The moderating effect of gender on the relationship between socialization and internalizing problems in early childhood

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Abstract

Researchers in the field of Autism Spectrum Disorders (ASD) have confirmed that comorbidities are a common problem among diagnosed individuals. Current estimates suggest that more than two thirds meet criteria for an additional diagnosis of psychopathology. In particular, rates of several internalizing problems (i.e., anxiety, depression) appear to be greater for individuals with ASD than their typically developing counterparts. However, little research has been conducted examining factors apart from autism symptomatology that could influence this prevalence difference. Additionally, it is well established that anxiety and depression are more common in females than males in the general population. However, few studies have analyzed gender differences in internalizing problems in persons with ASD. Researchers who have examined this potential difference have obtained discrepant results. The current study aims to examine the interactions between autism symptomatology, comorbid internalizing problems, and gender by analyzing the potential moderating effect of gender on socialization differences predicting the development of internalizing problems in toddlers with autism.
The Moderating Effect of Gender on the Relationship Between Socialization and Internalizing Problems in Early Childhood

Autism Spectrum Disorder is a neurodevelopmental disorder of varying degree of severity but consistent symptomatology, which is commonly diagnosed in infancy or early childhood (Matson, Wilkins, & Gonzalez, 2008). Until May 2013 with the introduction of new diagnostic criteria, this spectrum disorder was conceptualized as five separate syndromes: Autistic Disorder, Asperger’s Disorder, Rett’s Disorder, Childhood Disintegrative Disorder, and Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS; American Psychiatric Association [APA], 2000; Matson, 2007; Matson, Nebel-Schwalm, & Matson, 2007). The controversial Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM5) collapsed the separate diagnoses into a single umbrella term, encompassing a similar set of symptoms: deficits in communication and socialization abilities, as well as the presence of repetitive or restricted interests and behaviors (APA, 2013; Matson, 2007). Symptoms are pervasive, meaning they are present across settings and persist throughout the lifetime.

Although not included in diagnostic criteria, internalizing problems are frequently comorbid with ASD (Howlin, 1997; Kim, Szatmari, Bryson, Streiner, & Wilson, 2000; Skokauskas & Gallagher, 2010). In early childhood (i.e., birth to 3 years), internalizing problems, generally conceptualized as emotional or mood disruptions, primarily include difficulties with anxiety, depression or withdrawal, fears, and shyness or inhibition (Briggs-Gowan, Carter, Bosson-Heenan, Guyer, & Horwitz, 2006). Psychopathology appearing in this period seems to continue to affect the individual throughout development and can be predictive of future problems (Briggs-Gowan et al., 2006; Carter et al., 2010; Keenan, Shaw, Delliquadri, Giovannelli, & Walsh, 1998; Mathiesen & Sanson, 2000; Mesman & Koot, 2001).

Although experts agree that these issues are common for individuals with ASD regardless of age, there is no good consensus regarding prevalence rates. For instance, for anxiety specifically, researchers have claimed rates from 7 to 84%, with the average around 40-50% (Helverschou & Martinsen, 2011). Although interest in psychopathology experienced by toddlers is growing, this topic has not been widely studied among a population of this age with comorbid ASD.

Furthermore, despite increasing interest in comorbid psychopathology among individuals diagnosed with ASD, an insufficient amount of exploration has been conducted regarding the interactions between these disorders, ASD symptomatology, and demographic characteristics. The purpose of the present study is to examine the potential influence of a particular demographic characteristic, gender, on the predictive relationship between a
specific domain of symptomatology of ASD, socialization deficits, and one set of comorbid problems, internalizing behaviors.
Autism Spectrum Disorders

History

Leo Kanner, an American child psychiatrist, is credited with the first recorded identification of children with autism in his work ‘Autistic Disturbances of Affective Contact,’ published in 1943. In his publication, Kanner (1943) described extensively eight boys and three girls he had encountered whose behavior and various idiosyncrasies were substantially different than anything illustrated previously. Within the cases’ descriptions, Kanner effectively portrayed three domains in which we presently recognize differences in persons with ASD including communication, social interaction and relationships, and restricted and repetitive behavior and interests. He also noted that the syndrome first became evident in infancy, which set it apart from previously explained cases of schizophrenia and child psychosis. He also remarked that the children appeared to be of normal intelligence (Kanner, 1943). Several years later, he came to call such a constellation of symptoms “early infantile autism” (Kanner, 1951). His choice of terminology was meant to emphasize the time of onset and the quintessential characteristic of restricted accessibility (Kanner, 1965). His parsed down description included several over-arching characteristics including marked social withdrawal, an obsessive insistence of sameness, and language without interpersonal communicative purpose.

The children who composed Kanner’s sample all exhibited what many researchers came to consider the hallmark of autism: extreme isolation. Kanner (1943) even described one boy as seeming to “draw into his shell and live within himself” (p 218). The children seemed self-satisfied even as infants, not likely to notice or care whether or not their parents were present. Instead, they seemed to regard persons as obstacles and even intrusions and preferred to have as little contact with them as possible. So severe was the desire for aloneness that anything brought in from the external world could cause anxiety, even rage and tantrum behavior.

Beyond their desire for seclusion, these children were described as necessitating consistency to an extreme degree. Their insistence on sameness was not limited to daily routine, but rather even to the minute details of the objects in their presence. Kanner (1943) described several of the children as becoming distraught when seeing something that was broken or incomplete. He noted that this “anxiously obsessive desire for the maintenance of sameness” was accompanied by the children’s reduced amount of varied spontaneous activity (Kanner, 1943, p. 245). The majority of the children behaved monotonously throughout the day with as little deviation from routine as possible, demanding the same of anyone who attempted to interact with them.

Use of language without communicative purpose was another common occurrence among the children in Kanner’s case study. Their unusual use of language included echolalia, often delayed and in similar intonation as
the source, and pronominal reversals, as the children would substitute ‘you’ for ‘I’ when speaking about themselves. Appropriate inflection was absent, and some of the children spoke in a singsong manner. Verbal rituals were not uncommon, representing another instance of their strict insistence on sameness. Additionally, many of the children exhibited impressive rote memory skills with their abilities to recite poems and other memorized information, a fairly function-less behavior parents often encouraged. Overall, rarely did the children use language properly to communicate with caregivers or clinicians.

Kanner’s publication was a rare exception for that era, in that he based his development of diagnostic criteria on his own observations of a child sample rather than modifying those criteria already in existence for adults (Rutter & Schopler, 1988). As a result, autism became recognized as the earliest psychosis known to appear in childhood (Eisenberg, 1956).

Around the same time as Kanner’s noteworthy publication, Hans Asperger was studying a similar group of children, whom he described in his 1944 thesis “Autistic Psychopathy’ in Childhood” (Frith, 1991). Because his work was written in German and was not translated until 1991 by Uta Frith, Asperger did not receive the same amount of credit for recognition of autism among those in English-speaking countries as Kanner did (Wing, 1997). Asperger described the children as having a ‘personality disorder’ that consisted of disturbances in their appearance, expressive abilities, and behavior. However, Asperger felt that their lack of social integration overshadowed the other difficulties he observed. Unlike Kanner, Asperger noted in his original publication on the topic that autism could occur in children of all intellectual levels, including in those with intellectual disability (ID). Other than this significant distinction, Asperger’s and Kanner’s descriptions were remarkably similar. Asperger detailed the children’s problems in numerous areas, including but not limited to: an absence of facial expression and nonverbal communication; stereotypic, not spontaneous, movements; abnormal use and inappropriate intonation of speech, when present; impressive rote memory; incomprehension and rejection of social figures, such as caregivers; desire for isolation and negative reaction to intrusions; abnormal reactions to tactile and auditory sensation; relationship with objects; extreme desire for consistency and routine. This extensive list overlapped significantly with those characteristics described by Kanner.

Many years before such illuminating publications, children who likely could have been diagnosed with autism were considered everything from saints to wild children raised by wolves (Wolff, 2004). In the era when Kanner and Asperger were developing their theories, children with autism were likely considered to have an emotional disturbance or ID (Wing, 1997). Additionally, many were assumed to be deaf due to their tendency to ignore persons around them (Eveloff, 1960). In fact, Rutter (1972) argued that an absence of response to auditory
stimulation was the first symptom noticed in children later diagnosed with autism. Other researchers claimed that a lack of anticipatory pose when being lifted by a caregiver and the absence of a smile response were the earliest signs (Eveloff, 1960). Regardless of the first indication, children who eventually developed these abnormal patterns of behavior had been recognized as atypical long before the disorder’s ‘discovery’ in the 1940s.

Although Kanner is credited with the discovery of autism, the word ‘autism’ first surfaced in 1908, when Swiss psychiatrist Eugen Bleuler coined the term to describe a disconnect from reality, as seen in patients with schizophrenia (Bleuler, 1913; Rutter, 1978). Although children with autism were sometimes thought to have childhood schizophrenia, a concept that will be discussed further, Kanner’s use of the word autism to describe children in 1943 was disparate from its original meaning as defined by Bleuler. Whereas Bleuler’s meaning of the word included a detachment from social relationships and reality into a rich fantasy life, Kanner’s autism described a failure to ever connect interpersonally and a seeming lack of imagination (Rutter, 1978). In concordance with Kanner, Asperger noted that although both persons with schizophrenia and autism show a shutting off with the outside world, the person with schizophrenia suffers from a loss or disintegration of contact, whereas those with autism do not seem to ever have had such a connection (Asperger, 1944; 1991).

Beyond these major distinctions, other researchers supported differential diagnosis due to the presence of many dissimilarities between autism and schizophrenia, including: presence of relapses and remissions in schizophrenia, absent in autism; presence of hallucinations and delusions in schizophrenia, absent in autism; greater number of males than females with autism, a distribution not seen in schizophrenia; frequency of perinatal complications and epilepsy in autism, absent in schizophrenia; higher incidence of schizophrenia in relatives of those with the disorder, a pattern not seen in autism (Eisenberg, 1956; Rutter, 1972, 1978). Nevertheless, the varied use of the word ‘autism’ created diagnostic confusion. Rutter (1978) noted that the terms ‘childhood schizophrenia,’ ‘autism,’ and ‘child psychosis’ were often used interchangeably.

Autism was not included in the first or second editions of the Diagnostic and Statistical Manual of Mental Disorders (DSM), the official diagnostic manual of the APA and a commonly used resource for clinicians. The first edition of the DSM was created in 1951 to classify the varied presentations of psychopathology displayed by veterans of World War II (Cohen, Volkmar, & Paul, 1986). At the time of the development of the first edition (DSM-I; APA, 1952), autism had been identified but nonetheless was not included. In the Diagnostic and Statistical Manual of Mental Disorders, 2nd edition (DSM-II; APA, 1968), autism still was excluded, and those children who were recognized as having the disorder were classified instead with item 295.80, ‘Schizophrenia, childhood type’ (Kanner, 1971). Rutter was a proponent of discontinuing the use of the term ‘childhood schizophrenia,’ which he
claimed had served its purpose, which was to draw attention to the existence of psychoses in childhood (1972). Rutter suggested it was damaging to the field to suggest that all childhood psychoses were the same and could be thus grouped (1972).

However, due to heterogeneity among cases of the disorder, clinicians and researchers found it difficult to elucidate diagnostic criteria for autism. Researchers Rank and Szurek used the term ‘atypical child’ to broaden the criteria of autism in such a way that “schizophrenia, mental subnormality, organic brain disease and psychoneurosis” would all be encompassed (Rutter, 1968, p. 1). At the same time, there existed the opposing desire to divide autism and childhood schizophrenia into further sub-categories (Rutter, 1968). As a result, many different variations of categorization were created, inspiring Rutter (1978) to stress the importance of identifying the symptoms that were both universal and specific in order to pin down an accurate description of what he called the ‘hypothesized disease-state’ detailed by Kanner. The result of Rutter’s effort was diagnostic criteria very similar to Kanner’s original proposal; the symptoms included an inability to develop social relationships, delay in language and impaired understanding (including presence of echolalia and pronominal reversal), and ritualistic or compulsive behavior. Beyond these three primary characteristics, a few additional symptoms were seen in most but not all cases, including self-injurious behavior, stereotyped repetitive movements, a limited attention span, and delayed bowel control (Rutter, 1978). Even with the quintessential components of the syndrome elaborated, Rutter (1978) encouraged further research on the disorder’s relationship with cognitive functioning as measured by IQ, age of onset, effect on neurological status, and minutiae of symptomatology.

Since 1968, the United States government has used the International Classification of Diseases (ICD) by the World Health Organization (WHO) as its certified diagnostic manual. As a result, APA’s official diagnostic manual, the DSM, has remained in concordance with the ICD since its second edition (Cohen et al., 1986). A report published the following year (Rutter et al., 1969) provided insight into the work being done at the time to classify childhood disorders for the 1975 revision of the ICD by the WHO. The task force in charge of the revision proposed a tri-axial classification method. Rutter, an influential member of the task force, advised that it include “behavioral syndrome, intellectual level, medical conditions, and psychosocial situation” (Rutter, 1978, p. 156). Each of the aforementioned dimensions would then be included on a separate axis in order to elucidate communication of a thorough case conceptualization between clinicians. Specifically, the task force recommended the first axis denote ‘clinical psychiatric syndrome,’ including the categories ‘developmental disorders’ and ‘psychoses.’ They suggested including infantile autism under the psychoses heading, within a necessary sub-category established for ‘infantile psychosis’ that develops in the first two to three years of life. Prior to the utilization of this multi-axial
A technique, disorders were primarily classified by etiology. Such a classification method proved less than helpful in disorders like autism, when the etiology was still a mystery (Rutter, 1972). Their suggestions appeared in the ninth edition of the ICD, published in 1977 (WHO), marking a change in the way diagnosis was executed and a step forward for autism.

After ICD-9’s (WHO, 1977) inclusion of autism, infantile autism (IA) finally made an appearance in 1980’s *Diagnostic and Statistical Manual of Mental Disorders, 3rd ed.*, (DSM-III; APA) under the rubric of ‘pervasive developmental disorder,’ accompanying childhood onset pervasive developmental disorder (COPDD) and atypical pervasive developmental disorder (APDD; Cohen et al., 1986). In this edition, the IA criteria were a slightly modified version of Kanner’s original characterization of the disorder in 1943: lack of response to others, issues with language development, peculiar speech if present, unusual reactions to the environment, and a lack of delusions and hallucinations as would be displayed in schizophrenia with onset prior to age 30 months (Cohen et al., 1986; Volkmar, Cohen, & Paul, 1986). The COPDD diagnosis was reserved for children who displayed autistic symptoms (including difficulties relating to people and at least three other prominent differences from resistance to change to abnormal movements and speech patterns, etcetera) after the cut-off of 30 months but before 12 years of age. APDD was much like today’s PDD-NOS, a categorization of children who display symptoms of disorders under the ASD umbrella but do not fit precisely the criteria for infantile autism or COPDD (Cohen et al., 1986; Volkmar et al., 1986). Volkmar et al. (1986) noted that it was uncertain whether IA and COPDD were separate disorders or simply differences in severity of the same disorder along a yet determined continuum.

In the 1987 revision of the DSM-III, the *Diagnostic and Statistical Manual of Mental Disorders, 3rd edition, revised* (DSM-III-R; APA, 1987), a few noteworthy changes took place regarding the criteria for ASDs. The revision included changing “infantile autism” to “autistic disorder” and “atypical” to “not otherwise specified (NOS).” Additionally, this edition introduced 16 criteria in the domains of social development, communication, activities, and interests, and changed the age criteria to onset during infancy or early childhood. Researchers concluded that the DSM-III-R made the criteria overly inclusive and resulted in false-positive diagnoses. It also deviated from the criteria in the ICD-10, which was said to result in negative consequences in regards to aligning future research (Tidmarsh & Volkmar, 2003).

The *Diagnostic and Statistical Manual of Mental Disorders, 4th edition* (DSM-IV; APA, 1994) was the result of the necessity of aligning criteria with the ICD-10. The DSM-IV added Rett’s Disorder and Childhood Disintegrative Disorder (CDD), making the diagnostic systems for PDD in ICD-10 and DSM-IV comparable. Additionally, the DSM-IV criteria for the disorders more closely aligned with that of the ICD-10, although appeared
to be more inclusive (Tidmarsh & Volkmar, 2003). Autistic Disorder was clearly defined, but Asperger’s Disorder and PDD-NOS were less specific, making differential diagnosis challenging and creating false positives.

These criteria were again revised in the *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision* (DSM-IV-TR) in 2000. The DSM-IV-TR class of Pervasive Developmental Disorders (PDD) included Autistic Disorder, Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS), Rett’s Disorder, Childhood Disintegrative Disorder, and Asperger’s Disorder (APA, 1994).

**Autistic Disorder.** Autistic Disorder received the most attention in terms of symptom description and assessment development (Matson et al., 2007). As a result, its criteria were particularly well defined. According to DSM-IV-TR criteria (APA, 2000), to qualify for a diagnosis of Autistic Disorder, an individual must have had significantly impairing deficits in social interaction and communication, as well as restricted repetitive and stereotyped behaviors, interests, and/or activities. A total of at least six items from the core autism symptoms must have been endorsed. Additionally, delays or abnormality in social interaction, language as used in social communication, or symbolic/imaginative play must have developed before the individual reached three years of age.

More specifically, at least two of the six or more symptoms must have been impairments in social interaction, including the following: (a) marked impairment in the use of multiple nonverbal behaviors (e.g., eye-to-eye gaze, facial expression, body postures, gestures to regulate social interaction); (b) failure to develop peer relationships appropriate to developmental level; (c) a lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g., by a lack of showing, bringing, or pointing out objects of interest); (d) lack of social or emotional reciprocity.

At least one of the six or more symptoms must have been impairments in communication, including the following: (a) delay in, or total lack of, the development of spoken language (not accompanied by an attempt to compensate through alternative modes of communication such as gesture or mime); (b) in individuals with adequate speech, marked impairment in the ability to initiate or sustain a conversation with others; (c) stereotyped and repetitive use of language or idiosyncratic language; (d) lack of varied, spontaneous make-believe play or social imitative play appropriate to developmental level.

At least one of the six or more symptoms must have been in the area of restricted repetitive and stereotyped patterns of behavior, interests, and activities, including the following: (a) encompassing preoccupation with one or more stereotyped and restricted pattern of interest that is abnormal either in intensity or focus; (b) apparently inflexible adherence to specific, nonfunctional routines or rituals; (c) stereotyped and repetitive motor mannerisms
(e.g., hand or finger flapping or twisting, or complex whole-body movements); (d) persistent preoccupation with parts of objects.

Finally, the exhibited problems could not be better accounted for by Rett’s Disorder or Childhood Disintegrative Disorder.

**Asperger’s Disorder.** Asperger’s Disorder was often conceptualized as a variant of Autistic Disorder, and some researchers in the field even used the terms high functioning autism (HFA) and Asperger’s Disorder interchangeably. However, using these terms interchangeably was a controversial practice, which had drawn greater attention due to the change in criteria in the DSM5, which supports the view of Asperger’s Disorder being a degree of Autistic Disorder by removing the diagnosis of Asperger’s Disorder altogether (Matson et al., 2007). Specifically, classification as HFA indicated a history of language delay but no associated intellectual delay, whereas Asperger’s Disorder indicated a lack of significant delay in language OR cognitive development (Nayate et al., 2012).

According to DSM-IV-TR criteria (APA, 2000), to qualify for a diagnosis of Asperger’s Disorder, an individual must have had significantly impairing deficits in social interaction and the presence of restricted repetitive and stereotyped patterns of behavior, interests, and/or activities. Additionally, one could not have a significant delay in language or cognitive development.

More specifically, one must have had at least two of the following impairments in social interaction: (a) marked impairment in the use of multiple nonverbal behaviors (e.g., eye-to-eye gaze, facial expression, body postures, gestures to regulate social interaction); (b) failure to develop peer relationships appropriate to developmental level; (c) a lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g., by a lack of showing, bringing, or pointing out objects of interest to other people); (d) lack of social or emotional reciprocity.

Additionally, one must have exhibited at least one of the following restricted repetitive and stereotyped patterns of behavior, interests, or activities: (a) encompassing preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus; (b) apparently inflexible adherence to specific, nonfunctional routines or rituals; (c) stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping or twisting, or complex whole-body movements); (d) persistent preoccupation with parts of objects.

Finally, one could not be diagnosed with Asperger’s Disorder if he or she met criteria for a different PDD or schizophrenia.
Pervasive Developmental Disorder, Not Otherwise Specified. With less attention devoted to research of PDD-NOS among the other ASDs, the disorder was defined commonly with the conceptualization that it was not autism (Matson et al., 2007). According to the DSM-IV-TR (APA, 2000), a PDD-NOS diagnosis was reserved for cases in which an individual had a severe and pervasive impairment in the development of reciprocal social interaction or verbal and nonverbal communication skills, or when stereotyped behavior, interests, and activities were present, but the criteria were not met for a specific PDD, Schizophrenia, Schizotypal Personality Disorder, or Avoidant Personality Disorder. Furthermore, this included atypical autism, which was implicated when the individual did not meet criteria for Autistic Disorder due to late age of onset, atypical symptomatology, subthreshold symptomatology, or all three.

Current Criteria

Because potential biological markers of autism have yet to be identified, diagnoses remain based on developmental background, behavioral observations, and the use of standardized tests (Klin, Lang, Cicchetti, & Volkmar, 2000). In most cases, this information is considered and matched to DSM criteria using clinical judgment to determine a diagnosis. Researchers (Klin et al., 2000) found that the criteria were beneficial in providing structure and guidance for diagnosing by persons with less experience with ASDs, and are thus preferable over clinical judgment alone for less experienced professionals (e.g., psychologists who do not focus on treatment for or research on autism). Thus, careful study of the current version of the DSM is recommended in order to establish diagnoses most effectively and accurately.

The diagnostic criteria for ASD according to the newest edition of the DSM, DSM5, is provided following. Further, symptomatology is discussed in depth, in the Symptomatology of Autism Spectrum Disorders section. Please note that some of the research findings reference past diagnoses according to now outdated diagnostic criteria.

Autism Spectrum Disorder. To receive a diagnosis of ASD according to DSM5, the individual must currently, or by history, meet all of the following criteria.

The individual must have persistent deficits in social communication and interaction across contexts, as manifested by all three of the following: (a) deficits in social-emotional reciprocity; (b) deficits in nonverbal communicative behavior utilized for social interaction; and (c) deficits in developing and maintaining relationships.

The individual must exhibit restricted repetitive patterns of behavior, interests, or activities, as displayed by at least two of the following: (a) stereotyped or repetitive speech, motor movements, or use of objects; (b) excessive adherence to routines, ritualized patterns of verbal or nonverbal behavior, or excessive resistance to change; (c)
highly restricted, fixated interests that are abnormal in intensity or focus; (d) hyper- or hypo-reactivity to sensory input or unusual interest in sensory aspects of the environment.

These symptoms must have been present in early childhood, but may not become fully manifest until social demands exceed limited capacities. Additionally, the symptoms combined must limit and impair the individual’s everyday functioning.

**Prevalence**

Once believed to be rare, ASD is now in the spotlight of public awareness due to its relative frequency. In 2002, Fombonne reported what he claimed to be conservative estimations of the prevalence of ASD, as garnered from an in-depth review of studies in English language journals from 1966 to 2001: 10 in 10,000 and 27.5 in 10,000, respectively. However, he hypothesized that, if studies coming out at the time (in the early 2000s) were replicated, the prevalence would be more accurately reported as 60-70 in 10,000. In 2007, the United States Centers for Disease Control reported relatively similar rates, as high as 1 in every 152 children (White, Oswald, Ollendick, & Scainhill, 2009). Matson and Shoemaker (2009) concurred, estimating ASD occurs in about 1 in every 150 children.

Early in the conceptualization of autism, researchers found a predominance of males being diagnosed with the disorder. Kanner and Eisenberg (1957) estimated a male to female ratio of 4 to 1 in 1957. More recently, Fombonne (2002) reported a 4.2 to 1 male to female ratio across surveys included in his review, indicating that this gender difference in diagnoses has remained stable. This discrepancy in prevalence between genders exists even in toddlers (Worley, Matson, Mahan, Kozlowski, & Neal, 2011). The difference appears to be especially obvious for those with an IQ within a normal range and less so for those individuals with profound intellectual disability (Bailey, Phillips, & Rutter, 1996). Despite this noteworthy gender disparity, most researchers agree that occurrence of autism does not seem to be influenced by other demographic components, including economic, social, racial, or ethnic background (Bertoglio & Hendren, 2009).

It is questionable whether the increase in prevalence in recent years is due to an actual increase in instances of the disorder or because of increased awareness and/or changes in diagnostic practice, including prior relaxing of diagnostic criteria. Matson and Shoemaker (2009) suggested that some increase could be due to the frequent comorbidity of ID and ASD. As ASD is better recognized, individuals whose symptoms were once considered the result of ID alone may now be more accurately attributed to a comorbid developmental disability, thereby increasing prevalence rates. Finally, early reports of prevalence were based on clinical rather than community samples, leading to potential underestimates (White et al., 2009).
Etiology

With the identification of ASD, naturally questions of its origin and causes followed. Etiological ideas were perhaps even more varied than diagnostic criteria, ranging from proposals of ‘emotional refrigeration’ during early development, to underactive or over-aroused brain areas, to dearth of proper conditioning by guardians (Rutter, 1968). Kanner believed there were genetic factors involved but was also influenced by the popular psychoanalytic nature of psychology at the time. Consequently, Kanner’s idea that autism may result from rearing by cold, humorless, uncaring parents was widely accepted among professionals, in large part due to the theory’s popularization by psychologist Bruno Bettelheim, until the end of the 1950s (Severson, Aune, & Jodlowski, 2007). Additionally, Kanner was confident that there was no physical pathology involved (Wing, 1997), while Eisenberg (1956) similarly argued against an anatomical ‘locus’ of the disorder.

In his review of popular theories of the time, Rutter (1968) rejected the majority in support of the idea that autism was the result of language and perceptual abnormalities. He cited language problems, along with intellectual ability, as the most crucial prognostic factor, remarking that such issues regularly persist despite improvements in other domains. Later in his career, Rutter came to support indications of the involvement of genetic factors (Wing, 1997).

The 1990s brought more awareness of neuropathology that could be involved and shifted the focus of research. Particularly, scientists believed that abnormalities occurred at the cellular level from early on in development. Psychological aspects of ASD, such as atypicality in language and communication, were being researched. Finally, the importance of genetic factors was salient at this time as well (Wing, 1997).

At present, ASD is viewed as a neurodevelopmental disorder with a biological foundation. A small percentage of cases are linked to a medical condition, such as genetic conditions Tuberous Sclerosis (about 1.2% average rate, according to Fombonne, 2002) and Fragile X (Yates & Le Couteur, 2009). Further, some environmental factors relating to the disorder have been reported. Controversial theories of a link between autism and the MMR vaccine or the mercury-containing vaccine preservative thimerosal have remained scientifically unsupported but continue to be influential among laypersons (Yates & Le Couteur, 2009). Contemporary etiological research has shifted toward increased focus on neurobiological differences in persons with ASD, spanning from studies of variation in neurotransmitters to functional and structural differences in assorted areas of the brain (Yates & Le Couteur, 2009). Additionally, it has become apparent that there is a genetic component to the disorder, but specifics have not been determined thus far (Ozonoff, Goodlin-Jones, & Solomon, 2005). Overall, conclusive
etiological results remain elusive at present, as results across studies remain inconsistent and no unique ‘autistic anomaly’ yet has been discovered (Ozonoff et al., 2005).

In regard to social factors, many researchers (including Kanner and Asperger) have noted that the parents of children with autism they have studied were frequently of above-average intelligence and are financially well off (Rutter, 1972). Kanner and Eisenberg (1957) mentioned that the majority of the children in their sample came from “highly intelligent, obsessive, and emotionally frigid backgrounds” (p. 60). They elaborated to say that the parents of the children with autism are almost universally dramatically detached, obsessive, and cold. Often the children had relatives whom were described as recluses or remained unmotivated to interact socially with others. Despite these accounts, the authors did not appear convinced these characteristics predicted the disorder, as they proceeded to mention several contradictory indications. Notably, 10% of the parents in their sample did not have these characteristics, some parents with the characteristics raised other neurotypical children, and there existed parents fitting the description who did not raise children who developed autism. Later they concluded that these factors likely play a role, but are not sufficient in and of themselves, in the development of autism. Today, most researchers agree that occurrence of autism does not seem to be influenced by economic, social, racial, or ethnic background (Bertoglio & Hendren, 2009). However, social or demographic variables should continue to be considered in studies, as they could have implications for aspects of ASD other than etiology.
Symptomatology of Autism Spectrum Disorders

As previously mentioned, symptoms of ASD begin to appear in very early development, often in the first few years of life (Matson, Wilkins, & Gonzalez, 2008). Autism is considered a pervasive disorder partly because it persists throughout the lifetime. Nevertheless, the form and quality of symptoms in all three domains of function commonly change over time (Ozonoff et al., 2005). These changes in symptoms only add to the heterogeneity in characteristics found across ASD. Although symptom presentation differs greatly among individuals on the spectrum, there are three domains in which abnormalities occur commonly, specifically, socialization, communication, and restricted and repetitive behaviors and interests (RRBI). Symptomatology within each of the three aforementioned domains is described in detail following. Because socialization is the domain of function studied specifically in this analysis, more depth regarding this area is provided.

Socialization

Deficits in the socialization domain are common early symptoms of ASD. Social issues appear to manifest as early as during the first year of life, but differences seem to be most apparent in the second year. However, researchers assert that the majority of critical social behavior is established by 6 to 9 months of age (Ozonoff et al., 2010). Social dysfunction ranges from the most basic aspects of social interaction, such as joint attention, to the general difficulty of establishing and maintaining relationships (Matson & Wilkins, 2007).

One major indication of the presence of an ASD is lack of joint attention. This concept refers to the simultaneous sharing of focus of two or more persons on an external stimulus including initiation and response (Baldwin, 1995). Initiation includes eye contact, declarative pointing, and gesturing, whereas response is a reaction to attention-seeking behavior (Murray et al., 2008). Joint attention skills are believed to differ based on intellectual functioning and developmental level (Travis & Sigman, 1998). The deficit has serious implications, including a negative impact on language development, as responsiveness to joint attention bids appears to be one predictor of language development in persons with autism (Travis & Sigman, 1998).

Another key issue in persons with ASD is lack of theory of mind. According to Bailey and colleagues (1996), deficits in theory of mind are extremely common, apparent in 80% of individuals with ASD and verbal abilities. Theory of mind is the recognition that other people have thoughts, beliefs, and feelings. Without this realization, individuals with ASD treat humans and objects alike, which can lead to inappropriate behavior and difficulties interacting in a social setting. Furthermore, theory of mind is necessary to understand irony and sarcasm. In individuals with autism, misunderstanding of irony may stem from misinterpretation of the speaker’s attitude and
intent, which could occur easily in a person who lacks the ability to view things from the speaker’s perspective (Pexman, 2008).

Additionally, orienting attention to social stimuli appears to be impaired in persons with ASD. This skill typically appears quickly, sometimes within the first few months of development, and deficits could be a sign of autism even before shared attention deficits are noticed (Dawson, Meltzoff, Osterling, Rinaldi, & Brown, 1998). Dawson and colleagues (1998) described evidence of lack of social orienting when they reported that children with autism commonly failed to direct their attention to a person who called their names or clapped their hands. For those who did direct attention to these social stimuli, the response was delayed in comparison to controls. The preference of children with ASD for non-social stimuli has important clinical indications, as social orienting is a basic skill that needs to be addressed before more complex social behaviors can be established.

Another abnormality found in persons with ASD concerns their ability to initiate and respond to facial expressions. This deficit often is noticed in young infants who exhibit a lack of social smiling or decreased display of positive affect (Zwaigenbaum et al., 2005). Further, this population seems to display more flat or neutral expressions and sometimes even ‘blends’ of emotions, creating expressions not seen in typical populations (Travis & Sigman, 1998; Yirmiya, Kasari, Sigman, & Mundy, 1989). Other researchers noted that children with ASD commonly display the extremes of emotions, often inappropriate considering the time or setting (Ricks & Wing, 1975). Individuals with autism also have demonstrated insufficient responses to the emotional signals of others (Travis & Sigman, 1998).

Another common deficit in individuals with autism is their limited spontaneous make-believe or pretend play. Pretend play, according to the definition suggested by Baron-Cohen (1987), includes “using an object as if it were another object, attributing properties to an object which it does not have, and/or referring to absent objects as if they were present” (p. 140). Wing and Gould (1979) noted ‘problems of imagination’ as a common characteristic of children with autism, who appear to have difficulties with non-literal behavior. Compared to typically developing populations, children with autism demonstrate significantly less spontaneous pretend play, both frequency- and duration-wise (Baron-Cohen, 1987; Jarrold, 2003).

Considering the aforementioned social difficulties, it is not surprising that individuals with autism have difficulty establishing and maintaining relationships with others. Struggles with interpersonal relationships likely are due to impairment of basic social skills, such as emotional responsiveness and social understanding. Therefore, interventions aimed at improving social relationships can be successful if these building blocks are primary aims. Many children with ASD are able to establish relationships despite their deficits. However, researchers have noted
that, after 5 years of age, children with ASD often can improve their relationships with adults, but interpersonal difficulties with peers seem to persist (Travis & Sigman, 1998). Unfortunately, it seems that even those children who progress in connecting with other people find that their lack of social perceptiveness causes the experience to be unpleasant, and thus prefer isolation (Eisenberg, 1956; Kanner & Eisenberg, 1957).

**Communication**

Communication problems prominent in ASD encompass both verbal and nonverbal domains. Notably, delayed or atypical patterns of language development are the primary first concern reported by parents who are worried about abnormalities in their children’s development, as these deficits appear early in development and are persistent (Bailey et al., 1996; Tager-Flusberg, Paul, & Lord, 2005). Issues with communication are important to consider because language function is a dependable predictor of educational and social outcome, as well as strongly correlated with severity of behavioral symptoms and social and cognitive abilities (Bailey et al., 1996). The symptoms in this domain are numerous and vary widely among individuals.

Various studies report that one-quarter to one-half of those individuals with autism never acquire functional speech (Bailey et al., 1996; Bryson, 1996; Kasari, Paparella, Freeman, & Jahromi, 2008; Noens & Berckeaer-Onnes, 2005). Although symptoms of communication disorders overlap significantly with symptoms of ASD (Matson & Neal, 2010), one major disparity is that persons with ASD generally do not compensate for lack of verbal language using nonverbal communication techniques; pointing, gestures, eye contact, and facial expression are often impaired or absent in this population (Howlin, 1998; Mundy, Sigman, & Kasari, 1994). Instead, children with ASD sometimes opt for pre-symbolic contact gestures, such as pulling a caregiver’s hand to lead him or her to something the child wants (Noens & Berckeaer-Onnes, 2005; Ricks & Wing, 1975). Ricks and Wing (1975) remarked that persons with autism do not find it easy or natural to perform any aspect of nonverbal communication. They maintained that any of these skills displayed by an individual with autism was a result of rote memory rather than normal maturation.

In anywhere from an estimated 15 to 30% of cases of individuals with ASD, parents report their children had some words between ages 12 to 18 months but then lost them (Kurita, 1985; Pickles et al., 2009; Xi, Hua, Zhao, & Liu, 2010). Language regression appears to be specific to ASD and not found among children with specific language impairment or other developmental delays (Lord, Shulman, & DiLavore, 2004; Pickles et al., 2009). The regression appears to occur when children have relatively small expressive vocabularies, before advanced linguistic skills have been established (Tager-Flusberg et al., 2005).
Fortunately, it seems that as early intervention becomes more common, the proportion of individuals to develop at least some spoken language is increasing (Tager-Flusberg et al., 2005). Beyond the obvious positive implications of this finding, it seems that children who develop functional language before entering school (i.e., before age 5) have a more positive prognosis overall than those who do not (DeMyer et al., 1973). Additionally, enhancement of language skills appears to cause decreases in challenging behavior used as an alternative communication method, such as aggression toward others or self-injurious behavior (Goldstein, 2002).

Beyond the lack of development of speech altogether, it has been suggested that the main impairment in communication is in pragmatic skills rather than formal and semantic aspects of language. Specifically, vocal quality and prosody appear particularly abnormal (Noens & Berckelaer-Onnes, 2005). Prosody involves such components of speech as pitch, rhythm, and stress patterns. Together, these components allow the speaker to indicate intended meaning, and the listener to make linguistic decisions such as recognizing and understanding syntax (Diehl, Bennetto, Watson, Gunlogson, & McDonough, 2008). Intact prosody comprehension and expression are necessary for effective communication. Many children with ASD, regardless of age, are impaired in understanding and using prosody, which can cause barriers to social acceptance. Prosody production patterns in individuals with ASD have been described variously, from flat and monotonous, to pedantic, to having a singsong quality. Such deviations from typical prosody have been related to significantly lower ratings of social and communicative function (Paul et al., 2005).

Additionally, children with ASD who do develop speech often display an idiosyncratic use of language, including echolalia (immediate and delayed), pronoun reversal, and neologisms (Noens & Berckelaer-Onnes, 2005). Echolalia is the meaningless repetition of words spoken by others, which can include phrases, sentences, or even entire conversations at times (Ricks & Wing, 1975). Children with autism may echo speech without interest in content, often imitating inflection or other vocal qualities of the speaker (Ricks & Wing, 1975). At times, children may fasten on to a certain phrase or collection of words and repeat them habitually in a stereotyped manner (Ricks & Wing, 1975). Pronominal or pronoun reversal refers to an error made when ‘you’ or another pronoun is used in place of ‘I.’ In Kanner’s first qualitative descriptions of children with autism, he noted that many children repeated personal pronouns exactly (1943). Pronominal reversal has been attributed commonly to the use of echolalia and to problems with differentiation of the self (Fay, 1979). Neologisms are ‘non-words,’ linguistic creations outside of standard lexicon but that hold meaning for the speaker (Volden & Lord, 1991). Idiosyncratic language refers to unusual uses of words or phrases to convey particular meaning (Volden & Lord, 1991). Thus, the main difference between neologisms and idiosyncratic language is that neologisms are nonwords. All of the abnormalities
mentioned can be conceptualized as the results of a gestalt processing style (i.e., processing locally rather than globally), a technique hypothesized as used by children with ASD in early language learning (Prizant, 1983).

Beyond the aforementioned unique language components, individuals with ASD often have difficulty maintaining conversation on topics beyond their restricted interests (Bertoglio & Hendren, 2009). Though some individuals are able to talk at length about their circumscribed subjects of interest, they are unlikely to be able to really discuss them or consider new angles about the topic (Ricks & Wing, 1975). To make matters worse, they are unlikely to recognize boredom in others and thus will continue on their monologue without regard to their audience (Ricks & Wing, 1975).

**Restricted and Repetitive Behaviors and Interests (RRBI)**

A heterogeneous core feature of autism includes repetitive and stereotyped movements, preoccupations with parts of objects, interests that are abnormal in intensity or content, and rigid completion of nonfunctional rituals or routines (Richler, Bishop, Kleinke, & Lord, 2007). Overall, these behaviors are characterized by “repetition, rigidity, invariance, and inappropriateness” (Turner, 1999, p. 839). Although social and communication issues may be more readily detected in younger individuals, the present category of dysfunction appears to be manifested in persons as young as 16-18 months of age (Honey, Rodgers, & McConachie, 2012). Additionally, it is believed that age and level of function of the individual affect the quality and prevalence of such behaviors (Esbensen, Seltzer, Lam, & Bodfish, 2009). For example, researchers (Richler et al., 2007; Szatmari et al., 2006) suggested that those with more severe developmental delays exhibit a greater amount of repetitive sensory and motor behaviors.

Researchers debate whether the criteria of RRBI should constitute a single category, or whether the heterogeneity among the behaviors included suggests that it should be broken down into several smaller domains. For example, Szatmari and colleagues (2006) suggested a two-factor structure including ‘repetitive sensory and motor behaviors and interests’ such as hand and finger movements, body movements, and vocalizations, as well as ‘insistence on sameness’ or ‘resistance to change,’ such as difficulty coping with changes to routine or environment and compulsions and rituals (Szatmari et al., 2006). Other experts separate the behaviors into lower-order behaviors, including repetitive movements such as self-injurious behavior (SIB) and manipulation of objects, and higher-order behaviors, including complex behaviors such as repetitive language and atypical interests (Esbensen et al., 2009).

Circumscribed interests are argued to be the ‘highest-order’ behavior in this domain (Esbensen et al., 2009). Highest order behavior may include preoccupations that are unusual in intensity or focus. These can range from atypical preoccupations with certain objects, to more common interests exaggerated to an extreme degree, like
an intense obsession with trains or other topics. Such interests commonly do not transform into adaptive skills or functional hobbies without intervention (Boyd, Conroy, Mancil, Nakao, & Alter, 2007). Further, these obsessions frequently interfere with social gains. Individuals with autism either want to accumulate facts about or engage in their interest rather than interact with others, or they will insist on only discussing their topic of interest regardless of the desires of the person with whom they are interacting. Despite the negative consequences of circumscribed interests, some researchers believe that these preoccupations can be used as motivation for social interaction for some children (Boyd et al., 2007).

When referring to a population with autism, the phrase ‘insistence on sameness’ at times can be defined as more than solely resistance to change. Its utilization can refer to an almost obsessive concern with placement and arrangement of objects or people, a rigid approach to daily routine, or a strict diet of foods with similar textures, among many other explanations. Individuals with autism differ from other persons displaying an insistence in sameness in that theirs commonly extends beyond personal restricted behavior to that of other people (Turner, 1999). That is, they might insist that their caregivers follow routines as well. Additionally, they seem to showcase more severe reactions to an inability to follow their routines. Extreme distress and temper outbursts are common results of disruption in routine in individuals with ASD but not necessarily in individuals without ASD who follow routines (Turner, 1999). Thus, individuals with ASD present as more cooperative and calm when routine and predictability are maintained, and when advance preparation for change is provided (Lainhart, 1999). Szatmari and colleagues (2006) found that individuals with less typical communication skills were more likely to display insistence on sameness, and, further, that individuals with ASD had higher ratings of the quality as well.

The type of RRBI that commonly has the worst consequences is SIB, aggressive behaviors directed toward the self. This behavior frequently presents as head banging, biting, scratching, or hair pulling. Baghdadli, Pascal, Grisi, and Aussilloux (2003) reported that 53% of their sample of children with autism exhibited SIB. According to their results, lower chronological age, more impaired daily living skills, severity of autism, and associated medical conditions all increased risk for SIB. Of particular note was their finding that about 15% of cases rated the presenting SIB as severe. With high prevalence and high severity ratings, SIB among individuals with autism has negative implications for the child, the family, and consulting professionals. Specifically, severe SIB can result in injury and even death at its most serious, while less severe consequences include disruptions to daily functionality, educational opportunities, and access to the community.

RRBI seems to have been researched the least of the core features of autism. Of the behaviors included in this category, repetitive movements are the most commonly studied of the behaviors in this area, as they are easily
observed and quantified. Unfortunately, there is a dearth of literature addressing the etiology and function of the RRBI behaviors. RRBI behaviors are an important area to study because of the behaviors’ serious implications in the lives of those who demonstrate them. RRBI can result in difficulty with skill acquisition, decreased opportunities for positive social interactions, and can be stigmatizing (Honey et al., 2012). Moreover, SIB can result in serious injury and even death (Baghdadli et al., 2003). The lack of a precise conceptualization or definition causes problems for assessment, differential diagnosis, and treatment.
Psychopathology in Early Childhood

Infant and toddler mental health is a relatively new topic of interest (Carter et al., 2010). Evidence that psychopathology exists in early childhood (birth to 3 years) continues to grow. The idea that such problems are transient has been disproven by researchers who posit that early childhood mental health issues continue to affect the individual as he or she develops and/or predicts later problems (Briggs-Gowan et al., 2006; Campbell, 1995; Carter et al., 2010; Keenan et al., 1998; Mathiesen & Sanson, 2000; Mesman & Koot, 2001). For example, Mesman and Koot (2001) found that preschoolers with internalizing symptoms were three times more likely to have a later internalizing problem than preschoolers without such symptoms. This trend appears to occur across categories of symptoms, with at least moderate stability of internalizing and externalizing problems (Lavigne et al., 1998). The majority of research regarding persistence of psychopathology in early childhood has not included examination of gender differences. Nevertheless, some evidence has emerged that persistence of problems may differ between boys and girls, with internalizing disorders appearing more persistent in boys than girls, but no differences apparent regarding the persistence of externalizing disorders (Lavigne et al., 1998).

In young children, psychopathology is frequently thought of in terms of internalizing and externalizing categories of maladaptive behavior (Keenan & Shaw, 1997; Mesman, Bongers, & Koot, 2001). In general, the words ‘internalizing’ or ‘internalization’ signify disordered mood or emotion, as evidenced by behavior that directs feelings inward. In early childhood, internalizing problems may include difficulties with depression or withdrawal, anxiety, fears, and shyness or inhibition (Briggs-Gowan et al., 2006). Internalizing behavior is in opposition to externalizing behavior, which is the expression of feelings through overt behavior (Kovacs & Devlin, 1998). For infants and toddlers, externalizing behavior may include aggression, overactivity, impulsivity, and inattention (Briggs-Gowan et al., 2006).

Perspectives on internalizing problems in early childhood have shifted recently. Although this category of problems was oft neglected in research, interest in the topic has increased due to new knowledge that reflects the ability to study internalizing symptoms among infants and toddlers. For instance, evidence exists that not only are children capable of internalization, but that typical presentation of internalizing symptoms occurs in early childhood (Sterba, Prinstein, & Cox, 2007) and can be reliably identified in that period (Carter et al., 2010). As a result, excluding this age group from this topic of study should no longer be a methodological necessity (Carter et al., 2010; Sterba et al., 2007).

Identification of symptoms during early childhood is important not only to alleviate impairments throughout that period, but also to improve prognosis in later years. Earlier intervention for problems appearing during this stage of
development may be more effective because maladaptive behaviors are less entrenched and behavioral control is beginning to emerge and develop (Keenan & Wakschlag, 2000). Problems in early childhood can lead to deficits in social competence and academic functioning later in development (Keane & Calkins, 2004). Thus, early identification and analysis of psychopathology in infants and toddlers, followed by effective intervention, is important because problems during this time period in development can affect numerous aspects of later life.
Internalizing Problems

Diagnosing Comorbid Conditions

Individuals with ASD can have any developmental, medical, or mental health condition that typically developing individuals may face (Yates & Le Couteur, 2009). Persons with ASD may have psychiatric difficulties due to a number of factors, including significantly impairing symptoms of autism itself, cognitive deficits, comorbid psychiatric and medical disorders, difficult or negative experiences related to life with the disorder, or any combination thereof (Lainhart, 1999). Simonoff and colleagues (2008) found that 70% of their sample of children and adolescents with ASD had at least one comorbid psychiatric disorder, while 41% presented with two or more. However, it is controversial whether or not symptoms of these conditions, especially those of psychopathology, should be considered inherent to ASD or an aspect of a co-existing problem. Making a decision regarding this facet of differential diagnosis is important because of its implications for research and clinical practice.

Two potential methods of identifying psychopathology in persons with ASD are common and include diagnosing all symptom patterns that meet some diagnostic criteria or associating such patterns with ASD, the primary disorder (Simonoff et al., 2008). The latter method may result in “diagnostic overshadowing,” the tendency of a clinician to attribute certain symptoms to the primary diagnosis of developmental disability rather than a separate comorbid condition. The controversial issue of which technique to use will likely remain unresolved until applicable neurobiological and genetic discoveries are made. Additionally, because of the psychosocial and behavioral implications of ASD, it is possible for autism to “mask” symptoms of other comorbid disorders, making them difficult or impossible to identify (Ozonoff et al., 2005). Cognitive abilities may influence symptom presentation as well (Ozonoff et al., 2005).

Of course, in addition to creating diagnostic difficulties, emotional and behavioral disruptions have a number of other effects warranting their proper recognition. Such effects include creating stress and diminishing quality of life for the child and his or her caregivers and family, increasing the likelihood of social isolation for the child, and increasing the prospect of administration of psychotropic drug therapy for the child (Lecavalier, Gadow, Devincent, Houts, & Edwards, 2011). In fact, in some cases, the consequences of the presence of additional comorbid disorders are of even greater concern than the ASD symptoms exhibited (Pearson et al., 2006).

Internalizing Disorders

Although prevalence rates of internalizing disorders are lower in children than adult populations (Kovacs & Devlin, 1998), estimates suggest rates of depression range from 1% to 5.9% and prevalence of anxiety is about 8.9%
in samples of typically developing children (Costello, 1989). Few psychopathology prevalence studies have been conducted utilizing toddler samples; estimates of parent-endorsed social-emotional and behavior problems among toddlers range from 7% to 24% (Briggs-Gowan, Carter, Skuban, & Horwitz, 2001). Clearly, overall frequency estimates for internalizing disorders vary widely across studies, and such approximations are more difficult to ascertain than estimates for externalizing disorders (Merrell, 2008). Additionally, such studies frequently use a clinically referred rather than an epidemiological sample, which can alter representativeness (Lainhart, 1999). Finally, internalizing disorders commonly occur together and with externalizing disorders (Kovacs & Devlin, 1998), rendering it difficult to disentangle symptoms. Prevalence figures should be considered with these shortcomings in mind.

Much like the prevalence rates for typically developing populations, estimates for internalizing behaviors in the ASD population vary widely among the research literature and across age groups. Despite the discrepant data, researchers generally agree that internalizing problems such as anxiety (MacNeil, Lopes, & Minnes, 2009) are found more frequently in individuals with ASD than among their peers without ASD. Indeed, anxiety and avoidance problems appear to be more prevalent among infants and toddlers with ASD than among those who are typically developing (Davis III, Fodstad, et al., 2011). Differences in incidence of such issues exist even between atypically developing infants and toddlers with and without ASD. For instance, in an infant and toddler sample, all varieties of comorbid psychopathology as measured by the BISCUIt Part 2 were more pronounced among the group with ASD compared to the group with atypical development but no ASD diagnosis (Matson, Hess, et al., 2010). More specifically, Horovitz, Matson, and Sipes (2011) found that their ASD groups (Autistic Disorder and PDD-NOS) had significantly greater scores than their atypically developing group on the Anxiety/Repetitive Behavior and Avoidance subscales of the BISCUIt Part 2.

Furthermore, in a study by Volkmer et al. (2010), the authors compared typically developing children matched for age, gender, and ethnicity to individuals with high-functioning autism spectrum disorders (HFASD). They described the group with HFASD as being persons with HFA, Asperger’s Disorder, or PDD-NOS, who display relative strengths in cognitive ability and language levels. Their results indicated that typically developing participants and participants with HFASD were significantly different in terms of endorsements of anxiety and depression but not somatization, as indicated by scores on the Behavior Assessment System for Children, Second Edition (BASC-2; Reynolds & Kamphaus, 2004). Mahan and Matson (2011) used the same measure and reported slightly discrepant results, indicating that children and adolescents with ASD scored significantly higher on the depression and somatization subscales than their atypically developing counterparts. However, contrary to previous
research, they did not find significant differences in scores on the anxiety subscale or the internalizing composite. The researchers suggested that their lack of statistically significant findings for anxiety could be the result of some endorsements on the scale necessitating verbal communication by the child (e.g., items like “says I’m afraid I will make a mistake”), and 7.9% of their ASD group lacked ability to communicate verbally. Additionally, Mahan and Matson (2011) proposed that the presence of intellectual disability in a large percentage of their ASD group (18.4%) could limit potential for engaging in effective verbal communication. They noted that it is possible that the lack of significant difference in anxiety was responsible for the lack of significant difference in the internalizing composite as well.

Overall, ASD appears to make individuals especially vulnerable to internalizing problems (Mayes, Calhoun, Murray, Ahuja, & Smith, 2011a). Indeed, anxiety (Skokauskas & Gallagher, 2010) and depression are the most commonly reported comorbid conditions within the spectrum (Howlin, 1997) and are reported as more common than externalizing disorders in this population (Kim et al., 2000). Furthermore, the majority of children with ASD have anxiety symptoms, whereas about half have depression symptoms (Mayes et al., 2011a). These authors found that the most powerful combined predictors of anxiety and depression were autism severity, verbal IQ, and age. They proposed that this finding demonstrates that internalizing problems are fundamentally interconnected with autism (increasing with autism severity) and have a developmental aspect (increasing with age and IQ). Additionally, Kim and colleagues (2000) suggested that the increase of depression in autism may be a natural result of environmental stressors, which this population seems to face frequently, including major changes in educational programs/school or therapy, placement in and changes at a group home setting, parental marital discord, or the presence of psychopathology in the family (Ghaziuddin, Alessi, & Greden, 1995).

Compared to their typically developing counterparts, individuals with ASD appear to overlap significantly in how internalizing problems manifest behaviorally. However, there are minor differences in presentation, thus careful assessment is necessary. As in the general population, depressive symptoms among an ASD population may include social withdrawal, sleep problems, and appetite disturbance (Mayes et al., 2011b). In typically developing individuals, the characteristic indication of depression is a negative change in mood (Lainhart & Folstein, 1994). Although depressed mood is frequently reported among individuals with autism, other symptoms of depression such as feelings of worthlessness, reduced capacity to concentrate, and suicidal ideation are considered rare among this population (Stewart, Barnard, Pearson, Hasan, & O’Brien, 2006). Lainhart (1999) reported somewhat conflicting findings, noting that presenting complaints of depressed mood were unusual; instead, caregivers reported the occurrence of “new or worsened agitation, aggression, self-injurious behavior, temper outbursts, social withdrawal,
vegetative changes in sleep or appetite, increased compulsive behaviors, hypoactivity and inertia, or general deterioration in functioning at home, at school, or at work” (p. 287). Vegetative behaviors seemed to be the most easily recognized in individuals with autism, but these characteristics were difficult to interpret among those with chronic sleep or energy disturbances (Lainhart & Folstein, 1994). Furthermore, Lainhart and Folstein (1994) noted that during episodes of depression, individuals with autism appeared even more withdrawn, passive, and uncooperative than when measured at baseline. As discussed previously, level of functioning could be the source of the discrepancy between the common presenting symptoms reported by Stewart and colleagues (2006) and Lainhart (1998).

Anxiety is difficult to recognize in ASD populations because symptoms are often attributed to autism rather than anxiety (e.g., repetitive questioning; Kim et al., 2000). Indeed, some experts argue that problems such as social avoidance and ritualistic or compulsive behavior are not attributable to a separate anxiety disorder, but rather to ASD (Kerns & Kendall, 2012). However, conceptual differences appear to exist; for instance, individuals with ASD may withdraw from social situations as a result of non-social factors, such as loud noises, rather than due to overwhelming anxiety (Leyfer et al., 2006). Furthermore, distinguishing whether the issues are consistent deficits or dependent on situation and environment may help make distinctions between core ASD symptoms and anxiety-related problems. Stable behaviors are likely core ASD symptoms, whereas dynamic behaviors could be indications of a comorbid anxiety disorder (Kim et al., 2000). Additionally, anxiety may be manifested in different manners in individuals with autism than in their typically developing peers. For instance, symptomatic behavior among individuals with ASD may include obsessive questioning or insistence on sameness instead of more common symptoms like rumination of thoughts or somatic complaints (Ozonoff et al., 2005). The discrepancy in symptoms results in an additional degree of complication in recognizing anxiety in this population. Although challenging to accomplish, the distinction between ASD and anxiety is important to make, particularly for case conceptualization and treatment implications.

The most prevalent issues regarding anxiety among children with ASD include symptoms characteristic of specific phobias, GAD, separation anxiety disorder, OCD, and social phobia, with panic disorder the least prevalent (White et al., 2009). Anxiety symptoms appear to be present across all levels of cognitive functioning, but may vary with ASD symptom severity. MacNeil and colleagues (2009) addressed this issue; in four of the six studies they reviewed, there was an inverse relationship between severity of ASD symptoms and presence of anxiety issues. Indeed, much research supports a differentiation in internalizing symptoms according to autism symptom severity. Specifically, it seems that individuals with HFA have more internalizing problems than those with lower functioning
autism (LFA), persons with Autistic Disorder and intellectual disability. For example, Mayes et al. (2011a) reported that maternal ratings for 350 children with autism indicated that 79% of children with HFA (IQ ≥ 80) and 67% of children with LFA (IQ < 80) had anxiety, and 54% children with HFA and 42% of children with LFA had depression.

Finally, as discussed above, comorbid conditions such as internalizing problems create diagnostic difficulties, as well as have assorted other more practical, daily consequences. Specifically, internalizing problems including anxiety and mood issues may cause an increase in aggression in children with HFA (Kim et al., 2000). Additionally, these problems may limit parents’ or caregivers’ social activities, and may result in poorer relationships between these children and their teachers, peers, or family members (Kim et al., 2000). Considering these and other serious costs related to internalizing problems as comorbid conditions with autism, it is surprising that there is not yet substantially more research on these disorders and potential risk factors, such as gender, in the field. Findings on such topics could have important implications for treatment and result in greater quality of life in the autism population.
Gender Differences

Autism Severity and Symptomatology

Gender differences in ASD (other than prevalence disparities) have received minimal attention in the research field (Worley & Matson, 2011). The lack of work on this topic is likely due to the preponderance of males diagnosed with ASD as compared to females, which has resulted in a lack of separation of gender groups in, or even females’ exclusion from, research studies (Hartley & Sikora, 2009; Lord, Schopler, & Revicki, 1982). However, the current literature suggests several important, albeit at times subtle, gender differences that have implications for assessment and treatment and thus warrant discussion and further study.

Autism is generally identified earlier among females than males (Short & Schopler, 1988). Furthermore, research has shown that, overall, females with autism have more severe developmental delays, in that they have lower intelligence and adaptive functioning, when compared to males with autism (Lord et al., 1982). It is important to note that Lord et al. (1982) found that proportions of females at each intellectual level were fairly similar to those of males, but that actual scores on applicable measures were several points lower for the female participants. Short and Schopler (1988) supported this finding with their report of IQ differences between the males and females in their study. However, they noted that the aforementioned earlier onset in females could be a corollary of the IQ data, as more severe symptoms could have resulted in earlier identification. Relatedly, Carter et al. (2007) determined males showed more advanced development than females, including stronger verbal and motor skills. However, Hartley and Sikora (2009) indicated similarity between the genders on an overall developmental profile of strengths and weaknesses (i.e., visual reception and fine motor skills were better developed than language skills). Carter and colleagues (2007) found nearly an exact same pattern.

Most current research has indicated few if any major differences in ASD symptomatology between males and females. Solomon, Miller, Taylor, Hinshaw and Carter (2012) indicated similar autism symptom profiles in boys and girls with ASD. Hartley and Sikora (2009) also found only a few, subtle differences in autistic symptom patterns between genders. Specifically, their data supported previous findings that indicated males have more stereotyped and repetitive behaviors than females, which they argue is the most consistent gender discrepancy found thus far. Concurrently, Sipes, Matson, Worley, and Kozlowski (2011) discovered significant discrepancies between male and female toddlers only in the domain of RRBI. Any other significant differences found in other domains lost significance when developmental level and IQ were considered. That is, developmental level and IQ contributed to differences in other domains, with lower functioning associated with increased impairment in the majority of symptom areas (Sipes et al., 2011).
Despite the majority of the literature on this topic indicating only minor differences between the genders, even slight disparities could impact assessment and treatment practices and are thus worth consideration. Carter et al. (2007) determined that boys in their sample of toddlers with ASD had more advanced social functioning than girls, as determined by scores on the Vineland Adaptive Behavior Scales (Sparrow, Balla, & Cicchetti 1984) and the Infant-Toddler Social and Emotional Assessment (Carter et al., 2003). Both of the aforementioned measures consist of items specifically for the toddler age group examined in the study by Carter et al. (2007). In a different population, a sample of children and adolescents with HFA, McLennan, Lord, and Schopler (1993) found that as females reached older childhood and beyond, they had more severe impairments in social skills, especially in regard to peer relationships, than their male counterparts when matched for age and IQ. Unlike previous research, which indicated worse social reciprocity in males than females, Hartley and Sikora (2009) did not find such a difference. However, their sample included both children with HFA and LFA, and they note that this difference may be a result of HFA-only samples. Additionally, these researchers found that girls in their sample had significantly worse communication skills than boys, even after controlling for age and cognitive functioning, a pattern noted in prior research.

The above-described studies indicating poorer social skills among females with ASD seem to contrast patterns found in typically developing individuals. In a typically developing population, girls appear to have better social competence than boys (Briggs-Gowan et al., 2001). Furthermore, in early childhood, communication and social skills, emotional development, and self-regulation abilities appear earlier among typically developing females than their male counterparts (Keenan & Shaw, 1997).

**Internalizing Problems**

For the population as a whole, it is widely accepted that internalizing problems are more common in females beginning even before adolescence and throughout adulthood (Keenan & Shaw, 1997; Lewinsohn, Gotlib, Lewinsohn, Seeley, & Allen, 1998; Nolen-Hoeksema, 1990). However, in early childhood, males and females in the general population appear to exhibit similar rates of internalizing problems (Keenan & Shaw, 1997). Overall, compared to males, females seem to have less continuity in terms of psychopathology; patterns suggest that females demonstrate difficulties in early childhood, which remain stable or do not increase during childhood, followed by an escalation in internalizing problems during adolescence and adulthood (Keenan & Shaw, 1997).

Similar differences seem to exist between the genders among individuals with ASD, with females having greater difficulty with anxious or depressed affect than males (Hartley & Sikora, 2009). Likewise, Solomon and colleagues (2012) reported that adolescent girls with autism were significantly different from both boys and
typically developing girls in terms of internalizing problems, with 26% of adolescents self-reporting clinical levels of related symptoms (compared to 0% in the other two groups). Additionally, parent reports from this study indicated comparable findings. The aforementioned autism studies support the idea that female gender increases risk of developing symptoms of anxiety and/or depression comorbid with ASD.

Interestingly, other recent research has indicated that internalizing disorders are not affected by gender in some ASD samples (Mayes et al., 2011a; Worley & Matson, 2011). It is possible that the lack of difference in internalizing problems between genders is a result of more severe symptom presentation among females. As mentioned previously, the link between more anxiety symptoms present in higher functioning individuals with autism may be a fallacy, and the interaction between gender and severity of symptoms could be affected as well. Furthermore, Worley and Matson (2011) posited that their lack of significant differences in psychopathology among males and females might have been a result of a large age range. Specifically, these authors suggested that their inclusion of both children and adolescents might have masked differences usually thought to have adolescent onset. In particular, it is commonly thought that anxiety worsens in individuals with ASD as they reach adolescence because they are likely to increasingly notice their differences from peers. This resulting increased anxiety could compound overall social impairment, leading to further isolation and social difficulties. However, a recent study (Davis III, Hess, et al., 2011) reported that anxiety symptoms fluctuate across the lifespan in individuals with ASD. Specifically, according to these authors, symptoms appear to accumulate through childhood and decrease during adolescence and young adulthood. With this idea in mind, it remains possible that a broad age range influenced the results of the study by Worley and Matson (2011), as differences in symptoms across the ages of their sample remain proposed.

Among an early childhood population, the age group of focus in the present study, research results vary in regard to differences in internalizing issues between males and females. On a particular measure of maladaptive behavior, the Child Behavior Checklist for ages 1.5 through 5 years (CBCL; Achenbach & Rescorla, 2000), informant endorsements regarding the problems of toddlers with ASD demonstrated that girls had more sleep difficulties and issues with anxious or depressed affect than boys (Hartley & Sikora, 2009). However, on a scale that assesses comorbid psychopathology among toddlers with ASD, the BISCUIT Part 2, the three most frequently and the three least frequently endorsed items were identical for males and females (Horovitz et al., 2011). The latter results suggest that co-occurring problems among male and female toddlers with ASD are qualitatively similar.

Considering these vastly discrepant findings, further research needs to be conducted regarding the interaction between gender and psychopathology comorbid with autism. Such investigation should consider
individuals of varying ages. The aim of the present study is to examine the role of gender as a moderator of the interaction between a core domain of deficits in autism, socialization, and internalizing problems among an infant and toddler sample.
Socialization and Internalizing Problems in Early Childhood

Despite advances in public policy that mandate intervention services for early childhood socialization and emotional problems, these services have lagged behind the higher priority delays in cognition, language, and motor development (Briggs-Gowan, Carter, Irwin, Wachtel, & Cicchetti, 2004). This pattern is especially problematic considering that social-emotional competence and the persistence of emotional and behavioral problems appear to be related (Masten & Coatsworth, 1995). To illustrate, in a toddler sample, approximately 32% of participants with subclinical or clinical scores on the CBCL exhibited delayed social-emotional competence (Briggs-Gowan et al., 2001). Competence in this domain may help decrease persistence of early problem behaviors. Greater skill development in this area likely corresponds to “a broader and more adaptive repertoire of responses,” meaning the child with better social competence may be able to react more appropriately to or cope with challenges (Briggs-Gowan et al., 2001, p. 812). Further, with an increased ability to react appropriately, the child may receive more positive interpersonal interaction (Briggs-Gowan et al., 2001). Both improved coping skills and increased positive interaction could contribute to a decrease in problem behavior.

Mesman and colleagues (2001) note the relationship between socialization and psychopathology at school entry, when preschoolers are faced with new social demands, such as making friends. These authors stress that adaptation to unfamiliar social situations is crucial to further development of more than just interpersonal skills. Behavior and emotional problems present in early childhood can hinder successful social adaptation, which may subsequently lead to later development of additional internalizing and externalizing symptoms. As such, deficits in social skills may be a source, as well as a consequence, of internalizing problems. Mesman and colleagues (2001) presented a representative example: difficulty adapting to new social situations may result in sadness and negative self-evaluation, whereas decreased interest and energy may result in more difficulty with social function. Even with proposed relative socio-emotional strengths, females are not any more protected from developing early internalizing pathology than males (Sterba et al., 2007).

Furthermore, an additional pathway is posited in which externalizing problems, such as aggression, may hinder successful social adaptation, fostering internalizing problems, such as depressive symptoms and withdrawal. In these pathways, socialization plays a major role in the development or maintenance of internalizing issues. In their study, Mesman and colleagues (2001) found that deficits in social skills at school entry significantly influenced preadolescent psychopathology, but, interestingly, only among males.

Because typically developing females generally develop social skills earlier than their male peers and, relatedly, have better social and emotional abilities in early childhood, they may experience less adversity adapting
to new situations (Keenan & Shaw, 1997). As such, their easier passage through developmental challenges may
decrease their comparative likelihood to develop and maintain internalizing symptoms.
Purpose

Although currently researchers support the idea that internalizing problems are more prevalent in individuals with autism than their typically developing counterparts, prevalence rates vary widely and little elaborative research has been conducted. Most of the research that has been done on this topic relates to the influence of autism symptomatology on the development of internalizing disorders (e.g., the study by Davis III, 2012, which examined the effect of communication deficits among those with ASD on their development of comorbid anxiety). Additionally, researchers recognize that several internalizing problems (i.e., symptoms of anxiety and depression) are more common in women than men in the general population (Lewinsohn et al., 1998; Nolen-Hoeksema, 1990). However, gender differences as a whole among those with autism have received little attention (Worley & Matson, 2011), much less the effects of gender on comorbid conditions. As a result, it appears that researchers have not specifically looked at gender as a moderator of the association between autism symptoms and comorbid internalizing problems. Thus, the purpose of the present study is to delve into this topic with a preliminary examination of the influence of gender on the predictive relationship between socialization, as rated on the Battelle Developmental Inventory, Second Edition (BDI-2; Newborg, 2004), and internalizing problems, as measured by the BISCUIT Part 2 (Reynolds & Kamphaus, 2004), in a sample of toddlers with and without ASD. Socialization in particular was chosen as the symptomatology to study because it is often considered the defining characteristic or core deficit of ASD (National Research Council, 2001). Additionally, as described, social deficits and internalizing problems appear to be closely related.

Findings from this study could have important implications for the assessment and treatment of internalizing problems in individuals with ASD. If gender differentially affects the role of socialization in development of internalizing problems, this link may encourage differences to be made in terms of identification of and treatment for internalizing problems in males versus females with ASD. For instance, if socialization deficits put females more at risk for development of internalizing problems, females lacking appropriate social skills can be assessed for internalizing symptoms more in depth and earlier. As a result, intervention aimed at discouraging development of such problems could be initiated earlier and remain a primary focus. Increased consideration of gender as an influence on individual differences in symptomatology could lead to more appropriate assessment and treatment plans, improving outcomes and quality of life. Finally, if significant results are discovered, perhaps this additional evidence will encourage further inquiry regarding gender differences in individuals with ASD, an important topic that lacks sufficient research at present.
Based on extensive literature review, several hypotheses were formed regarding the results of the present study. First, the author expects that infants and toddlers with ASD will have greater endorsements of internalizing symptoms (i.e., combined Avoidance and Anxiety/Repetitive Behavior subscales of the BISCUIT Part 2) than their atypically developing peers without ASD diagnoses. Additionally, because socialization deficits are a characteristic symptom of ASD, the author predicts that infants and toddlers with ASD will have lower scores on the Personal-Social domain of the BDI-2 than their atypically developing peers without ASD diagnoses.

Although internalizing problems are more common among females in the general population and among the majority of autism populations (Hartley & Sikora, 2009; Solomon et al., 2012), researchers have indicated few differences between typically developing male and female toddlers in terms of internalizing issues (Keenan & Shaw, 1997). Findings vary regarding differences between the genders among an early childhood population with ASD, with some researchers suggesting females have more difficulty with anxiety (Hartley & Sikora, 2009), and others positing that problems at this age are qualitatively similar between the genders (Horovitz et al., 2011). Considering all of the above, the author predicts that atypically developing female infants and toddlers with and without ASD will have greater endorsements of internalizing symptoms than their male counterparts.

In a typically developing population, females gain social and emotional skills sooner and have better abilities in these areas during early childhood (Briggs-Gowan et al., 2001; Keenan & Shaw, 1997). In contrast, male toddlers with ASD appear to have more advanced social skills than their female counterparts (Carter et al., 2007). Based on these findings, the author of the present study predicts that the atypically developing females without ASD will have the highest socialization scores, indicating the most advanced social skills, but also that the males with ASD will have higher socialization scores than the females with ASD.

Development of social and emotional competence appears to influence maintenance of internalizing symptoms. In a typically developing population, this relationship likely affects the lack of continuity in psychopathology found among females; because girls have earlier-developing and greater social and emotional abilities, they adapt more readily to challenges and thus avoid continuation of internalizing symptoms. In contrast, among a population with ASD, males appear to be more skilled in the social domain than females. As such, the author of the present study predicts that male toddlers with ASD will have fewer internalizing symptoms than female toddlers with ASD due to their greater social-emotional competence. Thus, the author predicts that gender moderates the effect of socialization on internalizing problems, in that a larger effect of socialization on internalizing problems overall would be seen in females than in males. The addition of the interaction term (socialization x
gender) is hypothesized to account for significant unique variance in the outcome variable (internalizing problems), resulting in a two-way interaction.
Method

Participants

The participants for this study consisted of 2,338 infants and toddlers ranging from 17 to 36 months of age \((M = 25.67, SD = 4.63)\). The included infants and toddlers were recipients of services from EarlySteps. EarlySteps is Louisiana’s Early Intervention System under the Individuals with Disabilities Education Act, Part C, which provides services to infants and toddler and their families from birth to 36 months. Children qualify if they have a developmental delay (e.g., ASD or ID) or a medical condition likely to result in one (e.g., cerebral palsy, epilepsy, deafness, blindness, premature birth). A pediatrician or other health care specialist must make such a determination.

The included individuals were selected from a pre-existing database that contains demographic information and results of developmental and diagnostic assessment measures. The data is collected on an ongoing basis by EarlySteps service providers, as described in the following Procedure section. Of the original pool of individuals, participants in this study were included if their \textit{BISCUIT Part 1}, \textit{BISCUIT Part 2}, and \textit{BDI-2} scores were available and free of measurement errors. Of the included infants and toddlers, 71.5\% were males. Additionally, 51.4\% were Caucasian, 39.5\% were African American, 2.5\% were Hispanic, and 5.9\% were other ethnicities or did not specify. Further demographic information is provided in Table 1.

<table>
<thead>
<tr>
<th>Table 1 Overall Sample Demographics (n = 2338)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in years</td>
</tr>
<tr>
<td>Range</td>
</tr>
<tr>
<td>17-36 mo.</td>
</tr>
<tr>
<td>Mean</td>
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<tr>
<td>25.67</td>
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<tr>
<td>SD</td>
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<tr>
<td>4.63</td>
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<tr>
<td>Gender</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>71.5%</td>
</tr>
<tr>
<td>Female</td>
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<tr>
<td>28.5%</td>
</tr>
<tr>
<td>Race</td>
</tr>
<tr>
<td>Caucasian</td>
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<tr>
<td>51.4%</td>
</tr>
<tr>
<td>African-American</td>
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<tr>
<td>39.5%</td>
</tr>
<tr>
<td>Hispanic</td>
</tr>
<tr>
<td>2.5%</td>
</tr>
<tr>
<td>Other/Unspecified</td>
</tr>
<tr>
<td>5.9%</td>
</tr>
<tr>
<td>Unreported</td>
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<tr>
<td>0.7%</td>
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<tr>
<td>Diagnosis</td>
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<tr>
<td>ASD</td>
</tr>
<tr>
<td>15.6%</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>76.7%</td>
</tr>
<tr>
<td>No Diagnosis/Atypical</td>
</tr>
<tr>
<td>84.4%</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>70.5%</td>
</tr>
</tbody>
</table>

Note: \textit{SD} = Standard Deviation.

The included participants were partitioned into two groups, ASD (n = 365) and non-ASD (n = 1,973), according to informant endorsements of symptoms on the \textit{BISCUIT Part 1}, using an algorithm based on the criteria for ASD according to \textit{DSM-5} (APA, 2013). The non-ASD group was considered atypically developing, based on their referral to EarlySteps per the reasons above. These diagnostic assignments had a 96\% agreement rate with
previous diagnostic assignments made by a licensed psychologist with over 30 years of experience in the field of developmental disorders.

Measures

This study utilizes various scores from two assessment measures: the Battelle Developmental Inventory, Second Edition (BDI-2; Newborg, 2005) to measure socialization and the Baby and Infant Screen for Children with Autism, Part 2 (BISCUIT Part 2) to measure internalizing behaviors. Informant-reported endorsements on the BISCUIT Part 1 were utilized via an algorithm representative of DSM-5 criteria in order to form the two diagnostic groups, ASD and atypically developing. Descriptions of the measures and their psychometrics are provided.

Battelle Developmental Inventory, Second Edition (BDI-2).

The purpose of the BDI-2 is to assess development of adaptive, cognitive, communication, motor, and personal/social skills. The measure can be used on children from birth to 7 years 11 months of age (Newborg, 2005). There are three modes of administration: a structured, play-based activity using provided manipulatives; an observation conducted in the child’s natural setting; and a scripted interview with a parent, teacher, or caregiver serving as the informant. The administrator makes ratings of the child’s development on a Likert scale of zero to three based on observation or informant report for a total of 450 items. For each skill addressed, a score of zero corresponds to no ability, one corresponds to emerging ability, and three corresponds to ability present. Basal and ceiling levels of three items each (i.e., score of zero on three consecutive items and score of two on three consecutive items) should be established. The ratings are summed to create a raw score for each subdomain, which is then converted into a scaled score with a mean of 10, standard deviation of 3, and range of 1 to 19, as well as percentile ranks and age equivalencies. Each subdomain corresponds to a certain domain (i.e., adaptive, cognitive, communication, motor, and personal-social), creating domain scores. For example, scores corresponding to self-care and personal responsibility subdomains are combined into an adaptive domain score. All of the domain scores are transformed into a total sum of scaled scores, which is converted into an overall Developmental Quotient (DQ). The domain scores and DQ each have a mean of 100, standard deviation of 15, and range of 40 to 160, as well as percentile ranks and confidence intervals. For the present study, the total sum of scaled scores and the score on the overall Personal-Social domain were considered. This domain consists of three subdomains: adult interaction, peer interaction, and self-concept and social role.

The BDI-2 exhibits robust psychometric properties. Test-retest reliability was found above .80 for the total score and all domain scores, while internal consistency coefficients ranged from .98 to .99 (Newborg, 2005).
Furthermore, validity has been established for the population relevant to this study, individuals with ASD and other developmental delays (Newborg, 2005).

**Baby and Infant Screen for Children with Autism (BISCUIT).**

The Baby and Infant Scale for Children with Autism (BISCUIT) is a comprehensive assessment battery developed to measure symptoms of ASD and associated problems in infants and toddlers between the ages of 17 and 37 months. The battery is comprised of three informant-based components: (1) BISCUIT Part 1, which assesses for symptoms of ASD; (2) BISCUIT Part 2, which assesses for symptoms of comorbid psychopathology; and (3) BISCUIT Part 3, which assesses for challenging behavior. For the present study, only BISCUIT Part 1 and BISCUIT Part 2 were utilized.

**BISCUIT Part 1** has 62 items that describe symptoms characteristic of ASD. Parents or caregivers serving as informants rate their children in comparison to same-aged peers. A rating of zero corresponds to “not different; no impairment,” while a rating of one corresponds to “somewhat different; mild impairment,” and a rating of two corresponds to “very different; severe impairment.” The current cut-offs for BISCUIT Part 1 have not yet been updated to represent the changes made in DSM-5, and thus make reference to PDD-NOS and Autistic Disorder, rather than ASD. Accordingly, a score of below 17 suggests no diagnosis, a score of 17 to 38 suggests PDD-NOS, and a score of above 38 suggests Autistic Disorder (Matson, Boisjoli, Hess, & Wilkins, 2010). Internal consistency of BISCUIT Part 1 has been reported to be .97 (Matson, Wilkins, et al., 2009). Additionally, the scale was found to have good convergent validity with the M-CHAT and the Personal-Social domain of the BDI-2 (Matson, Wilkins, & Fodstad, 2011).

**BISCUIT Part 2** has 57 items that describe comorbid problems frequently faced by infants and toddlers with ASD. Similar to the BISCUIT Part 1, parent or caregiver informants make ratings of their children’s behavior on a scale of zero to three. Ratings indicate if the behavior described in the item has been a recent problem. A rating of zero corresponds to “not a problem or impairment,” while a rating of one corresponds to “mild problem or impairment,” and a rating of two corresponds to “severe problem or impairment.” A five-factor structure has been determined: Tantrum/Conduct Behavior, Inattention/Impulsivity, Avoidance Behavior, Anxiety/Repetitive Behavior, and Eat/Sleep Problems (Matson, Boisjoli, Hess, & Wilkins, 2011). Preliminary psychometric analyses suggest the BISCUIT Part 2 has excellent reliability with an overall internal consistency coefficient of .96 (Matson, Wilkins, et al., 2009).

For the present study, only two factors of the BISCUIT Part 2 were utilized: the Avoidance Behavior and the Anxiety/Repetitive Behavior factors. The former factor consists of 9 items (e.g., fear of being around others in...
school, at home, or in social situations; persistent fear that is not age appropriate), while the latter is composed of 11 items (e.g., repetition of actions or words to reduce stress; checking on play objects excessively). These factors most accurately represent the symptoms related to internalizing problems, primarily anxiety, among an early childhood population. When factor analysis was conducted, the Avoidance Behavior and Anxiety/Repetitive Behavior subscales were determined to have internal consistency alphas of .82 and .88, respectively (Matson, Wilkins, et al., 2009).

**Procedure**

The Louisiana State University Institutional Review Board and the state of Louisiana’s Office for Citizens with Developmental Disabilities (OCDD) approved the study. Prior to completing study protocol, the authors obtained informed consent from the parents and legal guardians serving as informants for the child participants. Child observations and informant interviews were conducted in the child participant’s home or daycare setting. Each of the approximately 175 test administrators held sufficient degree and certification or licensure to be eligible to provide services in the State of Louisiana’s EarlySteps program. Qualified assessors had licensures or certifications in a variety of areas, including occupational therapy, physical therapy, special education, social work, speech-language pathology, and psychology. Furthermore, administrators held degrees ranging from bachelor’s in early childhood education to doctoral degrees in psychology. Finally, all evaluators were proficient in evaluation and treatment of young children and had completed a full-day training on ASD, scale development, and test administration issues specific to the measures used for this study.

As aforementioned, infants and toddlers with any missing data points or with any measurement errors on the BISCUIT Part 1, BISCUIT Part 2, and BDI-2 scores were excluded. As previously explained, from the BISCUIT Part 1, individual items were selected to represent DSM-5 criteria for ASD, and diagnostic categories were assigned based on endorsements. From the BISCUIT Part 2, an Internalizing Problems score was calculated by combining total scores on two subscales representative of such issues, Avoidance Behavior and Anxiety/Repetitive Behavior. Greater scores on the Internalizing Problems variable indicate greater endorsements of internalizing symptoms (e.g., anxiety). From the BDI-2, the scaled score from the Personal-Social Domain was utilized to represent socialization skills. Greater scores on the Personal-Social Domain represent greater ability in this area. Additionally, the overall total sum of scaled scores (from which the DQ is derived; hereafter referred to as “BDI-2 total”) from the BDI-2 was considered, as the author hypothesized that differences in overall development might affect the outcome variables, necessitating its co-variation in the moderation model. This hypothesis was posited due to findings that IQ affected occurrence of internalizing problems (Mayes et al., 2011a),
and DQ is generally a good predictor of IQ among typically developing individuals and those with ASD (Lord & Schopler, 1989). Descriptive statistics for the included variables are provided in Table 2.

<table>
<thead>
<tr>
<th>D</th>
<th>Mean</th>
<th>SD</th>
<th>Minimum</th>
<th>Maximum</th>
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<tbody>
<tr>
<td><strong>Total Sample (n = 2338)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Internalizing Problems Score</td>
<td>2.13</td>
<td>3.50</td>
<td>0.00</td>
<td>24.00</td>
</tr>
<tr>
<td>Personal-Social Sum of Scaled Scores</td>
<td>20.41</td>
<td>8.56</td>
<td>2.00</td>
<td>81.00</td>
</tr>
<tr>
<td>Total Sum of Scaled Scores</td>
<td>86.38</td>
<td>30.16</td>
<td>9.00</td>
<td>187.00</td>
</tr>
<tr>
<td><strong>No ASD Group (n = 1973)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Internalizing Problems Score</td>
<td>1.37</td>
<td>2.45</td>
<td>0.00</td>
<td>20.00</td>
</tr>
<tr>
<td>Personal-Social Sum of Scaled Scores</td>
<td>21.62</td>
<td>8.06</td>
<td>3.00</td>
<td>81.00</td>
</tr>
<tr>
<td>Total Sum of Scaled Scores</td>
<td>90.62</td>
<td>28.89</td>
<td>10.00</td>
<td>187.00</td>
</tr>
<tr>
<td><strong>ASD Group (n = 365)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Internalizing Problems Score</td>
<td>6.20</td>
<td>5.13</td>
<td>0.00</td>
<td>24.00</td>
</tr>
<tr>
<td>Personal-Social Sum of Scaled Scores</td>
<td>13.85</td>
<td>8.20</td>
<td>2.00</td>
<td>76.00</td>
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<tr>
<td>Total Sum of Scaled Scores</td>
<td>63.47</td>
<td>26.32</td>
<td>9.00</td>
<td>166.00</td>
</tr>
</tbody>
</table>

Note: SD = Standard Deviation
Internalizing Problems Score from *BISCUIT – Part 2*; Personal-Social and Total Sums of Scaled Scores from *BDI-2*
Statistical Analyses

Using G*Power 3 (Faul, Erdfelder, Lang, & Buchner, 2007), an a priori power analysis was conducted to determine sample sizes required to achieve sufficient power. Field (2009) recommends that a power of .80 is adequate to detect a significant difference if one exists. Accordingly, alpha was set to .05, power was set to .80, and the effect size was set to .15 (medium), which is considered a medium effect size for a moderation (Cohen, 1988). With such requirements set, a total sample size of 68 was recommended for the following analyses. All individuals who met inclusion criteria were included as participants to increase power.

This study involved analyzing the ability of socialization differences (as indicated by the Personal-Social domain scaled score from the BDI-2) to predict internalizing problems (as indicated by Avoidance and Anxiety/Repetitive Behavior subscale scores, combined into an Internalizing Problems variable, on the BISCUIT Part 2), and gender’s role as a moderator, for toddlers with or without a diagnosis of ASD. As a result, moderation effects were tested, using multiple regression analysis, as suggested by Baron and Kenny (1986). Multiple regression analysis as described by Baron and Kenny (1986) is the most widely used method to assess moderation (MacKinnon & MacKinnon, 2008). Furthermore, statistical analysis procedures conducted in SPSS were based on guidance from Field (2009). All statistical analyses were conducted in SPSS 21.

Preliminary Analyses

Once participants with missing data or errors in measurement were eliminated, a sample of 2,338 individuals remained. Next, normality of each variable was assessed. Formal normality tests, such as the Kolmogorov-Smirnov test, may be unreliable for samples containing greater than 300 participants (Kim, 2013). As such, previously suggested guidelines for acceptable values of skewedness and kurtosis statistics were used. Absolute values of the skewedness statistic greater than 2.0 and of the kurtosis statistic greater than 7.0 suggest substantial departure from normality (Kim, 2013). All but the Internalizing Problems variable had acceptable values (skewedness = 2.46, kurtosis = 6.70). As such, a log plus one transformation was applied to this variable, resulting in acceptable absolute values of skewedness (.84) and kurtosis (.42).

A priori statistical analyses were conducted to determine if diagnostic groups differed significantly on demographic variables. First, several Chi-square analyses were conducted. No significant difference of ethnicity between diagnostic groups (i.e., atypically developing with and without ASD diagnosis) was found, \( \chi^2 (5) = 4.90, p = .428 \). However, diagnostic groups were found to differ significantly in terms of gender, \( \chi^2 (1) = 5.83, p < .05 \). Both groups were predominantly male, with females composing approximately 30% of the atypically developing with no ASD diagnosis group and approximately 24% of the atypically developing with ASD diagnosis group. The
A greater prevalence of males in the ASD group was not surprising, given the greater prevalence of males than females diagnosed with ASD (Fombonne, 2002). Additionally, an analysis of variance (ANOVA) was conducted to determine if the groups differed significantly in regard to age. Age was found to differ significantly between the groups, \( F(1, 2336) = 12.67, p < .001 \), with the no ASD diagnostic group average age older than the ASD diagnostic group average age. Finally, an ANOVA was conducted to determine if groups differed significantly in terms of overall development. Indeed, the total sum of scaled scores differed significantly between the groups, \( F(1, 2336) = 416.62, p < .001 \). As such, significantly different demographic variables were entered as covariates when not the variables being examined.

Following a priori statistical analyses to determine demographic variables that needed to be co-varied, simple ANOVAs were conducted to test each of the hypotheses presented in the Purpose section. Analyses were conducted across diagnostic groups or gender, depending on the hypothesis in question. The outcomes of such analyses are provided in the Results section below.

**Regression Analysis**

Before regression analyses were initiated, Pearson correlations were calculated to test if any demographic variables that were not predictor or outcome variables (i.e., age, *BDI*-2 total, ethnicity) correlated significantly with any of the predictor or outcome variables of the moderation analysis (i.e., gender, internalizing problems, social skills). Age was significantly correlated with Personal-Social Sum of Scaled Scores \( (r = .157, p < .01) \) and Internalizing Problems \( (r = .076, p < .01) \), but not gender \( (r = -.027, p = .190) \). The *BDI*-2 total was significantly correlated with Personal-Social Sum of Scaled Scores \( (r = .806, p < .01) \), Internalizing Problems \( (r = -.196, p < .01) \), and gender \( (r = .069, p < .01) \). Ethnicity was not significantly correlated with any of the predictor or outcome variables. Statistically significant correlations necessitated that age and *BDI*-2 total be included as covariates in the following multiple regression analysis.

Moderation was examined using hierarchical multiple regression analysis, following procedures outlined by Field (2009). The regression model was utilized to assess the potential effect of gender on the ability of socialization differences to predict internalizing problems, the dependent or outcome variable. Variables deemed necessary to include as covariates, *BDI*-2 total and age, were entered into Step 1 of the regression model as control variables. For Step 2, the previously centered Personal-Social domain scaled score was entered. For Step 3, gender was entered using dummy coding. Finally, for Step 4, an interaction term for Personal-Social domain scaled score by gender, created by multiplying these variables, was entered.
For steps entered in the model that resulted in significant change in $R^2$, effect size is presented. Specifically, Cohen’s $f^2$ (Cohen, 1988) is appropriate for calculating the effect size within a multiple regression model in which the independent variable of interest and the dependent variable are both continuous (i.e., socialization and internalizing problems, respectively, in the current study). Cohen’s $f^2$ is the most common measure of effect size for moderation analyses (West & Aiken, 1991). For the present study, the local effect size was computed using the equation advised by Selya, Rose, Dierker, Hedeker, and Mermelstein (2012). Cohen (1988) suggested that $f^2$ effect sizes of 0.02, 0.15, and 0.35 are considered small, medium, and large, respectively.

Does gender moderate the effects of socialization differences on the development of internalizing problems? If the regression coefficient of the interaction term is significantly different from zero, it can be concluded that gender and socialization differences interact in explaining variation in development of internalizing problems. Moderating effects of gender are indicated if there is a significant interaction of socialization differences and gender in explaining internalizing behaviors.
Results

Preliminary Analyses

Simple ANOVAs were conducted to test each of the aforementioned hypotheses. First, infants and toddlers with ASD were predicted to have greater endorsements of internalizing symptoms than their atypically developing peers without ASD diagnoses. Indeed, the ASD group had significantly greater internalizing symptoms ($M = 6.20$) than the non-ASD group ($M = 1.37$), $F(1, 2337) = 495.69, p < .001$, Cohen’s $d = 1.2$. Next, infants and toddlers with ASD were predicted to have lower scores on the Personal-Social domain of the BDI-2 than their atypically developing peers without ASD diagnoses. As expected, the ASD group had significantly lower socialization scores ($M = 13.85$) than the non-ASD group ($M = 21.62$), $F(1, 2337) = 12.88, p < .001$, Cohen’s $d = .96$.

Next, comparisons were made between gender groups but across diagnostic group. As such, a priori statistical analyses were conducted to determine if any variables would need to be co-varied in the following analyses. Gender groups did not differ in terms of age, $F(1, 2336) = 1.72, p = .19$. However, gender groups differed significantly in terms of ethnicity, $\chi^2 (5) = 11.29, p < .05$, and ASD diagnosis, $\chi^2 (1) = 5.83, p < .05$. Additionally, gender groups differed significantly on BDI-2 total, $F(1, 2336) = 18.49, p < .001$, with females having higher scores than males ($M = 89.66$ and $M = 85.07$, respectively). As such, in the following analyses, ethnicity, ASD diagnosis, and BDI-2 total were included as covariates.

First, atypically developing female infants and toddlers with and without ASD were predicted to have greater endorsements of internalizing symptoms than their male counterparts. Results indicated that there were no significant differences between gender groups in internalizing symptoms, $F(1, 2247) = .216, p = .642$. However, when the participants were separated by diagnostic group, significant differences emerged, but only for the ASD group. That is, the non-ASD group still did not exhibit significant differences in endorsed internalizing problems between gender groups, $F(1, 1894) = .114, p = .735$; however, among the ASD group, females exhibited significantly more internalizing problems ($M = .80$) than males ($M = .70$), $F(1, 352) = 4.64, p < .05$, Cohen’s $d = .21$.

Next, the author predicted that atypically developing females without ASD would have the highest socialization scores, indicating the most advanced social skills, but also that the males with ASD would have higher socialization scores than the females with ASD. Analyses indicated that atypically developing females without ASD did, indeed, have the highest socialization scores ($M = 22.59$). Furthermore, females showed significantly greater socialization scores ($M = 21.7$) than males ($M = 19.8$) in the overall sample, $F(1, 2247) = 5.96, p < .05$, Cohen’s $d =$
However, when divided into diagnostic groups, this discrepancy disappeared for the no diagnosis group, $F (1, 1894) = 45.65, p = .187$, but remained for the ASD group, $F (1, 352) = 6.30, p < .05$, Cohen’s $d = .24$.

**Regression Analysis**

Following these preliminary analyses, moderation was examined using hierarchical multiple regression analysis, following procedures outlined by Field (2009). The regression model was utilized to assess the potential effect of gender on the ability of socialization differences to predict internalizing problems, the dependent or outcome variable. The assumption of no multicollinearity was met, with no variance inflation factor (VIF) greater than 10 (Field, 2009). The variable deemed necessary to include as a covariate, age, was entered into Step 1 of the regression model as a control variable. For Step 2, the previously centered Personal-Social domain scaled score was entered. For Step 3, gender was entered using dummy coding. Finally, for Step 4, an interaction term for Personal-Social domain scaled score by gender, created by multiplying these variables, was entered.

Does gender moderate the effects of socialization differences on the development of internalizing problems? If the regression coefficient of the interaction term is significantly different from zero, it can be concluded that gender and socialization differences interact in explaining variation in development of internalizing problems. However, in the current analysis, this was not the case for the overall sample. The interaction term (i.e., Personal-Social domain scaled score by gender) did not significantly predict internalizing problems, $\beta = .033$, $t (2333) = 1.349, p = .177$. For the overall sample, age, as entered in Step 1, accounted for a significant proportion of variance in internalizing problems, $R^2 = .003, F (1, 2336) = 7.137, p < .05, f^2 = .003$. Further, age and the Personal-Social domain scaled score, as entered in Step 2, accounted for a significant proportion of additional variance in internalizing problem scores, $\Delta R^2 = .079, \Delta F (1, 2335) = 200.528, p < .001, f^2 = .089$. Following Step 2, Steps 3 and 4 did not account for any significant additional variance, $\Delta R^2 = .000, \Delta F (1, 2334) = .317, p = .573$, and $\Delta R^2 = .001, \Delta F (1, 2333) = 1.821, p = .177$, respectively. These findings indicate that the addition of gender and the interaction term, gender by Personal-Social domain scaled score, into the model did not explain any variance in internalizing problem scores beyond what was entered in Step 2. Additional information regarding the model for the overall sample is provided in Table 3.

The same regression analyses were conducted with the diagnostic groups separated. For the atypically developing with ASD group, the regression coefficient of the interaction term was not significantly different from zero, thus gender and socialization differences do not interact in explaining variation in internalizing problems. The interaction term (i.e., Personal-Social domain scaled score by gender) did not significantly predict internalizing problems, $\beta = .078, t (2333) = 1.037, p = .3$. For this group, age, as entered in Step 1, did not account for a
significant proportion of variance in internalizing problem scores, $R^2 = .008, F (1, 363) = 2.885, p = .09$.

Furthermore, age and the Personal-Social domain scaled score, as entered in Step 2, did not account for a significant proportion of additional variance in internalizing problem scores, $\Delta R^2 = .003, \Delta F (1, 362) = .957, p = .329$. Unlike in the overall sample, the addition of Step 3 (i.e., gender) to the model explained additional significant variance in the outcome variable. For Step 3, $\Delta R^2 = .011, \Delta F (1, 361) = 4.054, p < .05, f^2 = .021$. The addition of Step 4 to the model did not explain additional significant variance in the outcome, $\Delta R^2 = .003, \Delta F (1, 360) = 1.075, p = .3$.

Additional information regarding this model is provided in Table 4.

For the atypically developing without ASD group, the model is similar to the overall sample. The interaction term (i.e., Personal-Social domain scaled score by gender) did not significantly predict internalizing problems, $\beta = -.001, t (196) = -730, p = .465$. For this group, age, as entered in Step 1, did not account for a significant proportion of variance in internalizing problem scores, $R^2 = .000, F (1, 1971) = .150, p = .699$. Step 2 accounted for a significant proportion of additional variance in internalizing problem scores, $\Delta R^2 = .033, \Delta F (1, 1970) = 66.916, p < .001, f^2 = .034$. Following Step 2, Steps 3 and 4 did not account for any significant additional variance, $\Delta R^2 = .000, \Delta F (1, 1969) = .000, p = .991$, and $\Delta R^2 = .000, \Delta F (1, 1968) = .534, p = .465$, respectively. Additional information regarding this model is provided in Table 5.
Table 3
Summary of Hierarchical Regression Analysis for Variables Predicting Internalizing Problems in Overall Sample (N = 2338)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model 1</th>
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<th>Model 2</th>
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<th>Model 3</th>
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<td>β</td>
<td></td>
<td>B</td>
<td>SE</td>
<td>β</td>
<td></td>
<td>B</td>
<td>SE</td>
<td>β</td>
<td></td>
<td>B</td>
<td>SE</td>
<td>β</td>
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<tr>
<td>ΔF</td>
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<td>200.528**</td>
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<td>.1821</td>
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</table>

Note: * p < .05. ** p < .01.
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<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
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</table>

*Note: * $p < .05$. ** $p < .01$.*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
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<tbody>
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<td>SE B</td>
<td>β</td>
<td>B</td>
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<tr>
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<tr>
<td>Personal-Social x Gender</td>
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<td>-.001</td>
</tr>
<tr>
<td>$R^2$</td>
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<tr>
<td>$\Delta F$</td>
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<td>66.916**</td>
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<td>.534</td>
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*Note: * $p < .05$. ** $p < .01$.*
**Discussion**

The majority of the preliminary analyses conducted resulted in expected findings, providing further support for existing literature. Individuals in the ASD group had higher internalizing problems scores than their atypically developing counterparts. Such a pattern is consistent with previous research suggesting that individuals with ASD frequently suffer from co-occurring internalizing problems, such as symptoms of anxiety and depression, even more frequently than externalizing problems (Howlin, 1997; Kim et al., 2000; Mayes et al., 2011a; Skokauskas & Gallagher, 2010). More specifically, this pattern mirrors existing studies in which researchers have noted greater anxiety and avoidance problems among infants and toddlers with ASD than among those who are typically developing (Davis III, Fodstad, et al., 2011). Furthermore, individuals in the atypically developing group had higher scores on the Personal-Social domain scaled score, indicating greater social skills, than their ASD group counterparts. Such a result is unsurprising given social skills deficits are a core domain of the symptomology of ASD (APA, 2013; Matson, 2007; National Research Council, 2001).

Contrary to hypothesized patterns, no significant differences between gender groups in terms of internalizing problems were found in the overall sample. This finding was somewhat surprising, given the extensive body of research literature suggesting that internalizing disorders, such as anxiety and depression, are more common among females (Keenan & Shaw, 1997; Lewinsohn et al., 1998; Nolen-Hoeksema, 1990). For ASD populations, however, studies of gender differences in internalizing problems have resulted in a variety of findings that do not necessarily mirror the pattern found in the general population. Furthermore, the present sample as a whole is composed of individuals with atypical development (i.e., infants and toddlers referred to EarlySteps), to which patterns found in the general population and in an ASD-only population may not apply. Indeed, when gender differences were examined more closely with diagnostic groups separated, the atypically developing group without ASD exhibited the same lack of differences between genders. However, among the individuals with ASD, females exhibited more internalizing problems than males, reflecting the hypothesized pattern. Due to uneven sample sizes, the greater number of individuals with atypical development but no ASD may have masked differences among the individuals with ASD in the analysis of the overall sample.

In terms of scaled scores on the Personal-Social domain, atypically developing females without ASD had the highest scores, as predicted. Contrary to hypotheses and extant research studies (Carter et al., 2007; McLennan et al., 1993), the females with ASD had higher scores than the males with ASD, indicating greater social skills. In each of the aforementioned studies, the authors found that males with ASD have greater social skills. Because the authors considered different age groups (toddlers in the sample in Carter et al., 2007, children and adolescents in the sample in McLennan et al., 1993), their results indicate there may be a consistent weakness in social skills among females as compared to males across development prior to adulthood. As discussed, Hartley and Sikora (2009) suggested that their finding of a lack of difference in social reciprocity between the genders might have been a result of their inclusion of both high and low functioning individuals with ASD. Nonetheless, in the present study, the BDI-2 total score was co-varied in the preliminary analyses.
and in the moderation analysis to account for any effect that developmental level may have on the outcome variables. As such, the functioning of the individuals included would not have been a contributing factor to the difference in social skills found in the present study. Further research is needed on the topic of gender differences in socialization among the ASD population to elucidate the discrepant results of extant studies.

Additionally, the aforementioned lack of effects of gender on internalizing problems in the overall sample and in the no ASD group was somewhat surprising, considering much of the research literature indicates that typically developing females and females with ASD have more internalizing problems than their male counterparts (Keenan & Shaw, 1997; Lewinsohn et al., 1998; Nolen-Hoeksema, 1990). However, these differences have been found as less apparent and noteworthy early in development among typically developing populations, with males and females demonstrating comparable rates of internalizing problems (Keenan & Shaw, 1997). Similarly, as discussed, Horovitz and colleagues (2011) found that differences in internalizing problems might be fairly minute and inconsequential among an atypical infant and toddler population, with the discrepancy between genders increasing with age. As such, future research may replicate these findings, providing further evidence that discrepancies in internalizing issues do not begin until early childhood, rather than among infants and toddlers, in both typically developing and ASD populations.

As discussed, three separate moderation analyses were conducted. The interaction between socialization (i.e., Personal-Social domain scaled score) and gender did not significantly predict internalizing problems for any group. In the overall sample, age and age and Personal-Social score together explained significant additional variance in internalizing problems. In the atypically developing group, only age and Personal-Social score together explained significant additional variance in internalizing problems. Finally, in the ASD group, only age, Personal-Social, and gender together explained significant additional variance in internalizing problems. However, the effect sizes for each of these findings were negligible to small (i.e., no greater than .08; Cohen [1988] suggested .02 is a small effect size and .15 is a medium effect size for a multiple regression analysis). Thus, statistical significance was likely achieved due to large sample sizes, rather than due to the existence of clinically significant and notable interactions among the included variables. As such, statistically significant results should be interpreted with caution, and further research should be conducted to determine if gender may have a moderating effect on the relationship between socialization and the development of internalizing problems among an infant and toddler sample.

Uneven sample sizes may have influenced the results of the present study. The atypically developing group was significantly larger than the ASD group. Furthermore, males accounted for the majority of the participants in both the atypically developing and ASD groups. Differences in sample sizes may have resulted in overshadowing or masking of significant discrepancies in the outcome variables. Although various comparisons in the present study indicated differences that were statistically significant, effect sizes were negligible. As such, all results should be interpreted with caution. Additionally, although the BISCUIT Avoidance Behavior and Anxiety/Repetitive Behavior factors have face validity as
representation of symptoms of internalizing problems, they have not yet been statistically validated for this purpose. Thus, the author suggests that, following this preliminary analysis, this topic be examined using a scale specifically created and validated for the assessment of internalizing problems among children in order to assemble more conclusive findings.

Furthermore, future research on this topic should include a wide range of ages, divided into empirically based developmental groups, in order to consider gender differences in social skills and internalizing problems across the lifespan. Results of such studies can prove valuable in the understanding of gender differences in core ASD symptomatology and comorbid problems. Greater appreciation for the distinctions between males and females with ASD, including whether these distinctions change with development, will allow clinicians to better individualize treatment options, leading to better treatment outcomes. If the present findings are replicated, emphasis will not need to be placed on gender differences in comorbid internalizing symptoms among infants and toddlers in early intervention programs. However, if the relationship between socialization and internalizing problems is found to be moderated by gender in future research, clinicians may be able to lessen the frequency of development of comorbid internalizing problems by prioritizing social skills in treatment plans differentially based on gender. Further, if this moderating effect is found among older children and adolescents but not among infants and toddlers, clinicians can be cognizant of such differences and consider them as their clients reach an empirically indicated age range. Finally, because social competence and problem behavior, both internalizing and externalizing, appear to be related, future research should consider additional potential moderating variables other than gender. Results of such studies could help elucidate this relationship, providing knowledge valuable for the prevention of internalizing problems among individuals with and without ASD.
References


Appendix

Application for Exemption from Institutional Oversight

Unless qualified as meeting the specific criteria for exemption from Institutional Review Board (IRB) oversight, all LSU research projects using living humans as subjects, or samples, or data obtained from humans, directly or indirectly, with or without their consent, must be approved or exempted in advance by the LSU IRB. This form helps the PI determine if a project may be exempted, and it is used to request an exemption.

- Applicant: Please fill out the application in its entirety and include the completed application as well as parts A-F, listed below, when submitting to the IRB. Once the application is completed, please submit two copies of the completed application to the IRB Office or to a member of the Human Subjects Screening Committee. Members of this committee can be found at http://research.lsu.edu/CompliancePolicies/Procedures/InstitutionalReviewBoard%20IRB%20Home/247377.html

- A Complete Application Includes All of the Following:
  (A) Two copies of this completed form and two copies of parts B thru F.
  (B) A brief project description (adequate to evaluate risks to subjects and to explain your responses to Parts 1&2)
  (C) Copies of all instruments to be used.
  *If this proposal is part of a grant proposal, include a copy of the proposal and all recruitment material.
  (D) The consent form that you will use in the study (see part 3 for more information.)
  (E) Certificate of Completion of Human Subjects Protection Training for all personnel involved in the project, including students who are involved with testing or handling data, unless already on file with the IRB. Training link: (http://phhp.med.wisc.edu/irbtraining.com/users/login.php)
  (F) IRB Security of Data Agreement: (http://research.lsu.edu/files/item/26774.pdf)

1) Principal Investigator: Dr. Johnny L. Matson
Dept: Clinical Psychology
Ph: (225) 578-8745
Rank: Clinical Director
E-mail: johnmatson@aol.com

2) Co-Investigator(s): Please include department, rank, phone and e-mail for each
*If student, please identify and name supervising professor in this space

3) Project Title: Autism in Early Childhood

4) Proposal? (yes or no) No
   If Yes, LSU Proposal Number
   Also, if YES, either
   ○ This application completely matches the scope of work in the grant
   OR
   ○ More IRB Applications will be filed later

5) Subject pool (e.g. Psychology students) Young children assessed for developmental delay via EarlySteps
   *Circle any "vulnerable populations" to be used: (children < 18; the mentally impaired, pregnant women, the ages, other). Projects with incarcerated persons cannot be exempted.

6) PI Signature
   Date 9/19/13
   No per signatures

**I certify my responses are accurate and complete. If the project scope or design is later changes, I will resubmit for review. I will obtain written approval from the Authorized Representative of all non-LSU Institutions in which the study is conducted. I also understand that it is my responsibility to maintain copies of all consent forms at LSU for three years after completion of the study. If I leave LSU before that time the consent forms should be preserved in the Departmental Office.

Screening Committee Action: Exempted N
Signed Consent Waived: Y/N
Reviewer: Matthews
Signature: Matthews
Date: 9/19/13
Vita

Hilary Lynn Adams, a native of Houston, Texas, received her bachelor’s degree at Tulane University in 2010. Following a year off working as a research coordinator, she began graduate school in the Department of Psychology at Louisiana State University. She will receive her master’s degree in May 2014 and will continue in the clinical psychology doctoral program.