



Intact hedonic responses to sweet tastes in autism spectrum disorder



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ABSTRACT

The Sweet Taste Test (STT) is a standardized measure designed to index the ability to detect differences in sweet tastes (sweet taste sensitivity) and hedonic responses to sweet tastes (sweet taste liking). Profiles of response on the STT suggest enhanced hedonic responses to sweet tastes in psychiatric disorders characterized by dysfunctional reward processing systems, including binge-eating disorders and substance use disorders, and a putative mechanism governing STT responses is the brain opioid system. The present study examined STT responses in 20 adults with autism spectrum disorder (ASD) and 38 healthy control adults. There were no differences in sweet taste sensitivity or hedonic response to sweet tastes between the ASD and control groups. Within the ASD sample, ASD symptom severity was associated with sweet taste sensitivity, but not hedonic response to sweet taste. Results may ultimately shed light on brain opioid system functioning in ASD.

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1. Introduction

Autism spectrum disorder (ASD) is characterized by social communication impairments and restricted and repetitive behaviors and interests (APA, 2013). Despite the heterogeneity of symptom presentation in ASD, impaired reward-based processes have been proposed as a possible core deficit (Dawson et al., 2004; Dawson, Webb, & McPartland, 2005; Grelotti, Gauthier, & Schultz, 2002). Consistent with this conceptualization, there is emerging evidence for atypical reward processing of both social and nonsocial rewards in ASD across both behavioral and neurobiologic domains (e.g., Cascio et al., 2012; Damiano, Aloï, Treadway, Bodfish, & Dichter, 2012; Delmonte et al., 2012; Dichter, Richey, Rittenberg, Sabatino, & Bodfish, 2012b).

Reward processing may be decomposed into a number of distinct constructs mediated by distinguishable neurobiological mechanisms, including the anticipation of rewards (i.e., reward “wanting” or motivation toward future rewards) and the experience of obtaining rewards (i.e., reward “liking” or reward consumption; Berridge & Robinson, 2003; Krangelbach &

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Berridge, 2009). Amongst studies that have examined these two components of reward processing separately, there is considerable evidence for impairments in reward “wanting” in ASD (for review, see Dichter, Damiano, & Allen, 2012a). However, evidence for impairments in reward consumption or “liking” in ASD is less consistent (Kohls, Chevallier, Troiani, & Schultz, 2012). Indeed, these constructs are neurobiologically distinct; reward “wanting” is associated with mesolimbic dopaminergic brain circuitry, and reward “liking” is mediated by a medial prefrontal cortical network that is heavily innervated by neural opioid systems (Barbano & Cador, 2007; Berridge & Kringelbach, 2011).

Studies of reward processing in ASD to date have relied primarily on paradigms involving the presentation of visual stimuli (i.e., images representing monetary values or images of social or non-social rewards) which are secondary rewards and thus suboptimal for assessing hedonic “liking” responses. Notably, no study to date has directly examined responses to primary rewards in ASD. The current study sought to address this gap in the literature by investigating hedonic responses to sweet tastes in ASD.

Hedonic responses to sweet tastes are heritable and relatively stable across time and changes in metabolic state (Keskitalo et al., 2007; Looy & Weingarten, 1991; Mennella, Pepino, & Reed, 2005; Thompson, Moskowitz, & Campbell, 1976). A tendency to prefer more concentrated sweet solutions has been found across several different forms of psychopathology characterized by dysfunctional reward processing, including eating disorders and substance use disorders (Kampov-Polevoy, Eick, Boland, Khalitov, & Crews, 2006b; Kampov-Polevoy, Garbutt, Davis, & Janowsky, 2006c; Kampov-Polevoy, Tsoi, Zvartau, Neznanov, & Khalitov, 2001; Kampov-Polevoy et al., 2006d; Krahn et al., 2006; Wronski et al., 2007). The processing of sweet taste involves two distinct neural pathways: (1) a taste detection/discrimination pathway extending from the thalamus to the primary gustatory cortex, and (2) a taste hedonic processing pathway extending from the thalamus to the limbic system and other reward-related brain regions (Hajnal & Norgren, 2005; Kosar, Grill, & Norgren, 1986; Reilly, Grigson, & Norgren, 1993). This second pathway is centrally involved in sweet taste liking (Pecina & Berridge, 2005; Pecina, Smith, & Berridge, 2006) and is linked to endogenous levels of brain opioids (Calcagnetti & Reid, 1983; Garbutt et al., 2009; Leventhal & Bodnar, 1996; Miller, Barr, & Young, 1994; Pecina & Berridge, 2005; Pepino & Mennella, 2005). Specifically, brain opioids regulate sweet taste liking, sensitivity to the mood altering effects of sweet tastes, and coding for the valence of rewards more broadly (Berridge & Robinson, 1998; Kampov-Polevoy, Alterman, Khalitov, & Garbutt, 2006a; Smith & Berridge, 2007). In this regard, hedonic response to sweet tastes is a marker of opioid functioning related to the hedonic processing of a primary reward.

The purpose of the current study was to investigate potential differences between individuals with ASD and controls in sweet taste sensitivity and sweet taste liking, as well as to examine associations between these measures and ASD symptom severity. These constructs were indexed using a standardized measure of sweet taste sensitivity and liking, the Sweet Taste Test (STT), which has been used extensively in previous studies of both clinical and nonclinical populations (e.g., Dichter, Smoski, Kampov-Polevoy, Gallop, & Garbutt, 2010; Kampov-Polevoy et al., 2006c; Lange, Kampov-Polevoy, & Garbutt, 2010). Given the growing literature on reward processing in ASD and evidence that perception of sweet tastes is intact in ASD (Bennetto, Kuschner, & Hyman, 2007), we hypothesized that the ASD group would not differ in *sensitivity to sweet tastes* but only in *sweet liking* (i.e., *the hedonic response to sweet tastes*) and that, within the ASD group, *sweet liking* would be related to ASD symptom severity.

2. Method

2.1. Participants

The ASD group included 21 adults with ASD diagnoses provided by experienced licensed clinicians and confirmed with the Autism Diagnostic Observation Schedule–Generic (ADOS–G) using standard clinical cutoffs (Lord et al., 2000). The ADOS–G is a semi-structured observational assessment used to evaluate symptoms of ASD across five behavioral domains: Language and Communication, Reciprocal Social Interaction, Play or Imagination/Creativity, Stereotyped Behaviors and Restricted Interests, and Other Abnormal Behaviors. The ADOS–G has demonstrated strong psychometric properties, including good test–retest reliability, interrater reliability, and internal consistency (Lord et al., 2000). Participants in the ASD group were recruited via an ASD research registry maintained by the Carolina Institute for Developmental Disabilities. The control group included 40 adults without ASD recruited from databases of undergraduate students participating in research for course credit and from databases of control participants maintained by the Duke–UNC Brain Imaging and Analysis Center. All participants had IQ scores ≥ 85 on the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999) and no known sensory deficits. The data from three control participants and one participant with ASD were discarded due to missing data, resulting in final samples of 20 participants with ASD (3 females, 17 males) and 37 control participants (4 females, 33 males). The final ASD group was 85% Caucasian, 10% African–American, and 5% Asian, and the control group was 78.4% Caucasian, 8.1% African–American, 8.1% Asian, 2.7% Native American, and 2.7% Hispanic. All participants provided written informed consent.

Groups did not differ in Verbal, Performance, or Full Scale IQ, all p 's $> .10$ (see Table 1). In addition, groups did not differ in gender or race/ethnicity distributions, all p 's $> .10$. However, ASD participants were significantly older on average ($M = 25.95$, $SD = 7.96$) than control participants ($M = 20.42$, $SD = 5.64$), t -test for unequal variance: $t(29.31) = 2.76$, $p = .01$. Thus between-groups analyses were conducted both with and without age as a covariate.

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