8-2019

Influences of Autism Spectrum Disorder on Sensory and Emotional Responses to Smell and Taste Cues

Asmita Singh
University of Arkansas, Fayetteville

Influences of Autism Spectrum Disorder on Sensory and Emotional Responses to Smell and Taste Cues

A thesis submitted in partial fulfillment
of the requirements for the degree of
Master of Science in Food Science

by

Asmita Singh
Sam Higginbottom University of Agriculture, Technology and Sciences
Bachelor of Technology, 2017

August 2019
University of Arkansas

This thesis is approved for recommendation to the Graduate Council.

Han-Seok Seo, Ph.D.
Thesis Director

Navam Hettiarachchy, Ph.D.
Committee Member

Suzanne Kucharczyk, Ph.D.
Committee Member

ABSTRACT

Autism spectrum disorder (ASD) is a developmental disability that causes social, communication, behavioral and sensory challenges. The prevalence has been on a rise, with the latest reports stating 1 in 59 children is diagnosed with ASD. These challenges play a significant role in feeding behavior, leading to reduced nutrition among individuals. Much research in this field has been attributed to children, however, this study was focused on the adult population, in an attempt to improve their quality of life. Building on previous findings and knowledge gaps, the objectives of this thesis were two-fold: To better understand the sensory experiences of adults with ASD and their responses toward food and beverages and 2) to determine whether ASD influences sensory and emotional responses to smell and taste stimuli. Participants with ASD reported abnormal and non-uniform sensory experiences, which combined with environmental factors, influenced their food choices and eating behavior. Odor identification and odor discrimination ability were reduced in adults with autism, as compared to their control counterparts. Additionally, the taste identification ability of adults with autism was also reduced. The perception of odors, in terms of arousal and intensity also differs among the two groups. Increased sensitivity to sweet taste and decreased liking of sour taste was observed. It seemed that both odors and tastes with a sour quality were perceived as more intense by the test group. Moreover, the emotions evoked by taste solutions differed among the two groups, people with ASD reported a lesser number of emotion attributes evoked by tastes and a higher number of negative emotions for sweet and sour tastes. In conclusion, ASD affects the olfactory and taste abilities of people.

Keywords: *Autism spectrum disorder, Sensory perception, Emotion, Eating behavior, Sensitivity.*

ACKNOWLEDGMENTS

This journey would not have been possible without the support of my professors, family, and friends. I would like to extend sincere gratitude to my advisor, Dr. Han-Seok Seo (Associate Professor, Department of Food Science) for his support and instructive guidance throughout my graduate career. I am appreciative of my committee members, Dr. Navam Hettiarachchy (Professor, Department of Food Science), and Dr. Suzanne Kucharczyk (Assistant Professor, Department of Special Education) for their vital contributions. In addition, I am thankful to my lab mates at the Sensory Service Centre, Ms. Tonya Tokar (Former Lab Manager), Mr. Kyle Buffin (Lab Manager), Ms. Victoria Hogan (Research Specialist), Ms. Peggy Pang (Lab Technician), Ms. Tina McMaster (Lab Technician), Dr. Shilpa Samant (PhD graduate), Mr. Matthew Chapko (Master graduate), Ms. Sara Jarma Arroyo (PhD student), Ms. Ragita Pramudya (PhD student), Mr. Thadeus Beekman (PhD student), Mr. Reza Moltaji (Master student) and Ms. Shady Jeesan (Master student), for their help and support throughout my program of study. To my family, thank you for encouraging me to follow my dreams and constantly being there for me. Last, but not the least, my heartfelt thankfulness to Lifestyles Inc. (Fayetteville, AR) for helping me with recruitment and the enthusiastic participants, without whom this project would not be complete.

TABLE OF CONTENTS

LIST OF TABLES

CHAPTER 2

CHAPTER 4

CHAPTER 5

LIST OF FIGURES

CHAPTER 4

CHAPTER 1

GENERAL INTRODUCTION

Autism spectrum disorder (ASD) is characterized as a developmental disability that can cause significant social, communication, and behavioral challenges (Centers for Disease Control and Prevention, 2016). The five diagnostic criteria of ASD, as described in the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM-5) by the American Psychiatric Association (2013), are as follows: Firstly, there are persistent deficits in social communication and social interaction across multiple contexts, currently or by history. Secondly, there are restricted, repetitive patterns of behavior, interests, or activities, currently or by history. Thirdly, symptoms must be present in the early developmental period (but may not become manifest until social demands exceed limited capacities or may be masked by learned strategies in later life). Fourthly, symptoms cause clinically significant impairment in social, occupational, or other important areas of current functioning. Finally, these disturbances are not better explained by intellectual disability (intellectual developmental disorder) or global developmental delay. Intellectual disability and ASD frequently co-occur which can cause further difficulty in attaining a diagnosis (Autism Speaks, 2013).

The prevalence of ASD reported in the US has been increasing: 1 case per 150 children in 2000, 1 in 88 of in 2008, and 1 in 68 children (1 in 42 boys and 1 in 189 girls) in 2012 (Centres for Disease Control and Prevention 2016). The latest reports by Centres for Disease Control and Prevention (2019) state that the prevalence of Autism as of 2014 is 1 in 59 children. There is ongoing research to identify the causes of ASD, and various speculations have been made. ASD maybe a family of diseases with common phenotypes linked to a series of genetic anomalies, each of which is responsible for no more than 2–3% of cases. The total fraction of ASD that can be attributed to genetic inheritance may be about 30–40% (Landrigan et al., 2012).

Although incurable, symptoms of ASD seem to reduce with increasing age. In a study by Shattuck et al. (2007), a greater proportion of the sample experienced declines than increases in their level of ASD symptoms. ASD commonly co-occurs with other developmental, psychiatric, neurologic, chromosomal, and genetic diagnoses patients (Abdul-Rehman & Hudgins 2006). There may be coexisting psychiatric symptoms in individuals with ASD, including depression, mania, hyperactivity, inattention, aggression, obsessive-compulsive disorder, Tourettes disorder, specific phobias, and generalized anxiety (Ghaziuddin et al., 1998).

Children with ASD have problems with modulating sensory input (Ornitz, 1985). Differences in sensory processing may cause core features of ASD, such as language delay (auditory processing) and difficulty with reading emotion from faces (visual processing) (Marco et al., 2011). Sensory processing problems can be present in people with ASD in several forms, a child or adult with ASD can be under-responsive to one stimulus and over-responsive to another (Rogers & Ozonoff, 2005). These sensory difficulties can predict communication competence and maladaptive behavior (Lane et al., 2010). Researchers support the use of sensory-based interventions in the remediation of communication and behavioral difficulties in autism.

There is a strong relationship between atypical sensory responsiveness and social impairment for both typically developing children and for those with High Functioning ASD (Hilton et al., 2010). These sensory processing problems may lead to feeding issues that are prevalent in people with ASD. Children with ASDs show more food refusal and exhibit a more limited food repertoire than typically developing children (Bandini et al., 2010). Children with

ASD are significantly more likely to refuse foods based on texture/consistency, taste/smell, mixtures, brand and shape (Hubbard et al., 2014). Schreck et al. (2006) showed that food refusals were primarily related to food presentation, specific utensil requirements, food texture, and oral motor problems. Whiteley et al. (2000) have also indicated that brand, product name, and packaging information might affect food selectivity.

The atypical sensory response among toddlers is often one of the first red flags for children with autism (Hilton et al., 2010). Being able to identify them may facilitate early diagnosis and intervention. There are knowledge gaps in our understanding of what impacts the eating behaviors of people with ASD. Firstly, many of the studies regarding sensory and feeding issues are focused on children, not adults. Secondly, less attention has been paid to chemosensory systems than to visual, tactile, and auditory systems (Luisier et al., 2015). Thirdly, the limited research that has been conducted to determine whether ASD influences multisensory interactions among five sensory cues, particularly smell and taste, has given contradicting results. Finally, little is known about whether and how ASD affects emotional responses toward single or multisensory cues of food and beverages.

Building on previous findings and knowledge gaps associated with the effects of ASD on sensory perception and eating behavior, the objectives of this thesis were two-fold: 1) To better understand the sensory experiences of adults with ASD and their responses toward food and beverages and 2) to determine whether ASD influences sensory and emotional responses to smell and taste stimuli.

REFERENCES

Abdul-Rahman, O. A., & Hudgins, L. (2006). The diagnostic utility of a genetics evaluation in children with pervasive developmental disorders. Genetics in Medicine, 8, 50.

American Psychiatric Association (2013). Diagnostic and statistical manual of mental disorders, 5th Edition. Washington, DC : American Psychiatric Publishing.

Autism Speaks. DSM 5 Diagnostic Criteria. 2013. https://www.autismspeaks.org/what-autism/diagnosis/dsm-5-diagnostic-criteria \ Accessed on March 15 2018.

Bandini, L. G., Anderson, S. E., Curtin, C., Cermak, S., Evans, E. W., Scampini, R., & Must, A. (2010). Food selectivity in children with autism spectrum disorders and typically developing children. The Journal of Pediatrics, 157, 259-264.

Centre for Disease Control and Prevention. Data and Statistics on Autism Spectrum Disorder. 2019. https://www.cdc.gov/ncbddd/autism/data.html\ Accessed on May 29, 2019.

Centre for Disease Control and Prevention. Facts about autism. 2016. https://www.cdc.gov/ncbddd/autism/facts.html \ Accessed on March 15, 2018.

Ghaziuddin, M., & Greden, J. (1998). Depression in children with autism/pervasive developmental disorders: a case-control family history study. Journal of Autism and Developmental Disorders, 28, 111-115.

Hilton, C. L., Harper, J. D., Kueker, R. H., Lang, A. R., Abbacchi, A. M., Todorov, A., & LaVesser, P. D. (2010). Sensory responsiveness as a predictor of social severity in children with high functioning autism spectrum disorders. *Journal of autism and developmental disorders*, *40*(8), 937-945.

Hubbard, K. L., Anderson, S. E., Curtin, C., Must, A., & Bandini, L. G. (2014). A comparison of food refusal related to characteristics of food in children with autism spectrum disorder and typically developing children. Journal of the Academy of Nutrition and Dietetics, 114, 1981-1987.

Landrigan, P. J., Lambertini, L., & Birnbaum, L. S. (2012). A research strategy to discover the environmental causes of autism and neurodevelopmental disabilities. Environmental Health Perspectives, 120, a258.

Lane, A. E., Young, R. L., Baker, A. E., & Angley, M. T. (2010). Sensory processing subtypes in autism: Association with adaptive behavior. *Journal of autism and developmental disorders*, *40*(1), 112-122.

Luisier, A. C., Petitpierre, G., Ferdenzi, C., Clerc Bérod, A., Giboreau, A., Rouby, C., & Bensafi, M. (2015). Odor perception in children with autism spectrum disorder and its relationship to food neophobia. Frontiers in Psychology, 6, 1830.

Marco, E. J., Hinkley, L. B., Hill, S. S., & Nagarajan, S. S. (2011). Sensory processing in autism: a review of neurophysiologic findings. Pediatric Research, 69, 48R-54R.

Ornitz, E. M. (1985). Neurophysiology of infantile autism. Journal of the American Academy of Child Psychiatry, 24(3), 251-262.

Rogers, S. J., & Ozonoff, S. (2005). Annotation: What do we know about sensory dysfunction in autism? A critical review of the empirical evidence. Journal of Child Psychology and Psychiatry, 46, 1255-1268.

Schreck, K. A., & Williams, K. (2006). Food preferences and factors influencing food selectivity for children with autism spectrum disorders. Research in Developmental Disabilities, 27, 353-363.

Shattuck, P. T., Seltzer, M. M., Greenberg, J. S., Orsmond, G. I., Bolt, D., Kring, S., ... & Lord, C. (2007). Change in autism symptoms and maladaptive behaviors in adolescents and adults with an autism spectrum disorder. *Journal of autism and developmental disorders*, *37*(9), 1735-1747.

Whiteley, P., Rodgers, J., & Shattock, P. (2000). Feeding patterns in autism. Autism, 4, 207-211.

CHAPTER 2

REVIEW OF LITERATURE

1. Background

Autism was first described in 1943 by Leo Kanner. The children in his study showed extreme aloneness, were unaffected by people, did not respond to things from the outside world and established relations to objects instead (Kanner, 1943). In his article, he noted that infants with autism look at others less frequently and do not orient to their names as often as infants with mental retardation. Both infants with ASD and those with mental retardation use gestures and look to objects held by others less frequently and engage in repetitive motor actions more frequently than typically developing infants (Osterling et al., 2002). Approximately 5% of parents suspect a problem before their child is one year of age (Ornitz et al., 1977), and most parents express concern to their pediatrician by the time their child is eighteen months of age (Siegel et al., 1988). Pediatric health care providers are suggested to administer two ASD screenings, at ages eighteen and twenty-four months, using a valid and reliable screening tool (Johnson & Myers, 2007).

The increase in the prevalence of ASD is dependent on various factors. Identification and broadening of the diagnostic criteria and general awareness contribute to it (Gernsbacher et al., 2005). A study showed that ASD prevalence was significantly higher among non-Hispanic white children aged 8 years (15.5 per 1,000) compared with non-Hispanic black children (13.2 per 1,000), and Hispanic (10.1 per 1,000) children aged 8 years. These differences show that treatment and service to children with ASD might be lacking or delayed for some groups (Christensen et al., 2016). For the Hispanic population, poor access to care due to poverty, limited English proficiency, lack of empowerment to take advantage of services, lack of awareness of ASD and stigma associated with disability, may be the factors that reduce the identification of ASD among Hispanic children (Zuckerman et al., 2014).

2. Causes of ASD

There have been many speculations about the cause of ASD for a long time and the perspective of people changed as the research in this field continued.

2.1. Refrigerator mother theory

Kanner (1949) observed a small sample of children from well-educated families and stated that children with ASD were more likely to be born into highly intellectual families, due to mothering style being "cold". He coined the term "refrigerator mother" (Project Autism, 2018). Bettleheim (1950) claimed that ASD developed in some children because of psychological harm by their mothers and believed that ASD was an emotional disorder. These theories were dismissed after further research.

2.2. Vaccines

Wakefield et al. (1998) reported a link between MMR vaccines and autism. This report was later found to be a result of misconduct and was retracted by The Lancet in 2010. Since then, there are still many controversies about vaccines and ASD, especially since many parents cite the normal development of their children until they receive vaccines at about the age of 18 months (Lewine et al., 1999) and this is the stage where the first signs of autism become apparent. Vaccines have been extensively proven not to be an environmental risk factor for ASD, and there are multiple reports denying the association of ASD with vaccines. (Bailey et al., 1995; DeStefano, 2007; DeStefano et al., 2013; Institute of Medicine, 2004).

2.3. Heavy metals

Thimerosal, an organo-mercury compound has been implicated as a cause of ASD. Major symptoms of ASD are documented in cases of mercury poisoning, and biological abnormalities in ASD are very similar to the side effects of mercury poisoning itself (Bernard et al., 2001). These include psychiatric disturbances (like impairments in sociality, stereotypic behaviors, depression, anxiety disorder, and neuroses), increases in incidences of allergies and asthma, increases in the presence of IgG autoantibodies against brain and myelin basic proteins, reductions in natural killer cell function, and increases in neopterin levels which is indicative of immune activation (Bernard et al., 2001). However, nine research studies conducted under the supervision of Centres for Disease Control and Prevention reported that thimerosal was a preservative but had not toxic roles in vaccines (Institute of medicine, 2004).

2.4. Proteins

A theory called the ‘‘the Opioid-Excess Theory’’ (Whiteley et al., 1999) suggests that ASD is the consequence of incomplete breakdown and excessive absorption of peptides with opioid activity, derived from foods that contain casein and gluten. This causes disruption to biochemical and neuro-regulatory processes. Some researchers have shown interventions based on the Opioid-Excess Theory. Some of the research suggests that it may be possible to reduce ASD symptoms by providing a gluten and casein-free diet to individuals with ASD (Knivsberg et al., 1999). However, evidence also exists that disturbances of the gastrointestinal system are not more common in children with ASD than in the general population of children (Pastor & Reuben, 2008).

2.5. Environment

U.S. National Academy of Sciences (NAS) estimated that 3% of neurobehavioral disorders are caused directly by toxic environmental exposures and another 25% are caused by interactions between environmental factors (National Research Council, 2000). There are signs that show ASD is a neuropsychiatric disorder (Chakrabarti & Fombonne, 2001; Zwaigenbaum et al., 2002), and a theory of ASD suggests that it is not simply a characteristic of the individual but reflects a “disordered relationship between the person and the environment” (Loveland, 2001). Hence, changing the environment may also cause changes in behaviors.

2.6. Polyunsaturated fatty acid (PUFA)

A review by Van Elst et al. (2014) shows that n-6/n-3 PUFA ratio disturbances during early life can affect major processes in brain development and induce aberrant behavior. They reported PUFA changes might influence brain maturation and synaptic development. These changes in brain development processes and experimental PUFA ratio changes have clearly shown to induce changes in behavioral expression. They described that these behavioral changes are mainly observed in the domains of anxiety, locomotor activity, learning, and memory. The n-6/n-3 ratio, i.e., the introduction of vegetable oils and the removal of cholesterol, maybe an environmental factor in the increase of ASD related problems.

2.7. Mitochondrial dysfunction

There is increasing evidence of mitochondrial dysfunction in individuals with ASD, without the classic features associated with mitochondrial disease. It has also been determined that ASD can be caused by an underlying predisposition to mitochondrial dysfunction (Child Health Safety, 2010). Mitochondrial dysfunction could be caused by environmental toxins, and it could contribute to the altered energy metabolism in the brains of children with ASD (Chugani et al., 1999). Classical mitochondrial diseases occur in a subset of ASD cases, and they are usually caused by genetic or mitochondrial respiratory pathway abnormalities (Pons et al., 2004; Rossignol & Bradstreet, 2008). Some patients with autistic phenotypes clearly have a genetic-based primary mitochondrial disease (Haas, 2010). The lowered cellular energetics and deficient reserve mitochondrial energy capacity could lead to cognitive impairment and language deficits, both common in individuals with ASD.

2.8. Age of parents

There are well-documented effects of aging on human genetic traits, especially those that have their effects in early embryonic life (Strickberger, 1968). It is believed that the age of parents may be a causative factor of autism. A study of singleton children (n = 139,419) documented advanced maternal and paternal ages to be independently associated with the risk of ASD (Croen et al., 2007).

2.9. Genetics

Folstein and Rutter (1977) published the first twin study in ASD and showed that the concordance rate in identical twins was much higher than in non-identical twins. This twin study also suggests that genes may be at least in part, a cause for ASD. Genetic factors are now thought to account for 7–8% of ASD cases, but this fraction will likely increase as genetic research advances (Landrigan, 2010). ASD may be a family of diseases with common phenotypes linked to a series of genetic anomalies, each of which is responsible for no more than 2–3% of cases. This way, the total fraction of ASD that can be attributed to genetic inheritance may be about 30–40% (Landrigan et al., 2012). Other recent studies also show that ASD has a strong hereditary component (Buxbaum and Hof, 2011; Sakurai et al., 2011). Evidence from a study by Cusco et al. (2009) suggests multiple gene defects along with an environmental catalyst may be a cause of ASD. Rodier (2000) reported that there is indisputable evidence for a genetic component in ASD. Another twin study reported 60% concordance for classic ASD in monozygotic twins versus 0% in dizygotic twins; the higher monozygotic concordance attests to genetic inheritance as the predominant causative agent (Muhle et al., 2004). Data supports greater ASD concordance in monozygotic (MZ) vs. dizygotic (DZ) twins, higher functioning, psychiatric comorbidity, and Asperger syndrome concordance among affected MZ vs. DZ twins may also suggest differential heritability for different ASDs (Rosenberg et al., 2009).

3. Theories of ASD

There is ongoing research in order to identify causes of ASD, however, there is clear evidence that genes are at least partly responsible for causing ASD. There are some theories that have been put forth to understand the characteristics of ASD, and how the probable difference in gene expression could be explained.

3.1. Executive functioning theory

The theory of executive dysfunction stems from the belief that both people with ASD and people who have had frontal lobe injury, both have impaired executive functioning. Executive dysfunction can be seen to underlie many of the key characteristics of ASD, like rigidity and perseveration and initiation (Hill, 2004).

3.2. Theory of mind

Difficulty in understanding other minds is a core cognitive deficit of ASD conditions. Theory of mind is to be able to reflect on the contents of one's own and other's minds (Frith, 2005). This hypothesis proposes that a fault in just one of the many components of the social brain can lead to an inability to understand certain basic aspects of communication (Baron-Cohen et al., 2000). Lack of theory of mind could lead to not putting oneself in another person's shoes, not recognizing what another person feels, not being able to predict what another person will do based on what they know, think and feel. It explains poor pretend play, non-literal language understanding (jokes) and less trusting ability (Baron-Cohen, 2001).

3.3. Weak central coherence theory

This means that people with ASD look at the detail in things and their way of processing information gives more emphasis to tiny details than overall meaning. This theory explains why they can have superior rote memory, look at details, have narrow interests, store information differently, have perfect pitch and their insistence on sameness (Hape & Frith, 2006).

4. Characteristics of ASD

The severity of ASD can be defined by several different characteristics such as hyperirritability, self-injurious behaviors, cognitive level, or the presence of seizures or dysmorphic features. In studies defining the severity of ASD, the composite score on a rating instrument like the Autism Behavior Checklist (Krug et al., 1980) or the Childhood Autism Rating Scale (Schopler et al., 1980) have been used. Although incurable, studies have shown that the symptoms of ASD seem to reduce with increasing age. (Shattuck et al., 2007; Seltzer et al., 2003). A study by Duerden et al. (2012) attempted to explain this phenomenon. Children and adolescents, as compared to adults with autism have increased grey matter in regions of the brain responsible for social cognition and limbic processing regions which may underlie the emotional regulation that improves with age in this population.

4.1. Neurological aspects

Magnetic resonance imaging studies evidence that cerebellar anatomical maldevelopment in ASD is present before the end of the first year of life (Courchesne et al., 1987). Certain brain regions, including the limbic system, particularly the hippocampus, amygdala, and cerebellum, have been implicated in the clinical expression and pathophysiologic mechanism of ASD (Wakefield et al., 1998). Children with ASD have enlarged cerebral volumes in comparison with Typically Developing and Developmentally Delayed children and cerebral enlargement observed was independent of IQ (Sparks et al., 2002). The amygdala plays a crucial role in behavioral responses to emotional stimuli and in emotional learning (Piven et al., 1995). This shows that behavioral problems arise due to brain abnormalities. Hashimoto et al. (1995) studied the development of the brainstem and cerebellum in patients with ASD and found that although the brainstem and cerebellum significantly increased in size with age, these structures were significantly smaller in patients with ASD than in controls. Another study on brain development from five-year-old to adult brains of people with ASD showed that neurons are small, mini-columns are narrow and underdeveloped, efficacy is reduced for at least some neurotransmitter systems and connectivity is abnormal (Courchesne et al., 2004).

4.2. Comorbidities of ASD

Comorbidity is the study of the association between two or more conditions. The co-occurring conditions may or may not be causally related. A variety of coexisting psychiatric symptoms have been commonly reported in individuals with ASD, including depression, mania, hyperactivity, inattention, aggression, obsessive-compulsive disorder, Tourettes disorder, specific phobias, and generalized anxiety (Gadow et al., 2004; Ghaziuddin et al., 1998; Green et al., 2000; Lecavalier, 2006; Leyfer et al., 2006; Sverd, 2003). In fact, ASD commonly co-occurs with other developmental, psychiatric, neurologic, chromosomal, and genetic diagnoses (See Table 1). Many genetic and chromosomal disorders with a possible causative relationship to ASD have been identified.

Table 2-1: Common co-morbidities of Autism Spectrum Disorder

Medical comorbidity	References
Intellectual disability	Croen et al. (2017); Alexeeff et al. (2017)
Sleep problems	Croen et al. (2017); Alexeeff et al. (2017); Jones et al. (2016)
Seizure disorder	Croen et al. (2017); Alexeeff et al. (2017); Aldinger et al. (2015); Guinchat et al. (2015); Doshi-Velez et al. (2014); Hung (2016)
Gastrointestinal disorder	Croen et al. (2017); Alexeeff et al. (2017); Jones et al. (2016); Aldinger et al. (2015); Doshi-Velez et al. (2014)
Mitochondrial disorders	Rossignol and Frye (2012)
Hormone dysfunction	Bauman (2010); Baron-Cohen et al. (2015)
Motor disorders	Alexeeff et al. (2017); Guinchat et al. (2015)
Language delay	Alexeeff et al. (2017)
Tic disorder	Alexeeff et al. (2017); Gilberg et al. (2016)
Cerebral Palsy	Alexeeff et al. (2017)
Neoplasm	Alexeeff et al. (2017)
Obesity	Jones et al. (2016)
Allergy	Aldinger et al. (2015)
Asthma	Aldinger et al. (2015)
Psychiatric disorders	Guinchat et al. (2015); Doshi-Velez et al. (2014); Alexeeff et al. (2017)
Encaphalitis	Kern et al. (2016)
Sotos syndrome	Timonen-Soivio et al. (2016)
Neurofibromatosis	Timonen-Soivio et al. (2016)

5. Sensory processing in ASD

In their latest edition, the Diagnostic and Statistical Manual of Mental Disorders has added sensory processing problems as one of the diagnostic criteria of ASD. This was done in the light of abundant evidence that sensory processing impairments can be highly prevalent in autism but are not universal (Baranek et al., 2006; Liss et al., 2006).

Whether sensory symptoms are related to other social-communicative impairments in ASD or not, has been under study for a long time. It was hypothesized that people with ASD have problems modulating sensory inputs (Ornitz, 1985). We now know that the expression of these sensory abnormalities differs from individual to individual (Klintwall, 2007). It may be that sensory impairment is an additional primary impairment, but not an ASD-specific impairment (Rogers et al., 2003). Children with ASD show impairments in the processing of dynamic noise, motion coherence, and form-from-motion detection (Annaz et al., 2010). In a community study, Bromley et al. (2004) found that among 75 children with ASD, 71% were hypersensitive to sound; 52% to touch; 41% to smell, and 40% to taste. Kern et al. (2006) showed that in their study, persons with ASD had abnormal auditory, visual, touch, and oral sensory processing that was significantly different from controls. There was a significant interaction in low threshold auditory and low threshold visual.

Recently it has been considered that at least some of these high-level deficits could be explained in terms of lower-level sensorial and perceptual abnormalities (Behrmann et al., 2006). Leekam et al. (2007) suggested that sensory inputs cause behavior problems in individuals with ASD who are unable to describe their distress. They found that children with ASD were more likely to have sensory abnormalities, and across multiple sensory domains when compared to Typically Developing children. Tomcheck and Dunn (2007) showed a significant correlation between processing modalities for high and low thresholds of visual, oral and touch sensations, suggesting that sensory disturbance correlates with the severity of ASD. Then, Marco et al. (2011) also suggested that differences in sensory processing may cause core features of ASD such as language delay (auditory processing) and difficulty with reading emotion from faces (visual processing).

5.1. Sensory Integration in ASD

Sensory integration is the ability to take in information through the senses of touch, movement, smell, taste, vision, and hearing, and to combine the resulting perceptions with prior information, memories, and knowledge already stored in the brain, in order to derive coherent meaning from processing the stimuli. The sensory integration problems seen in ASD are of several types. Hyperresponsiveness is an exaggerated behavioral response to sensory stimuli e.g., covering ears to sounds or avoidance of touch (Baranek et al., 2006). Also known a sensory over-responsivity (SOR), it is a common and impairing feature found in more than half of children with ASD, and SOR has been linked to anxiety in children with ASD (Pfeiffer et al., 2005). Anxiety disorders are common in children with ASD and can increase the functional impairment of these children (White et al., 2009). Children with SOR often react negatively to noisy or visually complex environments, are bothered by tags or seams on their clothing, or may dislike being touched unexpectedly (Liss et al., 2006). These reactions are possibly due to pain felt by them under these situations. According to Leekam et al. (2007), anxiety contributes to SOR as generalized hyperarousal and hypervigilance focus attention on a specific type of sensory stimulus. The threat-based emotion regulation associated with anxiety makes it more difficult for children to regulate their emotional and physiological reactions to stimuli. The researchers also address the possibility that a risk factor such as amygdala abnormalities may contribute independently to each condition because amygdala plays a role in fear and anxiety, and may also be related to SOR through overestimation of the threat value of a sensory stimulus which triggers an enhanced response to that stimulus.

Hypo responsiveness refers to lack of response, or insufficient intensity of response to sensory stimuli e.g., diminished response to pain (Baranek et al., 2006). A sensory craving may be a result if hypo responsiveness, seen in people with ASD. Grandin (1996), an animal scientist who has been diagnosed with ASD, talked about the beneficial effects of holding in some children are due to desensitization to touch of the autistic child's nervous system. She described a squeeze machine she constructed to satisfy her craving for the feeling of being held. The machine was designed so that she could control the amount and duration of the pressure. It was lined with foam rubber and applied pressure over a large area of the body. Using the machine enabled her to tolerate another person's touching. She thinks it is important to desensitize a child with ASD so that he/she can tolerate a comforting touch. Several squeeze machines are now in use at sensory integration clinics in the United States.

These conditions can be present in people with ASD in several forms. A person with ASD can be under-responsive to one stimulus and over-responsive to another. Hence, it is difficult to draw a line between these conditions. A study of parental reports by Dickie et al. (2009) is another good example of this. The parents described negative experiences in sound, visual, touch, and movement. Some children with ASD can have strong reactions to bright light and sunlight. Various sounds were examples of stimuli that provoked unpleasant sensory experiences, particularly sounds that were too loud. These are characteristics of over-responsivity. Most of the positive touch accounts were interpersonal touch cuddling or snuggling with a parent, having a back rub, being tickled, or engaging in rough play with an adult. This could mean that even though the children longed for social contact, they were not very comfortable with touch due to problems in sensory registration. Reports of pleasure related to movement usually involved speed, spinning, bouncing, and/or change of body position. Some children with ASD were described as loving to chew on things. Reports focused on behaviors such as hand flapping, having to chew on things, and not responding to extreme cold. Parents described some of these activities as self-stimulating and linked them to their child's diagnosis. This demonstrates sensory craving as the children were trying to stimulate themselves, which may be due to sensory under responsivity.

There have been a few theories to identify these sensory processing problems. Among somatosensory sub-modalities (primarily touch, temperature, and pain) that may contribute to tactile hypersensitivity in ASD, a class of unmyelinated tactile mechanoreceptors has been identified in humans. These receptors, known as CT-afferents, are unmyelinated C fibers that respond to light (low force), slowly moving, stroking stimuli (Vallbo et al., 1999; Olausson et al., 2002). In humans, these low-threshold unmyelinated afferents are distributed primarily in the hairy skin and the face, but not in the hairless, glabrous skin of the palm that is highly innervated with myelinated tactile afferents, known to be important for sensory discrimination (Kakuda, 1992). It is hypothesized that, with their distribution in hairy skin and their response preference for pleasant, stroking touch, this class of unmyelinated afferents constitute an affiliative, social touch system (Olausson et al., 2002; Valbo et al., 1999; Wessberg et al., 2003). Such a system is a prime candidate for the tactile hypersensitivity associated with ASD.

Psychophysical tactile studies have looked at thresholds and sensitivity using vibrotactile stimuli. They found that adults with ASD showed hypersensitivity in the Pacinian corpuscles receptor pathway, which is responsible for the vibrotactile stimulus (Blakemore et al., 2006). Tactile hypersensitivity was also shown to thermal stimuli in adults with ASD (Cascio et al., 2008). In contrast, in a small sample of children with ASD, there were no tactile perceptual threshold differences for vibrotactile detection (Guclu et al., 2007). These contrasting results show that even though tactile sensitivity is a symptom of ASD, it is not universal and can have varying degrees.

5.2. Interventions to improve sensory symptoms

King (1989) has reported that wrapping a child with ASD in a gym mat produces a calming effect. A case report involved the sensor motor effect of deep pressure and tactile input on Bob, a thirteen-year-old nonverbal boy with ASD with severe mental retardation and self-injurious behavior, including pinching, biting, and rubbing of his head, neck, trunk, and upper and lower extremities. During the observation periods when the patient received deep pressure and tactile input from the woven elastic bandage wraps on his extremities, he exhibited less self-stimulatory behavior, no self-injurious behavior, and, in general, an increase in the number of interactions with others (McClure & Holtz-Yotz, 1991). The children who received deep pressure in a study by Edelson et al. (1999) demonstrated a significant decrease on the Tension scale and a marginally significant decrease on the more general Anxiety scale and the benefits were those who had the highest initial levels of physiological arousal.

In another study, some people with ASD have found that sessions in ‘sensory rooms’, areas that provide soft cushions for sitting or lying on, pleasant displays of colored lights, soft sounds or music clips have a calming effect and are much enjoyed (Leekam et al., 2007). Another intervention method, Student Intervention Team, a clinic-based, child-centered intervention originally developed by Ayres (1972), provides play-based activities with enhanced sensation to elicit and reinforce the child adaptive responses. It focuses on the therapist–child relationship and uses play-based activities that challenge the children while enhancing self-regulation, for example, promoting optimal arousal, and increasing appropriate behaviors maybe the primary change-producing elements.

6. Feeding issues in ASD

Bandini et al. (2010) defined food selectivity to comprise three separate domains: food refusal; limited food repertoire; and high-frequency single food intake. They found that children with ASD showed more food refusal and exhibited a more limited food repertoire than did typically developing children. They also showed that dietary “pickiness” is not outgrown with age. Approximately 25% of all children experience eating problems during the early years of life, but this number may rise to as high as 80% in children with developmental difficulties (Manikam & Perman, 2000; Jacobi, 2003). One of the reasons may be that these children have specific developmental delays that may also affect eating. Difficulties with socialization may have an impact on the pleasure of eating in the company of others. This problem may also make learning by imitation and accepting nutritionally balanced meals more difficult. Similarly, having limited interests may restrict intake to known and familiar foods (Nadon et al., 2011). Children with ASD often exhibit a strong emotional response when presented with non-preferred food, including crying, disruption, and aggression during meals (Sharp et al., 2013).

Kerwin et al. (2005) stated that although more than 60% of parents interviewed reported that their children with ASD had strong food preferences, only 6.7% of them described their children as not having an appetite. Cermak et al. (2010) also showed feeding problems in children with ASD. Klein and Nowak (1999) found that children with ASD were reluctant to try new foods. An association between feeding problems and sensory defensiveness has been shown in otherwise typically developing children by Smith et al. (2005). The problems describe children with ASD as being “picky” eaters, eat few vegetables, rarely eat the same meal as the rest of the family, do not want different foods to touch each other, have aversions to certain tastes and textures, refuse some foods because of their smell, and do not like extremes of temperature. Ayres (1964) described sensory defensiveness in the tactile domain as an over-reaction to certain experiences of touch, resulting in an observable aversion or negative behavioral response to certain tactile stimuli. This tactile sensitivity could also be related to aversion from certain food textures. Dietary patterns of children with ASD often involve strong preferences for starches and snack foods coinciding with a bias against fruits and vegetables (Cornish, 2002).

There have been many studies on the feeding problems of children with ASD. According to parental reports for food refusal, children with ASD were significantly more likely to refuse foods based on texture/consistency, taste/smell, mixtures, brand, shape, appearance, taste, smell, and temperature, as well as reluctance to try new foods and a small repertoire of accepted foods (Hubbard et al., 2014; Williams et al., 2005). Similar results were found by other researchers, where children with ASD exhibited more general feeding problems including refusing foods, requiring specific presentations of foods and specific utensils, eating only low texture foods, and eating a narrow variety of foods than children without ASD. 70% of children with ASD selected what they ate according to texture compared to 11% of children without ASD. Although the children with ASD ate significantly fewer foods from each of the food groups than did children without ASD, this finding did not extend to their families (Schreck et al., 2004, Whiteley et al., 2000; Schmitt et al., 2008; Bandini et al., 2010). Klein and Nowak (1999) found that more than half (53%) of their subjects with ASD were unwilling to try new foods.

A study on mothers who nursed their children with ASD was done by Provost et al. (2010), where 47% reported difficulties with breast-feeding. Only 12% of the children with ASD ate a variety of food and only 21% had no difficulty eating in any setting. Almost one half preferred certain food temperatures. 33% of the children with ASD showed a preference for food colors and 25% had preferences for food packaging. Most children did not prefer eating in loud, crowded and unfamiliar places. Mealtime behaviors reported were leaving the table frequently and resisting sitting at the table, throwing/dumping the food, and frequent tantrums. 50% of the children with ASD required food prepared in a special way, became upset if a mealtime routine was broken. 33% of the children with ASD were reported to stuff their mouths and cheeks, and 25% had problems with gagging. 54% of the children with ASD were reported to mouth non-food items and 6 (25%) to swallow these items. Parents of children with ASD are more likely to report mealtime behavioral problems, spousal stress at meals, and that their child's food preferences influenced what other family members ate (Curtin et al., 2015). These factors cause difficulty in building family relationships further and decrease the quality of life.

6.1. Behavior questionnaires to identify feeding problems among children with ASD

A functional analysis involves the systematic manipulation of antecedent and consequent variables to determine their influence on problem behavior (Skinner, 1953). Functional analyses can be useful in identifying reasons why problematic feeding behavior may be maintained and in determining function-based treatments to reduce the feeding problems. In order to answer some of these questions, certain assessment tools were created. Archer et al. (1991) developed the

Children's Eating Behavior Inventory (CEBI) to address the contribution of children, parents, and family factors to eating and mealtime problems for children from a broad age span who had a variety of developmental and medical conditions. The questionnaire included food preferences, motor skills, and behavioral compliance of the children. Matson and Kuhn (2001) developed the Screening Tool of Eating Problems to assess the presence of feeding problems in adults with ASD, including aspiration risk (vomiting and rumination), selectivity (selectivity by food type, texture, temperature, feeder, and the meal setting), feeding skills (swallowing ability, the ability to chew, the ability to feed independently, and the need for adaptive feeding equipment), food refusal (mealtime refusal or termination and behaviors such as spitting out food, self-injury during meals, and aggression associated with mealtime), and nutrition-related problems (over- and under-eating as well as pica and food stealing). Hendy et al. (2009) developed the Parent Mealtime Action Scale (PMAS) to identify both child and parent mealtime behavior and the frequency that the parents eat and serve certain foods.

6.2. Causes of feeding issues in ASD

There have been several speculations about the causes of feeding issues. Maenner et al. (2012) reported that when there are no identifiable organic factors (abnormal sensory processing, oral motor disorders, or gastrointestinal problems), food selectivity can be the manifestation of the restricted interests and the behavioral rigidity characteristic of ASD. They demonstrated that certain behaviors associated with ASD, such as food refusal, were significantly associated with gastrointestinal disorders. Problems related to motor anticipation in children with ASDs have also been reported. Nausea, vomiting, and/or choking could be secondary to sensory disorders and, food refusal could be an adaptive behavioral response (Overland, 2011; Brisson et al., 2012).

In a study by Nadon et al. (2011), they found that some children took medications (Ritalin, Concerta, Adderall, Keppra, and Strattera) that may suppress food intake. These studies suggest that certain sensory modalities may influence the number of feeding problems more than others, like tactile sensitivity. Cermak et al. (2010) explained oral over-responsiveness (defensiveness) may result in difficulty with food textures and therefore food selectivity. Oral seeking behavior may result in the child putting everything in his/her mouth for oral stimulation.

6.3. Alternative diets for ASD

Cornish (1998) reported inadequate nutrient intakes in children with ASD based on a three-day dietary recall and a food frequency checklist. Inadequate intakes of iron, vitamin D, vitamin C, niacin, riboflavin, and zinc were found in one or more children. The majority of children did not consume adequate amounts of fruit and vegetables, but 94% of the children ate foods daily that the authors considered to be in the “fatty” and “sugary” food groups. There is evidence that poor dietary diversity in ASD may increase the risk of nutritional and/or related medical issues, including vitamin and mineral deficiencies (Bandini et al. 2010; Zimmer et al. 2012), poor bone growth (Hediger et al., 2008), visual loss and optic atrophy (Pineles et al., 2010).

In order to compensate for the feeding problems, and to supplement nutrition, over the years many parents have adapted alternative diets that may or may not have an evidence base. A survey of 552 parents of children with ASD found that alternative diets had been implemented with 9.9% of children with Aspergers syndrome, 29.4% of children with mild ASD, and 32.2%

of children with severe ASD (Green et al., 2006). Several studies have proven that alternative diets may not be an effective way to reduce feeding issues. Rather, these diets cause harm to the health of individuals. Arnold et al. (2003) collected plasma amino acid profiles of children with ASD and found them more likely to have nutritional deficiencies and lower plasma levels in essential amino acids. Children on the Gluten-Free Casein-Free diet were significantly more at risk than the rest of the children with ASD in this study. This difference may be due to the lack of proteins specifically found in gluten and casein and/or the increase in food refusal, which may follow the implementation of a GFCF diet. In the review by Mulloy et al. (2010), they concluded that published studies do not support the use of GFCF diets in the treatment of ASD. There were also increased rates of constipation among children with ASD (Ibrahim et al., 2009) and diet-related diseases (e.g. obesity and cardiovascular disease) into adolescence and adulthood (Ho et al., 1997). It is important to investigate the factors associated with food selectivity and sensory sensitivity in children with ASD as it affects the nutritional adequacy directly. Spelling out causes of food selectivity can be a step towards solving the issue.

7. Autism in adulthood

The past few decades have seen some insightful and much-needed research in the field of autism. General awareness is increasing, and society has made many changes to accommodate the needs of autism. Due to this, children are diagnosed with autism early in their life and accordingly, interventions can be used to help reduce symptoms. However, there is a major section of this population that did not get a diagnosis early on in life in order to make use of early intervention and are aging to be adults. Some research has revealed contradicting results that sensory sensitivities increased over age for people with autism (Talay-Ongan & Wood, 2000).

As more children with ASD are becoming adults, there is an increasing need for research on services for the growing population of adults with an ASD (Howlin, 2008). Especially because health concerns for people with autism is on a rise and many get inadequate care. A study done in Scotland with over 6500 adults with ASD revealed that adults with autism spectrum disorders have substantially poorer general health than other adults, across the entire adult life course (Rydzewska, 2019). Utilization and costs of health care services are significantly higher among adults with ASD than among adults with ADHD or adults with neither condition, even after controlling for medical and psychiatric comorbidities (Zerbo et al., 2019). Only about thirty-six percent of medical professionals in a survey reported receiving some training about caring for adults with ASD. This is a surprisingly low number considering the special needs of people with autism (Bruder et al., 2012). It is imperative to conduct research in the genetic field to understand ASD better, nevertheless, researchers have suggested emphasizing on research addressing quality of life for adults with autism, to improve living standards in this community (Gerhardt & Lainer, 2011).

REFERENCES

Aldinger, K. A., Lane, C. J., Veenstra-VanderWeele, J., & Levitt, P. (2015). Patterns of risk for multiple co-occurring medical conditions replicate across distinct cohorts of children with autism spectrum disorder. Autism Research,8, 771-781.

Alexeeff, S. E., Yau, V., Qian, Y., Davignon, M., Lynch, F., Crawford, P., & Croen, L. A. (2017). Medical conditions in the first years of life associated with future diagnosis of asd in children. Journal of Autism and Developmental Disorders, 47, 2067-2079.

Annaz, D., Remington, A., Milne, E., Coleman, M., Campbell, R., Thomas, M. S., & Swettenham, J. (2010). Development of motion processing in children with autism. Developmental Science, 13, 826-838.

Archer, L. A., Rosenbaum, P. L., & Streiner, D. L. (1991). The children's eating behavior inventory: reliability and validity results. Journal of Pediatric Psychology, 16, 629-642.

Arnold, G. L., Hyman, S. L., Mooney, R. A., & Kirby, R. S. (2003). Plasma amino acids profiles in children with autism: potential risk of nutritional deficiencies. Journal of Autism and Developmental Disorders, 33, 449-454.

Ayres, A. J. (1964). Tactile functions. their relation to hyperactive and perceptual motor behavior. The American journal of occupational therapy: official publication of the American Occupational Therapy Association, 18, 6-11.

Ayres, A. J. (1972). Sensory integration and learning disorders. Western Psychological Services.

Bailey, A., Le Couteur, A., Gottesman, I., Bolton, P., Simonoff, E., Yuzda, E., & Rutter, M. (1995). Autism as a strongly genetic disorder: evidence from a British twin study. Psychological Medicine, 25, 63-77.

Bandini, L. G., Anderson, S. E., Curtin, C., Cermak, S., Evans, E. W., Scampini, R., & Must, A. (2010). Food selectivity in children with autism spectrum disorders and typically developing children. The Journal of Pediatrics, 157, 259-264.

Baranek, G. T., David, F. J., Poe, M. D., Stone, W. L., & Watson, L. R. (2006). Sensory experiences questionnaire: discriminating sensory features in young children with autism, developmental delays, and typical development. Journal of Child Psychology and Psychiatry, 47, 591-601.

Baron-Cohen, S. (2001). Theory of mind in normal development and autism. Prisme, 34(1), 74-183.

Baron-Cohen, S., Auyeung, B., Nørgaard-Pedersen, B., Hougaard, D. M., Abdallah, M. W., Melgaard, L., & Lombardo, M. V. (2015). Elevated fetal steroidogenic activity in autism. Molecular Psychiatry, 20, 369.

Baron-Cohen, S., Ring, H. A., Bullmore, E. T., Wheelwright, S., Ashwin, C., & Williams, S. C. R. (2000). The amygdala theory of autism. *Neuroscience & Biobehavioral Reviews*, *24*(3), 355-364.

Bauman, M. L. (2010). Medical comorbidities in autism: challenges to diagnosis and treatment. Neurotherapeutics: The Journal of the American Society for Experimental NeuroTherapeutics, 7, 320–327.

Behrmann, M., Thomas, C., & Humphreys, K. (2006). Seeing it differently: visual processing in autism. *Trends in cognitive sciences*, *10*(6), 258-264.

Bernard, S., Enayati, A., Redwood, L., Roger, H., & Binstock, T. (2001). Autism: a novel form of mercury poisoning. Medical Hypotheses, 56, 462-471.

Bettelheim, B. (1950). Love is not enough; the treatment of emotionally disturbed children.

Blakemore, S. J., Tavassoli, T., Calò, S., Thomas, R. M., Catmur, C., Frith, U., & Haggard, P. (2006). Tactile sensitivity in asperger syndrome. Brain and Cognition, 61, 5-13.

Bromley, J., Hare, D. J., Davison, K., & Emerson, E. (2004). Mothers supporting children with autistic spectrum disorders: Social support, mental health status and satisfaction with services. *Autism*, *8*(4), 409-423.

Brisson, J., Warreyn, P., Serres, J., Foussier, S., & Adrien-Louis, J. (2012). Motor anticipation failure in infants with autism: a retrospective analysis of feeding situations. Autism, 16, 420-429.

Bruder, M. B., Kerins, G., Mazzarella, C., Sims, J., & Stein, N. (2012). Brief report: the medical care of adults with autism spectrum disorders: identifying the needs. *Journal of Autism and Developmental Disorders*, *42*(11), 2498-2504.

Buxbaum, J. D., & Hof, P. R. (2011). The emerging neuroscience of autism spectrum disorders.

Cascio, C., McGlone, F., Folger, S., Tannan, V., Baranek, G., Pelphrey, K. A., & Essick, G. (2008). Tactile perception in adults with autism: a multidimensional psychophysical study. Journal of Autism and Developmental Disorders, 38, 127-137.

Cermak, S. A., Curtin, C., & Bandini, L. G. (2010). Food selectivity and sensory sensitivity in children with autism spectrum disorders. Journal of the Academy of Nutrition and Dietetics, 110, 238-246.

Chakrabarti, S., & Fombonne, E. (2001). Pervasive developmental disorders in preschool children. Journal of American Medical Association, 285, 3093-3099.

Child Health Safety. Vaccination causes Autism. 2010. https://childhealthsafety.wordpress.com/2010/06/30/vaccination-causes-autism-

%E2%80%93-say-us-government-merck%E2%80%99s-director-of%C2%A0vaccines Accessed on March 15, 2018.

Christensen, D. L., Bilder, D. A., Zahorodny, W., Pettygrove, S., Durkin, M. S., Fitzgerald, R. T., & Yeargin-Allsopp, M. (2016). Prevalence and characteristics of autism spectrum disorder among 4-year-old children in the autism and developmental disabilities monitoring network. Journal of Developmental & Behavioral Pediatrics, 37, 1-8.

Chugani, D. C., Muzik, O., Behen, M., Rothermel, R., Janisse, J. J., Lee, J., & Chugani, H. T. (1999). Developmental changes in brain serotonin synthesis capacity in autistic and nonautistic children. Annals of Neurology, 45, 287-295.

Cornish, E. (1998). A balanced approach towards healthy eating in autism. Journal of Human Nutrition and Dietetics, 11, 501-509.

Cornish, E. (2002). Gluten and casein free diets in autism: a study of the effects on food choice and nutrition. Journal of Human Nutrition and Dietetics, 15, 261-269.

Courchesne, E., Hesselink, J. R., Jernigan, T. L., & Yeung-Courchesne, R. (1987). Abnormal neuroanatomy in a nonretarded person with autism: Unusual findings with magnetic resonance imaging.

Courchesne, E., Redcay, E., & Kennedy, D. P. (2004). The autistic brain: birth through adulthood. Current Opinion in Neurology, 17, 489-496.

Croen, L. A., Najjar, D. V., Fireman, B., & Grether, J. K. (2007). Maternal and paternal age and risk of autism spectrum disorders. Archives of Pediatrics & Adolescent Medicine, 161, 334-340.

Croen, L. A., Shankute, N., Davignon, M., Massolo, M. L., & Yoshida, C. (2017). Demographic and Clinical Characteristics Associated with Engagement in Behavioral Health Treatment Among Children with Autism Spectrum Disorders. Journal of Autism and Developmental Disorders, 47, 3347-3357.

Curtin, C., Hubbard, K., Anderson, S. E., Mick, E., Must, A., & Bandini, L. G. (2015). Food selectivity, mealtime behavior problems, spousal stress, and family food choices in children with and without autism spectrum disorder. Journal of Autism and Developmental Disorders, 45, 3308-3315.

Cusco, I., Medrano, A., Gener, B., Vilardell, M., Gallastegui, F., Villa, O., & Perez-Jurado, L. A. (2009). Autism-specific copy number variants further implicate the phosphatidylinositol signaling pathway and the glutamatergic synapse in the etiology of the disorder. Human Molecular Genetics, 18, 1795-1804.

DeStefano, F. (2007). Vaccines and autism: evidence does not support a causal association. Clinical Pharmacology & Therapeutics, 82, 756-759.

DeStefano, F., Price, C. S., & Weintraub, E. S. (2013). Increasing exposure to antibody-stimulating proteins and polysaccharides in vaccines is not associated with risk of autism. *The Journal of Pediatrics*, *163*(2), 561-567.

Dickie, V. A., Baranek, G. T., Schultz, B., Watson, L. R., & McComish, C. S. (2009). Parent reports of sensory experiences of preschool children with and without autism: A qualitative study. American Journal of Occupational Therapy, 63, 172-181.

Doshi-Velez, F., Ge, Y., & Kohane, I. (2014). Comorbidity clusters in autism spectrum disorders: an electronic health record time-series analysis. Pediatrics, 133, e54-e63.

Duerden, E. G., Mak-Fan, K. M., Taylor, M. J., & Roberts, S. W. (2012). Regional differences in grey and white matter in children and adults with autism spectrum disorders: an activation likelihood estimate (ALE) meta-analysis. *Autism Research*, *5*(1), 49-66.

Edelson, S. M., Edelson, M. G., Kerr, D. C., & Grandin, T. (1999). Behavioral and physiological effects of deep pressure on children with autism: A pilot study evaluating the efficacy of Grandin's Hug Machine. American Journal of Occupational Therapy, 53, 145-152.

Folstein, S., & Rutter, M. (1977). Infantile autism: a genetic study of 21 twin pairs. Journal of Child psychology and Psychiatry, 18, 297-321.

Frith, C., & Frith, U. (2005). Theory of mind. *Current biology*, *15*(17), R644-R645.

Gadow, K. D., DeVincent, C. J., Pomeroy, J., & Azizian, A. (2004). Psychiatric symptoms in preschool children with PDD and clinic and comparison samples. Journal of Autism and Developmental Disorders, 34, 379-393.

Gerhardt, P. F., & Lainer, I. (2011). Addressing the needs of adolescents and adults with autism: A crisis on the horizon. *Journal of Contemporary Psychotherapy*, *41*(1), 37-45.

Gernsbacher, M. A., Dawson, M., & Hill Goldsmith, H. (2005). Three reasons not to believe in an autism epidemic. *Current directions in psychological science*, *14*(2), 55-58.

Ghaziuddin, M., Weidmer-Mikhail, E., & Ghaziuddin, N. (1998). Co morbidity of Asperger syndrome: a preliminary report. Journal of Intellectual Disability Research, 42, 279-283.

Gillberg, I. C., Helles, A., Billstedt, E., & Gillberg, C. (2016). Boys with Asperger syndrome grow up: psychiatric and neurodevelopmental disorders 20 years after initial diagnosis. Journal of Autism and Developmental Disorders, 46, 74-82.

Grandin, T. (1996). My experiences with visual thinking sensory problems and communication difficulties. Center for the Study of Autism.

Green, J., Gilchrist, A., Burton, D., & Cox, A. (2000). Social and psychiatric functioning in adolescents with Asperger syndrome compared with conduct disorder. Journal of Autism and Developmental Disorders, 30, 279-293.

Green, V. A., Pituch, K. A., Itchon, J., Choi, A., O'Reilly, M., & Sigafoos, J. (2006). Internet survey of treatments used by parents of children with autism. Research in Developmental Disabilities, 27, 70-84.

Güçlü, B., Tanidir, C., Mukaddes, N. M., & Ünal, F. (2007). Tactile sensitivity of normal and autistic children. Somatosensory & Motor Research, 24, 21-33.

Guinchat, V., Cravero, C., Diaz, L., Périsse, D., Xavier, J., Amiet, C., & Consoli, A. (2015). Acute behavioral crises in psychiatric in patients with autism spectrum disorder (ASD): recognition of concomitant medical or non-ASD psychiatric conditions predicts enhanced improvement. Research in Developmental Disabilities, 38, 242-255.

Haas, R. H. (2010). Autism and mitochondrial disease. Developmental Disabilities Research Reviews, 16, 144-153.

Happé, F., & Frith, U. (2006). The weak coherence account: detail-focused cognitive style in autism spectrum disorders. Journal of Autism and Developmental Disorders, 36(1), 5-25.

Hashimoto, T., Tayama, M., Murakawa, K., Yoshimoto, T., Miyazaki, M., Harada, M., & Kuroda, Y. (1995). Development of the brainstem and cerebellum in autistic patients. Journal of Autism and Developmental Disorders, 25, 1-18.

Hediger, M. L., England, L. J., Molloy, C. A., Kai, F. Y., Manning-Courtney, P., & Mills, J. L. (2008). Reduced bone cortical thickness in boys with autism or autism spectrum disorder. Journal of Autism and Developmental Disorders, 38, 848-856.

Hendy, H. M., Williams, K. E., Camise, T. S., Eckman, N., & Hedemann, A. (2009). The Parent Mealtime Action Scale (PMAS). Development and association with children's diet and weight. Appetite, 52, 328-339.

Hill, E. L. (2004). Evaluating the theory of executive dysfunction in autism. Developmental Review, 24, 189-233.

Ho, H. H., Eaves, L. C., & Peabody, D. (1997). Nutrient intake and obesity in children with autism. Focus on Autism and Other Developmental Disabilities, 12, 187-192.

Howlin, P. (2008). Redressing the balance in autism research. *Nature Clinical Practice Neurology, volume* 4, page 407.

Hubbard, K. L., Anderson, S. E., Curtin, C., Must, A., & Bandini, L. G. (2014). A comparison of food refusal related to characteristics of food in children with autism spectrum disorder and typically developing children. Journal of the Academy of Nutrition and Dietetics, 114, 1981-1987.

Hung, K. L. (2016). Epilepsy Comorbidity of Autism in Children. Epilepsy J, 2, e011.

Ibrahim, S. H., Voigt, R. G., Katusic, S. K., Weaver, A. L., & Barbaresi, W. J. (2009). Incidence of gastrointestinal symptoms in children with autism: a population-based study. Pediatrics, 124, 680-686.

Institute of Medicine (US) Immunization Safety Review Committee. (2004). Immunization safety review: vaccines and autism. In *Immunization Safety Review: Vaccines and Autism*. National Academies Press (US).

Institute of Medicine. (2004). https://www.cdc.gov/vaccinesafety/pdf/cdcstudiesonvaccinesandautism.pdf\ Accessed on May 29 2019.

Jacobi, C., Agras, W. S., Bryson, S., & Hammer, L. D. (2003). Behavioral validation, precursors, and concomitants of picky eating in childhood. Journal of the American Academy of Child & Adolescent Psychiatry, 42, 76-84.

Johnson, C. P., & Myers, S. M. (2007). Identification and evaluation of children with autism spectrum disorders. Pediatrics, 120, 1183-1215.

Jones, K. B., Cottle, K., Bakian, A., Farley, M., Bilder, D., Coon, H., & McMahon, W. M. (2016). A description of medical conditions in adults with autism spectrum disorder: A follow-up of the 1980s Utah/UCLA Autism Epidemiologic Study. Autism, 20, 551-561.

Kakuda, N. (1992). Conduction velocity of low-threshold mechanoreceptive afferent fibers in the glabrous and hairy skin of human hands measured with microneurography and spike-triggered averaging. Neuroscience Research, 15, 179-188.

Kanner, L. (1943). Autistic disturbances of affective contact. Nervous child, 2(3), 217-250.

Kanner, L. (1949). Problems of nosology and psychodynamics of early infantile autism. *American journal of Orthopsychiatry*, *19*(3), 416.

Kern, J. K., Geier, D. A., Sykes, L. K., & Geier, M. R. (2016). Relevance of neuroinflammation and encephalitis in autism. Frontiers in Cellular Neuroscience, 9, 519.

Kern, J. K., Trivedi, M. H., Garver, C. R., Grannemann, B. D., Andrews, A. A., Savla, J. S., & Schroeder, J. L. (2006). The pattern of sensory processing abnormalities in autism. Autism, 10, 480-494.

Kerwin, M. E., Eicher, P. S., & Gelsinger, J. (2005). Parental report of eating problems and gastrointestinal symptoms in children with pervasive developmental disorders. Children's Health Care, 34, 217-234.

King, L. (1989). Facilitating neurodevelopment. Proceedings of the Autism Society of America, 117-120.

Klein, U., & Nowak, A. J. (1999). Characteristics of patients with autistic disorder (AD) presenting for dental treatment: a survey and chart review. Special Care in Dentistry, 19, 200-207.

Klintwall, L., Holm, A., Eriksson, M., Carlsson, L. H., Olsson, M. B., Hedvall, Å., ... & Fernell, E. (2011). Sensory abnormalities in autism: a brief report. *Research in developmental disabilities*, *32*(2), 795-800.

Knivsberg, A. M., Reichelt, K. L., & Nødland, M. (1999). Dietary intervention for a seven year old girl with autistic behaviour. Nutritional Neuroscience, 2, 435-439.

Krug, D. A., Arick, J., & Almond, P. (1980). Behavior checklist for identifying severely handicapped individuals with high levels of autistic behavior. Journal of Child Psychology and Psychiatry, 21, 221-229.

Landrigan, P. J. (2010). What causes autism? Exploring the environmental contribution. Current Opinion in Pediatrics, 22, 219-225.

Landrigan, P. J., Lambertini, L., & Birnbaum, L. S. (2012). A research strategy to discover the environmental causes of autism and neurodevelopmental disabilities. Environmental Health Perspectives, 120, a258.

Lane, A. E., Young, R. L., Baker, A. E., & Angley, M. T. (2010). Sensory processing subtypes in autism: Association with adaptive behavior. Journal of Autism and Developmental Disorders, 40, 112-122.

Lecavalier, L. (2006). Behavioral and emotional problems in young people with pervasive developmental disorders: Relative prevalence, effects of subject characteristics, and empirical classification. Journal of Autism and Developmental disorders, 36, 1101-1114.

Leekam, S. R., Nieto, C., Libby, S. J., Wing, L., & Gould, J. (2007). Describing the sensory abnormalities of children and adults with autism. Journal of Autism and Developmental Disorders, 37, 894-910.

Lewine, J. D., Andrews, R., Chez, M., Patil, A. A., Devinsky, O., Smith, M., & Chong, B. (1999). Magnetoencephalographic patterns of epileptiform activity in children with regressive autism spectrum disorders. Pediatrics, 104, 405-418.

Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., & Lainhart, J. E. (2006). Comorbid psychiatric disorders in children with autism: interview development and rates of disorders. Journal of autism and developmental disorders, 36, 849-861.

Liss, M., Saulnier, C., Fein, D., & Kinsbourne, M. (2006). Sensory and attention abnormalities in autistic spectrum disorders. Autism, 10, 155-172.

Loveland, K. A. (2001). Toward an ecological theory of autism. In Burack, J. A., Charman, T., Yirmiya, N., & Zelazo, P. R. (Eds.). (2001). The development of autism: Perspectives from theory and research. Routledge.

Maenner, M. J., Arneson, C. L., Levy, S. E., Kirby, R. S., Nicholas, J. S., & Durkin, M. S. (2012). Brief report: Association between behavioral features and gastrointestinal problems among children with autism spectrum disorder. Journal of Autism and Developmental Disorders, 42, 1520-1525.

Manikam, R. (2000). Current Lliterature: Pediatric Feeding Disorders. Nutrition in Clinical Practice, 15, 312-314.

Marco, E. J., Hinkley, L. B., Hill, S. S., & Nagarajan, S. S. (2011). Sensory processing in autism: a review of neurophysiologic findings. Pediatric Research, 69, 48R-54R.

Matson, J. L., & Kuhn, D. E. (2001). Identifying feeding problems in mentally retarded persons: Development and reliability of the screening tool of feeding problems (STEP). Research in Developmental Disabilities, 22, 165-172.

McClure, M. K., & Holtz-Yotz, M. (1991). The effects of sensory stimulatory treatment on an autistic child. American Journal of Occupational Therapy.

Muhle, R., Trentacoste, S. V., & Rapin, I. (2004). The genetics of autism. Pediatrics, 113, e472-e486.

Mulloy, A., Lang, R., O'Reilly, M., Sigafoos, J., Lancioni, G., & Rispoli, M. (2010). Gluten-free and casein-free diets in the treatment of autism spectrum disorders: a systematic review. Research in Autism Spectrum Disorders, 4, 328-339.

Nadon, G., Feldman, D. E., Dunn, W., & Gisel, E. (2011). Association of sensory processing and eating problems in children with autism spectrum disorders. Autism Research and Treatment, 2011.

Nadon, G., Feldman, D. E., Dunn, W., & Gisel, E. (2011). Mealtime problems in children with autism spectrum disorder and their typically developing siblings: A comparison study. Autism, 15, 98-113.

National Research Council. (2000). Scientific frontiers in developmental toxicology and risk assessment. National Academies Press.

Olausson, H., Lamarre, Y., Backlund, H., Morin, C., Wallin, B. G., Starck, G., & Bushnell, M. C. (2002). Unmyelinated tactile afferents signal touch and project to insular cortex. Nature neuroscience, 5, 900.

Ornitz, E. M. (1985). Neurophysiology of infantile autism. Journal of the American Academy of Child Psychiatry, 24, 251-262.

Ornitz, E. M., Guthrie, D., & Farley, A. H. (1977). The early development of autistic children. Journal of Autism and Childhood Schizophrenia, 7, 207-229.

Osterling, J. A., Dawson, G., & Munson, J. A. (2002). Early recognition of 1-year-old infants with autism spectrum disorder versus mental retardation. Development and Psychopathology, 14, 239-251.

Overland, L. (2011). A sensory-motor approach to feeding. SIG 13 Perspectives on Swallowing and Swallowing Disorders (Dysphagia), 20, 60-64.

Pastor, P. N., & Reuben, C. A. (2008). Diagnosed attention deficit hyperactivity disorder and learning disability: United States, 2004-2006. Vital and Health Statistics. Series 10, Data from the National Health Survey, 237, 1-14.

Pfeiffer, B., Kinnealey, M., Reed, C., & Herzberg, G. (2005). Sensory modulation and affective disorders in children and adolescents with Asperger's disorder. American Journal of Occupational Therapy, 59, 335-345.

Pineles, S. L., Avery, R. A., & Liu, G. T. (2010). Vitamin B12 optic neuropathy in autism. *Pediatrics*, *126*(4), e967-e970.

Piven, J., Arndt, S., Bailey, J., & Havercamp, S. (1995). An MRI study of brain size in autism. The American Journal of Psychiatry, 152, 1145.

Pons, R., Andreu, A. L., Checcarelli, N., Vilà, M. R., Engelstad, K., Sue, C. M., & DiMauro, S. (2004). Mitochondrial DNA abnormalities and autistic spectrum disorders. The Journal of Pediatrics, 144, 81-85.

Project Autism. History of Autism. 2018. http://projectautism.org/history-of-autism\ Accessed January 23, 2018.

Provost, B., Crowe, T. K., Osbourn, P. L., McClain, C., & Skipper, B. J. (2010). Mealtime behaviors of preschool children: Comparison of children with autism spectrum disorder and children with typical development. *Physical & occupational therapy in pediatrics*, *30*(3), 220-233.

Rodier, P. M. (2000). The early origins of autism. Scientific American, 282(2), 56-63.

Rogers, S. J., Hepburn, S., & Wehner, E. (2003). Parent reports of sensory symptoms in toddlers with autism and those with other developmental disorders. Journal of Autism and Developmental Disorders, 33, 631-642.

Rosenberg, R. E., Law, J. K., Yenokyan, G., McGready, J., Kaufmann, W. E., & Law, P. A. (2009). Characteristics and concordance of autism spectrum disorders among 277 twin pairs. Archives of Pediatrics & Adolescent Medicine, 163, 907-914.

Rossignol, D. A., & Frye, R. E. (2012). Mitochondrial dysfunction in autism spectrum disorders: a systematic review and meta-analysis. Molecular Psychiatry, 17, 290–314.

Rossignol, D. A., and Bradstreet, J. J. (2008). Evidence of mitochondrial dysfunction in autism and implications for treatment. American Jourbal of Biochemistry and Biotechnology. 4, 208–217.

Rydzewska, E., Hughes-McCormack, L. A., Gillberg, C., Henderson, A., MacIntyre, C., Rintoul, J., & Cooper, S. A. (2019). General health of adults with autism spectrum disorders–A whole country population cross-sectional study. *Research in Autism Spectrum Disorders*, *60*, 59-66.

Sakurai, T., Cai, G., Grice, D. E., & Buxbaum, J. D. (2011). Genomic architecture of autism spectrum disorders. Textbook of Autism Spectrum Disorders (Hollander E, Kolevzon A, Coyle JT. eds). Washington, DC: American Psychiatric Publishing, 281-298.

Schmitt, L., Heiss, C. J., & Campbell, E. E. (2008). A comparison of nutrient intake and eating behaviors of boys with and without autism. Topics in Clinical Nutrition, 23, 23-31.

Schopler, E., Reichler, R. J., DeVellis, R. F., & Daly, K. (1980). Toward objective classification of childhood autism: Childhood Autism Rating Scale (CARS). Journal of Autism and Developmental Disorders, 10, 91-103.

Schreck, K. A., & Williams, K. (2006). Food preferences and factors influencing food selectivity for children with autism spectrum disorders. Research in Developmental Disabilities, 27, 353-363.

Schreck, K. A., Williams, K., & Smith, A. F. (2004). A comparison of eating behaviors between children with and without autism. Journal of Autism and Developmental Disorders, 34, 433-438.

Seltzer, M. M., Krauss, M. W., Shattuck, P. T., Orsmond, G., Swe, A., & Lord, C. (2003). The symptoms of autism spectrum disorders in adolescence and adulthood. Journal of Autism and Developmental Disorders, 33, 565-581.

Sharp, W. G., Berry, R. C., McCracken, C., Nuhu, N. N., Marvel, E., Saulnier, C. A., & Jaquess, D. L. (2013). Feeding problems and nutrient intake in children with autism spectrum disorders: a meta-analysis and comprehensive review of the literature. Journal of Autism and Developmental Disorders, 43, 2159-2173.

Shattuck, P. T., Seltzer, M. M., Greenberg, J. S., Orsmond, G. I., Bolt, D., Kring, S., & Lord, C. (2007). Change in autism symptoms and maladaptive behaviors in adolescents and adults with an autism spectrum disorder. Journal of Autism and Developmental Disorders, 37, 1735-1747.

Siegel, B. R. Y. N. A., Pliner, C. A. R. O. L. B. A., Eschler, J. A. M. I., & Elliott, G. R. (1988). How children with autism are diagnosed: difficulties in identification of children with multiple developmental delays. Journal of Developmental and Behavioral Pediatrics: JDBP, 9, 199-204.

Skinner, B. F. (1953). Science and human behavior. Simon and Schuster. 35-38

Smith, A. M., Roux, S., Naidoo, N. R., & Venter, D. J. (2005). Food choices of tactile defensive children. Nutrition, 21, 14-19.

Sparks, B. F., Friedman, S. D., Shaw, D. W., Aylward, E. H., Echelard, D., Artru, A. A., & Dager, S. R. (2002). Brain structural abnormalities in young children with autism spectrum disorder. Neurology, 59, 184-192.

Strickberger, M. W. (Ed.) 1968. Genetics. New York, NY: The Macmillan Company.

Sverd, J. (2003). Psychiatric disorders in individuals with pervasive developmental disorder. Journal of Psychiatric Practice®, 9, 111-127.

Talay-Ongan, A., & Wood, K. (2000). Unusual sensory sensitivities in autism: A possible crossroads. International Journal of Disability, Development and Education, 47(2), 201-212.

Timonen-Soivio, L., Vanhala, R., Malm, H., Hinkka-Yli-Salomäki, S., Gissler, M., Brown, A., & Sourander, A. (2016). Brief report: Syndromes in autistic children in a Finnish birth cohort. Journal of Autism and Developmental Disorders, 46, 2780-2784.

Tomchek, S. D., & Dunn, W. (2007). Sensory processing in children with and without autism: a comparative study using the short sensory profile. American Journal of Occupational herapy, 61, 190-200.

Vallbo, Å. B., Olausson, H., & Wessberg, J. (1999). Unmyelinated afferents constitute a second system coding tactile stimuli of the human hairy skin. Journal of Neurophysiology, 81, 2753-2763.

van Elst, K., Bruining, H., Birtoli, B., Terreaux, C., Buitelaar, J. K., & Kas, M. J. (2014). Food for thought: dietary changes in essential fatty acid ratios and the increase in autism spectrum disorders. Neuroscience & Biobehavioral Reviews, 45, 369-378.

Wakefield, A. J., Murch, S. H., Anthony, A., Linnell, J., Casson, D. M., Malik, M., & Valentine, A. (1998). RETRACTED: Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children.

Wessberg, J., Olausson, H., Fernström, K. W., & Vallbo, Å. B. (2003). Receptive field properties of unmyelinated tactile afferents in the human skin. Journal of Neurophysiology, 89, 1567-1575.

White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. Clinical psychology review, 29, 216-229.

Whiteley, P., Rodgers, J., & Shattock, P. (2000). Feeding patterns in autism. Autism, 4, 207-211.

Whiteley, P., Rodgers, J., Savery, D., & Shattock, P. (1999). A gluten-free diet as an intervention for autism and associated spectrum disorders: preliminary findings. Autism, 3, 45-65.

Williams, K. E., Gibbons, B. G., & Schreck, K. A. (2005). Comparing selective eaters with and without developmental disabilities. Journal of Developmental and Physical Disabilities, 17, 299-309.

Zerbo, O., Qian, Y., Ray, T., Sidney, S., Rich, S., Massolo, M., & Croen, L. A. (2019). Health care service utilization and cost among adults with autism spectrum disorders in a US integrated health care system. *Autism in Adulthood*, *1*(1), 27-36.

Zimmer, M. H., Hart, L. C., Manning-Courtney, P., Murray, D. S., Bing, N. M., & Summer, S. (2012). Food variety as a predictor of nutritional status among children with autism. Journal of Autism and Developmental Disorders, 42, 549-556.

Zuckerman, K. E., Sinche, B., Cobian, M., Cervantes, M., Mejia, A., Becker, T., & Nicolaidis, C. (2014). Conceptualization of autism in the Latino community and its relationship with early diagnosis. Journal of Developmental and Behavioral Pediatrics: JDBP, 35, 522.

Zwaigenbaum, L., Szatmari, P., Jones, M. B., Bryson, S. E., Maclean, J. E., Mahoney, W. J., & Tuff, L. (2002). Pregnancy and birth complications in autism and liability to the broader autism phenotype. Journal of the American Academy of Child & Adolescent Psychiatry, 41, 572-579.

CHAPTER 3

PERCEPTION OF, AND RESPONSES TO FOOD AND BEVERAGE PRODUCTS AMONG ADULTS WITH AUTISM SPECTRUM DISORDER

1. Introduction

Research over the years has allowed for a better understanding of the characteristics of autism spectrum disorder (ASD). It is a popular notion that symptoms of ASD present themselves differently among different individuals and no two autistic-people can be expected to be the same. However, we do find some similarities among people with autism, in terms of sensory processing. It may thus be beneficial to study sensory processing in autism. Moreover, it is now being considered that some behavioral and feeding issues could be explained in terms of lower-level sensory and perceptual abnormalities (Behrmann et al., 2006; Leekam et al., 2007).

Children/adolescents with ASD show lower phonetic-iconic congruency response patterns than neurotypical controls, pointing to poorer multisensory integration capabilities (Occelli et al., 2013). There is an inability to engage selective attention to ignore non-salient irrelevant distractor stimuli in ASD. Socially meaningful auditory stimuli are noticed by neurotypical and people with ASD similarly across visual perceptual loads, however, people with ASD have a greater detection rate of the non-socially meaningful auditory stimuli under the high load (Tyndall et al., 2018). Awareness about these sensory issues is increasing among the public and there have been studies regarding perception in each sense. For example, Khalfa et al. (2004) found an increased perception of loudness, indicating hyperacusis in subjects with autism. Talay-Ongan and Wood (2000) reported that the most frequently reported incidents were hearing planes, trains, alarms, or television before others could hear them.

Studies have found that participants with ASD have impaired performances of odor identification and have a higher odor detection threshold (Galle et al., 2013; Luisier et al., 2015; Suzuki et al., 2003). Similarly, few studies like Bennetto et al. (2007) and Tavassoli and Baron-Cohen (2012) studied the taste perception in ASD and found that adults with ASD were less accurate in identifying tastes overall (irrespective of medication or age). In addition, people with autism can have a range of visual abnormalities, for example, disliking dark/bright lights, sharp flashes of light or liking reflections and brightly colored objects (Bogdashina, 2003). The focus of attention and enhancement of perception are sharper in people with ASD than in matched controls (Robertson et al., 2013). Adults with autism show increased sensitivity to vibration and thermal stimulation (Cascio et al., 2008). They tend to show diminished responses to pleasant and neutral tactile stimuli and exaggerated responses to unpleasant stimuli (Cascio et al., 2012).

These sensory abnormalities can influence eating behavior. For example, Williams et al. (2000) indicated that the parents who complained of food selectivity said that it was determined by texture, appearance, taste, smell, temperature, as well as reluctance to try new foods and a small repertoire of accepted foods. In another study, (Nadon et al., 2011) nearly fifteen percent of the participating children with ASD but none of the siblings had oral motor difficulties with chewing, moving their tongue or swallowing. However, it should be noted that most of these studies that have looked at multisensory integration through interviews have been done with parents who reported sensory abnormalities for their children, based on their own perception of it. This can pose a bias because these accounts are not firsthand. There are a few studies (Robertson & Simmons, 2013; Tavassoli et al., 2014; Crane et al., 2009) that asked adults in either a self-reported questionnaire form or a focus group discussion, about their sensory behavior. These studies have found that adults with ASD experience sensory over-responsivity, in at least one sensory function. They have suggested that individuals with ASD could experience very different, yet similarly severe, sensory processing abnormalities.

Some research has shown that sensory sensitivities increased over age for people with autism (Talay-Ongan & Wood, 2000). In our efforts to increase research among adults with ASD addressing quality of life to improve living standards in this community, we propose this one on one interview study with adults with ASD. This study aims to determine the perception and sensory experiences of people with ASD and to understand how it could affect eating behavior.

2. Materials and Method

This study was approved by the Institutional Review Board of the University of Arkansas and written consent was obtained from each participant.

2.1 Design of study

The objective of this study was to understand the perception of adults with autism spectrum disorder, toward food and beverages. This study utilized the structured form of interviews, in which questions were always asked in the same order. The complete interview was divided into three sections: sensory experiences, eating behavior and demographics. Within the sensory experiences section, subgroups of questions were formed which included sight, smell, taste, hearing, and touch.

2.2 Participants

For this study, twenty-three adult participants (fourteen males) were recruited from the Northwest Arkansas community, based on an existing diagnosis of autism spectrum disorder. The mean age of the participants was Mean ± Std Dev = 26 ± 8.5 years. The mean age of diagnosis of ASD for the participants was 12.3 ± 11.6 years. For two participants with reduced verbal abilities, the caregiver answered questions during interviews.

2.3 Data collection and analysis

The interviews were held in a quiet room. Interviews were audio-recorded and transcribed verbatim by the researcher. The questions that were asked in the sensory experience category for each of the five senses were for example:

1. Do you think that you are more sensitive in smell function than other people?
2. How important are odors, aromas, and flavors of food and beverages for your eating and drinking?
3. Are there any smells that you find extremely intense?
4. Are there any specific smells of food or beverage items that you like?
5. Are there any specific smells of food or beverage items that you don’t like?
6. Have you encountered any trouble in smelling food or beverage items?
7. When you consume food or beverage items, do you also consider smells from other sources such as persons or environments?

The eating behavior questions included preference to eat alone or with others, eating out frequency and preference to new foods or restaurants. The demographics questions were asked to identify any factors that could be related to their eating behavior.

A summative data analysis was used to understand the information gathered. It starts with identifying and quantifying certain words to understand the contextual use of the words. This quantification is an attempt not to infer meaning but, rather, to explore usage (Hsieh & Shannon, 2005).

3. Results

Six participants received their diagnosis by two years of age. All but one participant lived with their parent/s, with the number of family members being Mean ± Std Dev = 3.2 ± 1.3. Nine participants reported that they did not help with cooking meals at home. Two participants mentioned concerns of safety during cooking as being one of the main reasons. Eleven people said they did not eat at the cafeteria in school, rather brought their own lunch every day. Thirteen participants reported not receiving any kind of therapy to help with their symptoms of autism. Six participants received Applied Behavior Analysis as behavior therapy, three participants received speech therapy and one participant received music therapy. One participant mentioned studying in a special needs classroom.

When asked if they preferred eating alone or with others, eleven participants said they liked eating with others, reasoning that they liked socializing (4), talking with others (5) or listening to others (1), and avoid being alone (1). Seven participants said they prefer to eat alone because they dislike loud surroundings (2), they thought people judge them for the way they eat (2), like personal space (1), dislike socializing (1) and force of habit (1). The other five participants said that they liked doing both. Fourteen participants said that they liked going out to eat regularly, one mentioned only going during late or "quiet hours". Eight said they did so sometimes. One participant mentioned not liking to go out to eat at all. On average, participants in this study went out to eat three times in a month. Seven participants said they dislike trying new foods or going out to new restaurants. One participant liked trying different food, but in the same restaurant, and one said they like trying different restaurants, but not different foods. One participant mentioned making others try the food before eating to avoid surprises. Six participants mentioned having trouble in sensory function during their lifetime, including poor sight, over-responsivity to touch, hearing what other people cannot hear, being overly sensitive, having skin rashes and inability to taste when young.

We asked the participants about which the most important sense for them in their daily life is, ten reported sight, six hearing, five touch, four taste, and four smell. People rated sight because being able to see was most important to them, whereas people who reported touch reasoned liking to feel things. The most sensitive sense, compared to other people, was hearing for fifteen participants because loud noises bothered them, they could hear the slightest voices and get distracted by them. Seven people said that they did not think they were less sensitive than other people in any sensory function *("If someone pats me gently on my back it hurts me.")*. One participant mentioned: *"My hearing is most sensitive; I can sometimes hear something that others do not hear."*

3.1 Hearing

Twenty participants said they are more sensitive in their hearing function as compared to other participants, while three others think they are average or less sensitive. Eleven participants mentioned problems with loud places, and it hurts their ears. One participant used noise-canceling headphones most of the time to avoid pain. Four participants said they can hear things that others cannot which made it difficult to concentrate or sleep. Fourteen participants reported that sounds elicited by foods are important for their eating experience. Particularly, ten of them mentioned disliking chewing sounds of others, while seventeen mentioned they disliked people talking while eating.

"*I do not like chomping and loud chewing sounds, it is very annoying to me and I get goosebumps*."

"I *dislike background chatter. Eating is relaxing time, so I don't like distractions. I enjoy music sometimes*."

In terms of sounds elicited by food, most intense was crunchy for five participants, carbonation for two and gummy or sticky for two. The crunchy sound was pleasing to fourteen participants whereas the sounds of food with soft texture was disliked by six.

"*Some (sounds) can be annoying, some are fun. I dislike crunching of broccoli. I like crunch of toasted bread. I get annoyed by people chewing loud.*"

3.2 Sight

Fourteen adults said they are sensitive in their sight function as compared to other participants, while nine others think they are average or less sensitive. Nine participants mentioned that brightness bothers them. Three participants mentioned that they see details in things, that others often cannot see.

"I can be more sensitive than other people in sight, sometimes when it is very sunny it hurts my eyes. It is hard to drive without sunglasses."

"I can catch things that others do not see; I have to lower the brightness on my phone."

Eighteen participants thought that the appearance of food is important to them, while five others thought it was not an important part of their food.

"I am very particular about colors. I like things to match in color. The food presentation is very important, I don't like if my food touches other food on my plate"

"I like eating colorful foods"; "I like my plate to look pretty and be colorful"

In terms of food, most intense colors for ten participants were red/orange, while blue and green were most intense for six. The most liked color was red (8), while brown and black were most disliked (4). Food shaped round, and triangle were liked by four and three participants respectively. The visual texture of food was mentioned by two participants:

"I dislike the mashed potato and gravy visual texture"

"I dislike food that has big raisins in it. I dislike nuts in food like pecans."

When asked about the light conditions in the room where they eat, both liking and disliking of bright lights seemed to be evenly distributed, with the former being preferred by eight participants and later by seven. Four participants mentioned being particular with the utensils and having specific utensils that they liked.

"I am very picky about my utensils; I like clean plates without prints on them. I like square plates with a dark color. I like smoother-looking things. I like bright rooms, I do not like the dark."

3.3 Smell

Eleven participants said they are more sensitive in smell function as compared to other participants, while twelve others think they are average or less sensitive. Twenty participants thought that the smell of food was important in their perception. Four people said that if they smell something they do not like, they experience a gag reflex.

"It (being more sensitive to smells) bothers me because I smell things other people do not. I can smell even if someone is sick, body odor is especially bad."

Five participants mentioned that the smell of sour foods was very intense for them and 4 others thought the smell of spicy food was intense. The smell of sweet food was liked by four participants, whereas the smell of pizza and lasagna was favored by six. The smell of eggs and fish was disliked by three and four participants respectively. Fifteen participants mentioned that the smell of heavy perfume or other smells in the environment during eating bothers them and can give several of these participants a headache.

"smoking bothers me, smell of grass bothers me, some perfumes really aggravate me, it has gotten worse with time, now I prefer unscented products."

3.4 Taste

Eleven participants said they are more sensitive in taste function as compared to other participants, while twelve others think they are average or less sensitive. Twenty-one participants said that the taste of the food is very important to them in their food choices.

"somethings I taste are stronger than what other people think."

In terms of most intense tastes, bitter was selected by twelve participants while sour by six. Seven participants said the umami taste was their most liked and sweet was most liked for thirteen. Eight participants disliked the sour taste the most while six disliked the bitter taste.

3.5 Touch

Seventeen participants said they are more sensitive in tactile function as compared to other participants, while six others think they are average or less sensitive. Several participants mentioned they like to touch and feel things and that certain textures can be calming. Two participants were reported to dislike big chunks of food and taking little bites. Eighteen participants said that the texture of food was important to them in their food preferences.

"I like touching things and feeling them always. It does not bother me. I am sensitive in my mouth texture, like chips, it hurts me."

"I am more sensitive in hand-feel, I think I am on max overload all the time, I used to have issues sleeping because of how things feel, the bedsheet, I do not like microfiber in sheets."

Among the food textures that were mentioned as intense were hard, chewy, hot (spicy) and fizzy. The rubbery texture of meat and gummies was liked by seven participants, while the crunch of chips and cookies was liked by four. Six participants reported disliking the grainy texture of food, three disliked the mouthfeel of sour foods and two disliked mushy textures. Some participants also mentioned being particular about the temperature of food.

"I do not like hot food, liver texture bothers me, it is stringy and dry like white meat. I prefer dark meat."

"Very hard and solid foods, that require effort to chew, like gum. Heat and really cold is very intense."

4. Discussion

This study attempted to understand the sensory difficulties of people with ASD through one on one interviews. To our knowledge, it is a very extensive study done with adults, addressing all domains of sensory sensitivities. We found that the average age when the group received their ASD diagnosis was twelve years, which is later than what most early intervention programs suggest. Moreover, thirteen participants did not receive any therapy, which may be linked to their late diagnosis. About half the participants recalled bringing their own lunch to school. This finding is consistent with that of (Nadon et al., 2011), who found that more children with ASD than their siblings did not eat at daycare, school, in restaurants, with the extended family or with friends. Our participants also mentioned that they did not like going out to a restaurant, with the participants who did like going out, preferred to go to the same places or eat the same food. This could also be related to their responses that some of them prefer to eat alone because they dislike loud surroundings, think people judge them for the way they eat and like having personal space.

Hearing was the most sensitive sense for twenty people, both loud and softer voices can bother or distract them. The sounds elicited by food was also important to them, including being bothered by loud chewing sounds. These results are complementary to a previous study (Robertson and Simmons, 2015) where participants noted that loud sounds can sometimes cause pain, and noises with low intensity could also cause discomfort. We found that "Crunchy" sound was pleasing to fourteen participants, which was also found by Shea (2015) and Knox et al. (2012), even though those studies were done with only 1 participant each.

Some participants mentioned that brightness bothers them. For light conditions in dining rooms, both liking and dislike of bright lights seemed to be evenly distributed. This discrepancy was also noted by Bogdashina (2003), who said that some people who may be hyposensitive to light, liked looking at lights and colors, while others who were hypersensitive disliked it. Participants in our study reported that the appearance of food and serving utensils is important to them. These results have been previously seen in numerous studies for food preferences, where children refused foods based on appearance, visual texture, brands, etc. (Williams et al., 2000; Schreck & William, 2006).

About half the participants said that they were more sensitive in taste and smell function while the other half said that they were less sensitive. Acuity in odor and taste identification has been found to be reduced in people with ASD (Galle et al., 2013; Bennetto et al., 2007). Interestingly, sour smell quality was found to be more intense and sour taste was most disliked. This is in line with the study by Schreck and Williams, 2006 that found that children with ASD may be sensitized to sour and bitter tastes. In terms of most intense tastes, bitter was selected by twelve participants while sour by six. Seven participants said the umami taste was their most liked and sweet was most liked by another thirteen. Eight participants disliked the sour taste most while six disliked the bitter taste. Participants mentioned the smell of perfume or other smells in the environment during eating bothers them. Pellicano (2013) also found that their participants were hypersensitive to strong smells of perfume, cigars, damp wool caps or gloves.

Seventeen participants said they are more sensitive in tactile function. Eighteen participants said that the texture of food was important to them in their food preferences. One of our participants mentioned light patting being hurtful. Similar findings were mentioned in a paper by Robertson and Simmons (2015) where the participants said that people lightly brushing past and hugging could cause physical pain. Most people in their study mentioned textures of food being uncomfortable, including anything that “bursts” and the mixture of textures was also an issue. Among our participants, the food textures that were mentioned as intense were hard, chewy, hot (spicy) and fizzy. They liked the rubbery texture of meat and gummies, the crunch of chips and cookies, whereas disliked grainy texture, sour foods, and mushy textures. Two participants were reported to dislike big chunks of food and taking little bites. Kerwin et al. (1995) advanced a similar argument, suggesting that larger bite-sizes increase the response effort (acceptance; swallowing) and consequently the ‘cost’ of accepting (bite size). Cascio et al. (2012) noted that people with ASD show diminished responses to pleasant and neutral stimuli, and exaggerated limbic responses to unpleasant stimuli, which diminish social reward associated with touch.

5. Conclusion

To summarize, we found that people with ASD reported abnormal and non-uniform sensory experiences. Both hypersensitivity and hyposensitivity were reported by participants in each sensory function. Participants reported heightened responses to loud places, sour and bitter tastes, sour food smells, brightness, and touch. Participants reported food choices, driven by taste, smell, texture, appearance, and sound. We also found that environmental factors influence eating behavior in persons with ASD, including the brightness of a room, types of utensils, odors of other people or the room, background noise and food evoked sounds. In conclusion, we found that increased sensitivity to sensory stimuli, combined with unfavorable environmental factors lead to a reduces eating experience for people with autism. Further research is required to generate interventions to help with the difficult eating environment, to improve the overall quality of life for people with ASD.

REFERENCES

Behrmann, M., Thomas, C., & Humphreys, K. (2006). Seeing it differently: visual processing in autism. *Trends in cognitive sciences*, *10*(6), 258-264.

Bennetto, L., Kuschner, E. S., & Hyman, S. L. (2007). Olfaction and taste processing in autism. *Biological psychiatry*, *62*(9), 1015-1021.

Bogdashina, O. (2003). Sensory Perceptual Issues in Autism and Asperger Syndrome. Different Sensory Experiences. *Different Perceptual Worlds, Jessica Kingsley, London, UK*.

Cascio, C., McGlone, F., Folger, S., Tannan, V., Baranek, G., Pelphrey, K. A., & Essick, G. (2008). Tactile perception in adults with autism: a multidimensional psychophysical study. *Journal of autism and developmental disorders*, *38*(1), 127-137.

Cascio, C. J., Moana-Filho, E. J., Guest, S., Nebel, M. B., Weisner, J., Baranek, G. T., & Essick, G. K. (2012). Perceptual and neural response to affective tactile texture stimulation in adults with autism spectrum disorders. *Autism Research*, *5*(4), 231-244.

Crane, L., Goddard, L., & Pring, L. (2009). Sensory processing in adults with autism spectrum disorders. *Autism*, *13*(3), 215-228.

Galle, S. A., Courchesne, V., Mottron, L., & Frasnelli, J. (2013). Olfaction in the autism spectrum. *Perception*, *42*(3), 341-355.

Hsieh, H. F., & Shannon, S. E. (2005). Three approaches to qualitative content analysis. *Qualitative health research*, *15*(9), 1277-1288.

Kerwin, M. E., Ahearn, W. H., Eicher, P. S., & Burd, D. M. (1995). The costs of eating: A behavioral economic analysis of food refusal. Journal of Applied Behavior Analysis, 28(3), 245–260.

Khalfa, S., Bruneau, N., Rogé, B., Georgieff, N., Veuillet, E., Adrien, J. L., Catherine, B. & Collet, L. (2004). Increased perception of loudness in autism. *Hearing research*, *198*(1-2), 87-92.

Knox, M., Rue, H. C., Wildenger, L., Lamb, K., & Luiselli, J. K. (2012). Intervention for food selectivity in a specialized school setting: Teacher implemented prompting, reinforcement, and demand fading for an adolescent student with autism. *Education and Treatment of Children*, *35*(3), 407-418.

Leekam, S. R., Nieto, C., Libby, S. J., Wing, L., & Gould, J. (2007). Describing the sensory abnormalities of children and adults with autism. Journal of Autism and Developmental Disorders, 37, 894-910.

Luisier, A. C., Petitpierre, G., Ferdenzi, C., Clerc Bérod, A., Giboreau, A., Rouby, C., & Bensafi, M. (2015). Odor perception in children with autism spectrum disorder and its relationship to food neophobia. *Frontiers in psychology*, *6*, 1830.

Nadon, G., Feldman, D. E., Dunn, W., & Gisel, E. (2011). Mealtime problems in children with autism spectrum disorder and their typically developing siblings: A comparison study. *Autism*, *15*(1), 98-113.

Occelli, V., Esposito, G., Venuti, P., Arduino, G. M., & Zampini, M. (2013). The Takete—Maluma Phenomenon in Autism Spectrum Disorders. Perception, 42, 233-241.

Pellicano, E. (2013). Sensory symptoms in autism: A blooming, buzzing confusion?. *Child Development Perspectives*, *7*(3), 143-148.

Robertson, A. E., & Simmons, D. R. (2015). The sensory experiences of adults with autism spectrum disorder: A qualitative analysis. *Perception*, *44*(5), 569-586.

Robertson, C. E., Kravitz, D. J., Freyberg, J., Baron-Cohen, S., & Baker, C. I. (2013). Tunnel vision: sharper gradient of spatial attention in autism. *Journal of Neuroscience*, *33*(16), 6776-6781.

Schreck, K. A., & Williams, K. (2006). Food preferences and factors influencing food selectivity for children with autism spectrum disorders. *Research in developmental disabilities*, *27*(4), 353-363.

Shea, E. (2015). Understanding and Managing Eating Issues on the Autism Spectrum'. *Network Autism*, *3*.

Suzuki, Y., Critchley, H. D., Rowe, A., Howlin, P., & Murphy, D. G. (2003). Impaired olfactory identification in Asperger's syndrome. *The Journal of neuropsychiatry and clinical neurosciences*, *15*(1), 105-107.

Talay-Ongan, A., & Wood, K. (2000). Unusual sensory sensitivities in autism: A possible crossroads. *International Journal of Disability, Development and Education*, *47*(2), 201-212.

Tavassoli, T., & Baron-Cohen, S. (2012). Taste identification in adults with autism spectrum conditions. *Journal of autism and developmental disorders*, *42*(7), 1419-1424.

Tavassoli, T., Miller, L. J., Schoen, S. A., Nielsen, D. M., & Baron-Cohen, S. (2014). Sensory over-responsivity in adults with autism spectrum conditions. *Autism*, *18*(4), 428-432.

Tyndall, I., Ragless, L., & O'Hora, D. (2018). Effects of perceptual load and socially meaningful stimuli on crossmodal selective attention in Autism Spectrum Disorder and neurotypical samples. Consciousness and Cognition, 60, 25-36.

Williams, P. G., Dalrymple, N., & Neal, J. (2000). Eating habits of children with autism. *Pediatric nursing*, *26*(3), 259.

CHAPTER 4

INFLUENCES OF AUTISM SPECTRUM DISORDER ON SENSORY RESPONSES TO OLFACTORY CUES

1. Introduction

Olfaction serves the function of identifying noxious substances that are to be avoided, helps us to enjoy the hedonic pleasure of food and plays a role in our social life. There is an association between personality traits and attitudes toward the sense of smell. A study showed that people who were able to lie more often used olfactory cues in social communication (Seo et al., 2013). Olfaction is different from other senses in the way that it does not have a thalamus relay, which means there's a shorter pathway for olfactory stimuli. (Smythies, 1997). Perhaps the functions that it serves is the reason for this anomaly. The smell function is closely related to autobiographical memory and this memory, when triggered by olfactory information is older than verbal and visual cues (Willander & Larsson, 2006). An impairment in autobiographical memory exists among people with autism (Crane and Goddard, 2008). A study by Crane et al. (2009) demonstrated that both people with and without ASD could distinguish between self-defining and everyday memories, however, the people with ASD could recall fewer specific memories overall. They were not able to cite meaning from their narratives as well as the other group, which suggests a failure in using past experiences to update the self among people with ASD.

The olfactory functions have been understudied in people with ASD (Luisier et al., 2015) and the findings of olfactory functions, specifically, odor identification performance, have been conflicting. For example, in multiple studies, it has been found that participants with ASD had impaired performances of odor identification. The study by Galle et al. (2013) used control and

Asperger participants, and compared to them, participants in the ASD group performed worse. Identification of pleasant odors was better than for the unpleasant odors in typically developing children, but not children with ASD (Luisier et al., 2015). Suzuki et al. (2003) also confirmed that adults with Asperger syndrome showed impaired olfactory identification. However, Dudova et al. (2011) reported that children with ASD showed almost normal performance of odor identification as compared to the control group; odor identification ability correlated significantly with age in the control group, but not in the ASD group.

In terms of odor detection threshold, Suzuki et al. (2003) found that adults with ASD performed normally relative to typically developed individuals. Interestingly, a study reported that people with ASD detect odors at a mean distance larger than the control group, who detected them at a significantly shorter mean distance (Ashwin et al., 2014). In contrast, Dudova et al. (2011) reported that people with Asperger's syndrome and high functioning ASD, in comparison with healthy controls, were significantly impaired in odor detection threshold. These contrasting results might be caused by inconsistency between verbal reports by people with ASD, specific diagnosis of people used (e.g., Asperger's, PDD-NOS, low functioning and high functioning autism) or methodology being used.

There is a difference in the behavioral and implicit measures of olfactory processing in children with ASD who are less likely to match their facial expressions to the verbal expression of their affective states (Luisier et al., 2015). It is worth noting that in a study with only ASD children, Dudova and Hrdlicka (2013) found no significant correlation between autism severity and odor detection, odor pleasantness ratings or odor identification ability. Among children with ASD, results found that the less they discriminated hedonically (especially for pleasant odors), the more neophobic they were. This is consistent with another finding that an advantage in odor identification ability for non-neophobic people exists over more-neophobic participants (Dematte et al., 2013). These results suggest that one of the main causes of greater food selectivity in children with ASD may lie in their sensory functioning (Matson & Fodstad, 2009). Woo and Leon (2013) exposed three12-year-old ASD children to either daily olfactory/tactile stimulation along with sensory and cognitive exercises (enrichment group), or to standard care (control group) and observed that the severity of autistic traits was significantly lower in the enrichment group than in controls. They suggested that improvement in olfactory ability could help in reducing symptoms of ASD, for which, understanding of this mechanism is necessary. A study with parental reports (Lane et al., 2014) found that tactile sensitivity was not associated with picky eating and problem eating behaviors, but the taste and smell sensitivity was.

This study aims to determine whether ASD influences olfactory performances (odor discrimination and odor identification tasks), hedonic ratings, intensity, arousal, pleasantness, familiarity, and edibility of everyday odors. We also aim to investigate the differences in usage and importance of odors in everyday lives between people with ASD and without ASD.

2. Materials and Methods

This study was approved by the Institutional Review Board of the University of Arkansas and written consent was obtained from each participant. Participants were compensated with $30 in the form of gift cards, upon successful completion of the study.

2.1 Participants

Participants were recruited from the Northwest Arkansas community. A survey was sent out to the prospective participants which included questions about health, allergies, and demographics. Participants who qualified were over eighteen years of age, had no food allergies, had no clinical history of major diseases and did not smoke. Twenty participants (twelve males; mean age ± standard deviation = 29.5 ±12.5) were recruited for the test group, using University of Arkansas Sensory Service Center consumer database, based on a diagnosis of Autism Spectrum Disorder, which was confirmed using the Autism Spectrum Quotient (AQ) developed by Baron–Cohen et al. (2001). This screening tool can be administered for 5-10 min. The participants in the control group (twelve males, mean age ± standard deviation = 29.5 ±12.4) did not have a diagnosis of Autism Spectrum Disorder and were selected to match the age, gender, body mass index of the participants in the test group.

2.2 Samples

The participants were provided with an odor discrimination test kit called "Sniffin' Sticks" (Hummel et al., 1997) which are pen-shaped odor dispensers. It is a set of sixteen triplets of odorants. The "Sniffin' Sticks" odor identification kit was used for the second half of this study. There are sixteen common food and non-food odorants in this kit: Orange, shoe leather, cinnamon, peppermint, banana, lemon, licorice, turpentine, garlic, coffee, apple, cloves, pineapple, rose, anise, and fish.

2.3 Measurement of responses

Participants rated odor intensity, liking, pleasantness, arousal, familiarity, and edibility of the samples on a 9-point categorical scale. They were asked to identify each odor from four alternate forced choices. For the odor discrimination test, number of correctly identified odors were recorded.

2.4 Procedure

This study was conducted in one session that lasted for approximately one hour. Participants were asked to fill out a demographics form upon arrival. Following that, their body mass index was measured using a weighing scale and a stadiometer. Thereafter, they were explained the procedure of this study and made familiar with the scales.

Odor discrimination was tested using 16 sets of odorants. The participant was provided with three odorants and was asked to identify the sample that had a different smell. Participants were blindfolded during this test to avoid visual bias. The presentation of triplets was separated by thirty second intervals. After this test, the participant was given a ten-minute break. Following the break, participants were presented with sixteen odors in the Sniffin' Sticks odor identification kit. They sniffed each odor for ~three seconds and rated odor intensity, liking, pleasantness, arousal, familiarity, and edibility. Thereafter, they identified each odor from four different choices. The presentation of odors was separated by one-minute breaks.

Thereafter, each participant filled out an "Importance of Odors" questionnaire. This questionnaire was developed by Croy et al. (2010). It attempts to understand how people perceive everyday odors in terms of application, assessment, consequence, and aggravation. This is a 20-item questionnaire, with a four-point rating scale (1 = totally disagree, 2 = mostly disagree, 3 = mostly agree, and 4 = mostly disagree) for each question.

2.5 Data Analysis

Data was analyzed using JMP Pro (version 14.1, SAS Institute Inc., Cary, NC, USA) and SPSS (Version 25.0. IBM Corp., Armonk, NY, USA). For the odor discrimination test, a total number of correct responses was recorded and used for data analysis. The scale values for odor intensity, liking, pleasantness, arousal, familiarity, and edibility were assigned a number from 1 to 9. These values were used for analysis. Similarly, the responses on the “Importance of odors” ballot were assigned numbers from 1 to 4 and used for analysis. The normality assumption was tested for the dataset using the Shapiro–Wilk W test. The test showed that odor identification, discrimination, intensity, liking, pleasantness, arousal, familiarity, and edibility were not normally distributed ($P < 0.05$ for all attributes). Thus, Mann Whitney U tests were conducted to compare these variables among the two groups. This statistical test is used to compare the differences between two independent groups when the dependent variable is either ordinal or continuous. For each odor quality, the test groups were considered as factors and the ratings of intensity, liking, etc were considered as variables. A statistically significant difference was defined as when $P < 0.05$.

3. Results

The autism spectrum quotient (AQ) scores differed significantly between the control and test groups ($U = 3.00$, $P < 0.001$); as expected, the ASD group [mean ± standard deviation (SD) = 28.8 ± 5.3; mean rank = 30.35] showed higher AQ scores than control group (15.1 ± 4.3; mean rank = 10.65).

As shown in Figure 4-2, the ASD group (mean ± SD = 8.0 ± 1.9; mean rank = 10.65) showed a worse performance with respect to odor discrimination task than the control group (mean ± SD = 13.6 ± 1.4; mean rank = 30.35) ($U = 3.00$, $P < 0.001$). The ASD group (mean ± SD = 10.5 ± 3.3; mean rank = 16.15) also showed a worse perforamance in odor identification task than the control group (mean ± SD = 13.0 ± 1.6; mean rank = 24.85) ($U = 113.0$, $P = 0.018$).

As shown in Table 4-1, the two groups, ASD and control, showed no significant differences with respect to overall likings of everyday odors, except anise odor (U = 129.00, P = 0.046). The ASD group liked anise odor significantly more than control group. In addition, the ASD group perceived peppermint (U = 96.00, P =0.003), lemon (U = 129.50, P = 0.049), and pineapple (U = 128.00, P = 0.046) significantly more intense than the control group (Table 4-2). With respect to odor-induced emotions, significant differences between the ASD and control groups were found in several odors. More specifically, compared to the control group, the ASD group felt orange odor less pleasant (U = 122.50, P = 0.03) (Table 4-3). In addition, the ASD group felt peppermint (U = 127.50, P = 0.047), lemon (U = 120.50, P = 0.03), apple (U = 126.00, P = 0.04), clove (U = 91.50, P = 0.002), rose (U = 115.50, P = 0.02), and anise (U = 104.00, P = 0.007) odors significantly more arousing than the control group, as shown in Table 4-4.

There were no significant differences between the ASD and control groups with respect to edibility of everyday odors, except coffee odor (U = 123.50, P = 0.03). The coffee odor was perceived as lesser edible by the ASD group, compared to the control group (Table 4-5). Finally, there were significant differences between the ASD and control groups in terms of odor familiarity in several odors: shoe leather (U = 122.50, P = 0.03), cinnamon (U = 123.00, P = 0.03), and fish (U = 128.00, P = 0.04) odors. While the ASD group was found to be more familiar with shoe leather and cinnamon odors, the control group was more familiar with the fish odor, compared to the counterpart (Table 4-6).

For the importance of odors questionnaire, significant differences were not found in any of the three subscales of application, assessment and consequence.

4. Discussion

This study found that odor identification for adults with ASD was impaired when compared to adults without ASD. This is in line with the previous study by Galle et al. (2013) and who found that participants in ASD group were worse in odor identification than Asperger and control participants and suggested that the olfactory functions that involve verbalization are reduced in autistic individuals. In contrast, Suzuki et al. (2003) found odor identification impairment in participants with Asperger syndrome. Although we did not separate participants between ASD and Asperger's diagnosis, all our participants were verbal. Nevertheless, it is difficult to compare these results. Dudova et al. (2011) reported that children with ASD showed an almost normal performance of odor identification, and Brewer et al. (2008) reported the same for children with high functioning autism. However, the latter found a negative correlation of olfactory ability with age in the High Functioning Autism group, but not in the control group, which suggests that with age, olfaction could deteriorate in people with autism.

Odor discrimination also varied among the two groups, with ASD participants performing significantly worse than control participants. These results are contrasting with the results in the study by Galle et al. (2013), who did not find any differences in the odor discrimination ability. However, it is to be noted that their methodology of testing was different. Where our test involved identifying the "different odor" from sets of 3 odors, the previous paper asked whether a pair of smells were the same or different. Perhaps, this might be the key differentiator, where our methodology involved more verbalization of responses, and the existing verbal difficulty in ASD could have affected our results. In our study, liking and pleasantness of odors between the groups were very similar, which is consistent with previous findings by Tavassoli and Baron-Cohen (2011). The study by Hrdlicka et al. (2011) found that children with

Asperger Syndrome perceived the smell of cinnamon, pineapple, and cloves as significantly less pleasant than controls. These differences may be attributed to adaptation among adults, with repeated exposure.

Intensity ratings for lemon, peppermint, and pineapple were higher by the ASD group than the control group. Additionally, anise, apple, cloves, lemon, peppermint, and rose odors were perceived as significantly more arousing by the ASD group. This is a contrasting finding from the study by Galle et al. (2013). They used 8 odors on a scale from very weak to very strong. In this study, we used sixteen odors and a longer scale, ranging from extremely weak to extremely strong, which gives more space and could have been more sensitive to differences. Since some of these odorants may have a trigeminal sensation, like lemon, peppermint, cloves, pineapple, anise, there might be differences in perception of a trigeminal stimulus. This was also suggested by Luisier et al. (2015), who said that the stimulation of the fifth cranial nerve might be different in people with ASD. There is also evidence that people with ASD are cortically hyper-reactive to non-CT-targeted touch, while being hyporeactive to CT-targeted touch. Although trigeminal sensations cannot be classified as non-CT targeted touch one cannot eliminate the possibility of different mechanisms and physiology of nerves in people with autism. Muratori et al. (2017) suggested that hyper-responsivity to smell is due to the impairment in the odor identification ability, which makes the stimulus more intense. Pellicano and Burr (2012) had proposed that because people with autism did not have a reference point to compare stimuli, these odors might come as a surprise and be more arousing.

People with ASD rated less familiarity with shoe leather and fish odors, but more familiarity with cinnamon. These findings are inconsistent with Galle et al. (2013), and since they used a different scale and 8 undisclosed odors, these results are not comparable. We found similar ratings between edibility, with an exception in coffee, which might be the result of perception of coffee as bitter and the associated bitterness sensitivity in people with autism. We did not find any differences among the three sections of the Importance of Odors questionnaire.

5. Conclusion

To summarize, there were three major findings of this study. First, odor identification ability is diminished in adults with ASD. Second, the odor discrimination ability of people with ASD is lower as compared to people without ASD. Lastly, the perception of odors among people with ASD is different from people without ASD, specifically in terms of arousal and perceived intensity. It seemed that odors with sour quality and possible trigeminal sensation qualities were perceived as more intense by the ASD group. There were few differences in liking and pleasantness of odors among the two groups. In conclusion, ASD affects the olfactory abilities of people, and further research is necessary to identify emotions elicited by odors, in order to understand these discrepancies better.

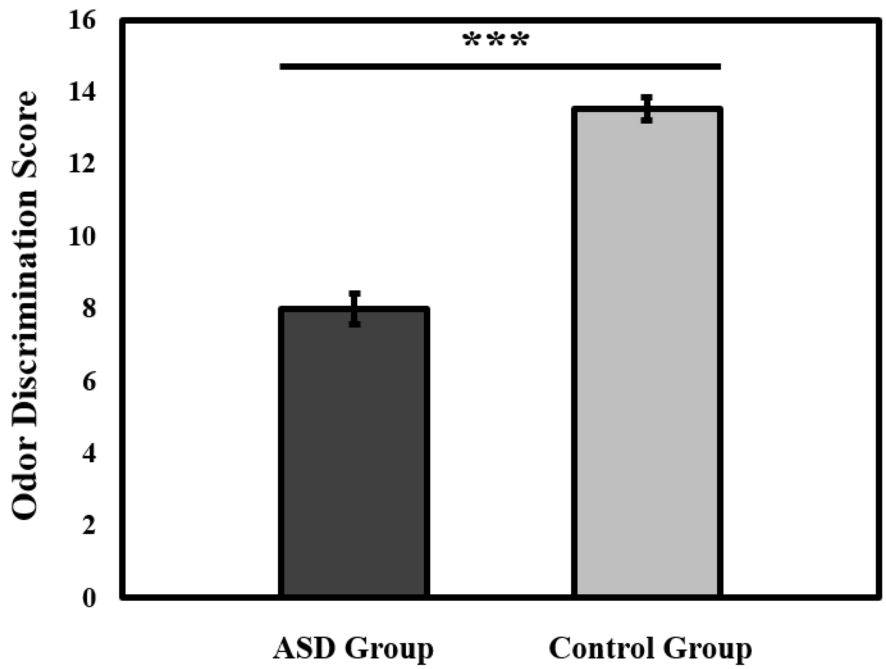

Figure 4-1: Comparison of odor discrimination scores between the ASD and Control groups. ******* represents a significant difference between the scores at $P < 0.001$. Error bars represent standard error of mean. ASD stands for Autism spectrum Disorder.

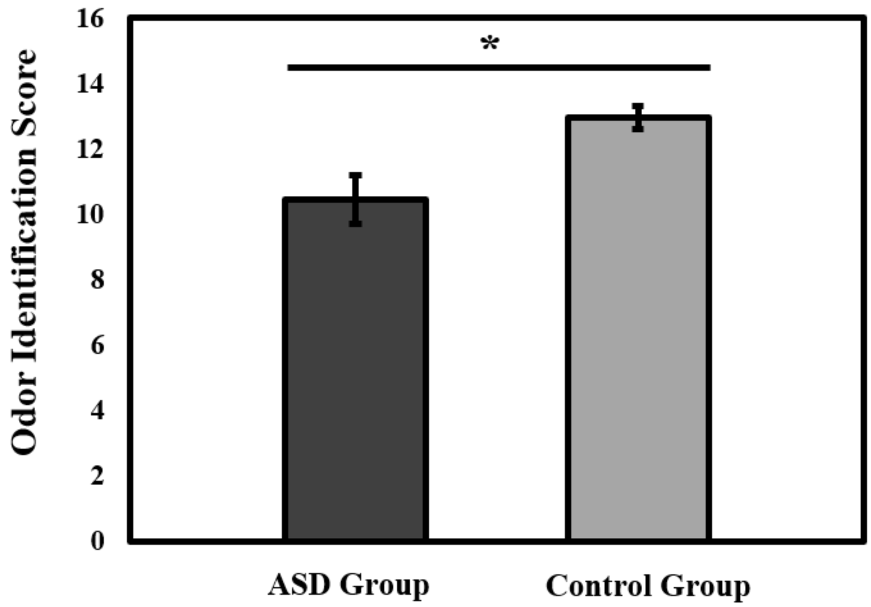

Figure 4-2: Comparison of odor identification scores between the ASD and Control groups. ***** represents a significant difference between the scores at $P < 0.05$. Error bars represent standard error of mean. ASD stands for Autism spectrum Disorder.

Table 4-1: Comparisons between the ASD and control groups with respect to likings of 16 everyday odors

	ASD Group	Control Group	U Values	P value
Orange	6.1 (±1.8)	6.6 (±1.3)	170.00	0.40
Shoe Leather	5.3 (±1.9)	5.1 (±1.6)	183.00	0.64
Cinnamon	6.3 (±2.5)	5.5 (±1.6)	139.50	0.10
Peppermint	7.5 (±1.6)	7.6 (±1.0)	186.50	0.70
Banana	6.6 (±2.2)	6.6 (±1.6)	190.00	0.78
Lemon	5.9 (±2.2)	6.6 (±1.0)	171.00	0.42
Licorice	5.6 (±2.6)	4.5 (±2.0)	143.00	0.12
Turpentine	4.5 (±2.5)	4.7 (±1.4)	187.00	0.72
Garlic	6.5 (±2.5)	5.5 (±2.1)	143.00	0.12
Coffee	6.6 (±2.3)	7.5 (±1.2)	163.00	0.31
Apple	7.0 (±1.4)	6.9 (±1.1)	190.00	0.78
Cloves	5.7 (±2.1)	5.7 (±1.9)	183.00	0.64
Pineapple	7.4 (±1.5)	7.6 (±1.0)	194.00	0.87
Rose	6.4 (±1.9)	6.4 (±1.6)	181.50	0.61
Anise	6.2 (±1.9)	5.3 (±1.2)	129.00	0.046
Fish	4.0 (±2.4)	3.0 (2.1)	150.00	0.17

Values represent mean (± standard deviation)
ASD stands for Autism Spectrum Disorder.

Table 4-2: Comparisons between the ASD and control groups with respect to perceived intensity of 16 everyday odors

	ASD Group	Control Group	U Values	P value
Orange	5.9 (±1.8)	5.4 (±1.1)	162.50	0.30
Shoe Leather	5.8 (±1.9)	5.0 (±1.5)	142.50	0.11
Cinnamon	6.7 (±1.6)	5.8 (±1.3)	133.50	0.07
Peppermint	8.0 (±1.2)	7.2 (±0.7)	96.00	0.003
Banana	6.2 (±1.8)	6.7 (±1.2)	174.50	0.48
Lemon	7.0 (±1.6)	5.8 (±1.7)	129.50	0.049
Licorice	6.4 (±1.9)	6.0 (±1.8)	187.00	0.72
Turpentine	6.4 (±2.2)	5.7 (±1.5)	163.50	0.32
Garlic	8.0 (±1.0)	7.7 (±0.8)	156.00	0.21
Coffee	7.2 (±1.5)	7.0 (±1.1)	185.00	0.67
Apple	6.4 (±2.1)	5.8 (±1.5)	147.50	0.14
Cloves	6.8 (±1.9)	6.4 (±1.5)	166.50	0.36
Pineapple	7.1 (±1.9)	6.4 (±1.2)	128.00	0.046
Rose	6.8 (±2.0)	5.8 (±1.6)	141.00	0.10
Anise	5.8 (±2.1)	5.4 (±1.6)	165.50	0.34
Fish	7.2 (±1.9)	7.9 (±1.3)	156.00	0.21

Values represent mean (± standard deviation)
ASD stands for Autism Spectrum Disorder.

Table 4-3: Comparisons between the ASD and control groups with respect to pleasantness of 16 everyday odors

	ASD Group	Control Group	U Values	P value
Orange	5.8 (±1.7)	6.8 (±1.3)	122.50	0.03
Shoe Leather	5.7 (±1.9)	5.1 (±1.7)	161.00	0.28
Cinnamon	6.0 (±2.1)	5.2 (±1.6)	153.00	0.20
Peppermint	7.5 (±1.1)	7.6 (±1.1)	190.00	0.78
Banana	6.4 (±2.2)	6.4 (±1.8)	199.50	0.99
Lemon	5.7 (±2.3)	6.7 (±1.3)	155.00	0.21
Licorice	5.4 (±2.4)	4.4 (±1.8)	147.00	0.14
Turpentine	4.7 (±2.4)	4.7 (±1.5)	195.50	0.90
Garlic	6.2 (±2.5)	4.9 (±2.1)	133.00	0.07
Coffee	6.5 (±2.2)	7.5 (±1.2)	151.50	0.18
Apple	7.0 (±1.5)	6.9 (±1.4)	192.00	0.83
Cloves	5.8 (±2.5)	5.3 (±2.1)	165.50	0.35
Pineapple	7.1 (±1.6)	7.5 (±1.1)	172.00	0.44
Rose	6.2 (±2.1)	6.3 (±1.6)	197.50	0.95
Anise	6.2 (±1.8)	5.2 (±1.6)	139.50	0.09
Fish	4.0 (±2.6)	2.8 (±2.1)	146.50	0.14

Values represent mean (± standard deviation)
ASD stands for Autism Spectrum Disorder.

Table 4-4: Comparisons between the ASD and control groups with respect to arousal of 16 everyday odors

	ASD Group	Control Group	U Values	P value
Orange	4.6 (±1.8)	4.5 (±1.4)	198.00	0.96
Shoe Leather	4.9 (±1.7)	4.9 (±1.4)	178.00	0.54
Cinnamon	5.7 (±2.2)	4.9 (±1.2)	130.00	0.06
Peppermint	6.1 (±2.6)	4.6 (±2.2)	127.50	0.047
Banana	5.7 (±1.9)	4.9 (±1.3)	143.00	0.12
Lemon	6.1 (±1.5)	4.9 (±1.6)	120.50	0.03
Licorice	5.4 (±2.0)	4.8 (±1.3)	159.50	0.25
Turpentine	5.9 (±1.8)	5.5 (±1.1)	155.00	0.20
Garlic	6.6 (±1.7)	6.0 (±1.3)	147.00	0.14
Coffee	6.6 (±1.8)	5.1 (±2.4)	132.00	0.06
Apple	5.4 (±2.1)	4.2 (±1.0)	126.00	0.04
Cloves	6.6 (±1.4)	5.2 (±1.3)	91.50	0.002
Pineapple	5.7 (±2.2)	4.5 (±1.5)	131.00	0.06
Rose	5.5 (±2.3)	4.1 (±1.4)	115.50	0.02
Anise	5.6 (±1.8)	4.4 (±1.0)	104.00	0.007
Fish	6.7 (±1.8)	5.9 (±1.4)	140.50	0.09

Values represent mean (± standard deviation)
ASD stands for Autism Spectrum Disorder.

Table 4-5: Comparisons between the ASD and control groups with respect to edibility of 16 everyday odors

	ASD Group	Control Group	U Values	P value
Orange	5.2 (±2.6)	6.0 (±2.4)	167.00	0.37
Shoe Leather	3.7 (±2.1)	2.7 (±1.5)	141.00	0.11
Cinnamon	5.9 (±2.6)	5.3 (±1.9)	160.00	0.27
Peppermint	6.9 (±2.5)	7.7 (±0.9)	186.00	0.69
Banana	6.0 (±2.5)	7.4 (±1.3)	137.50	0.08
Lemon	6.1 (±2.2)	6.0 (±1.7)	187.00	0.72
Licorice	5.3 (±2.7)	5.2 (±2.2)	193.50	0.86
Turpentine	4.1 (±2.6)	2.6 (±1.6)	131.50	0.06
Garlic	6.0 (±2.8)	7.0 (±1.6)	174.00	0.47
Coffee	5.9 (±2.8)	7.9 (±1.0)	123.50	0.03
Apple	6.8 (±2.0)	7.0 (±1.4)	194.50	0.88
Cloves	5.2 (±2.5)	5.7 (±2.2)	180.00	0.58
Pineapple	6.9 (±1.9)	7.3 (±1.5)	177.50	0.53
Rose	4.5 (±2.8)	3.4 (±2.0)	157.50	0.24
Anise	4.6 (±2.5)	4.1 (±2.0)	170.00	0.41
Fish	4.2 (±2.7)	3.8 (±2.9)	180.50	0.59

Values represent mean (± standard deviation)
ASD stands for Autism Spectrum Disorder.

Table 4-6: Comparisons between the ASD and control groups with respect to familiarity of 16 everyday odors

	ASD Group	Control Group	U Values	P value
Orange	6.6 (±2.1)	6.5 (±1.7)	181.50	0.61
Shoe Leather	6.4 (±1.6)	5.0 (±2.0)	122.50	0.03
Cinnamon	7.1 (±1.7)	5.8 (±2.0)	123.00	0.03
Peppermint	8.1 (±1.2)	8.4 (±0.8)	177.00	0.50
Banana	6.6 (±2.2)	7.7 (±1.1)	144.00	0.13
Lemon	7.7 (±1.3)	7.2 (±1.5)	158.50	0.25
Licorice	6.7 (±1.8)	7.0 (±1.5)	180.00	0.58
Turpentine	5.6 (±2.2)	4.7 (±2.1)	159.00	0.26
Garlic	7.5 (±2.0)	8.1 (±1.0)	177.00	0.51
Coffee	7.4 (±2.0)	8.3 (±0.7)	169.50	0.38
Apple	7.0 (±1.6)	6.6 (±1.5)	169.00	0.39
Cloves	6.4 (±1.9)	6.3 (±2.1)	197.50	0.95
Pineapple	6.8 (±2.3)	7.2 (±1.7)	192.50	0.84
Rose	6.6 (±2.5)	6.3 (±1.8)	169.50	0.40
Anise	5.5 (±2.4)	4.6 (±2.4)	148.00	0.16
Fish	6.6 (±2.3)	7.9 (±1.5)	128.00	0.04

Values represent mean (± standard deviation)
ASD stands for Autism Spectrum Disorder.

REFERENCES

Ashwin, C., Chapman, E., Howells, J., Rhydderch, D., Walker, I., & Baron-Cohen, S. (2014). Enhanced olfactory sensitivity in autism spectrum conditions. Molecular Autism, 5, 53.

Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001). The autism-spectrum quotient (AQ): Evidence from asperger syndrome/high-functioning autism, malesand females, scientists and mathematicians. Journal of Autism and Developmental Disorders, 31, 5-17.

Brewer, W. J., Brereton, A., & Tonge, B. J. (2008). Dissociation of age and ability on a visual analogue of the University of Pennsylvania Smell Identification Test in children with autism. *Research in Autism Spectrum Disorders*, *2*(4), 612-620.

Crane, L., & Goddard, L. (2008). Episodic and semantic autobiographical memory in adults with autism spectrum disorders. *Journal of autism and developmental disorders*, *38*(3), 498-506.

Crane, L., Goddard, L., & Pring, L. (2009). Sensory processing in adults with autism spectrum disorders. *Autism*, *13*(3), 215-228.

Croy, I., Buschhüter, D., Seo, H. S., Negoias, S., & Hummel, T. (2010). Individual significance of olfaction: development of a questionnaire. *European Archives of Oto-Rhino-Laryngology*, *267*(1), 67.

Demattè, M. L., Endrizzi, I., Biasioli, F., Corollaro, M. L., Pojer, N., Zampini, M., & Gasperi, F. (2013). Food neophobia and its relation with olfactory ability in common odour identification. Appetite, 68, 112-117.

Dudova, I., & Hrdlicka, M. (2013). Olfactory functions are not associated with autism severity in autism spectrum disorders. *Neuropsychiatric disease and treatment*, *9*, 1847.

Dudova, I., Vodicka, J., Havlovicova, M., Sedlacek, Z., Urbanek, T., & Hrdlicka, M. (2011). Odor detection threshold, but not odor identification, is impaired in children with autism. European Child & Adolescent Psychiatry, 20, 333-340.

Galle, S. A., Courchesne, V., Mottron, L., & Frasnelli, J. (2013). Olfaction in the autism spectrum. Perception, 42, 341-355.

Hrdlicka, M., Vodicka, J., Havlovicova, M., Urbanek, T., Blatny, M., & Dudova, I. (2011). Brief report: significant differences in perceived odor pleasantness found in children with ASD. *Journal of Autism and Developmental Disorders*, *41*(4), 524-527.

Hummel, T., Sekinger, B., Wolf, S. R., Pauli, E., & Kobal, G. (1997). 'Sniffin'sticks': olfactory performance assessed by the combined testing of odor identification, odor discrimination and olfactory threshold. Chemical Senses, 22, 39-52.

JMP®, Version <*14.1*>. SAS Institute Inc., Cary, NC, 1989-2019.

Lane, A. E., Geraghty, M. E., Young, G. S., & Rostorfer, J. L. (2014). Problem eating behaviors in autism spectrum disorder are associated with suboptimal daily nutrient intake and taste/smell sensitivity. *ICAN: Infant, Child, & Adolescent Nutrition*, *6*(3), 172-180.

Luisier, A. C., Petitpierre, G., Ferdenzi, C., Clerc Bérod, A., Giboreau, A., Rouby, C., & Bensafi, M. (2015). Odor perception in children with autism spectrum disorder and its relationship to food neophobia. Frontiers in Psychology, 6, 1830.

Matson, J. L., and Fodstad, J. C. (2009). The treatment of food selectivity and other feeding problems in children with autism spectrum disorders. Research in Autism Spectrum Disorders 3, 455–461. doi: 10.1016/j.rasd.2008.09.005

Muratori, F., Tonacci, A., Billeci, L., Catalucci, T., Igliozzi, R., Calderoni, S., & Narzisi, A. (2017). Olfactory processing in male children with autism: atypical odor threshold and identification. *Journal of autism and developmental disorders*, *47*(10), 3243-3251.

Pellicano, E., & Burr, D. (2012). When the world becomes 'too real': a Bayesian explanation of autistic perception. *Trends in cognitive sciences*, *16*(10), 504-510.

Seo, H. S., Lee, S., & Cho, S. (2013). Relationships between personality traits and attitudes toward the sense of smell. *Frontiers in psychology*, *4*, 901.

Smythies, J. (1997). The functional neuroanatomy of awareness: with a focus on the role of various anatomical systems in the control of intermodal attention. *Consciousness and cognition*, *6*(4), 455-481.

Suzuki, Y., Critchley, H. D., Rowe, A., Howlin, P., & Murphy, D. G. (2003). Impaired olfactory identification in Asperger's syndrome. *The Journal of neuropsychiatry and clinical neurosciences*, *15*(1), 105-107.

Tavassoli T, Baron-Cohen S, 2011 "Olfactory detection thresholds and adaptation in adults with autism spectrum condition" *Journal of Autism and Developmental Disorders* **42** 905–909

Willander, J., & Larsson, M. (2006). Smell your way back to childhood: Autobiographical odor memory. *Psychonomic bulletin & review*, *13*(2), 240-244.

Woo, C. C., & Leon, M. (2013). Environmental enrichment as an effective treatment for autism: a randomized controlled trial. Behavioral Neuroscience, 127, 487.

CHAPTER 5

INFLUENCES OF AUTISM SPECTRUM DISORDER ON PERCEPTION AND EMOTIONAL RESPONSES TO TASTE CUES

1. Introduction

Taste perception is one of the primary ways to enjoy food. Our liking of a certain taste drives our food choices which indirectly affect our health. There are five identified basic tastes: sweet, salty, sour, bitter and umami. Evidence suggests that taste perception can change based on genetic composition, which can result in different experiences of people to the same tastes. The taste perception in humans can differ depending on the number of taste buds and sensory sensitivity of individuals. (Duffy & Bartoshuk, 2000). There have been very few studies on the influences of ASD on taste perception. One of them, by Bennetto et al. (2007), investigated odor and taste perception in ASD. They found that compared to participants without ASD, those with ASD were significantly less accurate in identifying sour tastes and marginally less accurate for bitter tastes, but they were not different in identifying sweet and salty stimuli. The taste detection threshold using electrogustometry among both groups were equivalent. Tavassoli and Baron-Cohen (2012) performed a similar study on taste perception in ASD and found similar results. Adults with ASD were less accurate in identifying tastes overall (irrespective of medication or age). Specifically, adults with ASD had lower scores for identifying bitter, sweet, and sour tastes. However, they did not significantly differ with regard to detecting salty tastes. They noted that adults with ASD more often misidentified a taste as salty or as no taste (Tavassoli & Baron-Cohen, 2012). The results might be due to that different pathways are present for different tastes. The type two pathway is for sweet, bitter, salty and umami tastes, while type three is for sour taste (Trivedi, 2012). Thus, a better perception of salty taste and not sweet taste poses an intriguing question.

It is found that 6-n-propylthiouracil (PROP) is a compound that is used to detect supertasters (Bartoshuk et al., 1994), who have a higher density of taste buds as compared to rest of the population. When people with Alexithymia (subclinical inability to identify and describe emotions in the self) were given the PROP test, it was found that non-tasters had higher alexithymia scores than PROP tasters. Alexithymia is a subclinical phenomenon characterized by difficulties in recognizing, describing, and distinguishing feelings from the bodily sensations of emotional arousal (Nemiah et al., 1976). The researchers concluded that Alexithymia may play a role in responsiveness to the aversive and bitter taste of PROP and in combination with other personality traits, may provide important insights for better understanding food liking (Robino et al., 2016). Their data confirmed that genetic variation in the TAS2R38 gene is the main factor responsible for the capacity to perceive PROP, but that alexithymia can be a significant modifier of PROP bitter perception beyond the effects of the gene. In another study, it was reported that subjects with ASD were relatively impaired in both the appreciation and production of emotional expressions (Macdonald et al., 1989). These results suggest that due to the similarity in Alexithymia and certain ASD characteristics, it is possible that people with ASD might be non-tasters. Cole et al. (2017) found a relationship between bitter sensitivity from the TAS2R38 gene and feeding problems in healthy preschool kids. They associated the presence of the TAS2R38 gene to picky eating behavior, perhaps because of increased sensitivity. There is data that suggests a correlation between food selectivity and TAS2R38 genetics in ASD (Riccio et al., 2018). Although this study had a small sample size, there is compelling evidence that this gene should be further investigated in terms of eating behavior.

This study aims to determine whether ASD influences taste identification and discrimination performances, hedonic ratings, and emotional responses with respect to basic taste cues (sweet, sour, salty and bitter [as elicited by caffeine and quinine]). Two compounds for bitter taste were used because of a testable hypothesis published by Ghanizadeh (2010), that suggested that caffeine might have a role in ASD.

2. Materials and Methods

This study was approved by the Institutional Review Board of the University of Arkansas and written consent was obtained from each participant. They were compensated with $30 in the form of gift cards, upon successful completion of the study.

2.1 Participants

Participants were recruited from the Northwest Arkansas community, using the University of Arkansas Sensory Service Center consumer database. A survey was sent out to the database which included questions about health, allergies, and demographics. Participants who qualified were over eighteen years of age, had no food allergies, had no clinical history of major diseases and did not smoke. Twenty participants (thirteen males; mean age ± standard deviation = 28 ±12.1) were recruited for the test group, based on an existing diagnosis of Autism Spectrum Disorder, confirmed by Autism spectrum quotient (AQ). The Autism Spectrum Quotient (AQ) developed by Baron–Cohen et al. (2001). This screening tool can be administered for 5-10 min. The participants in the control group (thirteen males, mean age ± standard deviation = 27.3 ±11.9) did not have a diagnosis of Autism Spectrum Disorder and were selected to match the age, gender, body mass index of the participants in the test group.

2.2 Sample Preparation

Taste solutions were prepared using commercially available pure cane sugar (Great Value, Walmart Stores, Inc. Bentonville, AR), citric acid (Sigma Aldrich Fine Chemicals, St Louise, MO), caffeine (Aldrich Chemical Company, Inc., Milwaukee WI), quinine hydrochloride (Sigma Aldrich Fine Chemicals, St Louise, MO), and sodium chloride (Morton Salt, Inc., Chicago, IL). Springwater (Mountain Valley Springs Co., LLC, Hot Springs, AR) was used to prepare these taste solutions. The spring water was used as a warm-up sample. Each taste solution was prepared at three concentrations low, medium and high that correspond to 5, 7 and 10 rating on a 15 point intensity scale (Meilgaard et al. 2015). The conversion of these scale values to volumetric concentration are provided in Table 5-1. The samples were presented in 1oz. cups labeled with 3-digit random codes.

2.3 Measurement of responses

Taste intensity ratings were given on a General Labeled Magnitude Scale (Bartoshuk et al., 2004), which is a quasi-logarithmic line-scale containing labeled anchors from no sensation to strongest sensation imaginable of any kind. Participants rated the overall liking of the samples on a Labeled Hedonic Scale (Lim & Fujimaru, 2010), which is a line scale with common hedonic descriptors. The emotional responses were measured using explicit and implicit methods. The explicit responses were measured using a self-reported emotion questionnaire Essence 25 (Nestrud et al., 2016) which contains twenty-five terms of emotions on a 5 point scale labeled from not at all to extremely. This scale is a shortened version of the Essence profile which contains thirty-nine emotion terms on the 5 point scale (King& Meiselman, 2010). The implicit responses were measured using facial expression software iMotions (version 6.1, iMotions, Inc., MA). This software uses the principles of Facial Action Coding System and provides data based on head orientation, facial landmarks, and facial expressions. This software was used to analyze seven universal facial expressions: joy, anger, surprise, contempt, fear, disgust and sadness. Numerical scores are assigned to each emotion, called evidence values, which correspond to the degree of confidence that an emotion is present. (iMotion, 2018). A camera (C920 HD Pro Webcam, Logitech Europe S.A., Nijmegen, Netherlands) was mounted on top of the display screen in order to capture facial expression and was positioned correctly before each data collection session.

2.4 Procedure

This study was conducted in one session that lasted for approximately one hour. Participants were asked to fill out a demographics form upon arrival. Thereafter, they were explained the procedure of this study and made familiar with the scales. A warm up sample (spring water) was provided to the subject in order to demonstrate the procedure and answer potential queries. The participant was then provided with a total of ten samples (five taste solutions in low and high concentrations), randomized and presented in serial monadic fashion. For each taste solution, participants were instructed to completely pour the sample in their mouth, without swallowing it, and then look at the screen for 6 seconds. After that, participants were asked to expectorate the sample and rate taste intensity, evoked emotions and liking of the sample respectively. After rating, they were asked to cleanse their palate using unsalted crackers (Nabisco Premium, Mondelēz International, East Hanover, NJ) and spring water.

Once the participant tasted all ten samples, they were given a ten min break. Following that, participants performed a taste identification test, in which they were provided five taste solutions at medium concentration, randomized in a monadic sequential fashion. They were given five choices (salty, sweet, sour, bitter, and no taste) per sample. After five samples, participants were presented with an N-Propylthiouracil (PROP) taste strip (Precision Laboratories, Cottonwood, AZ) and asked to identify its taste, among the same five choices as above. This test is used to identify supertasters, individuals who perceive the taste strip as bitter are considered supertasters whereas individuals who perceive it as “bland” or no taste are considered non-tasters.

2.5 Data Analysis

Data was analyzed using JMP Pro (version 14.1, SAS Institute Inc., Cary, NC, USA) and SPSS (IBM Corp. Released 2017. IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY: IBM Corp.). The scale values for taste intensity were measured in centimeters and converted to a 100-point scale. Liking ratings made on the LHS were measured from the bottom of the scale and were translated into a range from -100 to +100. The ratings on the Essence25 were coded as 1 (not at all), 2 (slightly), 3 (moderately), 4 (very) and 5 (extremely). For facial expression analysis, the average values of emotions for 30 frames pre-tasting and 30 frames post tasting were used, to avoid the biased contribution of the anticipatory phase (Samant at al., 2017). The values of pre-tasting were subtracted from the values of post-tasting, and the resulting numerical values were used for analysis. The normality assumption was tested for the dataset using the Shapiro–Wilk W test. The test showed that the ratings of liking, intensity, Essence25, and facial expressions were not normally distributed ($P > 0.05$ for all attributes). Thus, Mann Whitney U tests were conducted to compare these variables among the two groups. This test is used to compare the differences between two independent groups when the dependent variable is either ordinal or continuous. For each taste quality, the test groups were considered as factors and the ratings of intensity, liking, emotions and facial expressions were considered as variables. A statistically significant difference was defined as when $P < 0.05$. Chi-square goodness of fit test was performed for PROP taster data since the values were either 1 or 0. A statistically significant difference was defined as when $P < 0.05$.

3. Results

The autism spectrum quotient for the test group and the control group differed significantly ($U = 371.50$; $P < 0.001$); as expected, the ASD group [mean ± standard deviation (SD) = 28.4 ±5.1] showed higher AQ scores than control group (17.0 ±5.4).

The ASD group (mean ± SD = 3.8 ± 1.1) showed a worse performance with respect to taste identification task than the control group (mean ± SD = 4.6 ± 0.5) ($U = 124.00$, $P < 0.05$). Additionally, it should be noted that caffeine was the only taste quality that was misidentified by the control group, however, participants in the test group misidentified among all five taste qualities. The results from the PROP taste strip test showed that seventeen individuals in the ASD group were super-tasters, whereas ten individuals in the control group were identified as supertasters ($P < 0.05$).

As shown in Table 5-2, the two groups, ASD and control, showed no significant differences in terms of liking of basic tastes except sour taste at a high concentration ($U= 122.50$; $P = 0.04$). Although both groups disliked this stimulus, the ASD group disliked it more than the control group. In addition, ASD group perceived sweet taste at a high concentration ($U = 117.50$; $P = 0.03$) significantly less intense than the control group (Table 5-3).

As shown in Table 5-4, the number of attributes reported were significantly different for all taste attributes at both concentrations among the ASD and the control group.The control group reported a greater number of emotions being felt as compared to the ASD group for caffeine at high concentration (U = 115.00, P = 0.02), caffeine at low concentration (U = 95.00, P = 0.004), sucrose at high concentration (U = 92.50, P = 0.004), sucrose at low concentration (U = 91.00, P = 0.003), sodium chloride at high concentration (U = 102.00, P = 0.008), sodium chloride at low concentration (U = 87.00, P = 0.002), quinine at high concentration (U = 69.00, P < 0.001), quinine at low concentration (U = 88.00, P = 0.002), citric acid at high concentration (U = 58.50, P < 0.001) and citric acid at low concentration (U = 54.00, P < 0.001). Additionally the control group reported feeling higher intensity of emotions (Table 5-5) for sodium chloride, quinine and caffeine taste qualities, but for sucrose at high concentration, where the ASD group reported feeling more aggressive (U = 122.50, P = 0.03) and more disgusted (U = 122.50, P = 0.03) than the control group and for citric acid at low concentration, the ASD group reported being more disgusted (U = 122.50, P = 0.03) than the control group.

As shown in Table 5-6, two taste qualities elicited differences in facial expressions. For citric acid at high concentration, the ASD group showed a higher sadness expression (U = 119.00, P = 0.03) and lower surprise facial expression (U =292.00, P = 0.01) than the control group. Moreover, for sucrose at high concentration the ASD group showed a higher sadness expression (U = 115.00, P = 0.02) and higher contempt expression (U = 123.00, P = 0.04) than the control group.

4. Discussion

We found that taste identification is impaired in people with autism, which was consistent with previous findings of Bennetto et al. (2007) and Tavassoli and Baron-Cohen (2012). Our results from the PROP test were contrasting than those found by Robino et al. (2016), for people with alexithymia. Thus, it can be said that the same mechanism does not exist for people with autism and people with alexithymia and further research is necessary to understand the differences. The perceived intensity of sweet taste was less in the test group compared to the control group. Damiano et al. (2014) found no differences in sweet taste sensitivity or hedonic response to sweet tastes between the ASD and control groups. Although we did not find differences in the liking of sweet taste, our results regarding sweet sensitivity were contrasting. Although Damiano et al. (2014) said that ASD symptom severity was associated with sweet taste sensitivity, we tested the perceived intensity and not the physiological sensitivity, thus these results cannot be directly compared. Sour taste liking was reduced in people with autism, which may be explained by a reduced ability to identify sour taste, thus leading to a reduction in liking, as hypothesized for odors by Muratori et al. (2017). These results are also complementary to the one on one interview and odor performance results, both of which sour qualities were perceived as more intense and less liked.

The results of facial expression and self-reported emotion have also highlighted sweet and sour tastes, particularly at higher concentrations. These taste qualities generally elicited negative emotions, both in terms of implicit and explicit measures. We found that the overall number of attributes reported by participants in the test group was less than the control group. It is known that the cerebral cortex and amygdala are smaller in volume among children with autism (Herbert et al., 2003), these regions are associated with processing, regulating and communicating emotions. Communication of emotions, thus, can be a significant difficulty in autism: communication of emotions can be a challenging task for them (Dennis et al., 2000).

These results suggest that similar emotions are not elicited by people with ASD as compared to others, which means that food appreciation might not be the same among the two groups. Emotions play a major role in food appreciation (Nederkoorn et al., 2000), and not being able to identify, express or feel these emotions elicited by food, would suggest differing food choices. Nevertheless, it should be noted that only a few facial expressions were different in the test group from the control group, while multiple self-reported emotions differed in both quantity and quality between the two groups. This suggests that the involuntary emotions elicited by both groups are similar, and facial expressions can prove to be a very useful tool in understanding perception and liking among people with ASD.

5. Conclusion

To summarize, we found that ASD affects taste abilities. Specifically, the taste identification ability of adults with autism was reduced. Increased sensitivity to sweet taste at low concentration and decreased liking of sour taste at a high concentration was noted. The majority of the ASD participants were tasters for PROP, which indicates that they might be generally more sensitive in terms of taste buds. Moreover, the emotions evoked by taste solutions differed among the two groups. People without ASD diagnosis reported a greater number of emotion attributes evoked by tastes, as compared to people with ASD. A higher number of negative emotions, both through implicit and explicit measures was noted by adults with ASD, for sour and sweet taste qualities. Thus, further investigation about the interactions of these taste qualities with the physiology of persons with ASD is recommended.

Table 5-1: Volumetric concentrations of basic taste solutions used in this study

	LOW INTENSITY	MEDIUM INTENSITY	HIGH INTENSITY
Caffeine	0.08%w/v	0.11% w/v	0.16% w/v
Sucrose	5% w/v	7% w/v	10% w/v
Sodium Chloride	0.35% w/v	0.44% w/v	0.55% w/v
Quinine Hydrochloride	0.004% w/v	0.006% w/v	0.008% w/v
Citric Acid	0.08% w/v	0.11% w/v	0.16% w/v

(Meilgaard et al. 2015)

Table 5-2: Comparisons between the ASD and control groups with respect to likings of the 10 taste stimuli

Stimulus	Control Group	ASD Group	U Value	P Value
Caffeine High	-25.4 (±24.8)	-13.2 (±45.2)	192.00	0.83
Caffeine Low	-5.8 (±20.9)	-8.3 (±39.7)	188.00	0.75
Sucrose High	37.7 (±26.4)	31.3 (±43.3)	196.50	0.93
Sucrose Low	26.7 (±24.9)	38.1 (±27.5)	152.50	0.20
Sodium Chloride High	-25.7(±26.7)	-18.3 (±40.5)	157.50	0.25
Sodium Chloride Low	-14.5 (±20.2)	-16.5 (±36.2)	186.50	0.72
Quinine High	-50.8 (±27.1)	-44.1 (±42.0)	183.50	0.66
Quinine Low	-43.7 (±28.4)	-48.1 (±35.0)	177.00	0.53
Citric Acid High	-2.8 (±34.4)	-22.9 (±42.8)	122.50	0.04
Citric Acid Low	4.3 (±25.5)	-12.6 (±44.6)	144.50	0.13

Values represent mean (± standard deviation)
ASD stands for Autism Spectrum Disorder.

Table 5-3: Comparisons between the ASD and control groups with respect to perceived intensity of the 10 taste stimuli

Stimulus	Control Group	ASD Group	U Value	P Value
Caffeine High	25.7 (±24.5)	33.7 (±24.5)	148.00	0.16
Caffeine Low	13.8 (±10.2)	21.5 (±23.1)	167.00	0.37
Sucrose High	40.6 (±15.0)	31.2 (±21.3)	117.50	0.03
Sucrose Low	34.2 (±17.4)	26.7 (±19.7)	140.50	0.11
Sodium Chloride High	36.2 (±20.7)	26.6 (±15.0)	144.00	0.13
Sodium Chloride Low	24.1 (±15.5)	19.4 (±15.3)	165.00	0.34
Quinine High	57.0 (±23.4)	46.5 (±24.2)	143.00	0.12
Quinine Low	52.0 (±26.7)	43.3 (±24.3)	159.50	0.27
Citric Acid High	30.9 (±16.7)	41.1 (±20.6)	135.00	0.08
Citric Acid Low	25.5 (±17.0)	30.9 (±19.6)	169.00	0.40

Values represent mean (± standard deviation)
ASD stands for Autism Spectrum Disorder.

Table 5-4: Comparisons between the ASD and control groups with respect to the number of emotional response terms reported on the EsSence25 scale

Stimulus	Control Group	ASD Group	U Value	P Value
Caffeine High	10.4 (±6.8)	5.9 (±6.6)	115.00	0.02
Caffeine Low	11.7 (±6.8)	5.3 (±5.9)	95.00	0.004
Sucrose High	15.9 (±4.4)	8.5 (±7.8)	92.50	0.004
Sucrose Low	15.2 (±5.7)	8.1 (±7.5)	91.00	0.003
Sodium Chloride High	11.8 (±6.8)	6.2 (±6.9)	102.00	0.008
Sodium Chloride Low	12.7 (±6.0)	6.2 (±7.3)	87.00	0.002
Quinine High	10.3 (±6.3)	3.9 (±3.8)	69.00	< 0.001
Quinine Low	11.1 (±6.8)	5.1 (±5.8)	88.00	0.002
Citric Acid High	12.5 (±6.4)	4.6 (±5.2)	58.50	< 0.001
Citric Acid Low	13.0 (±6.2)	4.5 (±4.7)	54.00	< 0.001

Values represent mean (± standard deviation)
ASD stands for Autism Spectrum Disorder.

Table 5-5: Comparisons between the ASD and control groups with respect to the intensity level of taste stimuli-evoked emotional responses reported on the EsSense25 scale

Emotion term	Control Group	ASD Group	U Value	P Value
Active	2.0 (±0.8)	1.5 (±0.9)	12326.00	< 0.001
Adventurous	1.8 (±0.9)	1.4 (±0.8)	13572.00	< 0.001
Aggressive	1.3 (±0.6)	1.3 (±0.9)	19603.50	0.60
Bored	1.4 (±0.8)	1.3 (±0.9)	16999.00	< 0.001
Calm	2.4 (±1.1)	1.6 (±1.0)	11287.00	< 0.001
Disgusted	1.9 (±1.3)	1.9 (±1.3)	19020.00	0.34
Enthusiastic	1.8 (±0.9)	1.4 (±0.9)	14657.00	< 0.001
Free	1.9 (±1.0)	1.4 (±0.9)	13408.50	< 0.001
Good	2.4 (±1.1)	1.7 (±1.1)	12325.00	< 0.001
Good-natured	2.3 (±1.2)	1.5 (±0.9)	11524.00	< 0.001
Guilty	1.1 (±0.5)	1.2 (±0.7)	19946.50	0.92
Happy	2.2 (±1.2)	1.5 (±1.0)	13302.50	< 0.001
Interested	2.4 (±1.0)	1.8 (±1.1)	12850.50	< 0.001
Joyful	2.0 (±1.1)	1.4 (±1.0)	14193.00	< 0.001
Loving	1.8 (±0.9)	1.4 (±0.9)	13799.50	< 0.001
Mild	2.0 (±1.0)	1.4 (±0.8)	12382.50	< 0.001
Nostalgic	1.4 (±0.8)	1.2 (±0.7)	17683.50	0.003
Pleasant	2.1 (±1.1)	1.4 (±0.9)	12674.00	< 0.001
Satisfied	1.9 (±1.1)	1.4 (±0.9)	14783.00	< 0.001
Secure	2.1 (±1.2)	1.4 (±0.9)	13143.50	< 0.001
Tame	2.0 (±1.1)	1.3 (±0.8)	12418.00	< 0.001
Understanding	2.1 (±1.1)	1.4 (±0.9)	12518.50	< 0.001
Warm	1.9 (±1.0)	1.5 (±1.0)	13991.50	< 0.001
Wild	1.4 (±0.7)	1.2 (±0.8)	17283.50	< 0.001
Worried	1.3 (±0.6)	1.3 (±0.8)	19329.50	0.39

Values represent mean (± standard deviation)
ASD stands for Autism Spectrum Disorder.

Table 5-6: Comparisons between the ASD and control groups with respect to facially expressed emotions to the 10 taste stimuli

Taste Quality	Facial Expression	Control Group	ASD Group	U Value	P value
Caffeine High	Joy	-0.5 (±1.9)	-0.1 (±1.7)	164.00	0.33
	Anger	0.5 (±0.7)	0.6 (±1.3)	161.00	0.29
	Surprise	-0.3 (±0.7)	-0.4 (±0.9)	175.00	0.50
	Fear	-0.2 (±0.6)	-0.3 (±0.8)	187.00	0.73
	Contempt	-0.2 (±0.5)	-0.2 (±0.6)	200.00	1.00
	Disgust	0.4 (±1.0)	0.3 (±1.0)	171.00	0.43
	Sadness	0.2 (±0.4)	0.4 (±0.7)	163.00	0.32
Caffeine Low	Joy	-1.2 (±1.8)	-0.8 (±2.0)	164.00	0.33
	Anger	0.7 (±0.8)	0.8 (±1.1)	159.00	0.27
	Surprise	-0.1 (±0.7)	-0.3 (±1.1)	162.00	0.30
	Fear	-0.2 (±0.5)	-0.3 (±0.4)	146.00	0.14
	Contempt	-0.1 (±0.6)	-0.4 (±0.6)	158.00	0.26
	Disgust	0.4 (±0.8)	0.3 (±1.1)	196.00	0.91
	Sadness	0.5 (±0.7)	0.5 (±0.7)	180.00	0.59
Sucrose High	Joy	-0.8 (±1.3)	-0.7 (±2.6)	165.00	0.34
	Anger	0.3 (±0.6)	0.6 (±1.4)	178.00	0.55
	Surprise	-0.1 (±0.5)	0.1 (±1.0)	152.00	0.19
	Fear	-0.1 (±0.3)	-0.2 (±0.8)	200.00	1.00
	Contempt	-0.3 (±0.3)	-0.0 (±0.7)	123.00	0.04
	Disgust	0.2 (±0.5)	0.0 (±1.1)	187.00	0.73
	Sadness	0.3 (±0.5)	0.9 (±1.1)	115.00	0.02
Sucrose Low	Joy	-0.7 (±1.2)	-0.6 (±2.8)	171.00	0.43
	Anger	0.3 (±0.6)	0.4 (±1.3)	196.00	0.91
	Surprise	-0.1 (±0.6)	-0. 3 (±1.0)	173.00	0.47
	Fear	-0.1 (±0.4)	-0.4 (±0.9)	161.00	0.29
	Contempt	-0.1 (±0.5)	-0.2 (±0.7)	190.00	0.79
	Disgust	0.3 (±0.7)	-0.2 (±1.3)	172.00	0.45
	Sadness	0.3 (±0.5)	0.5 (±0.9)	181.00	0.61

Table 5-6 (Cont.)

Taste Quality	Facial Expression	Control Group	ASD Group	U Value	P value
Sodium Chloride High	Joy	-0.6 (±1.5)	-1.0 (±3.0)	196.00	0.91
	Anger	0.6 (±0.4)	0.5 (±1.0)	172.00	0.45
	Surprise	-0.2 (±0.8)	-0.3 (±1.0)	188.00	0.75
	Fear	-0.1 (±0.7)	-0.5 (±0.6)	147.00	0.15
	Contempt	-0.2 (±0.6)	-0.4 (±0.8)	167.00	0.37
	Disgust	0.5 (±1.1)	0.1 (±1.2)	161.00	0.29
	Sadness	0.4 (±0.6)	0.3 (±0.6)	199.00	0.98
Sodium Chloride Low	Joy	-0.8 (±1.4)	-1.0 (±2.6)	184.00	0.67
	Anger	0.6 (±0.9)	0.6 (±1.3)	186.00	0.71
	Surprise	-0.3 (±0.6)	-0.1 (±0.7)	161.00	0.29
	Fear	-0.2 (±0.6)	-0.2 (±0.8)	194.00	0.87
	Contempt	-0.2 (±0.7)	-0.3 (±0.8)	193.00	0.85
	Disgust	-0.0 (±1.0)	0.1 (±1.1)	185.00	0.69
	Sadness	0.2 (±0.5)	0.5 (±0.7)	158.00	0.26
Quinine Hydrochloride High	Joy	-0.4 (±1.6)	-0.3 (±2.1)	177.00	0.53
	Anger	0.3 (±0.9)	0.8 (±1.0)	149.00	0.17
	Surprise	-0.5 (±0.8)	-0.3 (±1.1)	196.00	0.91
	Fear	0.0 (±0.6)	-0.0 (±1.0)	184.00	0.67
	Contempt	-0.3 (±0.7)	-0.3 (±0.8)	193.00	0.85
	Disgust	0.6 (±1.1)	0.4 (±1.4)	190.00	0.79
	Sadness	0.4 (±0.6)	0.6 (±0.9)	168.00	0.39
Quinine Hydrochloride Low	Joy	-0.3 (±1.6)	-0.7 (±2.0)	195.00	0.89
	Anger	0.6 (±0.7)	0.8 (±0.9)	173.00	0.47
	Surprise	-0.5 (±0.7)	-0.4 (±1.0)	193.00	0.85
	Fear	-0.2 (±0.7)	-0.3 (±0.9)	181.00	0.61
	Contempt	-0.3 (±0.5)	-0.5 (±0.6)	169.00	0.40
	Disgust	0.5 (±0.7)	0.6 (±0.9)	161.00	0.29
	Sadness	0.3 (±0.6)	0.6 (±1.0)	158.00	0.26

Table 5-6 (Cont.)

Taste Quality	Facial Expression	Control Group	ASD Group	U Value	P value
Citric Acid High	Joy	-0.8 (±1.5)	-0.3 (±2.1)	174.00	0.48
	Anger	0.2 (±0.5)	0.5 (±0.9)	167.000	0.37
	Surprise	0.2 (±0.7)	-0.3 (±0.9)	292.000	0.01
	Fear	-0.0 (±0.5)	-0.0 (±0.7)	207.00	0.85
	Contempt	-0.2 (±0.7)	-0.3 (±0.7)	216.00	0.67
	Disgust	0.3 (±0.7)	0.3 (±1.0)	186.000	0.71
	Sadness	0.3 (±0.6)	0.6 (±0.6)	119.000	0.03
Citric Acid Low	Joy	-1.2 (±1.6)	-0.7 (±2.1)	148.00	0.16
	Anger	0.5 (±0.8)	0.75 (±1.0)	159.00	0.27
	Surprise	-0.0 (±0.7)	-0.3 (±0.9)	191.00	0.81
	Fear	-0.2 (±0.5)	-0.3 (±0.8)	169.00	0.40
	Contempt	-0.2 (±0.6)	-0.2 (±0.7)	197.00	0.94
	Disgust	0.2 (±0.6)	0.3 (±1.0)	200.00	1.00
	Sadness	0.4 (±0.6)	0.5 (±0.7)	181.00	0.61

Values represent mean (± standard deviation)
ASD stands for Autism Spectrum Disorder.

REFERENCE

Bartoshuk, L. M., Duffy, V. B., & Miller, I. J. (1994). PTC/PROP tasting: anatomy, psychophysics, and sex effects. Physiology & Behavior, 56, 1165-1171.

Bartoshuk, L. M., Duffy, V. B., Green, B. G., Hoffman, H. J., Ko, C. W., Lucchina, L. A., ... & Weiffenbach, J. M. (2004). Valid across-group comparisons with labeled scales: the gLMS versus magnitude matching. *Physiology & behavior*, *82*(1), 109-114.

Bennetto, L., Kuschner, E. S., & Hyman, S. L. (2007). Olfaction and taste processing in autism. Biological Psychiatry, 62, 1015-1021.

Cole, N. C., Wang, A. A., Donovan, S. M., Lee, S. Y., & Teran-Garcia, M. (2017). Variants in chemosensory genes are associated with picky eating behavior in preschool-age children. *Lifestyle Genomics*, *10*(3-4), 84-92.

Damiano, C. R., Aloi, J., Burrus, C., Garbutt, J. C., Kampov-Polevoy, A. B., & Dichter, G. S. (2014). Intact hedonic responses to sweet tastes in autism spectrum disorder. *Research in autism spectrum disorders*, *8*(3), 230-236.

Dennis, M., Lockyer, L., & Lazenby, A. L. (2000). How high-functioning children with autism understand real and deceptive emotion. *Autism*, *4*(4), 370-381.

Duffy, V. B., & Bartoshuk, L. M. (2000). Food acceptance and genetic variation in taste. *Journal of the American Dietetic Association*, *100*(6), 647-655.

Ghanizadeh, A. (2010). Possible role of caffeine in autism spectrum disorders, a new testable hypothesis. *Journal of food science*, *75*(6), ix-ix.

Herbert, M. R., Ziegler, D. A., Deutsch, C. K., O'brien, L. M., Lange, N., Bakardjiev, A., ... & Kennedy, D. (2003). Dissociations of cerebral cortex, subcortical and cerebral white matter volumes in autistic boys. *Brain*, *126*(5), 1182-1192.

iMotions. (2018). https://imotions.com/facial-expressions/accessed on March 20,2018

King, S. C., & Meiselman, H. L. (2010). Development of a method to measure consumer emotions associated with foods. Food Quality and Preference, 21, 168-177.

Lim, J., & Fujimaru, T. (2010). Evaluation of the labeled hedonic scale under different experimental conditions. *Food quality and preference*, *21*(5), 521-530.

Macdonald, H., Rutter, M., Howlin, P., Rios, P., Conteur, A. L., Evered, C., & Folstein, S. (1989). Recognition and expression of emotional cues by autistic and normal adults. Journal of Child Psychology and Psychiatry, 30, 865-877.

Meilgaard, M. C., Civille, G. V., & Carr, B. T. (2015). Sensory Evaluation Techniques (5th ed.). Boca Raton: CRC Press, (Chapter 12).

Muratori, F., Tonacci, A., Billeci, L., Catalucci, T., Igliozzi, R., Calderoni, S., & Narzisi, A. (2017). Olfactory processing in male children with autism: atypical odor threshold and identification. *Journal of autism and developmental disorders*, *47*(10), 3243-3251.

Nederkoorn, C., Smulders, F. T. Y., & Jansen, A. (2000). Cephalic phase responses, craving and food intake in normal subjects. Appetite, 35, 45–55.

Nemiah, J., Freyberger, H., & Sifneos, P. E. (1976). Alexithymia: a view of the psychosomatic process. In, OW Hill (1976) Modern Tends in Psychosomatic Medicine 3: 430–439.

Nestrud, M. A., Meiselman, H. L., King, S. C., Lesher, L. L., & Cardello, A. V. (2016). Development of EsSense25, a shorter version of the EsSense Profile®. *Food Quality and Preference*, *48*, 107-117.

Riccio, M. P., Franco, C., Negri, R., Ferrentino, R. I., Maresca, R., D'alterio, E., ... & Bravaccio, C. (2018). Is food refusal in autistic children related to TAS2R38 genotype?. *Autism Research*, *11*(3), 531-538.

Robino, A., Mezzavilla, M., Pirastu, N., La Bianca, M., Gasparini, P., Carlino, D., & Tepper, B. J. (2016). Understanding the role of personality and alexithymia in food preferences and PROP taste perception. Physiology & Behavior, 157, 72-78.

Samant, S. S., Chapko, M. J., & Seo, H. S. (2017). Predicting consumer liking and preference based on emotional responses and sensory perception: A study with basic taste solutions. *Food research international*, *100*, 325-334.

Tavassoli, T., & Baron-Cohen, S. (2012). Taste identification in adults with autism spectrum conditions. Journal of Autism and Developmental Disorders, 42, 1419-1424.

Trivedi, B. P. (2012). Gustatory system: The finer points of taste. Nature, 486(7403), S2-S3.

CHAPTER 6

GENERAL CONCLUSION

To summarize, chapter 3 found that people with ASD have atypical and uneven sensory experiences, which can be either hypersensitivity or hyposensitivity to sensory stimuli. It was found that several participants were hyperreactive to one sensory stimulus while being hyporeactive to the other. Chapters 4 and 5 showed that participants with ASD had reduced abilities to identify taste and odor stimuli, but most participants in the ASD group were PROP tasters, as well as rated certain taste and odor stimuli as highly intense. Specifically, the sour quality was found to be very intense for persons with autism, which was confirmed in chapters 3, 4 and 5. It was also found that food choice in autism is driven by environmental factors, taste, smell, texture, appearance, and sound. Additionally, emotions evoked by taste stimuli were reduced for them, which could contribute to their food choice, paired with increased sensitivity. Odor discrimination and identification ability of people with ASD were found to be reduced, which confirms the results of previous studies, along with suggesting that odor identification ability might reduce with age for persons with ASD, but not for the control group.

In conclusion, this study attempted to study taste and smell abnormalities in autism, and how they are affected by it. This study confirmed some previously known results and found some new insight into this arena. We were able to study and report variances in food evoked emotions among people with and without ASD, through implicit and explicit measures. This study was one of the few that used an extensive number of subjects and controls. The researchers realize that this study might have some limitations. First, the asymmetric development of adult participants with ASD might have influenced some results. Second, the small sample size makes it difficult to see trends or significance. However, for practical reasons like few volunteers wanting to take part in such studies, it was difficult to recruit more panelists.

APPENDICES

APPENDIX 1

RESEARCH COMPLIANCE PROTOCOL LETTERS

UNIVERSITY OF ARKANSAS

To:	Han-Seok Seo FDSC N-215
From:	Douglas James Adams, Chair IRB Committee
Date:	05/08/2018
Action:	**Expedited Approval**
Action Date:	05/08/2018
Protocol #:	1804116930
Study Title:	Sensory and emotional responses to sensory cues
Expiration Date:	04/19/2019
Last Approval Date:	

The above-referenced protocol has been approved following expedited review by the IRB Committee that oversees research with human subjects.

If the research involves collaboration with another institution then the research cannot commence until the Committee receives written notification of approval from the collaborating institution's IRB.

It is the Principal Investigator's responsibility to obtain review and continued approval before the expiration date.

Protocols are approved for a maximum period of one year. You may not continue any research activity beyond the expiration date without Committee approval. Please submit continuation requests early enough to allow sufficient time for review. Failure to receive approval for continuation before the expiration date will result in the automatic suspension of the approval of this protocol. Information collected following suspension is unapproved research and cannot be reported or published as research data. If you do not wish continued approval, please notify the Committee of the study closure.

Adverse Events: Any serious or unexpected adverse event must be reported to the IRB Committee within 48 hours. All other adverse events should be reported within 10 working days.

Amendments: If you wish to change any aspect of this study, such as the procedures, the consent forms, study personnel, or number of participants, please submit an amendment to the IRB. All changes must be approved by the IRB Committee before they can be initiated.

You must maintain a research file for at least 3 years after completion of the study. This file should include all correspondence with the IRB Committee, original signed consent forms, and study data.

cc: Tonya Sue Priesmeyer Tokar, Investigator
Asmita Singh, Investigator

APPENDIX 2

IMPORTANCE OF OLFACTION QUESTIONNAIRE

		I totally agree	**I mostly agree**	**I mostly disagree**	**I totally disagree**
1	The smell of a person plays a role in the decision whether I like him/her.				
2	I smell foods to find out whether it is spoiled or not.				
3	I sniff on food before eating.				
4	Please imagine you visit a museum. There is an offer to get additionally smell-presentations to underline the overall impression for the price of $5.00. Would you take this offer?				
5	When I don't like the smell of a shampoo, I don't buy it.				
6	When I smell delicious food, I get hungry.				
7	Without my sense of smell, life would be worthless.				
8	I try to locate the odor, when I smell something.				
9	I feel rather quickly disturbed by odors in my environment.				
10	Certain smells immediately activate numerous memories.				
11	Before drinking coffee/tea, I intentionally smell it.				
12	When I buy tomatoes, I pay attention to their odor.				
13	If my partner has a nasty smell, I avoid kissing him/her.				
14	Certain smells immediately activate strong feelings.				
15	I smell my clothes to judge whether I have to wash them or not.				

APPENDIX 2 (Cont.)

16	When there is a nasty smell in the office/apartment of a colleague, I leave the room as soon as possible.				
17	Certain odors can stimulate my fantasy.				
18	To me it is more important to be able to smell than to be able to see or hear.				
19	Sometimes I smell a person (e.g. my partner or my child) to judge, if he/she has had alcohol or smoked.				
20	I cannot pass good smelling candles in a store without buying one.				