

1 Running title: Sensory sensitivity and food fussiness in neurodevelopmental disorders

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3 The relationship between sensory sensitivity, food fussiness and food preferences in
4 children with neurodevelopmental disorders

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21 Declarations of interest: none.

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27 **Abstract**

28 Heightened sensitivity to sensory information has been associated with food fussiness
29 in both atypical and typical development. Despite food fussiness and sensory
30 dysfunction being reported as common concerns for children with neurodevelopmental
31 disorders, the relationship that exists between them, and whether they differ between
32 disorders, has yet to be established. The current study aimed to examine sensory
33 sensitivity as a predictor of food fussiness in three different neurodevelopmental
34 disorders, whilst controlling for comorbidity amongst these disorders. Ninety-eight
35 caregivers of children with Attention Deficit Hyperactivity Disorder (ADHD; n=17),
36 Tourette Syndrome (TS; n=27), Autism Spectrum Disorder (ASD; n=27), and typical
37 development (TD; n=27) were compared using parental reports of child food fussiness,
38 food preferences and sensory sensitivity. Children with neurodevelopmental disorders
39 were reported to have significantly higher levels of both food fussiness and sensory
40 sensitivity, with children with ASD and TS also showing significantly less preference
41 for fruit than children with TD. Importantly, higher levels of taste/smell sensitivity
42 predicted food fussiness for all four groups of children. In addition, taste/smell
43 sensitivity fully mediated the differences in food fussiness between each group of
44 neurodevelopmental disorders compared to the TD group. The findings highlight that
45 food fussiness is similar across these neurodevelopmental disorders despite accounting
46 for comorbidity, and that greater sensitivity to taste/smell may explain why children
47 with neurodevelopmental disorders are more likely to be fussy eaters.

48

49 Keywords: Tourette syndrome; Attention Deficit Hyperactive Disorder; Autism
50 Spectrum Disorder; food fussiness; sensory sensitivity

51 **1. Introduction**

52 Neurodevelopmental disorders comprise a broad range of conditions emerging in
53 childhood and are primarily associated with impairments in the development of the
54 central nervous system and the brain (Posthuma & Polderman, 2013). These can
55 encompass a range of aetiologies, including a known mutation on a single gene (Fragile
56 X syndrome), disorders with polygenic or unknown genetic origins (e.g. Attention
57 Deficit Hyperactivity Disorder [ADHD], Autism Spectrum Disorder [ASD], Tourette
58 Syndrome [TS]), and disorders from early/perinatal factors (e.g. cerebral palsy; Cascio,
59 2010). Population-based twin studies have shown high genetic correlations between

60 ASD, tic disorders and ADHD (Lichtenstein, Carlström, Råstam, Gillberg &
61 Anckarsäter, 2010), and it is this group of disorders which formed the focus of the
62 current study.

63 Each of these three neurodevelopmental disorders, as defined in the DSM-5, are
64 characterised by a distinct set of diagnostic features, such that TS is characterised by
65 involuntary, repetitive and non-rhythmic motor and vocal tics, ADHD by excessive and
66 impairing inattentive, hyperactive and impulsive behaviour, and ASD by language
67 development, social and communication deficits (American Psychiatric Association,
68 2013). Despite this, these disorders have also been recognised as sharing many
69 overlapping features, in addition to being highly comorbid with each other. For
70 example, ADHD is diagnosed in 60% of individuals with TS (Freeman et al., 2000).
71 Recent studies have also documented that TS is comorbid with ASD (Cath & Ludolph,
72 2013), with research showing the presence of autistic symptoms in two thirds of
73 individuals with TS (Kadesjö & Gillberg, 2000), and the relationship between autism
74 symptomology in TS is particularly strong for individuals with early onset of TS
75 (Zappella, 2002). Furthermore, it is estimated that 50% of individuals with ADHD will
76 meet the criteria for diagnosis of ASD (Kochhar et al., 2011). Importantly in the context
77 of the current study, individuals with these disorders have also all been identified as
78 showing difficulties with feeding and eating concerns which extend beyond the
79 developmental stages of childhood, in which feeding and eating problems are very
80 common (Johnson, 2014). This raises the possibility that the comorbidity of these
81 disorders may explain their similarities in eating outcomes.

82 More specifically, the two eating outcomes which form the focus of this paper are food
83 preferences, understanding the likings of different food groups, and children's
84 behaviour towards food in terms of fussiness. Food fussiness can be defined as
85 consuming 'an inadequate variety of foods' (Galloway, Fioritio, Lee & Birch, 2005, p.
86 542). Caregivers commonly report food fussiness in children with typical development,
87 with around 46% of children reported as being picky eaters at some point between 1.5
88 and 6 years (Cardona-Cano et al., 2015). Although picky/fussy eating are relatively
89 stable traits (Mascola et al., 2010), a substantial proportion of children show reductions
90 in picky eating by 6 years (Cardona-Cano et al., 2015). In contrast, food fussiness is
91 more frequent and persistent in children with neurodevelopmental disorders (Bandini
92 et al., 2017; Suarez, Atchison & Lagerwey, 2014). For example, food fussiness has

93 been found to be one of the most commonly reported feeding problems in children with
94 ASD (Williams, Field & Seiverling, 2010), with a narrow range of foods being selected
95 based on food type, temperature, texture and colour (Seiverling, Williams & Sturmey,
96 2010). This is important, given the emotional and health implications of food fussiness
97 (Dovey, Staples, Gibson & Halford, 2008). Research has identified fussy eaters to
98 consume fewer foods containing vitamin E, C and fibre, likely due to their low intake
99 of fruit and vegetable. A lack of nutrient-rich foods can lead to nutritional deficiencies,
100 including magnesium and iron deficiencies, as well as slower growth patterns (Xue et
101 al., 2015; Antoniou et al., 2016). Food fussiness has also been associated with
102 additional stress and frustration for the child and their families (Rogers et al., 2012;
103 Curtin et al., 2015), along with difficulty eating in social environments (Nadon et al.,
104 2011). These various adverse effects highlight the need for interventions to increase
105 food intake, in terms of variety and healthy foods, in children with fussy eating.

106 For children with typical development, research has established several methods to
107 encourage children to reduce food fussiness and increase consumption of fruit and
108 vegetables. These include parent modelling, which can help increase consumption of
109 novel foods through observational learning, and repeated re-offering of food to help
110 familiarise the child with the foods until they are willing to try (Holley, Haycraft &
111 Farrow, 2015; Wardle, Carnell & Cooke, 2005). However, not only have children with
112 heightened levels of food fussiness been shown not to respond to these methods in the
113 same manner as typically developing (TD) children (Wolke, Schmid, Schreier &
114 Meyer, 2009), research addressing these interventions in children with
115 neurodevelopmental disorders is limited.

116 Recommendations often given to parents, such as only presenting family meals instead
117 of the child's preferred foods, have found to be ineffective for children with ASD who
118 have feeding problems and may lead to severe nutritional deficiencies (Rogers et al.,
119 2012). Behavioural interventions, such as behavioural momentum (Patel et al., 2007)
120 have been reported to be effective in reducing feeding problems and increasing food
121 consumption in children with ASD. However, these are single case studies and pre-post
122 intervention studies are needed to demonstrate the generalisability of treatment effects
123 (Matson & Fodstad, 2009). Ultimately, it is important to understand the underlying
124 aetiology of food fussiness in these clinical groups to devise the most effective
125 intervention strategy.

126 Given the comorbidity between TS, ASD and ADHD, it is important to understand
127 whether the underlying causes of food fussiness is syndrome specific or the shared
128 pathologies across the three neurodevelopmental disorders accounts for the high levels
129 of food fussiness evidenced in these groups of children (Smith, Rogers, Blissett &
130 Ludlow, 2019). One potential explanation for food fussiness is the associated
131 pathologies unique to the disorder. For example, some feeding difficulties may reflect
132 limited interests and difficulty in accepting change (Curtin et al., 2015). Evidence also
133 suggests that children with heightened motor impulsivity and reduced inhibitory control
134 and/or characteristics of ADHD weigh more. Children with ADHD are also more
135 susceptible to altered food intake patterns dependent on stressors, such as distress or
136 external cues including the time of day (Bennett & Blissett, 2017).

137 A common feature of all three neurodevelopmental disorders is an impairment in
138 sensory processing, which could provide an alternative explanation for food fussiness.
139 Impaired sensory processing may lead to over-responsiveness to stimuli (resulting in
140 more fussy eating), or under-responsiveness to stimuli (which may result in a desire for
141 more sweet, salty or fatty foods). Both sensory processing issues have been shown to
142 limit the range of food consumed and social enjoyment of eating (Johnson et al., 2014).
143 Furthermore, in children with both typical and atypical development, the perceived
144 sensory properties of food are often considered to underlie children's reasons for
145 rejecting food (Martins & Pliner, 2005). The focus of the current study is to explore
146 sensory over-responsiveness across these neurodevelopmental disorders in relation to
147 food fussiness.

148 Individuals with sensory over-responsiveness "respond to sensation faster, with more
149 intensity, or for a long duration than those with typical sensory responsivity" (Miller,
150 Anzalone, Lane, Cermak & Osten, 2007, p.136). This increased sensitivity to sensory
151 information, such as taste, smell and touch, has been identified as an inherent
152 characteristic that makes one particularly vulnerable to be a fussy eater. For example,
153 a reluctance to eat new foods and/or eat fruit and vegetables has been associated with
154 higher levels of tactile and taste/smell sensitivity (Coulthard & Blissett, 2009). For
155 example, tactile oversensitivity has also been shown to have an impact upon the eating
156 habits and food selection in children with and without atypical development (Cermak,
157 Curtin & Bandini, 2010). High levels of taste/smell sensitivity (Tomchek & Dunn,

158 2007), and difficulty with texture have been associated with a lack of variety in the diet
159 in children with ASD (Schmitt, Heiss, & Campbell, 2008), and TS (Smith et al., 2019).
160 Importantly, oral sensitivity which also forms part of tactile sensitivity, has also been
161 associated with food fussiness in children with ASD and ADHD (Chistol et al., 2018;
162 Ghanizadeh, 2013). Greater prevalence of sensory sensitivities have been found in
163 children with children with ASD (Bizzell, Ross, Rosenthal, Dumont, & Schaaf, 2019;
164 Simpson, Adams, Alston-Knox, Heussler, & Keen, 2019), ADHD (Ghanizadeh, 2011;
165 Lane, Reynolds, & Thacker, 2010), and TS (Belluscio, Jin, Watters, Lee & Hallett,
166 2011) compared to TD children. The relationship between sensory processing and food
167 fussiness in children with these neurodevelopmental disorders, without comorbidity
168 within these disorders, therefore warrants further investigation.

169 Despite the similarities in food fussiness found across these neurodevelopmental
170 disorders, there has been no research carried out directly comparing food fussiness
171 across different neurodevelopment disorders and/or examining which factors are
172 particularly associated with eating difficulties in children without comorbid
173 neurodevelopmental disorders. The current study therefore aimed to be the first study
174 to directly compare food fussiness and food preferences, along with the role of sensory
175 sensitivity in predicting these eating outcomes, amongst children with ADHD, ASD,
176 TS and TD children. It was hypothesised that children with neurodevelopmental
177 disorders would display higher levels of food fussiness and sensory sensitivity than TD
178 children, and that levels of food fussiness would be comparable across each
179 neurodevelopmental disorder despite no other comorbid disorder being present. It was
180 also predicted that sensory sensitivity would be a predictor of eating outcomes for all
181 groups of children.

182 **2. Method**

183 **2.1 Participants**

184 One hundred and thirteen mothers reported information on their children between
185 Spring 2017 and Autumn 2018. Children were screened for missing data ($N = 6$) and
186 comorbidity for other neurodevelopmental disorders ($N = 7$; 2 TS were removed for
187 having comorbidity with ASD, 1 TS with ADHD, 3 ASD with ADHD, and 1 TS with
188 ADHD and ASD) by asking caregivers for their child's full diagnosis and whether they
189 had been diagnosed with any additional disorders. Data from 98 mothers aged 25-67

190 years ($M=40$, $SD=1$) remained. Twenty-seven children had a diagnosis of TS, 27 had a
191 diagnosis of ASD, 17 had a diagnosis of ADHD, and 27 were typically developing
192 children with no known clinical diagnosis (5 females, 22 males) recruited through local
193 schools and forums. The groups did not differ in age, $F(3,98) = .64$, $p = .59$.

194 *2.1.1 Children with Tourette syndrome*

195 Twenty-seven children with a clinical diagnosis of TS (5 females, 22 males) were aged
196 between 6 years 7 months and 15 years 11 months. Caregiver report of a TS diagnosis
197 and the Premonitory Urge for Tics Scale (PUTS; Woods, Piacentini, Himle, & Chang,
198 2005) were used to confirm children's status in the TS group only. This measure reflects
199 the presence and frequency of premonitory urges, along with the relief that may be
200 experienced after tics have been performed, and is a tool used to estimate impact of
201 symptoms. A score above 31 indicates extremely high intensity with probable severe
202 impairments. In the current sample scores ranged from 9 to 35 ($M=22$, $SD = 5$). Of the
203 children with TS taking medication ($n = 13$), the most commonly reported was
204 melatonin ($n = 6$). Other prescription drugs recorded were sertraline ($n = 4$) and
205 clonidine ($n = 2$).

206 *2.1.2 Children with Autism Spectrum Disorder*

207 Twenty-seven children with a clinical diagnosis of an ASD (11 females, 16 males) were
208 aged between 6 years 7 months and 17 years. Children with ASD were required to meet
209 the appropriate cut off on the Autism Spectrum Screening Questionnaire (ASSQ;
210 Ehlers, Gillberg, Wing, 1999). The ASSQ comprises 27 items rated on a 3-point scale,
211 0 indicating normal, 1 some abnormality and 2 definite abnormality. The range of score
212 is 0–54. Eleven items tap topics regarding social interaction, six cover communication
213 problems and five refer to restricted and repetitive behaviour. The remaining five items
214 measure motor clumsiness and other associated symptoms including motor and vocal
215 tics. All children reached the cut-off scores of 19 or more ($M = 27$, $SD = 7$), with mean
216 scores on subscales: Social Interaction ($M = 11$, $SD = 7$), Communication ($M= 8$, $SD =$
217 3), Restrictive and Repetitive Behaviours ($M = 4$, $SD = 2$), and Motor skills and
218 clumsiness ($M =4$, $SD = 2$). Of the children with ASD taking medication ($n = 15$), the
219 most commonly reported was melatonin ($n = 6$). Other prescription drugs recorded were
220 Prozac ($n = 2$) and clonidine ($n = 2$).

221 ***2.1.3 Children with Attention Deficit Hyperactive Disorder***

222 Seventeen children with a clinical diagnosis of ADHD combined type. (7 females, 10
223 males) were aged between 6 years 2 months and 16 years 8 months. All of the children
224 met the required T-score of 65 or above on the Connors' Parent Rating Scale-Revised
225 (CPRS-R; Conners et al., 2008). Children's T-scores were reported as follows on the
226 content scales: Inattention ($M = 85, SD = 7$), Hyperactive/Impulsive ($M = 86, SD = 6$);
227 Learning Problems ($M = 77.57, SD = 10.29$), Executive Functioning ($M = 84, SD = 10$),
228 Aggression ($M = 77, SD = 16$), Peer Relations ($M = 86, SD = 6$), and for the symptom
229 scales: DSM-IV ADHD Inattention ($M = 84, SD = 9$), DSM-IV ADHD Hyperactivity-
230 Impulsive ($M = 87, SD = 6$), Conduct Disorder ($M = 70, SD = 19$), and Oppositional
231 Defiant Disorder ($M = 79, SD = 12$).

232 ***2.2 Measures***

233 Demographic variables collected included: child's sex, birth date, any clinical diagnosis
234 including comorbid disorders. Caregivers were asked to provide a measurement of their
235 child's weight and height, which was then converted to a BMI standard deviation score
236 (SDS). The Child Growth Foundation Package (1996) was used to standardise the
237 measurements for age and sex according to standardised norms for a UK sample.
238 Caregivers were also asked to describe their age, ethnicity and their relation to the child.
239 Finally, all caregivers were asked to complete the following questionnaires:

240 ***2.2.1 The Short Sensory Profile (SSP; McIntosh, Miller, Shyu & Dunn, 1999)***

241 The SSP is a 38-item an adapted version of the original Sensory Profile (Dunn, 1999)
242 designed to assess children's responses to sensory stimuli. The three subscales from the
243 questionnaire, which have been found to be common correlates of food fussiness, were
244 used to assess children's tactile sensitivity (e.g. avoids going barefoot, especially in
245 grass and sand), taste/smell sensitivity (e.g. avoids tastes or food smells that are
246 typically part of a child's diet), and visual/auditory sensitivity (e.g. covers eyes, or
247 squints to protect eyes from light). Caregivers responded to items on a 5-point Likert
248 scale ranging from 1 (always) to 5 (never) with lower scores indicating higher sensory
249 sensitivity. SSP total scores can range from a minimum of 38 (greatest frequency of
250 sensory symptoms) to 190 (no sensory symptoms). McIntosh et al., (1999) have shown
251 good psychometric properties internal consistency of the total and subscale scores
252 (Cronbach's alpha ranged from 0.68 to 0.92) with a discriminant validity of 95% in

253 distinguishing children with and without sensory modulation difficulties. In the current
254 study good to excellent internal reliability was found for the subscales used; tactile
255 sensitivity (Cronbach $\alpha=.88$), taste/smell sensitivity (Cronbach $\alpha=.95$), visual/auditory
256 sensitivity (Cronbach $\alpha=.90$).

257 *2.2.2 The Food Preference Questionnaire for children (FPQ; Fildes et al., 2015)*

258 The FPQ requires caregivers to rate their child's liking for 75 commonly consumed
259 individual foods from 6 food groups: fruit, vegetables, meat/fish, dairy, snacks and
260 starches. Originally developed using data from a cohort of United Kingdom twins born
261 in 2007 Gemini study (n=2686), the food items on the questionnaire are rated on a 5-
262 point Likert scale, ranging from 1 (dislikes a lot) to 5 (likes a lot), with an option of
263 'never tried' which is scored as a missing response. The mean score of items pertaining
264 to each subscale was calculated, with the higher the score indicating an increased like
265 towards the given food category. This measure has been used to understand the
266 children's food preferences longitudinally (Skinner, Carruth, Bounds & Ziegler, 2002)
267 and food preferences have been previously found to be a predictor of food consumption
268 (Drewnowski & Hann, 1999). In terms of psychometric properties, the current study
269 found a good to excellent internal reliability for the food groups; fruit (Cronbach
270 $\alpha=.95$), vegetables (Cronbach $\alpha=.93$), meat/fish (Cronbach $\alpha=.92$), snacks (Cronbach
271 $\alpha=.82$), dairy (Cronbach $\alpha=.74$), however the reliability for the starch subscale was
272 lower (Cronbach $\alpha=.66$).

273 *2.2.3 The Children's Eating Behaviour Questionnaire (CEBQ: Wardle, Guthrie,
274 Sanderson & Rapoport, 2001)*

275 The Children's Eating Behaviour Questionnaire (CEBQ) is a 35-item parent-report
276 questionnaire that assesses individual eating styles of children. The food fussiness
277 subscale of the CEBQ was used in the current study to assess parental perceptions of
278 their child's food fussiness behaviour (Sandvik, Ek, Eli Somaraki, Bottai & Nowicka,
279 2019). This subscale consists of six items and includes how difficult the child is to
280 please with meals; how often the child refuses to taste new foods subscale (food
281 neophobia) and the variety of foods the child will eat (picky eating). Caregivers rated
282 the frequency of which the child exhibits the behaviour on a 5-point Likert scale ranging
283 from 1 (never) to 5 (always). An average of the six food fussiness items was calculated.
284 A high score indicates that the child displays high levels of food fussiness.
285 Development of the questionnaire revealed good internal reliability coefficients

286 (Cronbach's alpha) for all the subscales, ranging from 0.74 to 0.91 (Wardle et al., 2001).
287 In the present study Cronbach's alpha for food fussiness was 0.68.

288 **2.3 Procedure**

289 Ethical approval for this research was obtained from the University of Hertfordshire
290 University Ethical Advisory Committee Protocol Number: aLMS/PGT/UH/02784(4)
291 and the research was performed in accordance with the Declaration of Helsinki.
292 Participants were recruited through Tourettes Action, National Autistic Society charity
293 online website, online forums, local organisations, and mainstream and Special
294 Education Needs (SEN) schools who agreed to advertise the study. Participants
295 volunteered to partake by clicking on the given link, which directed them to the online
296 survey. The online participant information sheet provided further details about the
297 study, and those wishing to continue were required to provide informed consent by
298 signing an online consent form. Following this, every participant was presented with
299 the questionnaires in the same order. Information on how to seek further advice if the
300 parents had any concerns regarding their child's eating behaviours was also provided.
301 The survey took approximately 25 minutes to complete and was active for two months.
302 Families were provided no incentive to take part. At the end of the study, participants
303 were provided details of support groups for any concerns around difficulties in their
304 child's eating behaviours and reminded how to they could withdraw their data from the
305 study.

306 **2. 4. Analysis**

307 A one-way ANOVA was first computed to compare differences in BMI SDS between
308 groups. Secondly, an independent t-test was conducted to examine whether there were
309 sex differences in outcome measures; Levene's test examined homogeneity of variance
310 and significance was reported appropriately. Subsequently, Two-tailed Pearson's
311 correlations were used to establish whether child age or BMI SDS were related to food
312 fussiness.

313 To investigate differences between the children with and without neurodevelopmental
314 disorders, a series of one-way ANOVAs and post-hoc tests were conducted for each of
315 the questionnaires (SSP, FPQ & CEBQ). To examine whether sensory sensitivity was
316 a predictor of eating outcomes in the four groups (TS, ASD, ADHD & TD), a series of

317 multiple linear regressions were carried with three of the sensory subscales (tactile,
318 taste/smell, visual/auditory) as a predictors of food fussiness.

319 Mediational analyses were used to evaluate differences between each group of
320 neurodevelopmental disorders compared to TD in relation to food fussiness, and to
321 examine whether sensory sensitivity mediated this relationship. Three separate
322 mediations were carried out using the procedure and macros provided by Preacher and
323 Hayes (2008). The effect of the group (coded 0 =TD, 1 = clinical group) was used as
324 an independent predictor of food fussiness in separate analyses for each clinical group,
325 including taste/smell sensitivity as a mediator of this relationship. Categorical data has
326 been shown to be appropriate to use as an independent variable in a mediation analysis
327 (Iacobucci, 2012). The recommendations of Hayes (2013) were followed, using dummy
328 coding to represent comparisons of interests and using the asymmetric bootstrap
329 Confidence Interval.

330 **3. Results**

331 ***3.1. Descriptive statistics***

332 A one-way ANOVA revealed no significant differences in BMI SDS between the four
333 groups of children, $F(3, 73) = 1.05, p < .38$. Across the total sample of children, two-
334 tailed Pearson's correlations indicated that child food fussiness was not significantly
335 associated with child age, $r(99) = -.115, p = .26$, or BMI SDS, $r(75) = -.059, p = .61$.¹
336 An independent samples t-test revealed no significant difference in food fussiness
337 between males and females when comparing the total sample of children, $t = (96) = .26$,
338 $p = .13$. Therefore, these measures were not controlled for in further analyses. Two-
339 tailed Pearson's correlations were also carried out to see if food fussiness was
340 associated with any of the symptom measures in children with neurodevelopmental
341 disorders. For children with TS, PUTS total was not significantly correlated with food
342 fussiness, $r(24) = -.24, p = .26$. For the children with ASD, the ASSQ total was not
343 significantly correlated with food fussiness, $r(26) = .17, p = .41$. For children with
344 ADHD, none of the subscales from Conner's parent rating scale significantly correlated

¹ Food fussiness did not correlate with age or BMI SDS when split into diagnostic groups.

345 with food fussiness, including DSM-IV Inattention, r (15) = .28, p = .31, DSM-IV
 346 Hyperactivity, r (15) = .28, p = .38.

347 ***3.2. Differences in food fussiness, food preference and sensory sensitivity***

348 To examine whether there were group differences in food fussiness, measured by the
 349 CEBQ, a one-way ANOVA was conducted. The results revealed significant differences
 350 between the groups on food fussiness, F (3, 97) = 6.29, p = .001. Tukey's HSD post
 351 hoc tests, as shown in Table 1, revealed children with TS (p = .004), children with ASD
 352 (p = .001), and children with ADHD (p = .02) to have significantly higher levels of
 353 food fussiness compared to children with TD. There were no significant differences in
 354 food fussiness between the three different clinical groups (TS vs ASD p = .99; ADHD
 355 vs TS p = .99; ADHD vs ASD p = .98). Mean scores and standard deviations are shown
 356 in Table 1.

357 *Table 1: Mean scores (standard deviation) for each of the questionnaires for children*
 358 *with neurodevelopmental disorders and typically developing children.*

	TD (n=27)	TS (n=27)	ASD (n=27)	ADHD (n=17)
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Demographics				
Age	9.7(2.4)	10.2(2.6)	10.4(3.2)	10.8(3.6)
Height	143.5(16.5)	146.7(17.6)	144.7(26.4)	147.8(28.0)
Weight	37.4(18.4)	39.4(17.8)	42.4 (19.2)	60.5(10.6)
BMI SDS kg/m ²	-.5(1.9)	.6(4.1)	.9(1.3)	.6(2.2)
CEBQ				
Fussiness	3(1.0)	4(1.3)	4(1.02)	4 (.9)
FPQ				
Meat/Fish	4(.5)	4(.8)	4(1.0)	4(.6)
Dairy	3(.6)	3(.8)	3(.8)	4(.8)
Starches	4(.6)	4(.7)	3(1.1)	3(.7)
Snacks	4(.7)	4(.6)	4(.5)	4(.6)
Fruit	4(.6)	3(1.0)	3(1.2)	4(.6)
Vegetables	3(.8)	3(1.0)	3(1.1)	3(.8)
Sensory Profile				
Tactile	32(5.0)	22(6.6)	23(6.7)	25(5.0)
Taste/Smell	18(3.1)	11(6.1)	11(5.5)	13(4.1)
Visual/Auditory	24(2.2)	16(5.9)	13(5.4)	19(5.0)
Overall	165(23.6)	114(30.2)	127(45.9)	112(20.9)

359

360 To examine whether there were group differences in preference to food categories, as
 361 defined by the FPQ, a series of one-way ANOVAs were conducted. The results

362 revealed significant differences between the groups on the following food categories:
363 Starch, $F(3, 97) = 4.97, p = .003$, Fruit, $F(3, 98) = 7.64, p < .001$ and Vegetables $F(3, 98) = 3.41, p = .02$. There were no significant differences for Meat, $F(3, 96) = 2.32, p = .08$; Dairy, $F(3, 97) = .62, p = .61$; Snacks, $F(3, 97) = .77, p = .52$. To further explore the group differences in preference for starch and fruit and vegetables, post-hoc Tukey's HSD tests were conducted. Children with TS ($p = .002$) and children with ASD ($p < .001$) had significantly lower preference for fruit than TD children. Children with ASD ($p = .011$) had significantly lower preference for vegetables than TD children. The children with TS ($p = .02$) and TD children ($p = .005$) had a significantly greater preference for starch than children with ASD. The remaining comparisons did not yield significant differences in these food categories, and there were no differences in preference for any food categories between children with ADHD and the other groups.

374 Finally, to examine whether there were group differences in sensory sensitivity, the
375 SSP total score and three selected subscales were analysed through a series of one-way
376 ANOVAs. The results revealed significant differences between the groups on overall
377 sensory sensitivity, $F(3, 97) = 14.25, p < .001$; taste/smell, $F(3, 96) = 11.07, p < .001$;
378 tactile $F(3, 96) = 14.52, p < .001$; and visual/auditory sensitivity, $F(3, 97) = 23.29, p <$
379 $.001$. Compared to TD children, post-hoc Tukey's HSD tests revealed children with TS,
380 children with ASD and children with ADHD showed greater overall sensitivity ($p < .001$), and greater sensitivity to the following: taste/smell (TS, ASD and ADHD $p < .001$),
381 tactile (TS $p < .001$; ASD $p < .001$; ADHD $p = .002$), and visual/auditory
382 information (TS $p < .001$; ASD $p < .001$; ADHD $p = .003$). There were no significant
383 differences between the three neurodevelopmental groups on any of the four sensory
384 measures tested.

386 **3.3. Multiple regressions**

387 Multiple linear regression analyses were carried out to explore the relationship between
388 the individual sensory subscales as predictors of food fussiness, and these were all
389 entered into the model in the same step. As shown in table 2, taste/smell sensitivity was
390 found to be the only significant predictor for food fussiness in all groups.

391 *Table 2: Standard Coefficients of the three sensory profile subscales predicting food*
392 *fussiness*

Tactile	Taste/	Visual/	R ²	F
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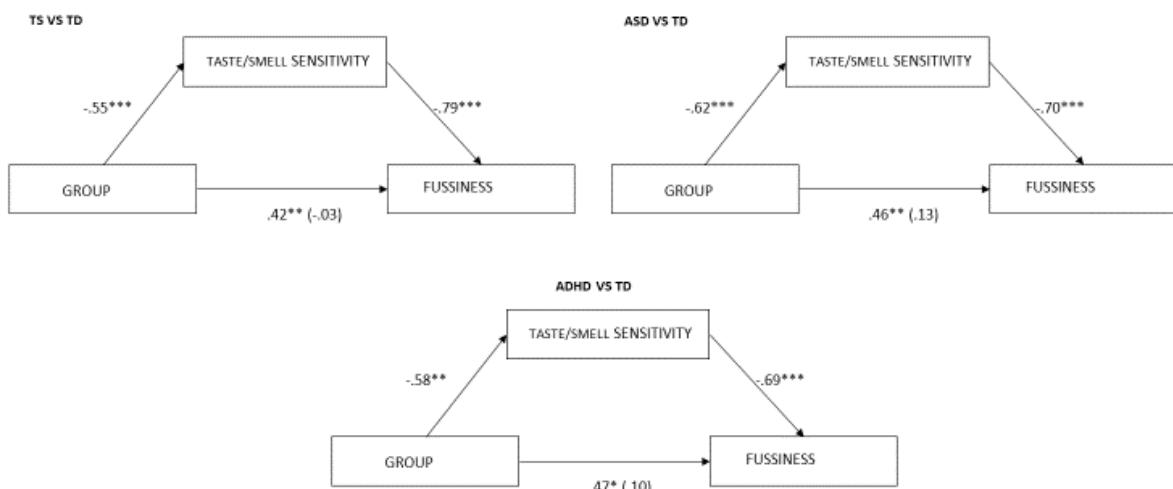
		Smell	Auditory		
TD					
	Food Fussiness	-.02	-.64***	-.46	.45
TS					
	Food Fussiness	.11	-.81***	-.13	.63
ASD					
	Food Fussiness	.33	-.55*	-.47	.39
ADHD					
	Food Fussiness	.13	-.75*	.14	.27
					2.95

393 Note: *** = $p < .001$, ** = $p < .01$, * = $p < .05$

394 **3.3. Mediation analyses**

395 The findings from the previous multiple regression analysis revealed only taste/smell
 396 sensitivity to be an independent predictor of food fussiness in all four groups of
 397 children. Therefore, we addressed whether the taste/smell sensitivity subscale was also
 398 an independent mediator of the relationship between food fussiness for each
 399 neurodevelopmental group compared to the TD group. Separate multiple linear
 400 regressions were used to explore the mediating role of taste/smell sensitivity on
 401 differences between food fussiness for each neurodevelopmental disorder compared to
 402 TD (e.g. TS vs TD, ASD vs TD, ADHD vs TD).

403 The difference in food fussiness between each neurodevelopmental group compared to
 404 TD was found to be mediated by taste/smell sensitivity. As Figure 1 illustrates, the
 405 standardized regression coefficient between group differences and taste/smell
 406 sensitivity was statistically significant, as was the standardized regression coefficient
 407 between groups and food fussiness. A significant Sobel test was found for each group
 408 analysis (TS vs TD, $Z = -4.07$, $p < .001$, ASD vs TD, $z = -3.63$, $p < .0013$, and ADHD
 409 vs TD $z = -3.02$, $p < .001$) showing differences existed between each
 410 neurodevelopmental group compared to TD, and that their food fussiness was fully
 411 mediated by the level of taste/smell sensitivity. These findings were confirmed using a
 412 bootstrapping method with 5,000 resamples and 95% confidence intervals (Hayes,
 413 2013). Results showed that the confidence interval did not include 0 in each of three
 414 separate analyses (TS vs TD LCI = -1.64: HCI = -.61; ASD vs TD LCI = -1.26: HCI =
 415 -.38; ADHD vs TD LCI = -1.23: HCI = -.36), confirming the indirect effect was
 416 statistically significant for each case.



417 Note *** $p < .001$ ** $p < .01$ * $p < .05$

418 *Figure 1: Mediation model of group and taste/smell sensitivity on food fussiness*

419 **4. Discussion**

420 The present study was the first to directly compare food fussiness across three different
 421 neurodevelopmental disorders, whilst excluding comorbidity among these disorders.
 422 The findings failed to differentiate levels of food fussiness between TS, ASD and
 423 ADHD, demonstrating heightened food fussiness and sensory sensitivity for each
 424 disorder compared to TD children. Importantly, greater taste/smell sensitivity was
 425 found to mediate the differences in food fussiness between the TD group compared to

426 the clinical groups, suggesting that it is greater sensitivity to sensory information in the
427 taste/smell domain which can account for why children with these neurodevelopmental
428 disorders have increased levels of food fussiness.

429 However, differences across the neurodevelopmental groups were also identified in
430 terms of food preferences. In contrast to previous research showing males with ADHD
431 to consume less fruit and vegetables than TD children (Ptacek et al., 2014), children
432 with ADHD in the present study did not display any preferential differences to food
433 categories. This research highlights the need to further explore contextual factors in
434 food preferences in children with ADHD children. Only children with ASD were found
435 to show a lower preference for vegetables compared to children with TD. Consistent
436 with previous research, children with TS and children with ASD were found to show
437 lower preference for fruit in comparison to children who were TD (Smith et al., 2019;
438 Maclin, Kandiah, Haroldson, & Khubchandani, 2017). Increased food fussiness and
439 reduced fruit and vegetable preference, across TS and ASD, could lead to an unvaried
440 diet with many adverse health implications. Low consumption of plant-based foods,
441 including both fruit and vegetables, have been associated with an increased risk of
442 cardiovascular disease, obesity and diabetes (Slavin & Lloyd, 2012; Aune et al., 2017),
443 and can lead to fatigue and deficiency in vital vitamins and minerals (Galloway et al.,
444 2005). This highlights the importance of exploring approaches to encourage
445 consumption of healthier foods, and the potential value of a focus on increasing fruit
446 and vegetable acceptance in children with neurodevelopmental disorders. However,
447 akin to existing literature on neurodevelopmental disorders, higher levels of food
448 fussiness were not found to be associated with children's BMI (Curtin, Bandini, Perrin,
449 Tybor & Must, 2005; Emond, Emmett, Steer & Golding, 2010).

450 Consistent with previous research, higher food fussiness was predicted by taste/smell
451 sensitivity in all groups of children (Cermak et al., 2010; Smith et al., 2019). This
452 provides further verification for the relationship between taste sensitivity and food
453 fussiness in children with both typical and atypical development (Cermak, et al., 2010).
454 However, unlike previous research that has suggested higher levels of tactile sensitivity
455 in ADHD (Ghanizadeh, 2013) and ASD (Schmitt et al., 2008) to be associated with
456 food fussiness, no significant relationship was established using the tactile measure,
457 although this may have been due to the small sample sizes. Instead, this is the first study

458 to show that greater taste/smell sensitivity may account for differences in food fussiness
459 between children with and without neurodevelopmental disorders.

460 The role of taste/smell sensitivity in food fussiness in children with these
461 neurodevelopmental disorders has important clinical implications, meaning pathways
462 of interventions should prioritise techniques which consider sensory sensitivity.
463 Repeated exposure techniques may be useful to gradually desensitise children and
464 increase their acceptance of different sensory experiences (Farrow & Coulthard, 2012).
465 Although, in children with neurodevelopmental disorders, such interventions may need
466 to be carried out over a lengthier time period than with TD children (Kim, Chung &
467 Jung, 2018), due to resistance to change food repertoire and unwillingness to try novel
468 foods identified in these clinical samples (e.g. Marí-Bauset, Zazpe, Mari-Sanchis,
469 Llopis-González, & Morales-Suárez-Varela, 2014). Additionally, children with ASD
470 have been found to explore foods for longer before making a hedonic decision than TD
471 children (Luisier et al., 2019). It is possible that providing children with
472 neurodevelopmental disorders with more time to explore their food could help them to
473 manage their sensory experiences independently and increase their familiarity with and
474 acceptance of exposed foods. In such cases, a multidisciplinary team, including
475 occupational therapists and registered dieticians, would be useful to individualise
476 interventions to the sensory characteristics of each child.

477 There are limitations of the current study that need to be noted. Firstly, while the food
478 preference questionnaire identified a reduced preference for fruit in the TS and ASD
479 groups, the absence of a food diary means specific detail on the types of foods children
480 with neurodevelopmental disorders eat, regarding both frequency and portion size, is
481 lacking (Day, McKeown, Wong, Welch & Bingham, 2001). The use of parent report
482 for height, weight and feeding problems has also been highlighted as not being the most
483 reliable method to gain information on children's BMI and feeding difficulties (for a
484 review see Arts-Rodas & Beniot, 1998). For example, parents may perceive even minor
485 feeding problems as major (Archer & Szatmari, 1990). There is a possibility that use of
486 parent-report for anthropometrics may also be discrepant to objective measures and
487 lead to miscalculation of BMI (Wedeen et al., 2013). Although objective measures are
488 optimal, where this is not feasible studies have highlighted strong and positive
489 correlations of parent-report of height and weight with objective measurements
490 (Haycraft & Blissett, 2012), and parents to be as accurate as a trained clinician in their

491 reporting (Chai et al., 2019). All children should also have been screened for other
492 neurodevelopmental symptomology; screening tools to assure correct inclusion in
493 diagnostic groups were used, but it is possible that screening all the children in the study
494 for each disorder of interest may have identified additional undiagnosed comorbid
495 problems. In the SSP, taste and smell sensory domains are combined into a single
496 subscale meaning there is an inability to differentiate between these characteristics
497 (Hubbard, Anderson, Curtin, Must & Bandini, 2014). Additionally, it is noted that there
498 are some items for the sensory taste/smell subscale that overlap with items for food
499 fussiness. Therefore, further work probably needs to make use of alternative measures
500 and observational sensory tests to confirm the importance and address the specific role
501 of sensory sensitivity in this domain for explaining food fussiness in children with
502 neurodevelopmental disorders.

503 The present study was the first to demonstrate that similar, high levels of food fussiness
504 are present across several individual neurodevelopmental disorders, thus indicating that
505 comorbid diagnoses do not underlie the effect, as previously suggested. It also suggests
506 taste/smell sensory domains may be responsible for similar patterns of food fussiness
507 that have been evidenced in children with neurodevelopmental disorders. It is clinically
508 important for future research to better understand how interventions, which take into
509 consideration taste/smell sensitivity impairments, may prevent or reduce food
510 fussiness.

511 **5. Acknowledgements**

512 We wish to thank Tourettes Action and the National Autistic Society for their support
513 with recruitment, and all the parents who kindly gave up their time to participate in this
514 research.

515

516 **6. Declarations**

517 This research did not receive any specific grant from funding agencies in the public,
518 commercial, or not-for-profit sectors.

519

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