

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.

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 acteristic 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

Group	Age (<i>SD</i>)	Sex (<i>m:f</i>)	Handedness (<i>SD</i>)	IQ (<i>SD</i>)
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.

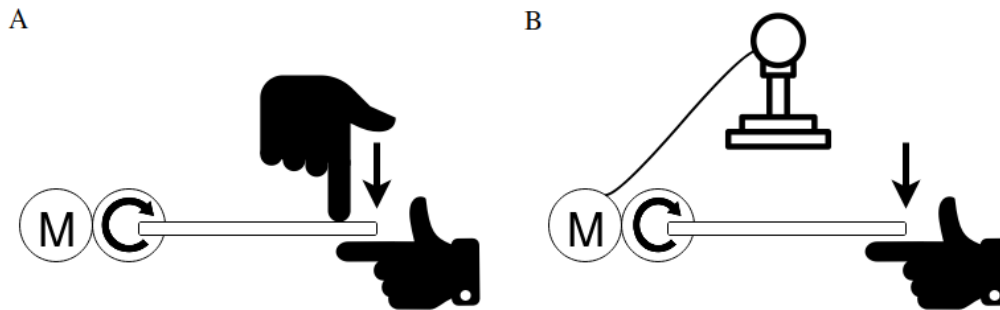


Figure 1: Illustration of the forcematching paradigm in which participants are asked to match a force applied to their left index finger via a lever. Participants had to reproduce the experienced force either by pushing down on the lever with their other index finger (A) or by moving a slider (B).

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 ν ($1-\infty$) 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) μ , standard deviation(s) σ and
197 the shared normality parameter ν) 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.

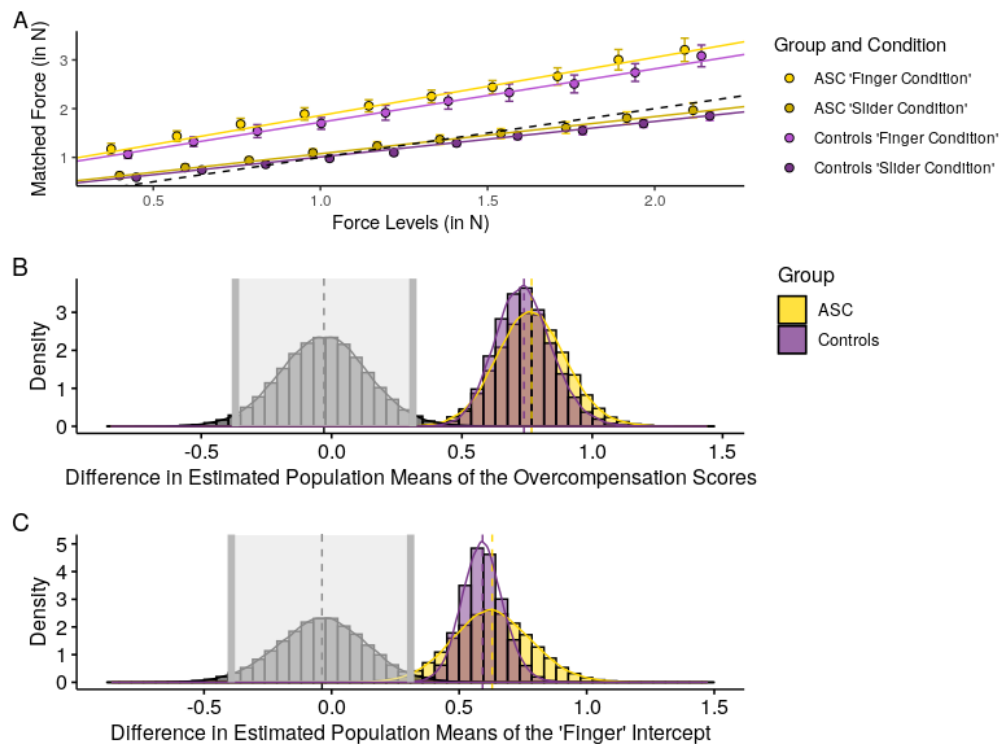


Figure 2: Main results for the forcematching task. **(A)** Mean linear regressions for the matched forces in the “finger” and “slider” conditions. Jitter was added to prevent overplotting. Error bars represent ± 1 standard error (SE) of the mean. Perfect matching performance is indicated by the dashed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

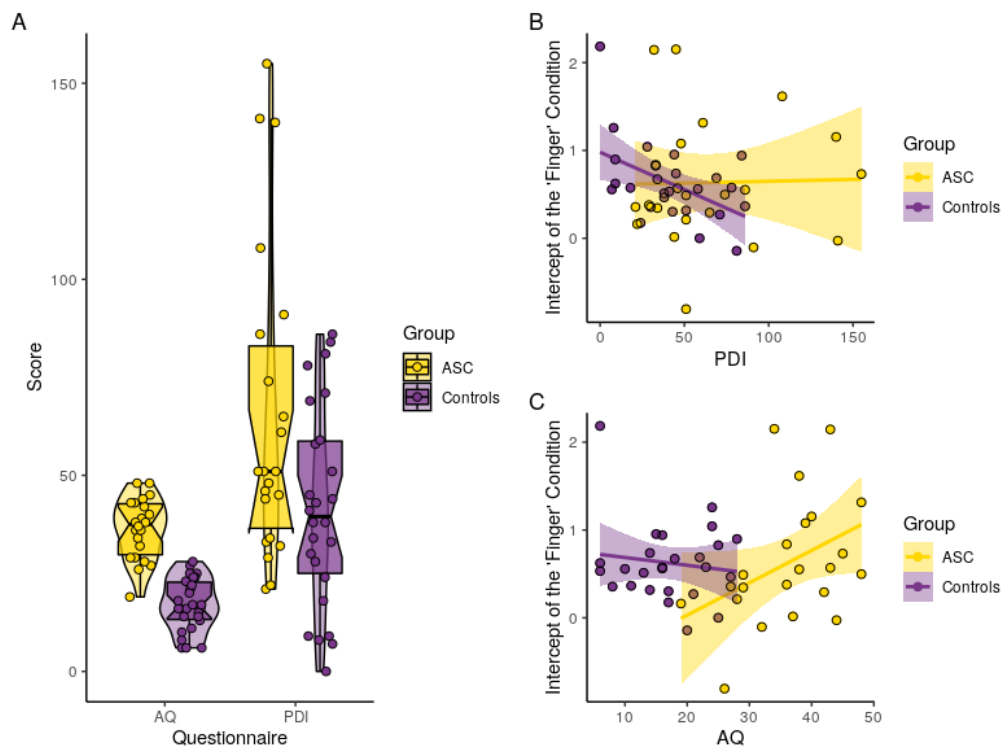


Figure 3: Results for the Questionnaire measures. **(A)** Plot showing the distribution of the questionnaire scores for both groups, including the median and interquartile ranges. **(B)** The correlation between sensory attenuation (as measured by the intercept in the “finger” condition) and the PDI.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

Group	Age (<i>SD</i>)	Sex (<i>m:f</i>)	IQ (<i>SD</i>)
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.

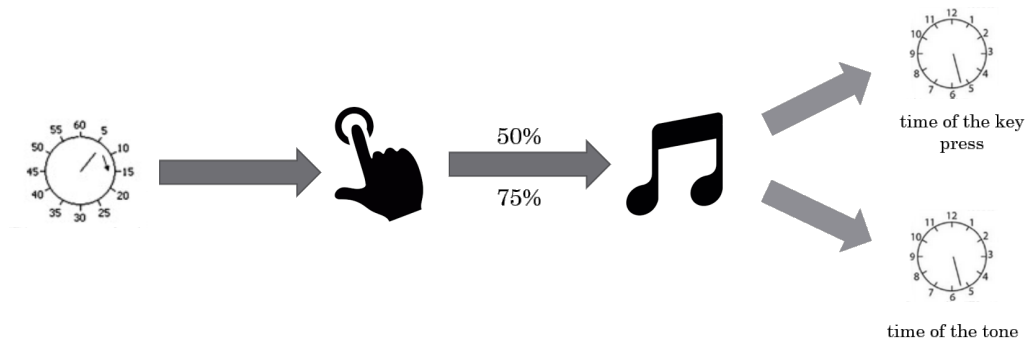


Figure 4: An illustration of the experimental procedure for IB with varying outcome contingencies

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).

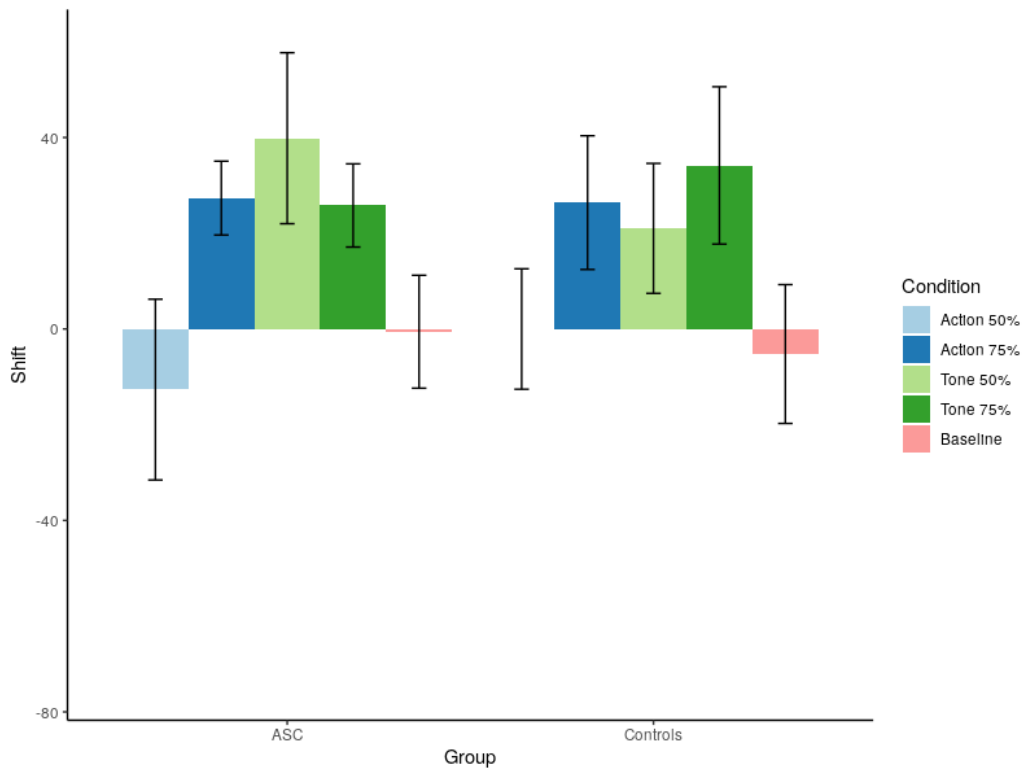


Figure 5: Baseline-corrected shift in the action estimates (ms) for each probability block in the “action only” and “tone only” conditions. Error bars represent ± 1 standard error (SE) of the mean.

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.

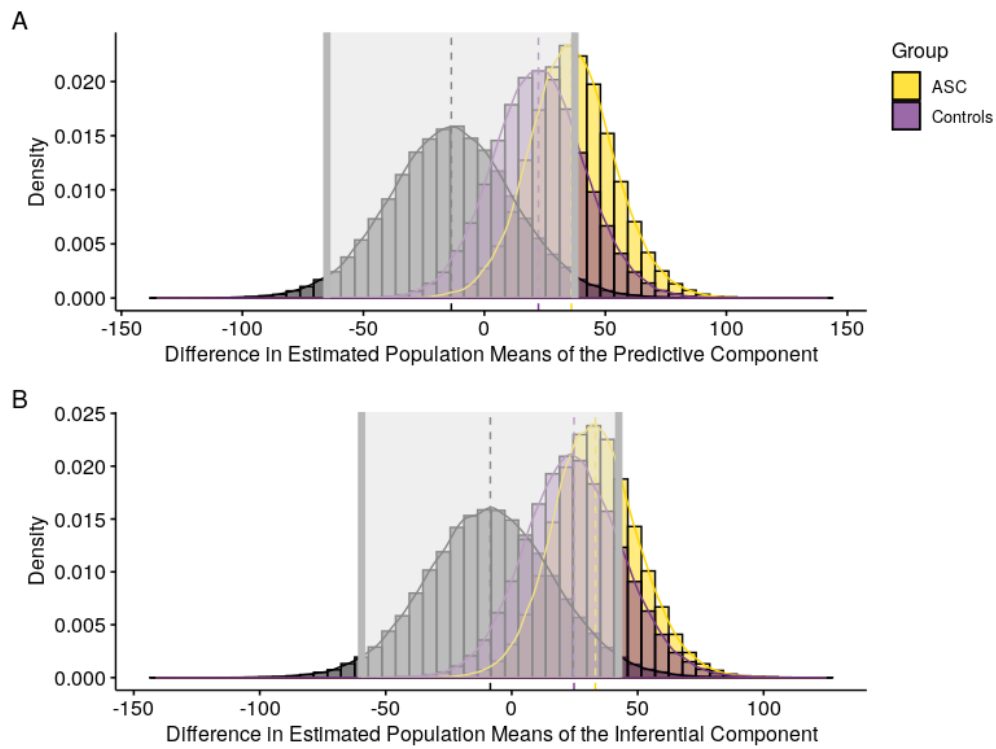


Figure 6: Posterior distributions for the difference in estimated population means of the predictive (A) and inferential (B) component of IB. The shaded area is the 95% Highest Density Interval (HDI).

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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