

IMPAIRED CLASSICAL CONDITIONING IN PERSONS WITH AUTISM SPECTRUM
DISORDERS

by

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ABSTRACT

Previous research has indicated that individuals with autism spectrum disorder (ASD) have difficulty with implicit learning; learning that occurs unconsciously and without intention. The present study utilized a classical fear conditioning paradigm to examine associative learning in individuals with ASD, which can be considered to be a simple form of implicit learning. Fifteen individuals diagnosed with ASD and 16 age, gender, and IQ-matched individuals with typical development participated in this study. Both participants with ASD and typical development were presented with a series of colors and sounds paired with an aversive loud noise while skin conductance responses (SCRs) were recorded. Following this task, an explicit memory test probed participants' awareness of the learning contingencies. Results from this study found that individuals with ASD demonstrate a general impairment in associative learning compared to individuals with typical development. Additionally, greater explicit awareness of the learning contingencies was related to greater associative learning in individuals with ASD. Implications for theories regarding associative and implicit learning impairments in ASD, such as the underconnectivity theory of autism and the Learning Compensation model are discussed.

DEDICATION

This manuscript is dedicated to my parents, my biggest supporters.

LIST OF ABBREVIATIONS AND SYMBOLS

<i>M</i>	Mean: the sum of a set of measurements divided by the number of measurements in the set
<i>SD</i>	Standard Deviation
<i>p</i>	Probability associated with the occurrence under the null hypothesis of a value as extreme as or more extreme than the observed value
<i>r</i>	Pearson product-moment correlation
<i>t</i>	Computed value of <i>t</i> test
SCL	Skin conductance level
SCR	Skin conductance response
CS+	Conditioned stimulus paired with the UCS
CS _{unpaired} ⁺	Conditioned stimulus presented without the pairing of the UCS
CS-	Neutral stimuli presented never paired with the UCS

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Impaired Classical Conditioning in Persons with Autism Spectrum Disorders

Autism is a developmental disorder usually diagnosed in early childhood. This disorder is typically defined by impairments in social and communication skills, abnormal language development, and a restricted range of behaviors and interests (American Psychiatric Association, 2000). Autism Spectrum Disorders (ASD) encapsulate varying degrees of these core symptoms, and includes diagnoses of autism, Asperger's Syndrome (AS), and pervasive developmental disorder not otherwise specified (PDD-OS). Many theories of ASD focus on explanations of social and communicative impairments (Baron-Cohen, 1997). However, there are other theories positing that the observed social impairments in ASD are caused by underlying cognitive impairments. Several studies, examining implicit learning, have shown that children with autism may have a basic impairment in implicit learning (Klinger & Dawson, 2001; Klinger, Klinger, & Pohlig, 2007). Klinger et al., (2007) suggested that abnormal development in ASD may lead to a basic cognitive impairment in implicit learning, which leads them to not learn about natural relationships in the world thereby manifesting into behavioral symptoms (e.g., communication impairments, repetitive and restricted behaviors).

Other theories of ASD have examined these cognitive impairments from a neurological perspective. The underconnectivity theory (Just, Cherkassky, Keller, & Minshew, 2004; Kana, Keller, Cherkassky, Minshew, & Just, 2006) suggests that the cognitive and social impairments observed in ASD are the result of connectivity differences between functional brain regions. Specifically, the underconnectivity theory posits that individuals with ASD may suffer from a dysfunction in the way areas of the brain communicate with one another. However, the

underconnectivity theory also theorizes that the cognitive strengths often found in ASD, such as hyperlexia, may be the result of the brains of persons with ASD compensating for a lack of integration by strengthening independent and free-standing brain structures (Just et al., 2004).

Also, abnormal neurological development in the amygdala has been suggested as another possible source of impairments (Baron-Cohen, 2000). In fact, one study has demonstrated impaired associative learning for individuals with ASD in a classical fear conditioning paradigm (Gaigg & Bowler, 2007). This study examined the abnormal production of fear in individuals with ASD which was thought to be the result of an abnormally functioning amygdala. Therefore, the current study aims to expand upon previous evidence of abnormal fear production in individuals with ASD at its relation to abnormal amygdala functioning and underlying cognitive impairments.

The amygdala theory of autism

Brothers (1990) proposed a network of neural regions that are heavily involved in social cognition, and identified the amygdala as an essential component of this neural network due to its role in emotional processing, and particularly fear processing. Bauman and Kemper (1985) observed abnormalities in the amygdala of individuals with ASD. Considering that social impairment is hallmark of ASD, these findings led Baron-Cohen et al. (2000) to propose an amygdala theory of autism in which they theorized that observed social impairments in ASD may be caused by an amygdala abnormality. Evidence for this theory has stemmed from studies examining the social behavior of monkeys after lesions to the amygdala (Kling & Brothers, 1992; Kling & Steklis, 1976). These studies demonstrated that amygdala-lesioned monkeys become more socially isolated as well as fail to initiate social interactions and respond appropriately to non-verbal gestures (Kling & Brothers, 1992; Kling & Steklis, 1976).

Following these animal lesion studies, Baron-Cohen et al. (1999) conducted a functional magnetic resonance imaging (fMRI) study to examine amygdala functioning in persons with ASD. Participants in this study were shown several pictures of men and women and asked to infer the mental states of the person in the photograph. Results from this study indicated that individuals with typical development displayed activation within the amygdala presumably due to the emotional processing involved. However, individuals with ASD show no activation within the amygdala. Interestingly, the ASD group tended to display greater activation in the temporal lobe structures, and therefore placed greater emphasis on verbal labeling of the photographs rather than inferring emotional processes. This finding suggests that individuals with ASD may engage in a unique type of emotional processing in order to compensate for abnormal amygdala functioning. Additionally, this finding also suggests that impaired amygdala functioning may result in abnormal fear acquisition in individuals with ASD.

Implicit Learning

Implicit learning refers to learning that occurs without awareness and or intention to learn. In contrast, explicit learning is an effortful and controlled process involving conscious awareness of what is being learned. The mental processes involved in implicit learning are often difficult to verbalize or explain in concrete terms (Reber, 1989). Previous studies have demonstrated that the development of implicit learning is largely independent of age and intelligence, whereas explicit learning is heavily dependent upon a person's age and intelligence (Atwell, Conners, & Merrill, 2003; Huang-Pollock, Maddox, & Karaluns, 2011; Weinert, 2009).

Additionally, implicit learning has been linked to language acquisition (Saffran, 1996) and social understanding (Heerey & Velani, 2010), which are amongst the most impaired areas in ASD. Previous studies examining implicit statistical learning through artificial grammar tasks

have found that typically developing infants are capable of discriminating between novel and familiar non-word pairings (Saffran, 1996) and grammatical and ungrammatical word strings (Gomez & Gerken, 1999). These results imply implicit understanding of the rules of language allowing children to communicate well before they are able to explicitly verbalize grammatical rules. Furthermore, implicit learning is considered important for understanding the unspoken rules elicited in social interactions. Implicit learning allows the subtleties of nonverbal behavior to become understandable in a social situation without having to be explicitly aware of these rules. In other words, implicit learning may give rise to much of people's social intuition (Libermann, 2000). Therefore, it is believed that the impairments in social and language communication typically observed in people with ASD may be related to impairments in implicit learning.

Learning compensation model.

Evidence for implicit learning impairments in ASD were first observed through implicit learning tasks such as prototype and artificial grammar tasks (Klinger & Dawson, 2001; Klinger et al., 2007). The prototype task is a category-learning task in which participants learn a category for which there are no rules of membership. Most people learn these categories by mentally averaging the examples they have seen (i.e. by forming a "typical" example or prototype). Once this prototype is formed, participants can then categorize new examples as a member of a category based upon whether the example closely matches the prototype (Posner & Keele, 1968). Klinger & Dawson (2001) used a prototype task involving cartoon animals to assess implicit learning impairments for children with autism and Down Syndrome. Participants in this study consisted of 12 individuals with autism, Down syndrome, and typical development (TD) that ranged in age from five years 10 months to 21 years three months. During this

experiment participants were exposed to eight different versions of a cartoon animal during the familiarization trial. Then on the test trial participants were asked to choose between two different cartoon animals based on similarity with a previously defined category (i.e., “Is this animal a member of this category?”). Klinger and Dawson found that typically developing children performed well above chance, correctly categorizing the cartoon animals 79% of trials. However, children with autism and Down Syndrome group successfully categorized the cartoon animals only 54% and 42% of trials, respectively. These results demonstrate that neither the ASD group nor Down Syndrome performed at levels that were different from chance, indicating a failure to form a prototype representation of the cartoon animal. However, it is interesting to note that when the autism group was given explicit rules on how to categorize the cartoon animal, their performance matched that of the typically developing group; which suggests that explicit learning is intact in individuals with ASD.

Although Klinger and Dawson (2001) demonstrated impaired implicit learning in an ASD population, other studies have shown intact prototype learning in ASD. For example, Molesworth, Bowler, and Hampton (2005) demonstrated that higher functioning individuals with ASD display intact prototype learning. In their study they used a prototype task similar to Klinger and Dawson and found that higher functioning individuals with ASD had intact prototype formation. However, there were key participant and methodological differences between Molesworth et al. and Klinger and Dawson. First, participants in Molesworth, et al.’s study were individuals with higher functioning ASD compared to Klinger and Dawson, whose participants had lower mental ages and greater intellectual disability. Therefore, the participants in Molesworth et al. may have been better at the task simply because of greater intelligence or mental age. Another difference between these two studies was that Molesworth et al. gave

participants a recall task asking if they had previously seen the cartoon animal, as opposed to Klinger and Dawson's study, which gave participants a forced-choice comparison between two cartoon animals. In an attempt to explain these contrasting findings, Klinger et al., (2007) postulated that higher functioning individuals may have higher verbal intelligence, thereby allowing them to engage in more explicit verbal processing for the prototype compared to lower functioning individuals with ASD.

To test the differences in methodology and participants between Molesworth et al. (2005) and Klinger and Dawson (2001), Klinger et al., (2007) conducted several studies to examine whether individuals with ASD demonstrate impairments in implicit learning, and whether higher functioning individuals engage in more explicit processing to compensate for these implicit deficits. In one study, participants were given the prototype task used by Klinger and Dawson in addition to an artificial grammar task (Reber, 1989) as a measure of implicit learning. In the artificial grammar task, participants were shown a sequence of shapes generated by a Markovian grammar chain, which applies a specific set of rules for the order in which the shapes must appear to conform to rules regulating valid sentence construction (Reber, 1989). During the familiarization trials, participants were shown a sequence of shapes, but were not aware that the sequence follows a set of rules. After the familiarization trials, participants were asked to judge which sequences looked most like the ones they previously saw. Participants typically choose the sequence of shapes that adhere to the grammatical rules compared to the sequences that contain a rule violation. This choice demonstrates that although participants cannot explicitly describe why they choose a particular sequence, they have learned the rules of proper sequence construction implicitly. The results from this study revealed that typically developing

participants performed significantly better in both the prototype and artificial grammar tasks than individuals with ASD, providing a replication of the results found in Klinger and Dawson.

Subsequent studies using implicit learning paradigms have found that higher functioning individuals with ASD perform equally as well as typically developing individuals (Barnes et al., 2008), which supports the findings of Moleswoth et al. (2005). Examining the results from Klinger et al., (2007) also revealed a strong relationship between mental age and implicit learning. In other words, younger individuals with ASD tended to display poorer implicit learning, whereas older individuals with ASD display similar levels of implicit learning as the typically developing group. This indicates that unlike individuals with typical development that demonstrate explicit and implicit learning independent from one another, individuals with ASD may recruit similar mental processes for both explicit and implicit learning. In other words, children with ASD may compensate for impairment in implicit learning by using explicit learning strategies (Klinger et al., 2007). Although these results suggest a relationship between explicit reasoning and prototype formation in individuals with ASD, it is unclear whether this relationship is observed in more simplistic forms of implicit learning such as the autonomic response developed during an associative learning task. Therefore, the current study examined whether explicit awareness of an associative learning paradigm was related to the autonomic responses of a conditioned stimulus. According to the learning compensation model, if individuals with ASD rely on explicit strategies when presented with an associative learning task, then we might expect more robust associative learning to be related to greater explicit awareness of the learning contingencies.

Associative Learning

It could be argued that one of the most basic forms of implicit learning is associative learning, specifically classical conditioning. Classical conditioning is an excellent example of implicit learning because it quite often entails an absence of awareness of what is being learned (Knight, Nguyen, & Bandettini, 2003). For example, many classical conditioning paradigms use an aversive stimulus (e.g., puff of air or blast of white noise) as the unconditioned stimulus (UCS) which in turn elicits an unconditioned startle response (UCR). During conditioning a relatively neutral stimulus (e.g., tone or red light) is paired with the UCS resulting in a learned association between the neutral conditioned stimulus (CS) and the UCS. After several pairings of the CS with the UCS, which elicits an UCR, participants begin to associate the CS with the UCR (e.g., fear). If participants demonstrate a fear response after the presentation of the CS, then it is assumed that the participant has developed a learned association between the CS and the UCR (Knight, Smith, Stein, & Helmstetter, 1999; Knight, Nguyen, & Bandettini, 2003). Because learning is simple and can occur largely outside the awareness of participants' conscious memory, classical conditioning provides an excellent process in which to explore a more basic implicit learning impairment in ASD.

Associative learning in ASD.

To date only two studies have examined classical fear conditioning in ASD. Bernier, Dawson, Panagiotides, and Webb (2005) used a classical fear conditioning paradigm in which they paired colored squares with an aversive puff of air. Participants consisted of individuals with ASD and individuals with typically development (TD) group aged 12 to 45 years. During the conditioning phase, participants were presented with only one visual-auditory pairing that consisted of a red colored square with an aversive airpuff displayed 20 times. The testing phase

consisted of six additional CS-UCS pairings to maintain learning, 12 presentations of an acoustic startle stimulus considered to be the “safe” condition, and 12 presentations of a CS and acoustic startle pairing considered to be the “threat” condition. The “safe” condition indicated to the participants the absence of the UCS, whereas the “threat” condition indicated the potential presence of the UCS. Participants’ startle responses were measured by eyeblink latency and magnitude using electromyographic (EMG) activity of the orbicularis oculi muscle. Bernier et al. (2005) found no group differences in the latency or amplitude of potentiated startle response suggesting that the ASD group does not display an atypical pattern of associative learning.

In contrast, Gaigg and Bowler (2007) found atypical fear discrimination in a classical fear conditioning paradigm. Both an ASD group and a typically developing group were shown colored squares in which one color was paired with an aversive sound (e.g., foghorn). Participants’ fear responses were measured by their skin conductance response (SCR) in the presence or absence of the aversive sound. Gaigg and Bowler found that the ASD group demonstrated attenuated fear learning to the conditioned stimulus. These results demonstrated that individuals with ASD may require more trials to learn the pairing between the conditioned and unconditioned stimulus.

Although Bernier et al. (2005) and Gaigg and Bowler (2007) found conflicting findings, several methodological differences may explain their findings. First, one difference was the particular paradigms used by Bernier et al. and Gaigg and Bowler. Bernier et al. employed a simple conditioning paradigm where only one CS was used, compared to Gaigg and Bowler’s use of a CS mixed with other neutral stimuli. The relative complexity of Gaigg and Bowler’s paradigm may have contributed to the difficulty of the ASD group distinguishing the CS from the other neutral stimuli. Another difference between these two studies was that Bernier et al.

used a 100% reinforcement schedule where the CS was always presented with the UCS. Gaigg and Bowler used a 50% reinforcement schedule in order to measure the difference in physiological response to the CS presented with the UCS and the CS presented without the UCS which, according to Gaigg and Bowler, resulted in the CS being a less reliable cue for the presence of UCS. Finally, Bernier et al. exposed participants to the CS for 2000 ms before the pairing of the UCS, whereas Gaigg and Bowler exposed participants to a 1000 ms presentation before the UCS pairing. The combination of the shorter presentation, along with the lower reinforcement schedule and the addition of neutral stimulus presentations may have limited participant's ability to consciously recognize the relationship between the CS and UCS, thereby making learning more difficult. The relative simplicity of the methodology in Bernier et al. may have made it easier for participants to explicitly learn the relationship between the CS and UCS. In other words, the ASD group in Bernier et al. may have been consciously aware of the relationship between CS and UCS. The possibility of greater explicit awareness in Bernier et al. may have contributed to the typical responses observed in the ASD group.

Ultimately Gaigg and Bowler's paradigm seems to be a more implicit measure of associative learning resulting in their observed impairment in implicit learning among an ASD population. Furthermore, Gaigg and Bowler demonstrated that the underlying impairment observed may suggest poor connectivity between regions commonly associated with a typical fear response such as the amygdala and cortical areas of the brain, which is the basis for the underconnectivity theory (Just et al., 2004).

The Underconnectivity Theory

The underconnectivity theory posits that individuals with ASD demonstrate dysfunctional integration within the circuitry involved in cognitive, perceptual and motor abilities (Just et al.,

2004). Moreover, this theory suggests that individuals with ASD lack general connectivity throughout the entire brain compared to typically developing peers. This lack of connectivity explains why individuals with ASD perform poorly on higher order cognitive tasks because a high degree of coordination and integration of different brain regions is needed for such tasks. Evidence for this theory has come from fMRI studies in which the brains of individuals with TD and individuals with ASD were compared during a variety of tasks (Just et al., 2004; Kana et al., 2006). In one of these studies, Just et al. found that individuals with ASD, compared to individuals with TD, not only performed poorly on a complex sentence task, but also showed less functional connectivity between ten regions of interest (ROIs) commonly associated with language processing. These results suggest reduced synchronization and integration of language information in persons with ASD. Additionally, Just et al. (2007) found that individuals with ASD not only demonstrated less functional connectivity within language areas, but also less functional connectivity between brain regions involved in an executive functioning task. These findings suggest reduced functional connectivity across several brain regions, and that these alterations in cortical connectivity may be a pervasive processing deficit in individuals with ASD.

The underconnectivity theory also provides a neurological explanation for the deficits observed in implicit learning tasks such as the artificial grammar task. This theory would explain that the difficulty individuals with ASD have in distinguishing between artificial sequences of grammatical versus ungrammatical strings may be due to an inability to integrate various different types of language-based processing. It is possible to hypothesize that due to underconnectivity, individuals with ASD may have had difficulty integrating the various language-based processing components necessary to identify grammatical versus ungrammatical

strings. However, Just et al. (2004) has also argued that though there is diminished interaction between different brain areas in ASD, persons with ASD adapt by developing and strengthening independent and free-standing brain structures. Therefore, we might expect that learning and perception that occurs within one modality (e.g., audition) might actually be better in individuals with ASD compared to individuals with TD. This prediction is consistent with Smith and Bennetto (2007) and Taylor et al. (2010) who found that individuals with ASD were less susceptible to the McGurk illusion, in which the perception of a speech sound is influenced by the simultaneous presentation of incongruent visual speech articulation, suggesting deficits in audio-visual integration. However, both of these studies found no diagnostic group differences for auditory accuracy, suggesting preserved auditory perception.

Both the learning compensation model and the underconnectivity theory can provide explanations for impaired implicit learning in ASD. However, neither the learning compensation model nor the underconnectivity theory has been directly applied to classical fear conditioning paradigms in an ASD population. Similarly, associative learning and classical conditioning can be seen as perhaps the most basic form of implicit learning, so impairments in associative fear learning could be caused by either abnormal amygdala functioning or a more broad implicit learning impairment. What is interesting about classical fear conditioning paradigms is that no explicit awareness of the learned association is required. Although we cannot rule out the possibility that explicit processes are engaged in a fear conditioning paradigm, by recording physiological reactions to aversive stimuli we can compare relatively automatic or implicit learning processes to more controlled or explicit learning processes. Accordingly, the underconnectivity theory could also provide an explanation for abnormal fear acquisition since most fear conditioning studies use different modalities (i.e., visual and auditory) as the

conditioned stimulus (e.g., a yellow square) and the unconditioned stimulus (e.g., a loud foghorn). However, the Learning Compensation model would predict that individuals with ASD might display similar associative learning patterns as compared to individuals with typical development if they engaged in more explicit –based processing.

The purpose of the present study was to examine whether individuals with ASD demonstrate intact associative learning when the associative learning occurs within a single modality (e.g., audition) and across modalities (e.g., audition and vision). Gaigg and Bowler (2007) examined atypical fear acquisition and discrimination across modalities (e.g., a visual CS and an auditory UCS), which provided evidence for poor connectivity between brain regions associated with learning a fear response to a visual stimulus. However, nothing was said of the functionality and connectivity within a particular modality, such as learning a fear response to an auditory stimulus. The current study sought to replicate Gaigg and Bowler using visual and auditory CSs. It was predicted that individuals with ASD would demonstrate abnormal fear conditioning specifically within the visual CS, auditory UCS condition. Additionally, we sought to test whether individuals with ASD would demonstrate impaired or intact conditioning within the auditory CS, auditory UCS condition. If learning is impaired in both the visual CS-auditory UCS condition and the auditory CS-auditory UCS condition, this would suggest a general impairment in implicit association learning. However, if learning is impaired in the visual CS-auditory UCS condition but not the auditory CS-auditory UCS condition, this would suggest a more specific impairment in connectivity across long-range connections in individuals with ASD.

Method

Design

This study used a 2 x 2 x 2 mixed factorial design. There were two diagnostic groups (ASD vs. TD), two types of stimulus modality (visual CS vs. auditory CS), and two pairings (unpaired CS vs. Neutral). Stimulus modality (visual vs. auditory) and pairing were within subject variables, and diagnostic group (ASD vs. TD) was a between-subjects variable. The dependent variable was SCR difference scores computed using a peak and valley method, wherein the valley of the first two seconds of activation was subtracted from the peak response typically observed within the last three seconds of each trial.

Protocol for the visual condition was based on the protocol used by Bechara et al. (1995) and Gaigg and Bowler (2007) and consisted of 12 habituation trials, 50 acquisition trials, and 10 extinction trials. Each subject received one colored square and one instrument sound paired with a loud foghorn (CS+) at 85 to 100 dB and three additional colored squares and three additional instrument sounds were used as neutral, control stimuli (CS-).

Participants

Fifteen adolescents and young adults with high functioning ASD (17-25 years old, 13 male, two female), and 16 adolescents and young adults with typical development (TD; 17-23 years old, 14 male, two female) were recruited for this study. Due to computer error two participants with ASD and one participant with typical development were excluded from all analyses. Participants with ASD were recruited through the University of Alabama ASD

Research Clinic and Mitchell's Place in Birmingham, AL. Participants with typical development were recruited through the University of Alabama's Psychology 101 subject pool, and local schools and churches.

In order to confirm a previous diagnosis for ASD, participants with ASD received the Autism Diagnostic Observation Schedule – Generic Module 4 (ADOS-G; Lord et al., 2000). All participants met the cutoff for combined social and communication scores of 7 or higher on the ADOS-G. Additionally, any TD participants with a neurological disorder (e.g., Obsessive Compulsive Disorder) or with a learning disability were excluded. Finally, any participant with ASD having a co-diagnosis of Fragile X, Down Syndrome, or other potentially confounding co-diagnosis was excluded.

The ASD and TD groups were matched on chronological age, verbal IQ, performance IQ, and full scale IQ. The means on these matching variables are presented in Table 1. The groups were compared using between group t-tests. As can be seen, the groups were well matched with no significant group differences on any matching variable (all p 's > .4).

Measures

Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999). The WASI is an abbreviated IQ measure designed for persons six to 89 years of age. This scale takes approximately 30 minutes to administer. The WASI has four subtests: vocabulary, similarities, block design, and matrix reasoning. The WASI provides a verbal IQ (vocabulary and similarities subtests), a performance IQ (matrix reasoning and block design subtests), and a full scale IQ. The WASI has good validity and reliability (reliability coefficients for adults range from .84 to .98).

Brief Autism Phenotype Questionnaire (BAPQ; Hurley, Losh, Parlier, Reznick, & Piven, 2007). The BAPQ is a 36-item questionnaire that assesses phenotypic expression and the genetic predisposition to autism. The questionnaire has the three symptom domains associated with ASD: aloof personality (social), pragmatic language difficulties (communication), and rigid personality (repetitive behaviors). There are two versions of the BAPQ available: a parent report and a self-report. This measure was used as a screening tool for typically developing adolescents and young adults as well as a measure of current ASD symptomatology for our participants with ASD. Only the self-report version of the BAPQ was used. This measure takes approximately 10-15 minutes to complete and has generally high sensitivity and specificity for individuals with an ASD (67% and 63%, respectively), along with inter-item reliability coefficients ranging from .85 to .95 for parent and self informants.

Autism Diagnostic Observation Schedule – Generic (ADOS-G; Lord et al., 1999).

The ADOS-G, Module 4, (35-40 minutes) is a semi-structured play assessment of social interaction, communication, and imaginative or symbolic play administered to adolescents and adults who presumably have ASD. The ADOS-G was administered to individuals with ASD to both confirm an ASD diagnosis and measure current autism symptomatology (i.e., social skills, and communication). Good criterion validity has been demonstrated, with 24 out of 25 clinically diagnosed children with autism also meeting criteria for autism on the ADI-R, with good reliability ratings, mean weighted kappa ($Mk_w = .66$), for items in module 4 (see Lord et al., 2000).

Demographic form. A demographic form was included to gather general information about the participants. The demographic form included information about the participant's age, gender, race, family income, and any current diagnoses. Also the date of birth was recorded on

this form to calculate appropriate standard scores for the ADOS-G and WASI. If the participant was under the age of 19, then we obtained demographic data from the parent or legal guardian.

Apparatus

Skin conductance response hardware and software. Skin conductance response was measured using a Biolog 3992 with two 7.5mm diameter Ag/AgCl surface electrodes attached with an electrolyte of 0.05 M NaCl inert ointment to the medial phalanges of the first and third digits of the right hand. A constant voltage device set at 0.5 V sampled at 12 Hz and BioLog's Downloading and Plotting Software (DPS) was used to record and assess data from electrodermal activity (BioLog, 2007).

Procedure

Participants were scheduled for a two and a half hour session. During this session, consent forms were signed, and participants were given an overview of the experiment. If the participant was under 19 years of age, they were be asked to sign the assent form and their parents were be given the consent form. During the session, participants completed the WASI and ADOS-G. After filling out all required forms, participants were then escorted to a semi-sound proof room for the conditioning task.

Participants were told that they were about to participate in a psychology experiment to examine simple learning of visual and auditory stimuli, and warned that they would hear some aversive sounds. The foghorn sound was initially presented at a moderate volume (85 dB) through headphones, and participants were allowed to adjust the volume so that the foghorn sound was as loud as possible without being painful. Prior to the attachment of the SCR electrodes, participants were asked to relax and find a comfortable sitting position approximately 50 cm away from the computer screen. Participants were asked to pay close attention to the

stimuli presented on the screen and over the headphones. Also participants were asked to remain as motionless as possible to reduce the possibility of excessive movement as a confounding variable in SCR recording. Presentation of the stimuli commenced once SCRs reached a stable baseline activity level. The experimenter was present during the entire duration of the experiment in order to monitor SCRs, observe any excessive anxiety to the aversive stimuli, and to ensure that participants were focused on the task. The experimenter was positioned approximately three feet to the side of the participant. Only the laptop and electrodes attached to the fingers were in the line of sight of the participant (*Figure 1*).

Protocol for the visual-auditory condition was based on the protocol used by Bechara et al. (1995) and Gaigg and Bowler (2007) and consisted of 12 habituation trials, 50 acquisition trials, and 10 extinction trials. Each trial contained three of four possible colored squares (red, yellow, green, or blue), and three of four possible instrumental sounds (e.g., piano, violin, guitar, and organ). All colors and instrument sounds were randomly presented during both habituation and extinction trials. However, in order to maintain learning during the acquisition phase, the first 40 trials were 100% reinforced and the last 10 trials presented the visual and auditory CS alone (i.e., without the UCS pairing). The colored square (CS_{visual}) and the instrument sound (CS_{auditory}) were paired with the foghorn sound (UCS) at 85 to 100 dB, presented through headphones. The square-foghorn pairing was denoted as $CS_{+\text{visual}}$ and instrument-foghorn was denoted as $CS_{+\text{auditory}}$. All other colored squares and instrument sounds were denoted as $CS_{-\text{visual}}$ and $CS_{-\text{auditory}}$. The onset of the foghorn sound occurred 5000 ms after the presentation of the colored square and 6000 ms after the presentation of the instrument sound. Both color square and foghorn were simultaneously presented for the duration of the foghorn sound (2000ms) and co-terminated at the end of the 2000 ms. However, the foghorn immediately followed the

presentation of the instrument sound due to the inability to distinguish between the instrument sound from the foghorn. Visual trials lasted 7000ms and auditory trials lasted 8000ms. The intertrial interval was 15 seconds in order for the participant's SCR to return to baseline prior to each trial. The color and instrument sound choice for the CS+ pairing was counterbalanced across all participants.

The stimuli used for the CS_{+visual} and CS_{+auditory} were presented two times each during the habituation phase and 15 times during acquisition. Furthermore, the first 12 occurrences of CS_{+visual} and CS_{+auditory} were paired with the UCS and the last three trials were presented without the UCS (3 CS_{+unpaired_visual} & 3 CS_{+unpaired_auditory}). The purpose of including the CS_{+unpaired} trials is to assess the physiological response (e.g., learned association) between the CS+ and CS- trials. Finally, during the extinction trials participants saw three random presentations of the CS_{+unpaired_visual} and CS_{+unpaired_auditory} and two neutral colors and instrument sounds (CS-). At the end of the experiment participants were given a brief memory test to probe their explicit knowledge of the task.

Data Processing

Data analysis was completed using SPSS IBM 19 statistical software. In order to account for random variance due to individual differences in SCL, a time series analysis was conducted.. A time series analysis can be conducted on data that is measured at successive and uniform time intervals (Cryer & Chan, 2008). The current data was sampled at a continuous 12 Hz, and therefore was appropriate for a time series analysis. This analysis is akin to the smoothing technique observed in fMRI data processing and was conducted in order to smooth out any anomalous data points that could have been the result of participant movement or the tendency for SCL reduction over time due to greater relaxation with the testing environment (i.e., sitting

still for an extended period of time) (Bach, Flandin, Friston, & Dolan, 2009) The time series analysis consisted of a centered moving average with a five sample window. The five sample window indicates that each data point was averaged with two data points collected prior to and directly after the current data point. Following this times series analysis, using standard criteria (see Gaigg and Bowler, 2007), SCRs was computed as the largest deflection during an eight second window after the presentation of the stimulus to provide a measure of autonomic response to that stimulus. In order to determine the SCL response amplitude we identified the valley within the first four seconds of the trial and subtracted this value from the peak response (typically observed during the last three seconds of each trial). Examining all CS⁺_{unpaired} trials and all CS⁻ trials, associative learning was calculated as the average response at the first onset of a trial (i.e., seeing a color or hearing a sound) subtracted from the response amplitude from the second interval response (peak response during 7 sec trial duration). Following this procedure, a range correction procedure was then computed on SCL amplitudes in order to reduce interindividual variance so that each SCL value was set in a proportion based upon each individual's SCL range. This correction was implemented based on the assumption that individual physiological differences, which do not directly relate to the psycho-physiological processes, could be eliminated (Levey, 1980). In order to compute this proportion, each individual's SCL amplitude was subtracted from their minimum SCL amplitude, then divided by the maximum SCL amplitude minus the minimum SCL amplitude (Appendix A).

Results

Before conducting our main analyses we compared the intensity of the UCS between diagnostic groups and found that the ASD group adjusted the intensity of the UCS lower ($M = 90.21\text{dB}$, $SD = 5.30$) than individuals with typical development ($M = 93.41\text{dB}$, $SD = 2.17$), $t(28) = 2.21$, $p = .04$. (It should be noted that we did not measure the decibel level for one participant with ASD who was therefore excluded from this analysis.)

Since individuals with ASD preferred a lower decibel setting to the UCS compared to individuals with typical development we compared the SCR to the UCS across both diagnostic groups to test whether both groups showed similar SCL reactivity to the UCS. This is important because if the two groups do not show similar reactivity to the UCS, their learning may be affected by these differences in sensitivity. Both diagnostic groups exhibited significant sensitivity to the trials on which the UCS was presented ($CS_{+paired}$) compared to the neutral trials ($CS-$). The ASD group showed an 8.0% peak increase in SCL to the $CS_{+paired}$ trials ($M = .08 \mu\text{S}$, $SD = .12$) and a 1.0% peak decrease to the $CS-$ trials ($M = -.01 \mu\text{S}$, $SD = .07$). This was a large, significant response to the UCS, $t(14) = 3.82$, $p < .01$, Cohen's $d = .99$. The group with typical development showed a 10% peak increase to the $CS_{+paired}$ trials ($M = .10$, $SD = .10$) and a 0.1% peak decrease to the $CS-$ trials ($M = .001$, $SD = .03$). This was also a large, significant effect response to the UCS, $t(15) = 4.50$, $p < .01$, $d = 1.12$). Furthermore, an ANOVA comparing these two trials for the two diagnostic groups showed that there was no significant interaction between

diagnostic group and pairing, $F(1, 29) = .004, p = .95$, suggesting that both diagnostic groups displayed similar reactivity to the UCS.

Following this we conducted a 2 (TD vs. ASD) x 2 (visual vs. auditory) x 2 (CS_{+unpaired} vs. CS₋) repeated measures ANOVA to compare differences between diagnosis, modality, and pairing. Trials that presented the CS without the presentation of the UCS were designated as CS_{+unpaired}, and presentations of non-conditioned stimuli were designated as CS₋. In order to compare the CR between CS_{+unpaired} and CS₋, we only analyzed the last three presentations of both the visual and auditory CS_{+unpaired} (which occurred at the end of the acquisition phase) and compared that to the CR for all presentations of the visual and auditory CS₋ (i.e., all visual and auditory neutral stimuli). The mean SCR difference scores for each condition are presented in Table 2. An overall greater response to CS_{+unpaired} trials than CS₋ trials was seen across groups. This main effect of pairing was significant and a large effect, $F(1, 29) = 25.12, p < .01, \eta_p^2 = .464$. However, this main effect of pairing was moderated by a significant interaction between diagnosis and pairing, $F(1, 29) = 7.90, p < .01, \eta_p^2 = .214$. As can be seen, the TD showed much larger effects of pairing (+.050) than the ASD group (+.014), suggesting significant differences between the two diagnostic groups in the amount of learning associated with the CS₊. There was no significant interaction between modality and pairing, $F(1, 29) = .14, p = .71$, modality and diagnosis, $F(1, 29) = .01, p = .93$, and no three way interaction between modality, pairing, and diagnosis, $F(1, 29) = .39, p = .54$.

Two additional follow-up 2 (visual vs. auditory) x 2 (CS_{+unpaired} vs. CS₋) repeated measures ANOVAs were conducted, one for each diagnostic group. Individuals with typical development demonstrated a main effect of pairing, $F(1, 15) = 32.13, p < .01, \eta_p^2 = .682$, and a non-significant interaction between modality and pairing, $F(1, 15) = .02, p = .89$, suggesting a

significant conditioned response to the CS_{+unpaired} trials and no differences in learning between the visual and auditory trials. In order to determine whether reliable conditioning was exhibited for visual and auditory trials, additional paired samples *t*-tests were conducted for CS_{+unpaired} versus CS₋ trials separately for visual and auditory trials. For individuals with typical development a significant difference was found for both the visual trials, $t(15) = 3.13, p = .007$, Cohen's $d = .78$ (*Figure 2.*) and auditory trials, $t(15) = 2.56, p = .02, d = .64$ (*Figure 3.*). These results suggest that individuals with typical development demonstrated learning in both visual and auditory trials.

Individuals with ASD showed a non-significant trend for pairing, $F(1, 14) = 2.31, p = .15$, no main effect for modality, and no interaction, $F(1, 14) = 1.64, p = .22$, suggesting that although individuals with ASD showed a slight increase in SCL amplitude to the CS_{+unpaired} trials, this was not statistically different from the CS₋ trials. On the follow-up *t*-tests, individuals with ASD demonstrated a non-significant difference within the visual trials, $t(14) = .45, p = .66, d = .12$ (*Figure 2.*), and a significant difference within the auditory trials, $t(14) = 2.17, p = .05, d = .56$ (*Figure 3.*). Thus, there was mixed evidence for learning in the ASD group. The ANOVA indicated no effect of pairing and no interaction of pairing with modality indicating no evidence of learning in these participants. However, the follow-up *t*-tests suggested that individuals with ASD demonstrated learning within the auditory trials but not the visual trials.

Explicit Memory Test

In line with the learning compensation model of ASD, we also predicted that individuals with ASD who demonstrated conditioning might also demonstrate greater explicit awareness of the learning contingencies within this paradigm (i.e., awareness that a particular color or instrument was always paired with the aversive sound). In order to assess participant's

awareness of the learning contingencies participants were asked eight questions pertaining to the visual and auditory stimuli. Participants were asked: (1) How many color/instruments did you see/hear? (2) What colors/instruments were they? (3) How many colors/instruments were followed by the loud noise? (4) What color(s)/instrument(s) was/were it/they? Scores were recorded out of four for both visual and auditory trials. Questions one through three were assigned a value of .5 and question four 2.5 points, since the final question was the most important indicator of explicit awareness (Bechara et al., 1995). All four questions were included in the initial analyses to examine explicit learning. However, upon further examination of the explicit memory test, it became clear that the most important question that determined whether participants exhibited explicit awareness was question four. Therefore, all subsequent analyses included only question four. A one sample *t*-test revealed that individuals with typical development showed a non-significant trend of greater explicit awareness ($M = 43\%$, $SD = 44\%$) $t(15) = 1.70$, $p = .11$ (chance = 25%), whereas individuals with ASD demonstrated explicit awareness greater than chance, ($M = 57\%$, $SD = 42\%$), $t(14) = 2.94$, $p = .01$. Following this analysis, a 2(visual vs. auditory) x 2(CS_{unpaired}⁺ vs. CS⁻) x 2(explicit memory correct vs. incorrect) repeated measures ANOVA was conducted on individuals with ASD to examine whether correctly identifying the CS resulted in differences between pairing and modality. This analysis revealed a significant interaction between pairing and explicit learning, $F(1, 13) = 8.13$, $p = .01$, and a non-significant three-way interaction between pairing, modality, and explicit learning, $F(1, 13) = .02$, $p = .89$. These results suggest that greater explicit learning resulted in greater learning of the paired association between CS_{unpaired}⁺ and CS⁻ trials, however there were no significant learning differences between modalities. Following this analysis, an independent samples *t*-test was conducted on each diagnostic group to compare auditory learning and overall

learning between individuals who answered question four correctly to those who answered incorrectly (*Figure 4*.) For individuals with typical development, no significant learning differences were found between those who answered question four correctly ($n = 7$) versus incorrectly ($n = 9$) for auditory learning, $t(14) = .34, p = .74$, and overall learning, $t(14) = .67, p = .51$. However, we did find that individuals with ASD that correctly identified the visual CS ($n = 8$) demonstrated greater auditory learning, $t(13) = 2.23, p = .04$, and overall learning, $t(13) = 2.85, p = .01$, compared to those who answered incorrectly ($n = 7$). There was also a marginally significant finding for visual learning, $t(13) = 2.02, p = .07$. These results are consistent with our prediction that individuals with ASD who displayed greater explicit awareness of associative learning contingencies would also display greater learning.

Explicit memory as a covariate. Because of the relationship between explicit awareness of the visual trials and learning, we wanted to explore whether explicit awareness of the visual trials accounted for auditory learning in the ASD group. We conducted an additional 2(vision vs. auditory) x 2 (CS_{unpaired} vs. CS₋) repeated measures ANOVA between each diagnostic group using explicit memory as a covariate. Individuals with typical development continued to demonstrate a main effect of pairing, $F(1, 14) = 21.30, p < .01, \eta_p^2 = .603$, and a non-significant interaction between pairing and explicit memory, $F(1, 14) = .45, p = .51$. However, individuals with ASD showed no main effect of pairing $F(1, 13) = .34, p = .57, \eta_p^2 = .03$, and a significant interaction between pairing and explicit memory, $F(1, 13) = 8.13, p = .01, \eta_p^2 = .385$. These results suggest that individuals with ASD demonstrating greater explicit awareness are more likely to exhibit associative learning.

ASD symptomatology measures and Learning

Correlational analyses were conducted to examine ASD symptomatology and its relationship to visual, auditory, and overall learning. ASD symptomatology was measured using both the self-report version of the boarder autism phenotype questionnaire (BAP-Q) and the autism diagnostic observation schedule generic (ADOS-G). This analysis revealed a marginally significant correlation between overall learning and the aloof subscale of the BAP-Q, $r(29) = -.33, p = .07$, and significant correlations between overall learning and the language subscale, $r(29) = -.49, p = .005$, rigidity subscale, $r(29) = -.50, p = .004$, total BAP-Q score, $r(29) = -.57, p = .001$ (*Figure 5*). However examining this relationship separately for each modality (visual or auditory), only auditory learning was significantly related to the language subscale of the BAP-Q, $r(29) = -.39, p = .03$, and the rigidity subscale, $r(29) = -.51, p < .05$. Similarly, scores from the repetitive behaviors subscale of the ADOS-G were also significantly correlated with auditory learning, $r(13) = -.53, p = .05$. No significant correlations were found between the BAP-Q or ADOS-G and visual learning. These results suggest that greater ASD symptom severity is associated with poorer auditory learning.

Explicit Learning and ASD Symptomatology.

Additionally, we found a significant correlation between explicit awareness of the visual trials and total BAP-Q scores, $r(13) = -.63, p = .01$. Participants with ASD who scored higher on the BAP-Q were less likely to explicitly identify which stimulus was paired with the UCS. This led us to explore the relationship between explicit learning, ASD symptomatology, and associative learning. Subsequently, we conducted a regression analysis to examine how explicit awareness of the visual trials and total BAP-Q scores predicted overall learning. Explicit awareness was entered at Step1, explaining 38.5% of the variance in overall learning, $F(1, 13) =$

8.13, $p = .02$. However, no additional variance was explained after the entry of the total BAP-Q scores, $F(1, 12) = .002, p = .97$. A follow-up Sobel's test of mediation indicated that explicit awareness mediated the relationship between ASD symptomatology and overall learning, Sobel = -2.02, $p = .04$, two-tailed.

Discussion

In the current study we examined autonomic conditioning of individuals with ASD and matched individuals with typical development to determine whether individuals with ASD displayed atypical conditioned responses across modalities. Previous studies have found both intact (Bernier et al. 2005) and impaired (Gaigg & Bowler, 2007) conditioning in adults with ASD. We explored whether individuals with ASD would display impaired conditioned response, relative to individuals with typical development, during both visual and auditory pairings. Significant differences were found between diagnostic groups across both visual and auditory CS pairings, suggesting that individuals with ASD demonstrated a reduced conditioned response to CS_{+unpaired} trials compared to the CS₋ trials. Analyses indicated that individuals with ASD did show a slight increase in SCL amplitude to both visual and auditory conditioned stimuli; however these differences were not statistically significant when analyses were collapsed across the two modalities. However, individuals with ASD demonstrated significant conditioning to the auditory CS when examined alone. This auditory conditioning effect was strongly related to explicit memory for pairing in the ASD group. Follow up analyses that controlled for explicit memory showed there was no longer a significant difference between the auditory CS and neutral stimuli.

It is important to compare how this study relates to previous studies examining associative learning in individuals with ASD. Previous studies have used only one conditioned stimulus to examine associative learning among individuals with ASD (Bernier et al., 2005;

Gaigg & Bowler, 2007), however our study included both a visual and auditory conditioned stimulus. Furthermore, in addition to using only one conditioned stimulus, Bernier et al. (2005) used at 100% reinforcement schedule and no additional neutral stimuli during the conditioning phase and found intact associative learning among individuals with ASD. Gaigg and Bowler (2007) used only one conditioned stimulus, but used a 50% reinforcement schedule and three additional neutral stimuli during the conditioning phase and found evidence of less reliable conditioning in individuals with ASD. Our study also included a 100% reinforcement schedule during the acquisition phase, but included two conditioned stimuli and six neutral visual and auditory stimuli. Therefore it seems that when using a relatively simple associative learning paradigm with only one conditioned stimulus and a 100% reinforcement schedule this may make it easier for individuals with ASD to explicitly learn resulting preserved associative learning. However, when you increase the number of stimuli or use a 50% reinforcement schedule this may make it more difficult to explicitly learn resulting in observed impairments in associative learning among individuals with ASD. This notion is supported by our results from the explicit memory test, although both diagnostic groups demonstrated explicit learning greater than chance, neither group was near 100% accurate. However, we found that individuals with ASD demonstrating greater explicit learning also demonstrate greater associative learning. Ultimately these results suggest that as tasks become more complex and difficult to explicitly learn, individuals with ASD will have greater difficulty learning compared to individuals with typical development. However, if we develop tasks that are relatively easy to explicitly learn, we ought to see similar learning between both diagnostic groups.

Another way to explore the relationship between explicit awareness and associative learning is to explore ways in which the presentation of the stimuli might influence an

individual's ability to associate the CS with UCS. Two common types of conditioning used in classical conditioning paradigms are delay and trace conditioning. Delay conditioning refers to the presentation of the CS which remains on until the UCS is presented then the two stimuli co-terminate. In trace conditioning the CS is presented and is followed by an empty (i.e., trace) interval followed by the UCS (Clark, Manns, & Squire, 2002). Previous studies using an amnesic population found that delay conditioning was intact in this population whereas trace conditioning was impaired (Gabrieli et al., 1995; McGlinchey, Capozzi, Fortier, & Disterhoft, 2008). These findings suggest that explicit awareness is important for trace conditioning, presumably due to the greater involvement of the hippocampus, whereas delay conditioning does not require explicit awareness. Furthermore, Weike, Schupp, & Hamm (2007) suggested that delay conditioning may, in fact, involve more implicit processes; whereas explicit awareness may be a prerequisite for learning in a trace conditioning paradigm. The current study utilized a delay conditioning paradigm in which the CS was presented and remained during the presentation of the UCS. Similar to previous studies (Weike, Schupp, & Hamm, 2007), our results indicated that for individuals with typical development, explicit awareness did not influence associative learning however, explicit awareness did influence associative learning in individuals with ASD. Therefore it seems that individuals with ASD who were more inclined to utilize explicit learning strategies may have demonstrated poorer associative learning because delay conditioning may rely on more implicit processes. It is interesting to speculate whether we would see a similar pattern as the present data if we were to use a trace conditioning paradigm in which explicit awareness may be more necessary for developing an association between the CS and UCS. We might expect that individuals with ASD might exhibit associative learning that is more similar to typically developing participants in a trace conditioning paradigm. However, it

is also possible that those individuals with ASD that do not utilize explicit strategies during a trace conditioning paradigm might display impaired associative learning as was seen in the present study. Future studies exploring the role of explicit awareness and how it relates to delay versus trace conditioning in persons with ASD should prove important to more fully understanding learning differences and why they occur in individuals with ASD.

Evidence of impaired associative learning of the visual-auditory pairings and auditory-auditory is somewhat consistent with the underconnectivity theory of ASD. That is, the communication between the long-range white matter tracts in the brains of individuals with ASD is impaired, which is demonstrated through evidence of impaired associative learning between visual and auditory CS-UCS pairings. However, according to the underconnectivity short-range connections within the brains of individuals with ASD are relatively intact, which presumably would have resulted in the ability of individuals with ASD to learn the association between an auditory CS and an auditory UCS. However, the learned association between auditory CS and UCS does not necessarily involve only the auditory cortex. It is also important to consider the role of the amygdala and other brain regions in classical fear condition paradigms. Therefore the lack of reliable conditioning observed in the auditory trials may be the result of impaired communication between the amygdala and the auditory cortex. This possibility is also consistent with Baron-Cohen's (2000) amygdala theory of autism, and suggests that the observed impairment in associative learning among individuals with ASD may be the result of abnormal functioning of the amygdala and its communication with associated cortices.

Although previous studies examining functional connectivity in ASD have largely relied on language-based tasks (Just et al., 2004; Kana et al., 2007), the current study suggests that the functional connectivity impairments observed in individuals with ASD may reflect a more

fundamental impairment in sensory integration and communication. Therefore the current results could also reflect evidence of sensory integration difficulties in individuals with ASD (Russo et al., 2010; Smith & Bennetto, 2007; Taylor, Issac, & Milne, 2010), particularly, sensory information that is also associated with emotionally-valenced stimuli. Similarly, the current task utilized simple associative learning principles unlike previous studies that have primarily examined language processing (Smith & Bennetto, 2007; Taylor, Issac, & Milne, 2010), and still found evidence of impaired associative learning. Therefore, the current study emphasizes a fundamental impairment in associative learning that may be independent of language development.

Implications for Implicit Learning and ASD

The learning compensation model of ASD also provides an explanation of the current results when one considers associative learning to be a simple form of implicit learning. The Learning Compensation model of ASD stems from evidence that individuals with ASD demonstrate impairments in implicit learning and seem to compensate by employing more explicit or rule-based strategies. Our current results indicate that individuals with ASD demonstrated a general learning impairment and greater explicit awareness was related to greater associative learning. Specifically, we observed impaired learning across both visual modalities. This finding is consistent with the Learning Compensation model which suggests that individuals with ASD have general impairments in implicit learning. Additionally, auditory implicit learning was related to explicit memory of the visual condition rules. This result fits nicely with the prediction that learning in individuals with ASD relies more on explicit learning than in individuals with typical development. These results suggest that persons with ASD are more

dependent upon explicit forms of learning than persons with typical development, and the results here directly follow from the Learning Compensation model.

It is also important to note how this task relates to other forms of implicit learning. As previously mentioned, our task used a delay conditioning paradigm and according to previous literature (Weike et al., 2007) this form of conditioning may rely on more implicit processes as compared to other types of conditioning paradigms (i.e., trace conditioning). A hallmark of implicit learning is the ability to learn without intention or awareness of what is being learned (Reber, 1989). Although we cannot rule out the possibility that our participants were not aware of what was being learned, we can assume that the conditioned response that we observed in our participants was largely a response that was automatic as compared to a controlled response. Therefore, what we have observed in our study is the ability of individuals with typical development to learn an association between two stimuli and demonstrate a response that is largely out of conscious control. However, individuals with ASD did not demonstrate the same response which suggests that this fundamental form of learning (i.e., the ability to link to ideas together) is impaired. This finding is consistent with other forms of implicit learning such as the artificial grammar task and prototype learning task used in previous studies (Klinger et al., 2007). What we seem to be observing in tasks that access implicit processes is that individuals with ASD have difficulty associating and combining different stimuli. Additionally, when we make implicit learning tasks more difficult by adding stimuli, we may see even greater impairments in learning. Therefore it is important to consider the difficulty of the implicit learning task when evaluating the ability of individuals with ASD to learn implicitly.

However, one difficulty with this finding is the lack of relationship between explicit memory of the auditory trials and auditory learning given that participants displayed similar

percent accuracy for both the visual and auditory questions. It is worth speculating why visual explicit learning was related to total learning and both visual and auditory learning using the SCR measure for participants with ASD. It is possible that visual explicit memory may be more strongly related to the use of actual explicit learning strategies than the auditory explicit learning. More participants with ASD explicitly learned the auditory pairing (10 of 15 participants) than explicitly learned the visual pairing (7 of 15 participants). The extra difficulty and cognitive processing necessary to learn the relation between the visual CS and UCS may reflect more extensive explicit reasoning and strategies for the participants with ASD. Also it may be the case that due to the greater accuracy within the auditory trials there may be somewhat of a ceiling effect preventing the ability to observe the relationship between explicit learning of the auditory trials and auditory learning. Further research that more directly examines the degree to which participants use explicit learning strategies and its relation to conditioning is important for more direct tests of this relation.

ASD Symptomatology and Learning

Our correlational analyses revealed a significant negative relationship between our two measures of ASD symptomatology (BAP-Q, ADOS-G) and auditory learning. These findings showed that greater ASD symptom severity was associated with more impaired auditory associative learning. However, we also found a significant relation between ASD symptomatology and explicit learning, indicating that greater ASD symptom severity was associated with less explicit learning. Our regression analysis revealed that the relationship between ASD symptomatology and associative learning is mediated by explicit learning. Although it is difficult to determine the direction of this relationship, Klinger et al. (2007) suggested that ASD symptomatology may be reflective of cognitive processing impairments.

As a result, the observed impairments in associative learning may be directly related to difficulties among individuals with ASD in utilizing explicit learning strategies. Therefore, one possible interpretation of these findings is that individuals with ASD exhibiting greater symptom severity may have difficulty employing explicit learning strategies, which then interferes with associative learning. These results also allow us to speculate that the impairments in language and social communication typically observed among individuals with ASD may be the result of a fundamental impairment in associative learning in addition to a difficulty of employing explicit learning strategies. However, a possible limitation of this finding is due to the use of self-report as opposed to parent-report on the BAP-Q. Individuals with ASD may have tendency to under-report their ASD symptoms due to difficulties with self-awareness which cause their scores on the BAP-Q to be lower than their actual symptom severity. However, this possibility would then make it more difficult to find a relationship between current symptomatology and associative learning. Given that a relationship between BAP-Q and associative learning was still observed, this suggests that this relation is likely very trustworthy. Future studies are needed to further examine more directly the relationship between ASD symptomatology and associative learning.

The current study also has important clinical implications. Given the importance of learning by automatic associations and its ubiquitous role in early learning, our findings provide important evidence for targeting associative and automatic learning in early intervention strategies. Many behavioral therapies for individuals with ASD such as Applied Behavioral Analysis (ABA) utilize basic associative learning principles in order to reinforce more appropriate behaviors. For instance, discrete trial training (DTT) relies on heavy reinforcement of behaviors that are repeated over and over at a very high rate. For children with ASD, greater intensity of training is needed to obtain a desired behavior than would be seen for children with

typical development. This kind of observation is very consistent with the results of this study. It is important to explore the rate of learning in children with ASD, compared to age and intelligence matched peers, to determine how many more trials are needed in order to demonstrate the same learning. Our findings also speak to the ability of individuals with ASD to compensate for implicit learning impairments by utilizing explicit learning processes. Therefore, use of more explicit or rule-based strategies to teach children with ASD may be a more effective approach towards developing a desired behavior. Finally, the current study examined high-functioning adolescents and young adults with ASD. Future research is needed to determine whether these findings are present in lower-functioning individuals with ASD, and the extent to which associative learning is related to ASD symptomatology.

In conclusion, the present study provides evidence of impaired associative learning in persons with ASD. This common observation may reflect a fundamental impairment in the way individuals with ASD associate various stimuli in their environment. Furthermore, associative learning principles are utilized early on in life, such as the development of attachment and joint attention. Therefore, evidence of impaired associative learning in individuals with ASD would likely have consequences that not only influence early learning, but also learning that continues throughout the lifespan. Future research using a greater age range are needed to determine the extent to which potential associative learning impairments influence the developmental trajectory of individuals with ASD.

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Table 1

Participant Characteristics: Mean and (Standard Deviation) of and Significance Levels of T-test Comparing the Groups (p)

	Typical (<i>n</i> = 16)	ASD (<i>n</i> = 15)	p
Chronological Age (years)	20.17 (1.50)	20.67 (2.53)	.51
Gender			
Male	14	13	
Female	2	2	
WASI			
Verbal IQ	109.50 (9.81)	106.80 (10.36)	.46
Performance IQ	106.43 (11.31)	106.60 (14.77)	.97
Full Scale IQ	108.81 (7.64)	107.40 (10.70)	.67

Table 2

Means and Difference Scores (CS+ - CS-) of SCL amplitudes (μ S)

	Visual Learning			Auditory Learning			Overall Learning		
	CS+	CS-	difference	CS+	CS-	difference	CS+	CS-	difference
TD	.049	-.003	+.052**	.053	.005	+.048*	.051	.001	+.050**
ASD	-.006	-.012	+.006	.010	-.012	+.022*	.002	-.012	+.014
Total	.023	-.007	+.030**	.033	-.003	+.036**	.028	-.005	+.033**

* $p < .05$, ** $p < .01$

Figure 1.

Skin conductance apparatus and testing environment.



Figure 2.

Skin conductance amplitudes between CS⁺_{unpaired_visual} and CS⁻_{visual} trials.

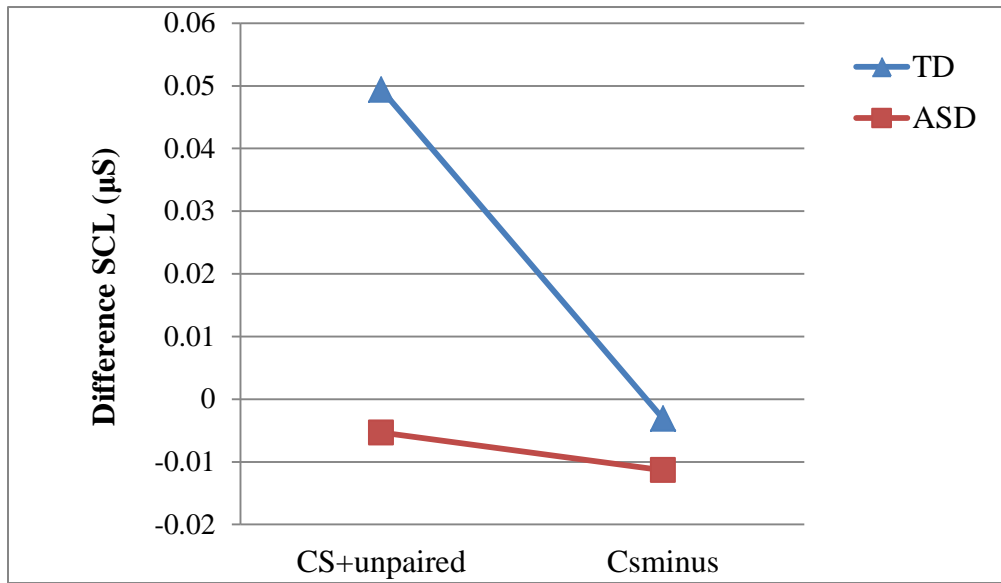


Figure 3.

Skin conductance amplitudes between CS⁺_{unpaired_auditory} and CS⁻_{auditory} trials.

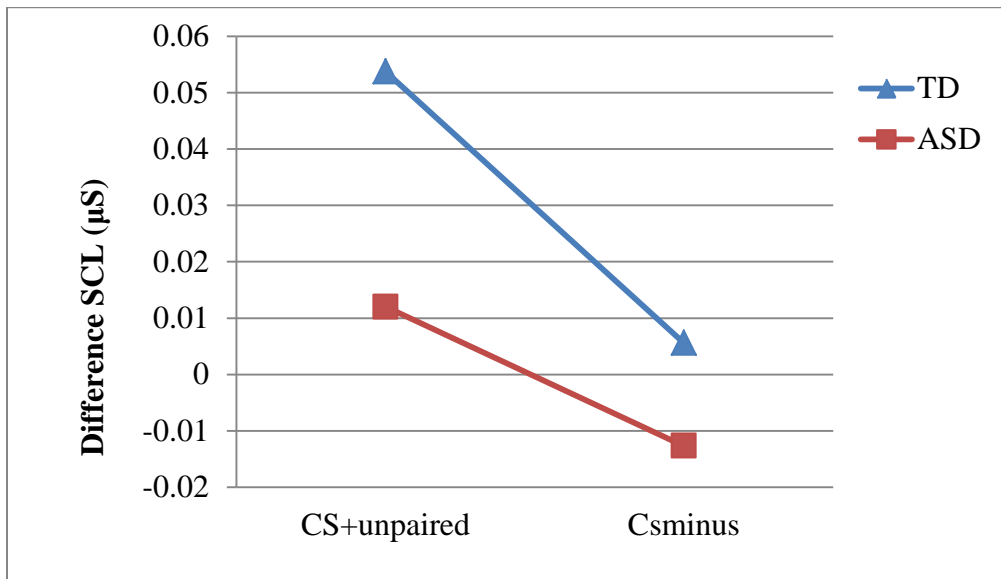


Figure 4.

Comparison of individuals with ASD and TD's explicit memory of visual trials and overall learning.

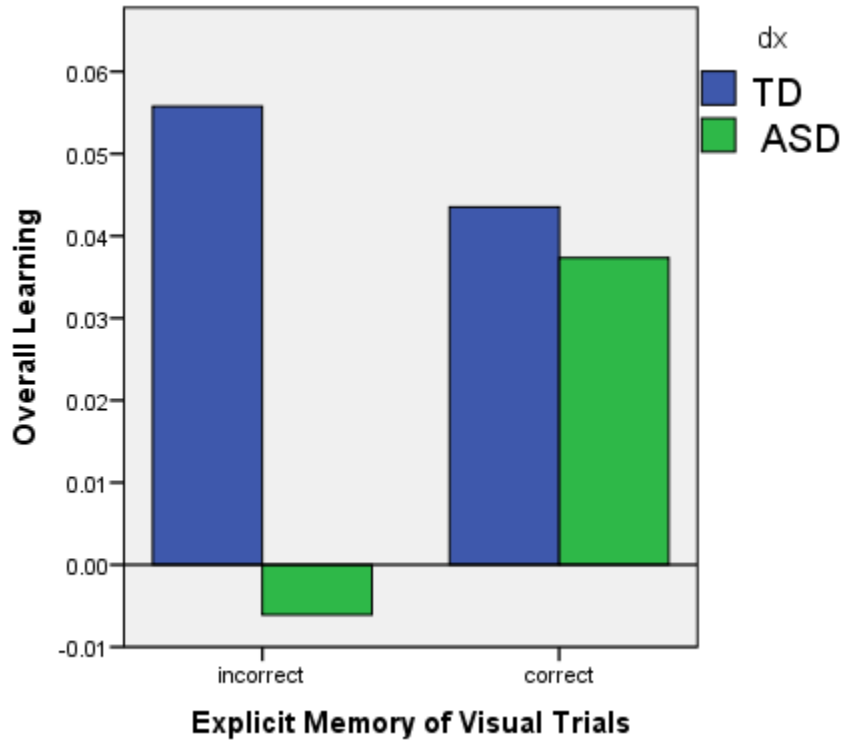
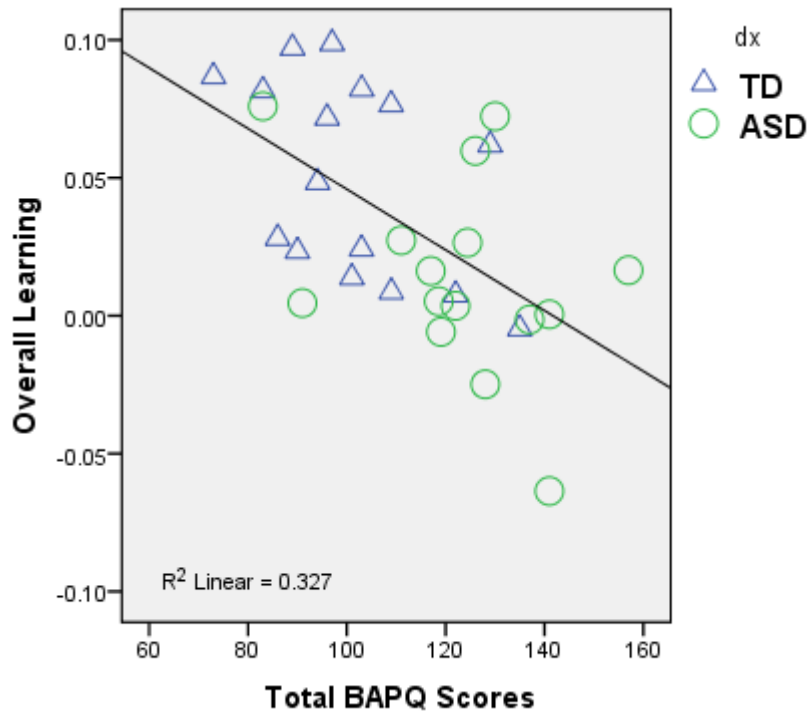


Figure 5.

Correlation between Broader Autism Phenotype Questionnaire (BAP-Q) and Overall Learning.



Appendix A

Range Correction Formula.

$$SCL_i' = \frac{SCL_i - SCL_{\min}}{SCL_{\max} - SCL_{\min}}$$

Where SCL_i is the uncorrected SCL value, SCL_i' is the range corrected SCL value at the time point i , while SCL_{\max} is the highest possible SCL value, and SCL_{\min} is the lowest possible value for each participant.