Neural correlates of taste reactivity in autism spectrum disorder

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ABSTRACT

Selective or 'picky' eating habits are common among those with autism spectrum disorder (ASD). These behaviors are often related to aberrant sensory experience in individuals with ASD, including heightened reactivity to food taste and texture. However, very little is known about the neural mechanisms that underlie taste reactivity in ASD. In the present study, food-related neural responses were evaluated in 21 young adult and adolescent males diagnosed with ASD without intellectual disability, and 21 typically-developing (TD) controls. Taste reactivity was assessed using the Adolescent/Adult Sensory Profile, a clinical self-report measure. Functional magnetic resonance imaging was used to evaluate hemodynamic responses to sweet (vs. neutral) tastants and food pictures. Subjects also underwent resting-state functional connectivity scans.

The ASD and TD individuals did not differ in their hemodynamic response to Gustatory stimuli. However, the ASD subjects, but not the controls, exhibited a positive association between self-reported taste reactivity and the response to sweet tastants within the insular cortex and multiple brain regions associated with gustatory perception and reward. There was a strong interaction between diagnostic group and taste reactivity on tautans response in brain regions associated with ASD pathophysiology, including the bilateral anterior superior temporal sulcus (STS). This interaction of diagnosis and taste reactivity was also observed in the resting state functional connectivity between the anterior STS and dorsal mid-insula (i.e., gustatory cortex).

These results suggest that self-reported heightened taste reactivity in ASD is associated with heightened brain responses to food-related stimuli and atypical functional connectivity of primary gustatory cortex, which may predispose these individuals to maladaptive and unhealthy patterns of selective eating behavior.


1. Introduction

Selective or 'picky' eating habits are a common feature of autism spectrum disorder (ASD) (Cermak et al., 2010; Diolordi et al., 2014; Kuschner et al., 2015; Williams et al., 2000). Nearly two-thirds of children with ASD exhibit some form of selective eating, including such traits as food neophobia, food refusal, and insistence on sameness while eating (Cermak et al., 2010; Williams et al., 2000). Compared to the general population, and other atypically developing groups, those with ASD are far more likely to exhibit these maladaptive eating habits (Berkman et al., 2007). While some degree of selective eating is common in young children, most outgrow this behavior at an early age (Birch, 1999). However, in many individuals with ASD, these atypical eating behaviors never fully resolve (Fodstad and Matson, 2008). This high degree of selective eating can lead to the development of poor diets in children with ASD (Sharp et al., 2013), which in turn can lead to inadequate or improper nutrition and poor health outcomes such as a higher risk for obesity (Phillips et al., 2014). Furthermore, these atypical eating behaviors constitute an added burden on families and caregivers, often leading to significant stress at mealtimes (Anderson et al., 2012).

However, despite the prevalence of these behaviors, very little is known about the neural mechanisms that underlie selective eating in ASD. Many of these behaviors are related to other frequently reported symptoms of ASD such as increased sensory reactivity, including heightened reactivity to taste, smell, and oral texture (Cermak et al.,...
Direct studies of taste perception in ASD demonstrate that the identification of specific tastes, such as sucrose, citric acid, or quinine, is impaired in ASD relative to typically-developing (TD) controls (Bennetto et al., 2007; Tavassoli and Baron-Cohen, 2012). In contrast, taste detection thresholds, measured via electrogustometry on the tongue, are comparable between ASD and controls (Bennetto et al., 2007), suggesting a central, rather than peripheral, deficit in taste perception in ASD. Other studies have identified that the perceived sweetness of sucrose, while not different between ASD and control subjects, is negatively related to the severity of ASD social symptoms (Damiano et al., 2014). These equivocal results are likely due to the wide heterogeneity of symptoms present in individuals with ASD. While atypical sensory processing has recently been incorporated as a diagnostic criterion of ASD in the latest edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), this symptom domain is still extremely broad, encompassing multiple sensory modalities, within which individuals may exhibit either hypo- or hyper-reactivity (Hazen et al., 2014). This highlights the need to examine more than simply group differences in studies of sensory perception in ASD, but also to identify significant relationships between behavioral symptoms specific to the sensory modality in question (e.g. taste or smell) and brain activity associated with the perception of that modality.

As such, heightened reactivity in other sensory domains in ASD, such as auditory, visual, or tactile, has been associated with heightened neural activation to those stimuli within the associated primary sensory cortices (Green et al., 2015; Green et al., 2013; Takarae et al., 2014). This suggests that heightened reactivity to taste in ASD would also be associated with heightened taste response within primary gustatory regions of the brain. One recent fMRI study that examined responses to pictures of palatable foods in fasting adolescents identified that ASD subjects exhibited abnormally increased hemodynamic activity to food pictures in the dorsal mid-insular cortex (Casco et al., 2012). In neuroimaging studies of TD subjects, the dorsal mid-insula has been identified as a region of primary gustatory cortex (Avery et al., 2015; Ogawa et al., 2005; Small, 2010), which also exhibits specific activation to pictures of foods (Simmons et al., 2005; Simmons et al., 2013; van der Laan et al., 2011) relative to other object categories. While these results are suggestive, much still remains unknown about the neural basis of taste reactivity in ASD. Most importantly, no prior neuroimaging study has examined whether the brain’s response to tastes is actually altered in ASD, and whether that response is related to individuals’ self-reported taste reactivity.

To address this gap in our knowledge of ASD, we used fMRI to assess brain hemodynamic responses to gustatory stimuli in individuals with ASD without intellectual disability. Additionally, atypical patterns of resting functional connectivity are one of the most frequently reported neuroimaging findings in ASD (Cerliani et al., 2015; Cheng et al., 2015; Gotts et al., 2012), and heightened sensory reactivity in ASD has been associated with heightened functional connectivity between limbic and cortical brain regions (Green et al., 2017; Green et al., 2015). As such, resting state fMRI data were also collected from these subjects to determine if atypical hemodynamic responses to gustatory stimuli in ASD subjects were reflected in atypical patterns of resting functional connectivity.

2. Methods and materials

2.1. Participants

A total of 21 males diagnosed with ASD and 21 TD males, between the ages of 15 and 29, were included in the study. Participants with ASD were recruited from the Washington, DC, metropolitan area and met Diagnostic and Statistical Manual of Mental Disorders – Fifth Edition (DSM-5) diagnostic criteria for ASD. Parents of participants with ASD received the Autism Diagnostic Interview (Lord et al., 1994); and participants with ASD were administered the Autism Diagnostic Observation Schedule (Lord et al., 2000), modules 3 or 4 by a trained, research reliable clinician. Based on these measures, all participants with ASD met the cutoff for the category designated as “broad autism spectrum disorders” according to criteria established by the National Institute of Child Health and Human Development/National Institute on Deafness and Other Communication Disorders Collaborative Programs for Excellence in Autism (Lainhart et al., 2006). In addition, IQ scores were assessed for all participants, and all full-scale IQ scores were ≥80 as measured by the Wechsler Abbreviated Scale of Intelligence –I or –II. Ethics approval for this study was granted by the NIH Combined Neuroscience Institutional Review Board under protocol number 10-M-0027. The institutional review board of the National Institutes of Health approved all procedures, and written informed assent/consent was obtained for all subjects and/or their parent/guardian, when appropriate. Participants were excluded from taking part in the study if they had any history of neurological injury, known genetic or medical disorders that may impact the results of cognitive testing and/or neuroimaging, prenatal drug exposure, severely premature birth or birth trauma, or any exclusion criteria for MRI. TD participants were also excluded if they had any past or present psychiatric conditions (e.g., depression or anxiety disorders), or current usage of psychotropic medications. See Table 1 for participant demographics.

2.2. Experimental design

Participants completed fMRI scans during two outpatient visits to the National Institutes of Health Clinical Center in Bethesda, MD. Participant sessions were split into two visits in order to accommodate the time required for multiple behavioral assessments, the taste assessment, and the imaging tasks. On the first day, subjects completed behavioral assessments followed by an anatomical MRI and an 8-minute eyes-open resting-state scan. On the second visit, subjects completed our Food Picture and Gustatory Mapping tasks during scanning.

2.3. Behavioral assessments

Prior to imaging, participants completed the Adolescent/Adult Sensory Profile (AASP), a self-report measure used to identify sensory processing patterns that impact everyday functioning (Brown, 2002). To measure taste reactivity specifically, four items from the taste/smell and touch sub-sections of the AASP were used (items 2, 5, 7, & 34, which assess food neophobia and reactivity to strong tastes and food textures). While these items assess different aspects of sensory seeking/responsiveness, previous research in an expanded population of ASD and TD adolescents/young adults indicated that ASD subjects self-reported greater food neophobia and texture sensitivity and lower preference for strong tasting or spicy foods than TD controls (Kuschner et al., 2010; Kral et al., 2015; Kuschner et al., 2015; Williams et al., 2000).

Table 1

<table>
<thead>
<tr>
<th></th>
<th>ASD (n = 21)</th>
<th>TD (n = 21)</th>
<th>t (ASD – TD)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>21 ± 3</td>
<td>22 ± 3</td>
<td>0.96</td>
<td>0.34</td>
</tr>
<tr>
<td>BMII</td>
<td>27 ± 6</td>
<td>23 ± 3</td>
<td>3.11</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>IQ</td>
<td>110.9 ± 13.8</td>
<td>119.52 ± 9.93</td>
<td>−2.32</td>
<td>&lt; 0.03</td>
</tr>
<tr>
<td>AASP – taste reactivity</td>
<td>11 ± 4</td>
<td>8 ± 3</td>
<td>2.67</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>AASP – sensory sensitivity</td>
<td>32 ± 8</td>
<td>25 ± 8</td>
<td>2.60</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Sucrose molarity</td>
<td>0.38 ± 0.11</td>
<td>0.39 ± 0.11</td>
<td>0.33</td>
<td>0.74</td>
</tr>
<tr>
<td>ASA24 – HEI</td>
<td>46 ± 17</td>
<td>50 ± 11</td>
<td>0.81</td>
<td>0.42</td>
</tr>
<tr>
<td>ASA24 – Kcal consumed</td>
<td>2760 ± 840</td>
<td>2378 ± 631</td>
<td>1.51</td>
<td>0.14</td>
</tr>
</tbody>
</table>

AASP – Adolescent/Adult Sensory Profile; ASA24 – Automated Self-Administered 24-hr dietary recall.
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