider was a potentiometer which transduced a force gain at the ra-  
158 tio of 0.5 N/cm. The target force was presented for 2.5 seconds (ramped  
159 up and down linearly over 0.25 seconds) after which an auditory go-signal  
160 indicated that participants should make their response to ensure that the  
161 matching took place within 2 seconds of the target force being withdrawn.  
162 After 3 seconds a second auditory signal indicated the end of each trial and  
163 instructed participants to lift their right index finger from the lever or move  
164 the slider back to the starting position. Mean force production was mea-  
165 sured between 2 and 2.5 seconds after the start of the matching period, as in  
166 previous studies (Voss et al. [47]). Within each condition 10 different force  
167 magnitudes between 0.5N and 2.75N, differing in steps of 0.25N were applied  
168 in randomised order. Each force magnitude was presented for a total of 8  
169 trials. Subjects first completed a 5-trial practice session for both conditions  
170 to ensure that they understood the task and were able to respond within the  
171 required time window. They then completed one “finger” and one “slider”  
172 block with 80 trials (160 trials in total). Invalid trials due to too slow or

173 fast responses were repeated until a total of 80 valid trials had been com-  
174 pleted. Practice sessions and test blocks were counterbalanced across both  
175 experimental groups.

### 176 *2.1.3. Data Analysis*

177 One ASC participant was excluded from further analysis as their perfor-  
178 mance in the “finger” condition was more than 9 standard deviations above  
179 the mean.

180 Basic force attenuation was indexed by calculating an overcompensation  
181 score based on the difference between the matched forces in the “finger” and  
182 “slider” condition (each normalised against the passively experienced force)  
183 for each force level (see Humpston et al. [68]). Individual regression lines of  
184 target force versus matched force for each subject were fitted for the “finger”  
185 and “slider” condition and then summarised as group regressions for both  
186 conditions. In addition to the basic overcompensation score, the slope and  
187 intercept of the regression lines can provide more detailed information about  
188 the matching performance of different groups (Wolpe et al. [45]).

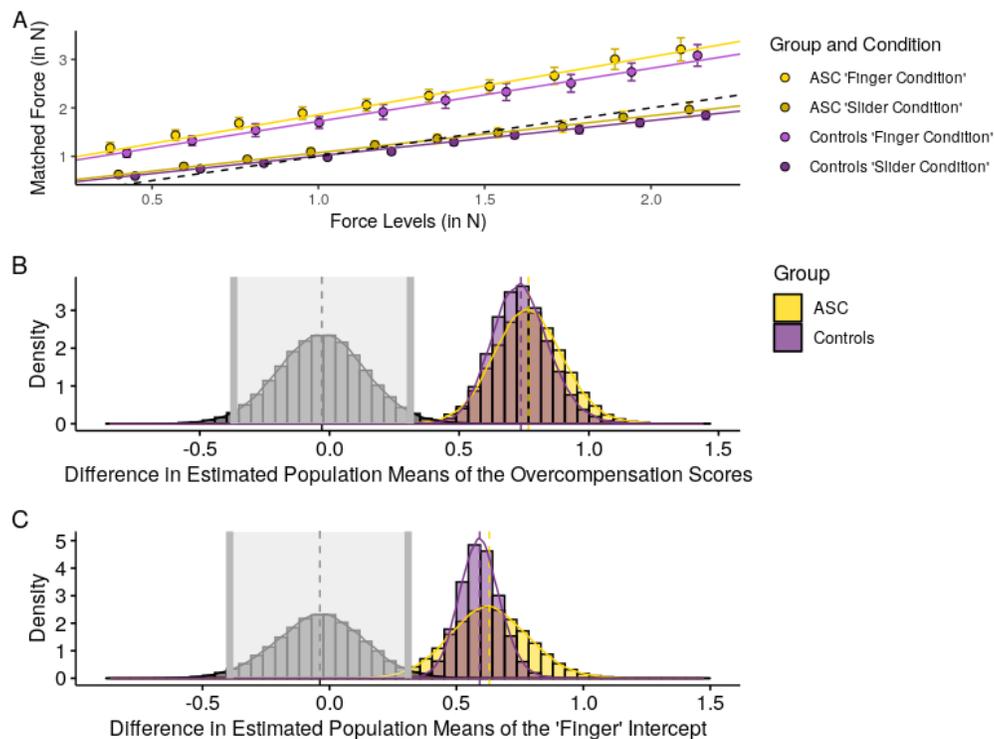
189 Group differences were evaluated with Bayesian estimation using Markov  
190 Chain Monte Carlo methods to generate samples of the relevant posterior dis-  
191 tributions. JAGS (Plummer [69]) was implemented to build a Gibbs sampler  
192 and the default non-informative priors of the R package *BEST* (Kruschke  
193 [70]) were implemented. The data is assumed to follow a t-distribution in  
194 *BEST* with  $\nu$  ( $1-\infty$ ) degrees of freedom controlling the width of the tails  
195 and thus acting as a measure of normality. The wide priors make the esti-  
196 mation of the posterior parameters (mean(s)  $\mu$ , standard deviation(s)  $\sigma$  and  
197 the shared normality parameter  $\nu$ ) very data driven. Convergence was as-  
198 sumed as long as the Brooks-Gelman-Rubin scale reduction factor (Gelman  
199 and Rubin [71], Brooks and Gelman [72]) was  $<1.1$ . Bayesian correlations  
200 were calculated using the *BayesianFirstAid* package in R.

## 201 **3. Results**

202 Both groups showed the characteristic force attenuation with the poste-  
203 rior estimates of the mean overcompensation scores being 0.73 (credible in-  
204 terval/CI: [0.51, 1.00], estimated effect size: 1.58) and 0.80 (CI: [0.52, 1.10],  
205 estimated effect size: 1.33) for the control and autism group respectively.  
206 Handedness was unlikely to be associated with the magnitude of sensory at-

207 tenuation (as measured by the overcompensation score) with an estimated  
208 correlation of  $r=-0.16$  and a 95% CI of  $[-0.45, 0.16]$ .

209 Plotting the mean linear regressions for matched forces in the “finger”  
210 and “slider” conditions did not suggest any group differences (Figure 2a).  
211 Congruously, Bayesian estimation yielded little evidence for a group differ-  
212 ence on the means of overcompensation scores (estimated difference of means:  
213  $-0.03$ , CI:  $[-0.37, 0.31]$ , estimated effect size:  $-0.08$ , Figure 2b) or intercept  
214 (estimated difference of means:  $-0.04$ , CI:  $[-0.39, 0.31]$ , estimated effect size:  
215  $-0.09$ , Figure 2c) of the “finger” condition.

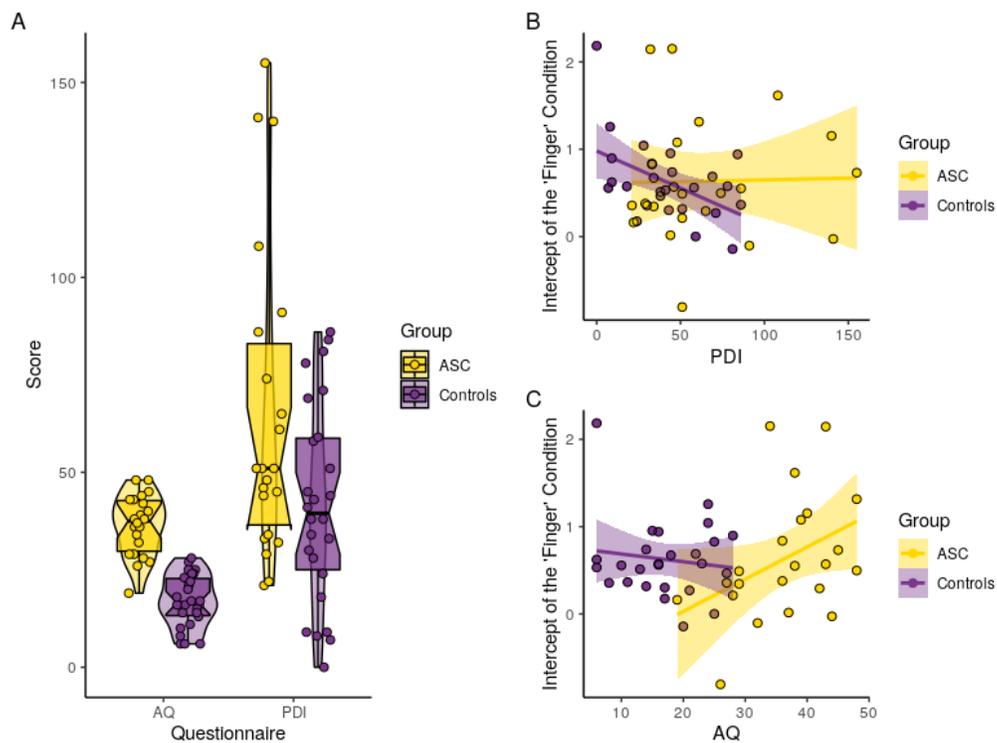


**Figure 2:** Main results for the forcematching task. **(A)** Mean linear regressions for the matched forces in the “finger” and “slider” conditions. Jitter was added to prevent overplotting. Error bars represent  $\pm 1$  standard error (SE) of the mean. Perfect matching performance is indicated by 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.

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

217 *3.0.1. Relationship between the Questionnaire Measures and Sensory Attenuation*  
218

219 As expected, posterior estimates for group means on the AQ indicated  
220 a difference (estimated difference of means: -19.49, CI: [-24.03, -15.06], es-  
221 timated effect size: -2.62) and perhaps more surprisingly there was also ev-  
222 idence in favour of the true difference in means on the PDI being non-zero  
223 (estimated difference of means: -21.50, CI: [-42.22, -0.58], estimated effect  
224 size: -0.65) (Figure 3a).



**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.

225 Using the intercept in the internal condition as the main measure of sen-

226 sory attenuation (see: Wolpe et al. [45]), in line with previous observations  
227 (Teufel et al. [48]; but see: Humpston et al. [68]), we found that the proba-  
228 bility that sensory attenuation has a negative relationship with schizotypy in  
229 the control group (probability: 98%, estimated correlation: -0.41, CI: [-0.73,  
230 -0.07]), whereas evidence in the ASC group suggested no significant relation-  
231 ship (estimated correlation: 0.04, CI: [-0.40, 0.45]). Conversely there did not  
232 seem to be an association between self-reported autistic traits on the AQ  
233 and sensory attenuation in the control group (estimated correlation: -0.01,  
234 CI: [0.42, 0.40]), but a trend for a positive relationship in the ASC group  
235 (estimated correlation: 0.36, CI: [-0.03, 0.70]), see Figure 3b and 3c.

### 236 *3.0.2. Summary*

237 Overall, we found no evidence of a deficit in the attenuation of self-  
238 produced sensory consequences in autism, which is in contradiction of ex-  
239 isting predictive processing models of the condition. A Bayesian analysis  
240 supported an absence of group differences in key measures of sensory atten-  
241 uation. Interestingly, not only AQ (as predicted) but also a measure related  
242 to schizotypy (PDI) was higher in the ASC group. Moreover, in line with  
243 previous work, correlative analyses of sensory attenuation with schizotypy  
244 showed an expected negative relationship in control participants. No such  
245 correlation was found in ASC. Conversely, AQ scores in the autism group  
246 correlated positively with sensory attenuation.

## 247 **4. Experiment 2 - Intentional Binding in Autism**

### 248 *4.1. Method*

#### 249 *4.1.1. Participants*

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

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

#### 256 *4.1.2. Experimental Procedure*

257 The basic structure of the task was similar to other intentional binding  
258 experiments (Haggard et al. [52]): Participants were instructed to press a key  
259 with their right index finger at a time of their own choosing which caused

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

<b>Group</b>	<b>Age (<i>SD</i>)</b>	<b>Sex (<i>m:f</i>)</b>	<b>IQ (<i>SD</i>)</b>
ASC ( <i>N=23</i> )	29.0 ( <i>6.1</i> )	11:12	105.2 ( <i>12.7</i> )
Controls ( <i>N=25</i> )	31.2 ( <i>5.7</i> )	10:15	104.6 ( <i>10.6</i> )

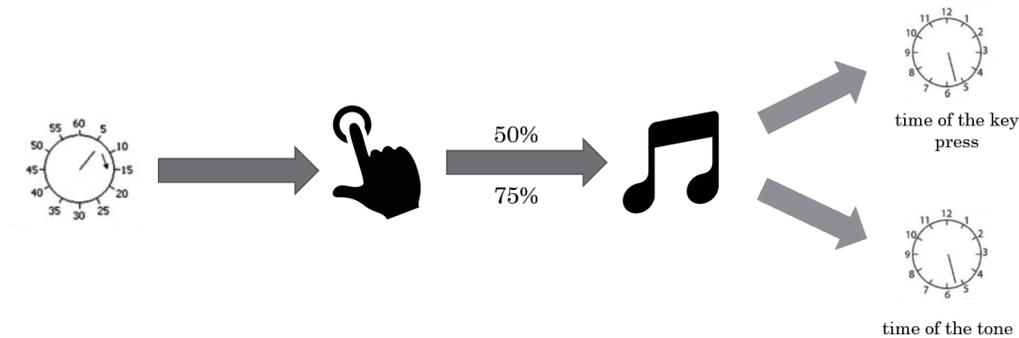
260 a tone 250ms later. While they were engaged in this task, a Libet clock  
261 (Libet et al. [73]) was visible in the middle of the screen with a clock-hand  
262 rotating at a rate of 2560ms per revolution. After the keypress, the clock-  
263 hand continued to rotate for a random amount of time. Participants were  
264 told to avoid pressing at “premeditated” clock positions.

265 In the “action block” condition, participants had to recall the time at  
266 which they pressed the key (i.e. recall where the clock-hand was pointing  
267 to when they performed the keypress) while in “tone blocks” participants  
268 were asked to enter the the clock-hand’s position when they heard the tone.  
269 As in Moore’s adapted version (Moore and Haggard [41]), the probability of  
270 the tone occurring was manipulated: In half of the blocks (2 per condition)  
271 the tone followed the key press 50% of the time while in the other half it  
272 happened 75% of the time (see Figure 4). When no tone occurred, partici-  
273 pants were asked to report a dummy value. Participants were informed of the  
274 response requirement (time estimation of the key press or tone occurrence)  
275 immediately prior to the blocks which otherwise did not differ visually from  
276 each other. The order of blocks was randomised for each participant.

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

280 Blocks with the 50% probability for tone occurrence had 50 trials whereas  
281 blocks with tones occurring 75% of the time had 40 trials. Baseline blocks had  
282 50 trials. Due to a technical error 2 control subjects had the trial numbers  
283 reversed and 3 controls and 7 ASC participants only completed 40 trials in  
284 the baseline task.

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



**Figure 4:** An illustration of the experimental procedure for IB with varying outcome contingencies

288 *4.1.3. Data Analysis*

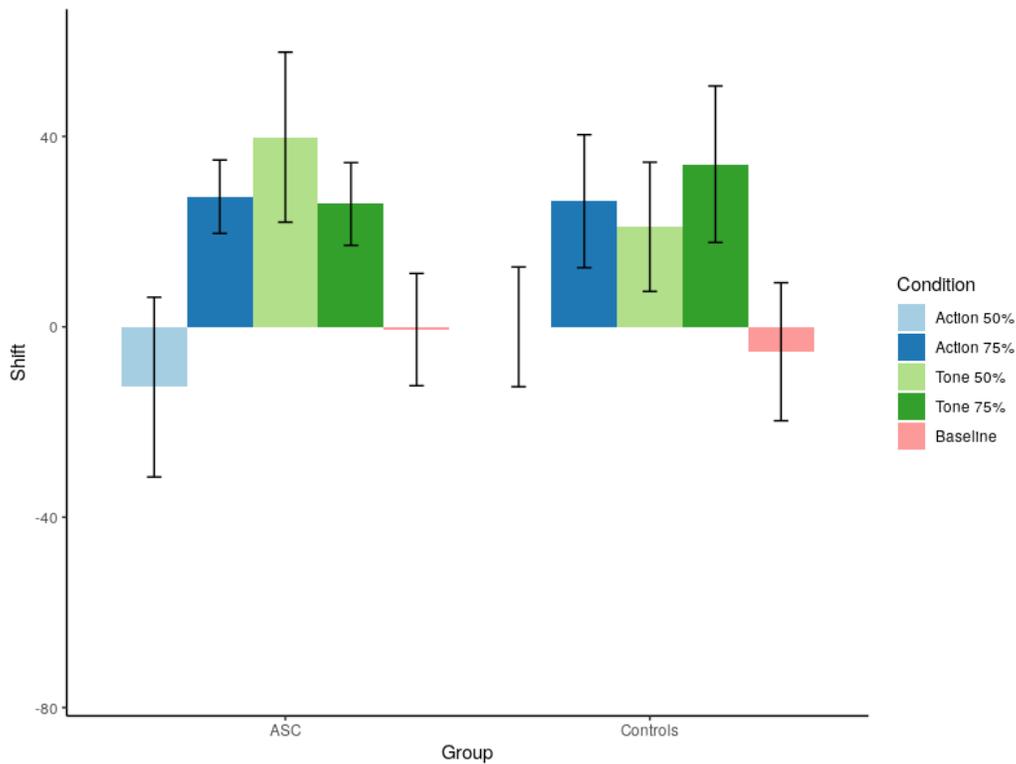
289 The analysis followed the typical protocol for IB studies. Initially, re-  
290 sponses were corrected against the mean of all baseline trials for each partic-  
291 ipant. For the purposes of the analysis, the first 10 trials of each block were  
292 not included as participants had to learn the contingencies. The reported  
293 shifts in the performed key presses were used as the measure of intentional  
294 binding. By convention, binding for actions is indicated by a positive differ-  
295 ence.

296 Based on Voss et al. [43], the predictive component to the intentional  
297 binding effect was calculated as the difference in overall shift between action  
298 only trials in the high probability blocks and action only trials in the low  
299 probability blocks (“action only” trials (75%) – “action only” (50%)). Since  
300 the tone is observed in neither condition, any difference in the strength of  
301 binding must be due to the higher predictive power of the “action only” 75%  
302 probability blocks. Analogously the inferential contribution was defined as  
303 the average shift in “tone only” trials in the 50% blocks. The authors describe  
304 the 50% contingency as subjectively “random”, so participants should not be  
305 able to form helpful predictions. Therefore any binding effect must be due to  
306 an inferential component that acts on the temporal estimation process after  
307 the tone occurs.

308 **5. Results**

309 *5.0.1. Basic Intentional Binding Effect*

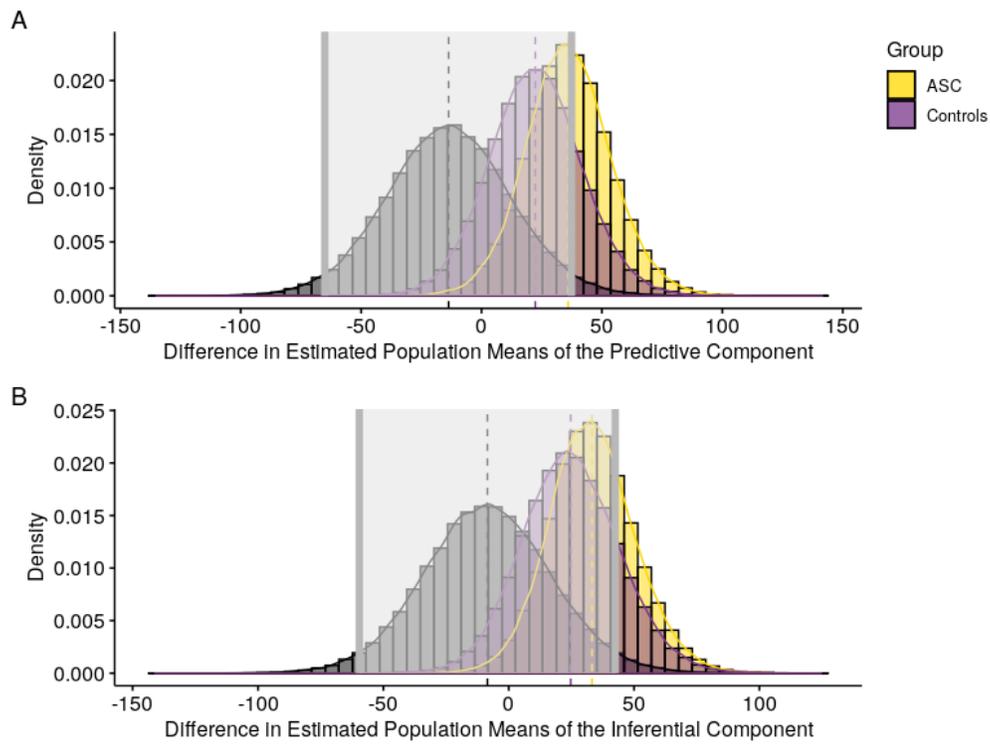
310 The resulting pattern resembled Moore and Haggard’s [41] results where  
311 intentional binding was observed in all conditions apart from the low-probability  
312 no-tone trials (see Figure 5).



**Figure 5:** Baseline-corrected shift in the action estimates (ms) for each probability block in the “action only” and “tone only” conditions. Error bars represent  $\pm 1$  standard error (SE) of the mean.

313 *5.0.2. Group Comparison on Predictive and Inferential Components of In-*  
314 *tentional Binding*

315 The Bayesian estimation of the group difference for the predictive compo-  
316 nent (estimated difference of means: -13.7, CI: [-65.1, 37.9], estimated effect  
317 size: -0.17, Figure 6a) and the inferential component (estimated difference of  
318 means: -8.49, CI: [-59, 42.5], estimated effect size: -0.11, Figure 6b) makes a  
319 difference unlikely for both parameters.



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

320 *5.0.3. Relationship between the Questionnaire Measures and Intentional Bind-*  
321 *ing*

322 There was little evidence that the AQ or PDI correlated with any of the  
323 measures; estimated correlations ranged between -0.22 and 0.23 and all CIs  
324 included 0.

325 *5.0.4. Summary*

326 Overall, therefore, in keeping with the findings from the force-matching  
327 task in experiment 1, we found no group difference in intentional binding.  
328 Both groups showed expected reductions in the subjective experience of  
329 action-outcome timing in both the predictive (tone absent) and postdictive  
330 (tone present) conditions.

331 **6. Discussion**

332 In the past decade, a number of prominent hypotheses have suggested  
333 that autism is primarily a disorder of atypical predictive processes and that  
334 the range of alterations, particularly in perceptual experiences can be ex-  
335 plained in terms of these atypicalities. However the empirical evidence sup-  
336 porting these hypotheses in the form of differences in low-level sensorimotor  
337 prediction has been lacking which led us to investigate sensory attenuation  
338 and agency-based temporal binding in adults with autism. In light of this  
339 theoretical work conceptualising autism as a “disorder of prediction” (Sinha  
340 et al. [16]), one would expect to find reduced perceptual attenuation in the  
341 autistic group and a reduction of the predictive component to the intentional  
342 binding effect. Neither of these observations were made and our experiments  
343 do not support the idea of a deficit in predictive processing in autism. Both  
344 ASC and control groups demonstrated sensory attenuation of self-generated  
345 stimuli with a magnitude consistent with previously reported results (Teufel  
346 et al. [48], Shergill et al. [40], Wolpe et al. [45]) and both groups exhibited  
347 the basic pattern of inferential and predictive binding reported by Moore  
348 and Haggard [41]. These findings indicate that global deficits in predictive  
349 processing cannot explain the observed cognitive, perceptual and motor dif-  
350 ferences in autism spectrum conditions.

351 However, one interesting group difference that emerged lay in the within-  
352 group relationship between odd or unusual beliefs, as measured by PDI and  
353 the magnitude of sensory attenuation. While we replicated the previous  
354 finding that an increase in the number of delusion-like beliefs was associated

355 with more accurate force-matching (i.e. reduced sensory attenuation), this  
356 relationship was not seen in autism. However there was some preliminary ev-  
357 idence that higher autistic traits in autistic individuals could be related to an  
358 increase in sensorimotor prediction as indicated by increased sensory atten-  
359 uation. The lack of correlation between attenuation and PDI in the autism  
360 group is intriguing. One possibility is that the PDI and AQ questionnaires  
361 do not measure the same underlying traits in autism as in controls (Murray  
362 et al. [74]). An alternative explanation would be that sensory attenuation  
363 is indeed modulated by different latent traits in autistic and non-autistic  
364 individuals.

365 Compared to the schizophrenia literature, evidence for disruptions of senso-  
366 ry gating and agency processing in autism is scant: Previous research on  
367 sensory attenuation in ASC has reported unimpaired cancellation of self-  
368 generated tactile stimulation in the form of self-tickling (Blakemore et al.  
369 [75]) and adults with autism are just as good as their matched controls at  
370 judging agency based on whether visual feedback matched their own hand  
371 movements or not (David et al. [76]). In contrast, Zalla et al. [77] showed a  
372 decreased use of sensorimotor cues in making judgments of agency in adults  
373 with autism which was correlated with performance on a Theory of Mind  
374 task. They conclude that autistic individuals experience their internal sig-  
375 nals as unreliable and might rely more on retrospective external cues (such  
376 as accuracy) to evaluate agency. Preliminary studies on interoceptive deficits  
377 in autism seem to support this claim (Noel et al. [78], Garfinkel et al. [79]).  
378 Similarly, Zalla and Sperduti [80] suggest that autism is characterised by  
379 an isolated impairment of predictive (but not postdictive) processes in the  
380 genesis of sense of agency. A recent study has indeed found an attenuated  
381 intentional binding effect in adults with autism when tested with visual, au-  
382 ditory and audio-visual action outcomes (Sperduti et al. [81]). In light of our  
383 diverging results the differences between the two experiments need to be ex-  
384 amined: The manipulation of the probability of the action effect occurring in  
385 the experiment that is presented here is unlikely to cause an enhancement in  
386 overall IB, as it should introduce more uncertainty and more spurious bind-  
387 ing effects. An obvious suggestion, given that Sperduti et al. employed three  
388 different delays between the action and action outcome, is that time estima-  
389 tion and temporal binding difficulties which are common in autism (Brock  
390 et al. [82], Maister and Plaisted-Grant [83]), impeded performance for the  
391 ASC group. As Maister and Plaisted-Grant [83] point out, impairments in  
392 estimating short time intervals between 0.5 and 2 seconds seem to be the

393 result of deficits in attentional control in autistic individuals, rather than  
394 indicative of a more global temporal processing deficit and thus might elude  
395 being captured by the proportion error scores used in Sperduti et al. [81].  
396 Other differences between the two studies include the smaller (N=15 for the  
397 autism group) all-male participant panel in Sperduti et al.'s experiment, the  
398 different estimation methods (Libet clock vs. analogue scale) and the fact  
399 that each condition (interval and modality) was only presented 10 times with  
400 180 trials in total by Sperduti et al. compared to  $\sim$ 460 trials in the current  
401 study. If autistic individuals are indeed more variable in their responses due  
402 to attentional deficits, a higher number of trials would be needed to obtain  
403 the expected effect.

404 The lack of phenotyping for sensory reactivity and abnormalities is cer-  
405 tainly a caveat of the present study and could be addressed more thoroughly  
406 in future investigations. Detailed assessments of sensory subtypes could also  
407 help to explain the commonly observed heterogeneity in task performance  
408 seen in the autistic group (Lane Alison E. et al. [84]) and it is possible that  
409 differences in predictive abilities might be domain-specific. As predictive  
410 attenuation is not unique to the tactile domain (Benazet et al. [85], Cardoso-  
411 Leite et al. [86], Desantis et al. [87], Hughes and Waszak [88]), an investiga-  
412 tion linking domain-specific sensory reactivity (like the frequently reported  
413 auditory defensiveness) to sensory attenuation might be better equipped to  
414 uncover potential differences. Furthermore, although it is sometimes claimed  
415 that these sensorimotor processes are well understood given the extensive re-  
416 search into central and peripheral nervous system mechanisms supporting  
417 sensory gating (Rushton et al. [89]), their relationship with the perceptual  
418 attenuative processes seen in the force matching task is not entirely clear  
419 and there is some evidence that the two processes are functionally distinct  
420 (Palmer et al. [90]).

421 A further limitation of the experiments presented here was the exclusion  
422 of younger populations for the experiments. As autism is a neurodevelop-  
423 mental disorder, it would be worth exploring if the trajectories for acquiring  
424 and refining internal models of the external world are different in autistic  
425 individuals even if performance is indistinguishable at a later developmen-  
426 tal stage. Since structural priors are likely to either emerge from long-term  
427 aggregation of individual experiences or as embedded constraints acting on  
428 bottom-up processes (Teufel and Fletcher [1]) - as opposed to the short-term  
429 learning of stochastic relationships for contextual priors - they supposedly are  
430 subject to developmental processes. As such the force-matching task would

431 be the best candidate for a developmental approach to predictive coding  
432 paradigms.

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

## 7. Bibliography

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