



UNIVERSITY OF PADOVA

Department of Developmental Psychology and Socialization

**Master's degree in Psicologia dello Sviluppo e dell'Educazione
Study track in Developmental and Educational Psychology**

Final dissertation

**The Association between Autistic Traits and Social Anxiety, and the Influence
of Familism**

Supervisor

Professor Irene Cristina Mammarella

Co-supervisor

PhD Ingrid Zugey Galán Vera

Candidate: Ufuk Özçidem

Student ID number: 2071363

Academic Year 2024/2025

Table of Contents

UNIVERSITY OF PADOVA.....	1
Chapter 1.....	1
Autism Spectrum Disorder.....	1
1.1 History of Autism Spectrum Disorder.....	1
1.2 Clinical Characteristics.....	2
1.2.1 Social Communication and Interaction.....	2
1.2.2 Restricted and Repetitive Behaviors.....	3
1.2.3 Sensory Features.....	3
1.2.4 Cognitive and Behavioral Flexibility.....	4
1.3 Diagnostic Criteria.....	5
1.3.1 Differential Diagnosis.....	6
1.4 Epidemiology.....	8
1.5 Etiology.....	9
1.5.1 Genetic Factors.....	9
1.5.2 Environmental Factors.....	10
1.5.3 Neurological Factors.....	11
1.6 Treatment and Prevention.....	11
Chapter 2.....	15
<i>The Association between Autistic Traits and Social Anxiety, and the Influence of Familism..</i>	15
2.1 Autistic Traits.....	15
2.2 Social Anxiety.....	16
2.3 The Association between Autistic Traits and Social Anxiety.....	19
2.4 Familism.....	21
2.5 The Association between Social Anxiety and Familism.....	22
2.6 The Association between Autistic Traits and Familism.....	24
Chapter 3.....	26
<i>The Present Study.....</i>	26
3.1 Aims and Hypotheses.....	27
3.2 Methods.....	27
3.2.1 Participants.....	27
3.2.2 Research Design.....	28
3.2.3 Materials.....	29
3.2.4 Procedure.....	31
Chapter 4.....	32

Results.....	32
4.1 Descriptive Statistics of the Sample.....	32
Table 1.....	32
Figure 1.....	33
Figure 2.....	34
Figure 3.....	35
Figure 4.....	36
Figure 5.....	37
Figure 6.....	38
4.2 Testing the First Hypothesis.....	39
Table 2.....	39
4.3 Hierarchical regression.....	40
Table 3.....	40
Table 4.....	41
Table 5.....	42
4.4 Testing the Second Hypothesis.....	43
Table 6.....	43
Figure 7.....	44
Chapter 5.....	46
Discussion.....	46
5.1 First Hypothesis.....	47
5.2 Second Hypothesis.....	48
5.3 Limitations.....	50
5.4 Future Research.....	50
6 References.....	52

Chapter 1

Autism Spectrum Disorder

Autism spectrum disorder (ASD) is a neurodevelopmental disorder with a multifactorial etiology (Sauer et. al., 2021). It is expressed by early-onset difficulties in social communication and uncommonly restricted, repetitive behavior and interests (Baron-Cohen, 2008).

1.1 History of Autism Spectrum Disorder

The concept of autism traces back to the early 20th century when Swiss psychiatrist Eugen Bleuler introduced the word "autism" to portray a characteristic feature of schizophrenia, relating to self-absorption and disengagement from social interaction (Moskowitz & Heim, 2011).

In 1943, psychiatrist Leo Kanner issued a groundbreaking paper study titled "Autistic Disturbances of Affective Contact," which marked the first systematic depiction of autism as a unique developmental disorder. In this case study, Kanner identified a group of eleven children presenting similar patterns of behavior, including social withdrawal, communication problems, and repetitive actions (Kanner, 1943).

Concurrently, Austrian pediatrician Hans Asperger independently identified related attributes in a group of children and introduced the concept of "autistic psychopathy" (Asperger, 1944). Like Leo Kanner did, Hans Asperger also used the word "autism" invented by Eugen Bleuler. In his case study paper, he emphasized the importance of giving specialized educational treatment to these newly identified special group of children. At the time, they observed that autistic children were almost always boys. Therefore, Asperger addressed "The autistic personality is an extreme variant of male intelligence" (Asperger, 1944). Asperger's work, however, remained

relatively hidden until it was rediscovered and translated into English in the 1990s by Uta Frith (Frith, 1991).

Throughout the mid-20th century, autism was often misunderstood and misdiagnosed, leading to confusion and controversy within the medical community. It wasn't until the 1980s that standardized diagnostic criteria for autism were established, most notably with the issuance of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III) in 1980 (American Psychiatric Association, 1980). Subsequent revisions of the DSM, including the DSM-IV in 1994 and the DSM-5 in 2013, suggested notable revisions to the categorization and diagnosis of autism, emphasizing the spectrum nature of the disorder and incorporating subclasses like Asperger's syndrome and pervasive developmental disorder not otherwise specified (PDD-NOS) (American Psychiatric Association, 2013).

Advancements in neuroimaging, genetics, and developmental psychology have benefited to a further comprehension of the biological and environmental factors underlying autism. Nowadays, autism is accepted as complex a neurodevelopmental disorder which involves a mixture of genetic and environmental determinants with a wide array of presentations and etiological factors, affecting individuals across the lifespan (Pardo & Eberhart, 2007).

1.2 Clinical Characteristics

ASD often becomes evident within the first two years of life, as it impacts several aspects of early development (Zwaigenbaum et al., 2013).

1.2.1 Social Communication and Interaction

Among the indications of ASD is ongoing difficulties in social communication and interaction (Johnson & Myers, 2007). Communication difficulties involve a delay or absence in

using gestures and developing spoken language, struggles in starting or keeping conversations, and unusual language patterns like repeating words (echolalia) or using them in unique ways (idiosyncratic use of words) (Johnson & Myers, 2007; Prelock & Nelson, 2012). Autistic individuals often exhibit challenges in understanding and appropriately responding to social cues, which can result in difficulties in establishing and maintaining relationships with others (Prelock & Nelson, 2012). Moreover, they often initiate interactions based on their needs and may struggle with following conversations due to communication difficulties (Kangas et al., 2011).

1.2.2 Restricted and Repetitive Behaviors

Restricted and repetitive behaviors (RRBs) are a key component of ASD, encompassing a range of symptoms such as stereotyped movements, repeated patterns in how objects are used and language is spoken, fixation on certain activities, strong preference for routines, and uncommon sensory experiences (Turner-Brown & Frisch, 2020). These behaviors are prevalent in ASD, with a study finding them in 94.7% of patients (Basit et al., 2022). Russell and Brosnan (2018) categorized restricted and repetitive behaviors as lower-order sensory-motor behavior repetitions or higher-order conceptual mental repetitions and mentioned RRB's as particularly prevalent during the early developmental years of autistic children. Richler et al. (2007) conducted research with 165 autistic children, 49 with non-spectrum developmental disorders, and 65 typically developing children, with a factor analysis finding support for a repetitive sensorimotor (RSM) factor and a tendency for insistence on sameness (IS). Although repetitive and restrictive behaviors are common, successful methods to lessen their occurrence and mitigate their effects on individuals and families have been recognized, such as with the help of behavioral and cognitive behavioral techniques (Turner-Brown & Frisch, 2020).

1.2.3 Sensory Features

Many autistic individuals display either an intensified or decreased sensitivity to sensory cues, a phenomenon often referred to as sensory processing differences. These sensitivities can manifest in various ways, such as hypersensitivity to loud noises, dislike to certain textures or tastes, or seeking out specific sensory experiences (Johnson & Myers, 2007; Lord et al., 2020). Sensory features can significantly impact daily functioning and may cause behavioral challenges and difficulties in sensory-rich environments, the hypersensitivity also can lead to heightened reactions to lights and smells. Conversely, some individuals with autism may demonstrate hyposensitivity, requiring more intense stimuli to elicit a response. These sensory processing differences can impact how individuals perceive and respond to sensory input (Johnson & Myers, 2007; Lord et al., 2020). Additionally, individuals with autism may engage in sensory avoidance or seeking behaviors, such as covering ears in response to loud noises or seeking out specific sensory experiences. The complexities of sensory sensitivities in autism underscore the significance of acknowledging personal variations in sensory processing and addressing sensory needs in interventions and support strategies for autistic individuals (Grapel et al., 2015; Hazen et al., 2014).

1.2.4 Cognitive and Behavioral Flexibility

Autistic people might also show limitations in cognitive and behavioral adaptability, that involves the ability to adapt to changes in routines, situations, or demands. For instance, having difficulties in shifting attention, changing from an activity to another one, and coping with unexpected events are frequent between autistic individuals (Geurts et al., 2009).

Researchers tend to believe that behavioral inflexibility challenges in autistic people are connected with cognitive flexibility deficits. For instance, Pouretmad (2022) found that executive dysfunction is associated with behavioral inflexibility in early-age children on the spectrum. However, according to Geurts et al. (2009), no homogenous data proves that there is a causal

relationship between cognitive and behavioral flexibility. After discovering more advanced techniques and using ecologically valid measures, the relationship between cognitive and behavioral flexibility is believed to be assessed better (Geurts et al., 2009). Additionally, neuroimaging studies have revealed atypical activation patterns in lateral frontoparietal and mid cingulo-insular networks during cognitive flexibility tasks in autistic individuals (Uddin, 2021). Finally, Memari et al. (2013) identified gender, lower language deficits, intelligence, education, and sleep time as potential correlates of cognitive flexibility impairments in autistic children.

1.3 Diagnostic Criteria

Many individuals with autism can receive a diagnosis between 16 and 36 months of age (Cervantes et al., 2016). Initial diagnosis of ASD in 2-year-old children is essential for ideal long-term outcomes and reducing the stressful diagnostic process for families (Zwaigenbaum et al., 2015).

In the diagnosis of ASD, different tools are employed to measure behavioral patterns and social communication impairments. These involve the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2000), the Autism Diagnostic Interview-Revised (ADI-R; Lord et al., 1994), the Childhood Autism Rating Scale (CARS; Schopler et al., 1986), the Diagnostic Interview for Social and Communication Disorders (DISCO; Wing et al., 2002), the Gilliam Autism Rating Scale (GARS; Gilliam, 2006), and the Developmental, Dimensional, and Diagnostic Interview (3di; Skuse et al., 2004). These tools involve a mixture of father, mother or caregiver interviews and firsthand monitoring of the children to accurately identify symptoms associated with ASD (Randall et al., 2018).

The standards used to diagnose ASD have undergone significant revisions over time, reflecting advances in our understanding of the condition and changes in diagnostic practices. The most recent version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), issued by the American Psychiatric Association (APA) in 2013, introduced several important modifications to the diagnostic standards for ASD. The DSM-5 defines ASD based on two core domains: ongoing difficulties with social interaction and communication and limited and recurring patterns in actions, preferences, or pursuits. The deficits mentioned have to be commenced in young childhood and must result in substantial disruption in social, work, or other essential life domains (American Psychiatric Association, 2013).

One of the additional evident changes in the DSM-5 is the consolidation of previous subtypes of ASD, like autistic disorder, Asperger's syndrome, and pervasive developmental disorder not otherwise specified (PDD-NOS), into a single diagnostic category of ASD. This shift reflects the recognition of autism as a spectrum disorder with changing levels of intensity and expression (Rosen et al., 2021).

Another notable alteration introduced in the DSM-5 is the inclusion of specifiers to describe the degree of the symptoms. The specifiers include degree of assistance required (i.e., needing assistance, needing considerable assistance, or needing intensive assistance) and the presence or absence of coexisting intellectual challenges and language impairment (American Psychiatric Association, 2013).

1.3.1 Differential Diagnosis

According to Baron-Cohen et al. (2000) and Stone, Coonrad, & Ousley (2000), the first method for conducting a differential diagnosis involves using a standardized autism assessment

tool, which is applicable to children as young as 18 months. Specifically, the Checklist for Autism in Toddlers (CHAT; Baron-Cohen et al., 2000) and the Screening Tool for Autism in Two-year-olds (STAT; Stone et al., 2000) have been designed for diagnosing children at 1.5 to 2 years old. Even though the evaluations by themselves should not be relied upon as the sole foundation for differential diagnosis, they currently represent the primary method supported by scientific evidence (Matson et al., 2007).

Communication disorders (CD), mild intellectual disability (MID), and attention-deficit hyperactivity disorders (ADHD) are the predominant definite clinical diagnoses in children exhibiting autistic traits (De Giacomo et al., 2021). Interestingly, communication disorders were particularly prevalent and often associated with a younger age group among the study participants. Furthermore, they concluded that the analysis of ADOS (Lord et al., 2000) domains and individual items do not guarantee a clear ability to distinguish between various neurodevelopmental disorders in the context of differential diagnosis (De Giacomo et al., 2021).

Research indicates that autistic individuals often present co-occurring conditions, such as anxiety disorders, depression, ADHD, and intellectual disabilities (Fombonne, 2003; Stewart et al., 2006; Magnússon & Sæmundsen, 2001). Autistic individuals with intellectual disability (ID), occurs with 70%, being one of the most common (Fombonne, 2003; Magnússon & Sæmundsen, 2001). Moreover, individuals with ASD might present other conditions such as schizophrenia, mood disorders, and anxiety disorders, being less frequent than intellectual disability (Trammell et al., 2013). Different types of anxiety are prevalent comorbid conditions in ASD, comprising social anxiety, generalized anxiety, and obsessive-compulsive disorder (Trammell et al., 2013). Depression is another frequent co-occurring condition in autistic individuals, presenting symptoms such as sleep disturbances, unhealthy eating behaviours, emotional disengagement, and

withdrawal from social interactions (Stewart et al., 2006). Clinicians face challenges in diagnosing depression in autistic individuals due to overlapping symptoms and the lack of standardized measures for this population (Trammell et al., 2013). Tools like the Beck Depression Inventory (BDI-II; Beck et al., 1996) and the Hamilton Depression Inventory (HDI; Kobak & Reynolds, 1999) can provide useful information. Schizophrenia is another condition that can present challenges in the differential diagnosis with ASD due to overlapping symptoms, such as social communication difficulties and stereotyped behaviors. Autistic individuals may exhibit less reciprocal communication and more stereotypical language compared to those with schizophrenia (Trammell et al., 2013).

1.4 Epidemiology

Autism is now recognized as a major global public health issue due to its increasing prevalence in recent years. In the last two decades, the number of 8-year-old children diagnosed with ASD has grown substantially. The prevalence has risen from 6.7 per 1,000 in 2000 to 23.0 per 1,000 in 2018. This upward trend indicates a growing awareness and improved screening practices for ASD (Maenner et al., 2023).

Furthermore, research indicates notable gender disparities in the prevalence of ASD. Although ASD is more often identified in males than in females, with a reported ratio between 2:1 and 5:1 (Brugha et al., 2016; Loomes et al., 2017), the exact reasons for this discrepancy remain under investigation (Loomes et al., 2017). Additionally, Van Wijngaarden-Cremers et al. (2014), discuss in their article that females with ASD exhibit fewer interests, repetitive behaviors, or stereotyped pattern compared to males with ASD, which in return, might cause a gender bias toward males in the clinical identification of ASD since it was historically developed based on observations of behaviors more commonly seen in males. As a result, females with ASD who may

exhibit different or less severe symptoms compared to males may fall short of the diagnostic threshold.

Geographical variability in ASD prevalence is notable, with rates varying across different states and monitoring sites. Understanding the contributing factors to this variability is crucial for informing interventions and addressing differences in ASD prevalence and outcomes (Maenner et al., 2023). Some studies have found that some migrant groups have higher level of ASD (Magnusson et al., 2012), however, in another research, minority groups and black population were found to have lower levels of ASD (McManus et al., 2016). The prevalence of ASD among 8-year-old white children was reported as lower compared to children of other racial backgrounds. In addition, the research also discovered that among autistic children, black children had a highly likelihood than white children of also having an intellectual disability (Maenner, 2023). In individuals diagnosed with ASD, comorbid intellectual deficiency is relatively common. Of the children diagnosed with ASD, 33% were categorized as having an intellectual disability ($IQ \leq 70$), 24% fell within the borderline IQ range (71–85), and 42% had an IQ above average ($IQ > 85$) (Maenner et al., 2020). In another study, the connection between high SES and ASD was noted between 2002-2010 (Durkin et al., 2017), however, this association was found to be more variable in 2018 (Maenner et al., 2021).

1.5.1 Genetic Factors

Considering the twin studies and family studies, it has been consistently proved that ASD is highly heritable, ranging from 40% to 90% (Lee et al., 2013; Gaugler et al., 2014). Recent genetic research has shown that ASD involves many rare genetic conditions, each contributing differently. Around 25% of people with ASD have rare genetic mutations that increase their risk. Studies also suggest that new genetic mutations and inherited genetic variations play a role in ASD. While some

common genetic variations have already been found to be related to ASD, it's challenging to replicate these findings due to the disorder's genetic diversity. Understanding ASD's genetic complexity is difficult since different genes affect brain development differently, and both common and rare genetic factors are involved (Geschwind & State, 2015). It has been studied that impaired methylation processes and mutations in *mecp2* have been linked to ASD and related conditions like Rett syndrome (Currenti, 2009). Furthermore, genetic variations in cytochrome P450 enzymes, including CYP27B1, play a role in the susceptibility to ASD. Additionally, cytogenetic abnormalities are also considered as inherited influences that play a role in the emergence of ASD (Gillberg, 1998).

1.5.2 Environmental Factors

Several environmental influences play a role in the risk of ASD development. Advanced parental age has been identified as one such factor, with studies showing a higher risk of ASD in children of older parents (Wu et al., 2017). Additionally, birth complications, particularly those involving a trauma or oxygen deprivation during delivery, have been strongly linked to ASD development (Modabbernia et al., 2017). Another interesting finding is that maternal immigration has also been linked to a higher likelihood of ASD, with elevated rates reported among children of migrant mothers compared to those of non-migrant mothers (Crafa & Warfa, 2015). Additionally, exposure to certain chemicals has been classified as another environmental potential cause for ASD (Rossignol et al., 2014). Conversely, certain environmental factors, such as vaccination and maternal smoking, have been found to be unrelated to the risk of ASD (Taylor et al., 2014). These findings provide reassurance regarding the safety of vaccinations and suggest that maternal smoking may not be a significant contributing factor to the development of ASD (Modabbernia et al., 2017).

1.5.3 Neurological Factors

The enzyme 5,10-methylenetetrahydrofolate reductase (MTHFR) is essential for transforming folic acid into a usable form for the body's methylation functions (Rogers, 2008). Research has shown that mutations in the MTHFR gene, particularly the C677T mutation, are linked to a higher likelihood of autism. Children with MTHFR mutations who do not receive adequate folic acid supplementation are more susceptible to neurological regression and therefore, being diagnosed with ASD (Currenti, 2009). In addition, research indicated that abnormalities in immune responses have been found similar in both autism and vitamin D deficiency (Cannell, 2008); since individuals with ASD have T cell abnormalities that can be similarly seen in malfunctioning immune system caused by vitamin D deficiency (Ashwood et. al. 2006). This suggests that adequate vitamin D intake is crucial for healthy neural development (Cui et al., 2007).

Disruption of pathways involved in synaptic protein synthesis, essential for communication between neurons, can result in cognitive deficits and neuronal impairments observed in autistic individuals. The interaction of genetic and environmental contributors can influence the development of neuronal networks, potentially leading to attention deficits and cognitive challenges commonly seen in ASD. Neurological regression, possibly linked to inefficient metabolism of vitamin D due to genetic mutations like CYP27B1, may contribute to the manifestation of ASD symptoms (Currenti, 2009).

1.6 Treatment and Prevention

As mentioned above, early diagnosis of ASD is crucial, and it can be established as early as 18-24 months. Research has shown that intervention programs based on applied behavior analysis are considered as the first-line treatment for ASD in early childhood. These programs,

such as the Early Start Denver Model (ESDM; Rogers, 2016) and Early Intensive Behavioral Intervention (EIBI; Klintwall & Eikeseth, 2014), have demonstrated efficacy in improving cognitive abilities, language development, and adaptive functioning in autistic children. These interventions focus on early development, provide one-on-one and small group instruction, and emphasize adult-directed methods to systematically address skill development. As examples to the techniques used in applied behavior analysis, positive reinforcement, shaping, fading, extinction, punishment, and differential reinforcement can be given. Additionally, interventions should include a family component, incorporating parent training to support the child's progress. Other common treatment approaches for autistic individuals may involve speech therapy to address language delays and behavioral therapies to manage challenging behaviors (Brentani et al., 2013). Dawson (2008) also underscores the effectiveness of early intensive behavioral interventions, and caregiver-based programs in enhancing results for autistic children. Additionally, he emphasizes the importance of understanding genetic and environmental influences, parental involvement, and transitioning interventions to naturalistic environments to enhance the efficacy of ASD treatments. Moreover, Dawson and Bernier (2013) also highlighted that applied behavior analysis therapies and comprehensive behavioral interventions are impactful in the improvement of intellectual functioning, language development, and social skills in children with ASD. Additionally, the importance of individualized support tailored to each child's needs is emphasized.

On the prevention side, Dawson & Bernier, (2013) pointed out that genetic guidance for families with a history of ASD, early intervention programs focusing on developmental milestones, and raising awareness about the early signs of autism are recommended strategies to diminish the impact of ASD symptoms and promote timely intervention. Gunby et. al. (2010) designed a study where they applied abduction-prevention abilities to autistic children through behavioral

competence training. Skills for preventing abduction were taught on a one-on-one basis to each child through verbal guidance, video demonstrations, live demonstrations, practice sessions with known adults and unfamiliar individuals, along with positive reinforcement and corrective feedback. The children were instructed on how to react to four common lure types: simple, incentive, authority, and assistance request. Despite the small number of participants (only three), during the 1-month follow-up assessment, the results showed that all children acquired the abduction-prevention skills, and the skills were maintained.

Related to prevention, Francis et al. (2021) discusses some strategies for ASD from a life course approach. The paper emphasizes the multifactorial nature of ASD, with genes interacting with environmental factors prenatally and postnatally, and preventions aim to manipulate these factors (Hertz & Picciotto et al., 2018) to prevent or reduce the occurrence of ASD. Therefore, primary prevention efforts focus on preconception and pregnancy to increase resilience and reduce ASD risk (D'Arcy & Meng, 2014). Secondary and tertiary prevention measures target altering developmental cascades in early childhood to prevent autistic symptoms and support adaptation and well-being throughout life (Dawson, 2008). Early interventions (before 3 years old) show promise in reducing ASD prevalence by returning development to normal trajectories. Particularly, by addressing environmental factors and genetic susceptibility during the preconception and perinatal periods, the risk of ASD can be reduced (Francis et al., 2021). For instance, informing parents about and addressing conditions related to toxic exposures, such as lead, mercury, pesticides, and air pollutants, as well as ensuring proper nutritional intake (e.g., folic acid, omega-3 fatty acids, and vitamins), and reducing the risk of high maternal stress, could be impactful in reducing the risk of ASD (Francis et al., 2021). Similarly, Dawson (2008) also points out that improvements in cognitive and affective developmental neuroscience, developmental

psychopathology, neurobiology, genetics, and applied behavior analysis lead to a more positive standpoint for autistic individuals; especially with the help of more improved early detection and treatment methods, altering the trajectory of early behavioral and neurological development to prevent the full manifestation of ASD will be possible.

Chapter 2

The Association between Autistic Traits and Social Anxiety, and the Influence of Familism

2.1 Autistic Traits

Autistic traits can be described as sub-threshold difficulties in socialization, communication, and restricted interests that do not fulfill the formal criteria for ASD (Constantino

and Todd, 2003). Research on autistic traits in the broader community is beneficial because the continuity of autism to autistic traits emphasizes the fact that autism is a form of human diversity. On the other hand, this should not mean that every individual possesses some type of autistic characteristics (Sasson & Bottema-Beutel, 2022). A clinical diagnosis is unnecessary while assessing autistic traits, its assessment typically done via self-reporting surveys (Gernsbacher et al., 2017). Autistic traits can be found in individuals with different developmental disabilities, moreover, sometimes even autistic people may not reach clinical cut-offs in autistic traits (Sasson & Bottema-Beutel, 2022). Additionally, according to a study carried out by Palmer et al. (2015), two distinct sub profiles of autistic traits among the broader population are identified: one characterized by greater social challenges and reduced attention to detail, and another marked by milder social difficulties and heightened detail orientation, suggesting that we can talk about different sub profiles of autistic traits in the general population.

Research on the heritability of autistic traits in the general population has revealed significant findings. Twin studies have shown that the genetic influence on autistic traits can range from 36% to 87%, depending on the assessment method and age group (Ronald & Hoekstra, 2011). Furthermore, the assessment of autistic traits in various age groups, from 2-year-olds to late adolescence, has consistently shown moderate to high genetic influence estimates for language (Stromswolts, 2001) and specific language impairment (Bishop, 2002), emphasizing the genetic influence on these traits (Ronald & Hoekstra, 2011).

Early signs of autistic traits may become noticeable as early as infancy, showing variances in social interaction, communication, and fine motor skills. These early indicators are linked to eventual ASD diagnosis and autistic traits in the broader population (Bolton et al., 2012). Additionally, autistic-like traits can emerge by the ages of 12 months in high-risk siblings of

individuals with ASD, with these traits being associated with more social-communication impairment, lower cognitive abilities, and more internalizing problems at the age of 3 (Georgiades et al., 2013).

Regarding the long-term stability of autistic traits in toddlers' typical development, a moderate level of stability over time has been observed across the broader population (Constantino et al., 2019). Additionally, a modest long-term stability in social autistic traits was identified in males, although no significant longitudinal associations for non-social autistic traits were found (Whitehouse et al., 2011). Furthermore, Robinson et al. (2011) found that autistic traits are highly stable in the general population from ages 7 to 13, with no significant change in mean scores for females and a slight decrease for males.

2.2 Social Anxiety

Social anxiety, referred to as social phobia, is a pervasive condition involving extreme apprehension and evasion of social encounters (Morrison & Heimberg, 2013). People with social anxiety typically endure intense stress and nervousness in social environments, causing notable disruptions in various aspects of their lives, therefor, referred as social anxiety disorder (SAD) or social phobia (Morrison & Heimberg, 2013). The fear of being in a position where an individual is negatively evaluated or judged by others is a central feature of social anxiety, which drives individuals to avoid social interactions or endure them with intense discomfort (Bögels et al., 2010).

The DSM-IV (American Psychiatric Association, 1994) criteria for social anxiety disorder (SAD) offer a structure to comprehend the diagnostic features of this condition. These criteria highlight the fear of social situations, the associated anxiety symptoms such as blushing, trembling,

and palpitations, and the overlap with avoidant personality disorder. The DSM-IV criteria emphasized the possibility of subtyping SAD based on feared social situations, such as performance anxiety or specific interactional fears. Individuals with social anxiety might show a range of symptoms, including excessive self-consciousness, fear of scrutiny, avoidance of social gatherings, difficulty speaking in public, and physical manifestations of anxiety like sweating or trembling.

The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5; American Psychiatric Association, 2013) has implemented various modifications to the diagnostic criteria for SAD, which affect both conceptual understanding and clinical practice. The DSM-5 emphasizes the nervousness about being evaluated negatively as a central aspect of SAD and considers the sociocultural context when determining the disproportionality of an anxious response (Heimberg et al., 2014). The DSM-5 has replaced the DSM-IV generalized subtype specifier with a performance-only specifier, although evidence for this change, especially in youth, is limited (Kerns et al., 2013). Furthermore, the DSM-5 version of SAD is believed to increase prevalence rates since in DSM-5, the individual's assessment of the fear as unreasonable or not is not required anymore. For instance, when DSM-IV criteria are applied, the 1-month prevalence of SAD in older adults is 2.5%, but when DSM-5 criteria are used, which do not require the individual's assessment of the fear as unreasonable, the prevalence increases to 5.1% (Karlsson et al., 2016).

Cultural factors such as individualism/collectivism and gender roles play a significant part in influencing the prevalence and expression of social anxiety and SAD (Hofmann et al., 2010). For example, individuals from collectivistic cultures, such as those in Asia, South America, the Pacific Islands, and Southern European countries, may experience social anxiety differently than those

from individualistic cultures due to the strict rules and expectations that individuals need to follow in certain social situations (Triandis et al., 1984; Argyle, 1986). Moreover, since norms are particularly predicting life satisfaction in collectivist societies, but not in individualist societies (Suh et al., 1998), there might be a link between the cultural positioning of an individual, norms that he/she possesses, and having a disorder such as SAD (Hofmann et al., 2010). As an example to the culture-specific expression of social anxiety, Taijin kyofusho, can be given. It is particularly observed in Japan and Korea, involves a fear of offending or embarrassing others through one's appearance, behavior, or body odors (Takahashi, 1989). SAD revolves around the fear of personal embarrassment or negative evaluation, whereas Taijin kyofusho is characterized by the fear of causing offense or embarrassment to others. This might reflect the cultural norms and values of these societies, where interpersonal relationships and social harmony are highly valued. Thus, understanding the cultural, racial, and ethnic background of an individual is crucial in assessing the level and the manifestation of social anxiety and SAD. (Hofmann et al., 2010).

How social anxiety and SAD differed between men and women in clinical samples has also been investigated. (Hofmann et al., 2010). Women are more likely to develop SAD than man, although there is a small difference (Kessler et al., 1994). Furthermore, it has been observed that adolescents with elevated degrees of social anxiety perceived diminished degree of acceptance and help from their peers, coupled with feeling less romantically attractive, with these associations being stronger for girls (La Greca & Lopez, 1998).

Cognitive-behavioral therapies and pharmacological treatments have both shown efficacy for social anxiety disorder. In a meta-analysis by Gould et al. (1997), both CBT and pharmacological treatments yielded better outcomes than the control conditions, with controlled effect sizes of .74 for CBT and .62 for pharmacological treatments. However, a meta-analysis by

Fedoroff and Taylor (2001) suggested that pharmacotherapies may achieve better outcomes than cognitive-behavioral treatments, particularly SSRIs (*Selective Serotonin Reuptake Inhibitor*) and benzodiazepines. CBT for social anxiety disorder commonly involves components such as psychoeducation, cognitive restructuring, exposure therapy, and relaxation training. On the other hand, which of these components is the most beneficial in terms of leading to positive outcomes in individuals with SAD is unknown (Rodebaugh et al., 2004).

There are certain barriers to prevent individuals with social anxiety being treated. The main obstacles to treatment identified by individuals with social anxiety in the study conducted by Olfson et al. (2000) included financial barriers and fear of judgment. According to the study, those experiencing social anxiety had a higher tendency to report functional impairment, social isolation, and suicidal thoughts as opposed to those not having social anxiety and less likely to deny having an anxiety disorder, moreover, uncertainty over where to go for treatment, insufficient acknowledgement of social anxiety by healthcare experts, and self-appraisal of illness severity and the belief that one could handle symptoms independently are mentioned as other barriers to treatment for individuals with social anxiety.

2.3 The Association between Autistic Traits and Social Anxiety

Several research points out that autistic people may show elevated degrees of anxiety (Bellini, 2004; Gillott, Furniss, & Walter, 2001). Moreover, Liew et al. (2015) found out that there is a positive association between autistic traits and symptoms of anxiety and depression.

Regarding the link between autistic traits and social anxiety, some of the findings revealed that clinically relevant degrees of autistic traits were associated to clinically significant degrees of

social anxiety (Freeth et al., 2013). Additionally, social skills were shown to be a stronger indicator of social anxiety in males compared to females. Conversely, limited attention-switching ability emerged as a more prominent indicator of social anxiety in females than in males, suggesting that gender differences may influence how individuals benefit from varied intervention approaches targeting specific aspects of the condition. It has also been reported that children diagnosed with anxiety disorders tend to score higher than typically developing peers on both early ASD traits and present-day ASD symptoms. Notably, a significant link was established between Social Anxiety Disorder symptoms and early ASD characteristics (van Steensel et al., 2013). Additionally, while a subset of children diagnosed with anxiety disorders showed a moderate ASD-like profile, the majority exhibited a milder pattern of ASD-like behaviors. Furthermore, the study found that communication difficulties were the most frequently reported symptoms among clinically anxious children compared to typically developing peers, which highlights a potential connection between autistic traits and anxiety disorders in children.

Similarly, Liew et al. (2015) discovered that autistic traits were positively correlated with various symptoms including social anxiety, obsessive-compulsive, worry, and depressive signs, with social competence mediating the link between autistic traits and social anxiety markers. In addition, Baiano et al. (2022) reported that particularly social autistic traits, as opposed to non-social traits, predict general anxiety sensitivity and concerns related to social and cognitive domains, highlighting the importance of differentiating between these traits.

According to Yang et al. (2021), the underlying mechanism behind the link between autistic traits and social anxiety has yet to be established. They examined coping styles and perceived security as potential explanatory factors, emphasizing the moderating role of the FKBP5 gene variant rs3800373. The study showed that autistic traits and negative coping were significant

positive predictors of social anxiety, while positive coping, interpersonal security, and sense of control were significant negative predictors. Moreover, The FKBP5 gene rs3800373 was shown to influence the association between autistic traits and social anxiety, with individuals carrying the CC genotype demonstrating reduced impact on the path from autistic traits to social anxiety compared to the ones with the carrier A genotype, meaning that this moderation effect suggests that genetic differences in the FKBP5 gene rs3800373 may have a major impact on how autistic traits play a role in the emergence of social anxiety (Yang et al., 2021).

2.4 Familism

Familism is a cultural value that highlight interdependent family relationships which are warm, close, and supportive (Campos et al., 2014). The first examples of the studies of familism were aimed to shed light on the differences between European and U.S. Latino culture (Keefe et al., 1979). Earlier, familism was strongly identified with characteristics such as loyalty, reciprocity, and solidarity among family members (Triandis et al., 1982). Since Latinos tend to fulfill family obligations and seek social support from their family members more compared Europeans, high level of familism is found apparent in Latinos (Fuligni et al., 1999). Moreover, other collectivist cultures, such as East Asians in the United States, have similar familism scores to U.S. Latinos (Schwartz et al., 2010). Therefore, according to (Campos et al., 2014) the concept of "familism" can be applied to a wide range of U.S. groups.

As examples to the advantages of familism, healthy pregnancy (Campos et al., 2008), higher well-being (Schwartz et al., 2010), decreased possibility of adolescent delinquency (Pabon E, 1998) and higher partner support and partner closeness (specific to the Latino sample) (Campos et al., 2016) can be given. As examples to the disadvantages, however, increased degree of distress (Schwartz et al., 2010) and symptoms of inflammation can be given (Fuligni et al., 2009). These

responses are believed to be stemmed from the tension of complying to family obligations and reactions to the family conflicts (Fuligni et al., 2009).

Familism has been found to function as a protective factor against mental health issues including depression and suicide, albeit with small effect sizes, however, no statistically significant association has been found with substance abuse and externalizing behaviors (Mora et al., 2016). Additionally, among various ethnicities, familism correlates with decreased feelings of loneliness, depression, and physical symptoms. It also functions as a defense against the adverse impacts of stress, by boosting self-esteem and subjective health (Corona et al., 2017). Familism can also influence youth psychological adjustment through parenting behaviors such as monitoring and discipline strategies, proposing a behavioral process model in which familism positively impacts family dynamics and youth development (Hernández & Bámaca-Colbert, 2016). Furthermore, Stein et al. (2014) emphasized that the protective effect of familism is most apparent during adolescence and mentioned the importance of studying the effects of familism across different developmental stages.

2.5 The Association between Social Anxiety and Familism

Broadly speaking, familism found to be linked to improved mental well-being and physical health, serving as a protective factor against stress among various ethnicities (Corona et al., 2016). For instance, in investigating the impact of familism on mental health, the role of familism in enhancing mental well-being through the promotion of closeness and social support has been emphasized, moreover, a positive association has been found between higher levels of familism, increased family closeness, greater social support and improved psychological health outcomes, in

different cultures (Campos et al., 2014). Familism served as a safeguard against the adverse impact of attachment anxiety on Social Self-Efficacy (SSE), suggesting that familism could offer a stable foundation for alleviating social anxiety within this cultural setting (Zamudio et al., 2020).

Family cohesion has been observed to be significantly related to social phobia in the aggregated Latino sample, with higher perceived family cohesion was associated with a reduced probability of meeting the diagnostic criteria for social phobia. Similarly, in the Mexican subgroup, family cohesion demonstrated a strong relationship with social phobia, where increased levels of family cohesion were associated with a lower likelihood of meeting the diagnostic criteria for social phobia. On the other hand, for all other Hispanic subgroup, no meaningful relationship was identified between family cohesion and social phobia (Priest and Denton., 2012).

While familism is often lauded for its contributions to familial support and cohesion within communities, the findings of the following study reveal a more complex relationship between familism and mental health outcomes. Familism, referred to in the research as allocentrism, reflects a cultural tendency to prioritize family values and interconnectedness, while idiocentrism emphasizes individual goals and autonomy over collective interests. Results indicated a positive association between family allocentrism and depression, anxiety, and stress, suggesting that individuals valuing family cohesion might experience heightened mental health challenges. In contrast, family idiocentrism showed a negative correlation, implying that a focus on individualistic values within the family may be linked to lower levels of these mental health problems (Mayorga-Muñoz et al., 2023)

2.6 The Association between Autistic Traits and Familism

To this point, the connection between autistic traits and familism has not been directly investigated. However, studies exploring the associations of autistic traits with similar constructs, such as family or social cohesion, have been undertaken.

A paper by Ten Hoopen et al. (2023) investigated the impact of autism traits in children on family functioning longitudinally. They found clear connections between autism traits and family functioning, showing specific patterns in their analysis. The study revealed a significant association between lower levels of autistic traits at the diagnostic assessment and improved family functioning twelve months later across the whole sample of children, regardless of the presence of internalizing (e.g., anxiety, depression) or externalizing (e.g., aggression, hyperactivity) behavioral problems. Research on Latinx parents of children experiencing developmental delays, including ASD, has highlighted the effect of affiliate stigma on parenting stress. Familism was significantly associated with stigma and stress experienced by parents. The association was strongest in families with reduced levels of familism values, indicating that familism could serve as a safeguard against self-stigma and parenting stress (Martin et al., 2022). Furthermore, research has shown that parents of autistic children often experience more psychopathology, marital difficulties, and family dysfunction in contrast to parents of children with typical development (Gau et al., 2012). Additionally, mothers of autistic children are inclined to display more psychopathology and marital maladjustment than fathers. Similarly, Factor et al. (2019) found that characteristics autistic children such as behavioral challenges and communication difficulties, significantly impact caregiver stress and mental health, leading to feelings of isolation, fatigue, and uncertainty within the family unit.

Chapter 3

The Present Study

The core of this study rests upon extensive empirical evidence highlighting the positive correlation between autistic traits and social anxiety. Research by Liew et al. (2015) underscores this association, revealing a positive link between autistic traits and various symptoms, including social anxiety, thereby necessitating an in-depth exploration of this relationship. Moreover, studies such as those conducted by Freeth et al. (2013) and Van Steensel et al. (2013) have demonstrated the predictive power of specific autistic traits in eliciting social anxiety symptoms, emphasizing the

need to identify these key predictors. Additionally, the study by Minami & Horikawa (2021) further supports the relationship between high ASD scores and Social Anxiety Disorder (SAD) in high school students, indicating the relevance of investigating this association across different developmental stages.

In addition to the connection between autistic traits and social anxiety, familism emerges as a crucial contextual factor that may influence this association. Familism, rooted in interdependent family relationships and characterized by warmth and solidarity, has been linked to enhanced psychological well-being and serves as a protective factor against mental health problems (Campos et al., 2014; Hernández & Bámaca-Colbert, 2016). These studies highlight the function of familism in promoting family closeness and positive parenting behaviors, which are integral to individuals' psychological adjustment and well-being. Considering the significance of familial support and dynamics in shaping mental health outcomes, understanding how familism interacts with autistic traits and social anxiety is essential.

3.1 Aims and Hypotheses

Building on previous research findings, this study, first aims to examine the connection between autistic traits and social anxiety among children aged 6-18, with a specific focus on identifying whether autistic traits can predict social anxiety. Second, it seeks to explore the influence of familism in moderating this relationship. For investigating this relationship, building upon previous research, it is hypothesized that elevated autistic traits will be positively associated with heightened levels of social anxiety (Bellini, 2004; Gillott, Furniss, & Walter, 2001; Liew et al., 2014; Freeth et al., 2013). Moreover, given the protective role of familism in promoting family

cohesion and support, it is hypothesized that familism will moderate the relationship between autistic traits and social anxiety. Specifically, higher levels of familism are expected to buffer the negative impact of autistic traits on social anxiety symptoms, leading to lower levels of social anxiety among individuals with elevated autistic traits (Corona et al., 2016; Campos et al., 2014; Zamudio et al., 2020).

These hypotheses serve as the guiding framework for the ongoing study, aiming to elucidate the complex interaction between autistic traits, social anxiety, and familism.

3.2 Methods

3.2.1 Participants

The participants were 202 parents from Italy with children aged 6-18 years. The inclusion criteria were being an Italian speaking parent of a typically developed child aged between 6-18 years. Moreover, the participant must voluntarily agree to participate in the research and acknowledge their understanding of the research objectives, the confidentiality of the data, the use of the data solely for educational and research purposes, and their freedom to leave the study at any point without facing any consequences. Participants are excluded if their children are not aged 6 to 18, are diagnosed with any developmental or psychological disorders, did not complete the survey, or did not provide informed consent to participate in the research.

3.2.2 Research Design

The current study was designed and conducted as part of a brother cross-cultural comparison, which aims to examine the interaction among variables such as autistic traits, social anxiety, emotional intelligence, and family cultural values within Mexican and Italian populations.

Ethical approval for the current research project was gathered from the relevant institutional review board at the University of Padua. All procedures and data collection methods adhere to the ethical guidelines outlined in the Italian legislation, including the D.Lgs.196/2003 on privacy and the EU GDPR 679/2016 on personal data protection, as well as the ethical standards stated in the Code of Conduct for Italian Psychologists. Participant confidentiality and privacy are strictly protected throughout the study, and data are assured to be handled anonymously and confidentially. The research team was committed to transparency and accountability in all aspects of the research process.

The current study adopted a correlational and non-experimental research design to examine the relationship between social anxiety, autistic traits, and the possible moderating role of familism in children aged 6-18 years old in Italy through an online parent survey.

Data for this study were collected using social media platforms, primarily Facebook, to reach parents of children aged 6-18 years old in Italy. A flyer including information about the university, researchers, participation requirements, survey, content, and duration with an informed consent has been created and used. The survey and flyer were regularly shared in a wide range of parent groups on Facebook for 3 months, reaching over 25,000 individuals. 317 participants filled the survey. From these, 202 participants completed it fully, also considering for inclusion in the study based on age criteria and absence of developmental disorders.

3.2.3 Materials

Participants completed a survey (158 items in total) through the Qualtrics platform (Qualtrics, Provo, UT), comprising various sections. The survey included Likert scales, meaning

that respondents rated their level of agreement with various statements on a scale, typically ranging from “strongly disagree” to “strongly agree”.

a. Demographic measures

The demographic data section (11 items) gathered information regarding the child's gender, age, presence of diseases or developmental conditions, respondent identity, parental demographic details (such as birthday, marital status, occupation, education level, religious affiliation, number of siblings, and place of residence).

b. Autistic traits

To assess autistic traits, participants completed the Social Responsiveness Scale (SRS-2; Constantino and Gruber, 2012), consisting of 65 items, with 5 subscales: social awareness, social cognition, social communication, social motivation, and autistic mannerisms, respectively. Participants' responses were evaluated on a 4-point Likert scale (0-1-2-3), with reversed items. The Italian version of SRS-2 was used in the study. The tool typically takes 15 to 20 minutes for parents to complete. The SRS-2 exhibits strong psychometric properties, such as internal consistency, test-retest reliability, and agreement between different raters across diverse cultural contexts and it correlates well with other recognized autism diagnostic tools like the Autism Diagnostic Observation Schedule (ADOS) and the Autism Diagnostic Interview-Revised (ADI-R), showing good convergent validity (Constantino et al., 2003; Bölte et al., 2008). Moreover, The SRS-2 has been found to be valid to be used in various cultures, including German, Taiwanese, and Iranian populations, therefore, the tool can effectively distinguish between ASD and other developmental disorders in diverse countries (Bölte et al., 2008). Examples of the items include: "Seems much

more restless in social situations compared to when alone," "Facial expressions are not congruent with what is being said," and "Seems comfortable when interacting with others."

c. Social Anxiety

Social anxiety was measured using the Social Phobia and Anxiety Inventory for Children (SPAI-C; Beidel et al., 1995), comprising 26 items. Answers were assessed using a 3-point scale (1-2-3). The Italian version of SPAI-C was used in the study. It is a tool designed to assess social anxiety in childhood and early adolescence, requires approximately 10 minutes to finish. It measures somatic, cognitive, and behavioral symptoms. A score of 18 or higher suggests the likely presence of social anxiety. The SPAI-C has undergone extensive reliability and validity testing globally, demonstrating strong internal consistency and reliability in clinical and research contexts (Beidel, 1996). It also shows good convergent validity with both self-report and clinician-administered (Beidel, 1996) social anxiety measures. Additionally, it effectively discriminates between children with social anxiety and those who have other anxiety disorders and is a reliable measure of treatment effectiveness (Beidel et al., 1995). Examples of the questions from the SPAI-C include: "Feels uncomfortable joining large groups of people," "Is afraid of being the center of attention," and "Feels uncomfortable when having to do something while others are watching."

d. Familism

Lastly, familism was assessed using the Mexican American Cultural Values Scale (MACVS; Cuéllar, Arnold, & González, 1995), consisting of 37 items. Responses were recorded on a 5-point Likert scale (1-2-3-4-5). The MACVS includes the following subscales: Familism (with 3 different subscales: support as 6 items, obligations as 5 items, and referent as 5 items, respectively), Religion, Independence & Self-Reliance, Competition & Personal Achievement,

and Traditional Gender Roles. The Italian adaptation of the MACVS was used in the study. Some examples to the items of subscales of familism can be given as follows: “Parents should teach their children that the family always comes first (support),” and “Children should be taught that it is their duty to care for their parents when their parents get old (obligation)”.

3.2.4 Procedure

Participants accessed the survey through the Qualtrics platform (Qualtrics.com), where they were presented with a series of questions covering demographic information and various questionnaires. The survey link and QR code to access the survey, along with an introductory paragraph and a specifically created flyer, were shared across social media platforms, particularly Facebook, within parent groups and communities targeting individuals with children aged 6-18 years old in different cities or regions of Italy. Before starting the survey, participants were presented with an informed consent form summarizing the objective of the research, confidentiality measures, and voluntary participation. It was emphasized that participation was voluntary, and participants were not paid for their involvement in the study. Upon completion of the survey, the collected data were exported from Qualtrics to an excel file and analyzed using R Studio (Posit team, 2023). Statistical analyses, including descriptive, correlation and regression analyses, were carried out to investigate the connections between variables of interest.

Chapter 4

Results

Once data collection was finished, all data from Qualtrics (www.qualtrics.com) was exported to a Microsoft Excel (Microsoft Corporation, 2018) spreadsheet. The analysis was performed using R Studio programming language (Posit Team, 2023). Initially, subjects who did

not meet the predefined exclusion criteria were excluded from the dataset. Then, an exploratory analysis was conducted to detect any outliers that could potentially skew the results. After removing these outliers, the final sample comprised 202 participants.

4.1 Descriptive Statistics of the Sample

Descriptive statistics were used to provide a comprehensive overview of the sample's characteristics (Table 1). Various statistical measures such as mean and standard deviation were calculated to summarize the data. This preliminary analysis was essential to understand the demographic background and key variables of the participants.

Table 1

Descriptive analysis of participants' and their children's age

	Age (offspring)		Age (father)		Age (mother)	
	Male	Female	Male	Female	Male	Female
Valid	103	99	103	99	103	99
Missing	0	0	0	0	0	0
Mean	12.134	12.226	47.680	46.629	44.251	44.060
Std. Deviation	3.552	3.600	6.804	7.480	6.051	6.885
Range	12.260	12.540	35.290	36.090	26.230	35.100
Minimum	5.320	5.270	33.260	26.720	30.550	26.030
Maximum	17.580	17.810	68.550	62.810	56.780	61.130

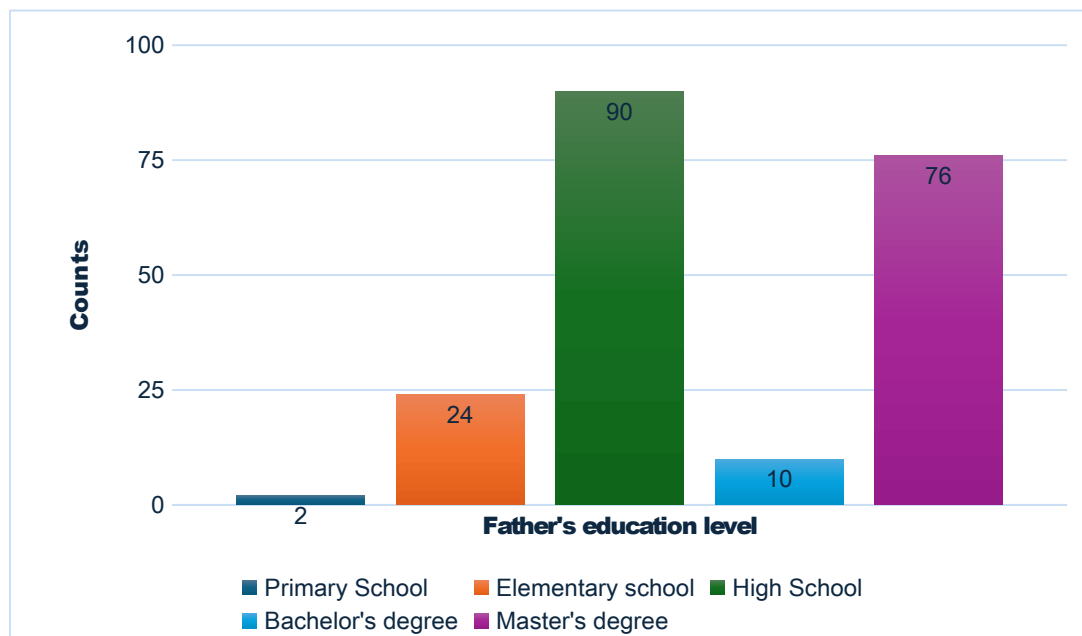
Note: Table 1 displays the descriptive statistics of the demographic data showing participants' and their children's age.

There were 103 male and 99 female children in the sample, with no missing data (317 participants had filled the questionnaire; however, 115 of them were excluded based on the

inclusion criteria). The average age of the children was 12.13 years (SD = 3.55) for males and 12.22 years (SD = 3.60) for females. The similarity of mean age and standard deviation indicates that gender does not cause significant biases at the age level. Fathers' average age was 47.68 years (SD = 6.80) for male children and 46.62 years (SD = 7.48) for female children. Mothers' average age was 44.25 years (SD = 6.05) for male children and 44.06 years (SD = 6.88) for female children. These data show general uniformity in ages for both genders, suggesting that age biases are not a potential confounding factor. The range represents the difference between the maximum and minimum age, with children's age range being about 12.26 years for males and 12.54 years for females.

Figure 1

Father's education level

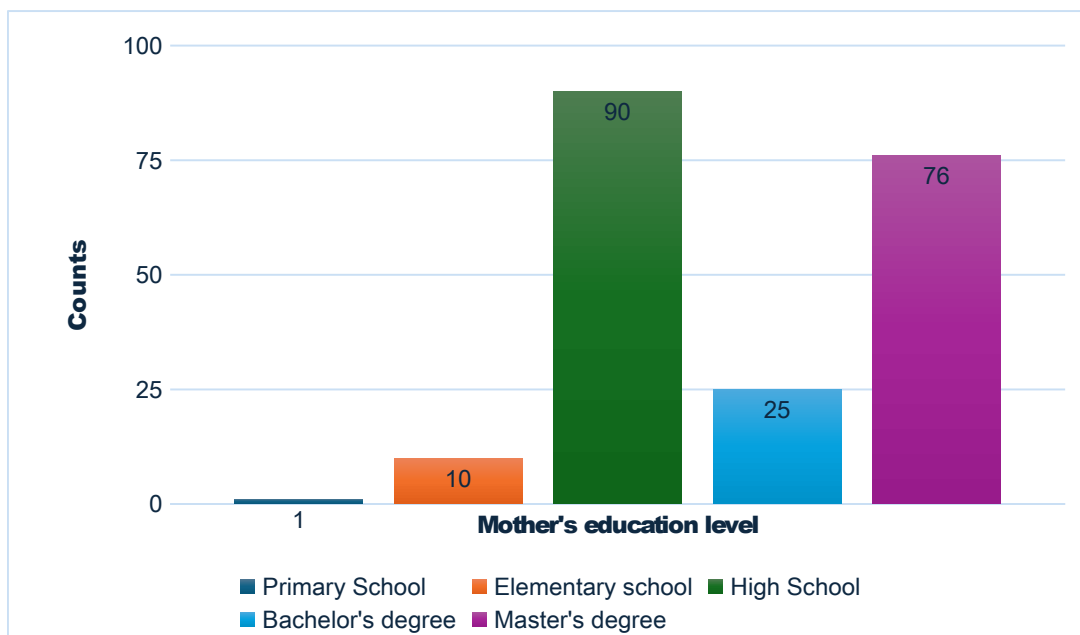


Note: The bar chart provides a visual representation of distribution of father's education levels. The numbers on top of the plots show the actual number of participants who graduated from the corresponding education level.

According to the demographics, of the 202 fathers, 76 hold a master's degree, 90 hold a high school degree, 10 hold a bachelor's degree, 24 graduated from elementary school, and 2 graduated from primary school (Figure 1).

Figure 2

Mother's education level

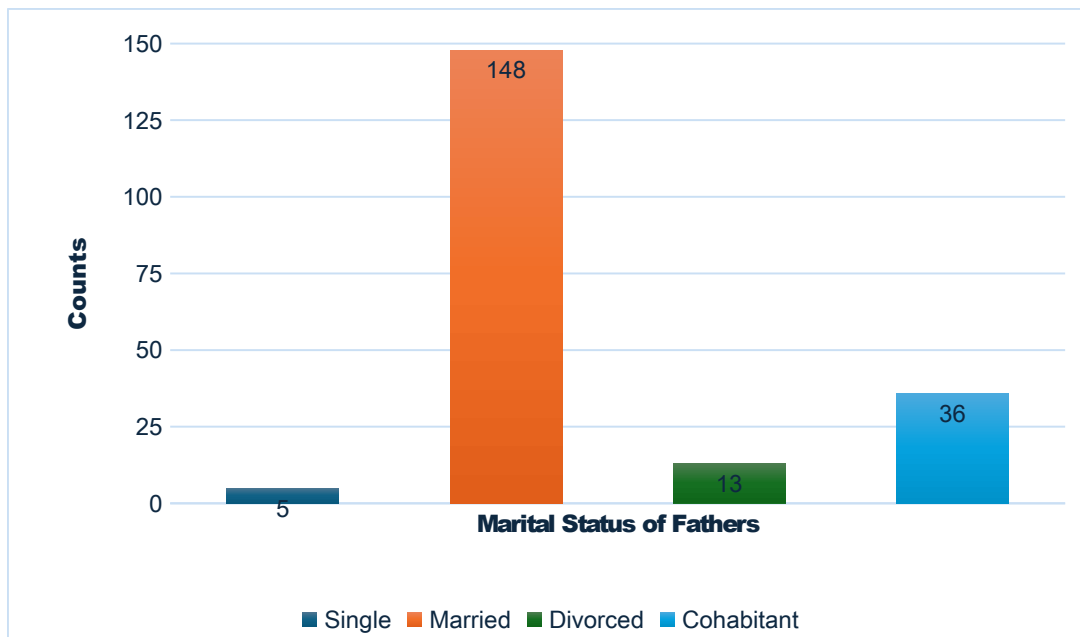


Note: The bar chart provides a visual representation of distribution of mother's education levels. The numbers on top of the plots show the actual number of participants who graduated from the corresponding education level.

Similar to the father's education level, of the 202 mothers, 76 hold a master's degree, 90 hold a high school degree, 25 hold a bachelor's degree, 10 graduated from elementary school, and 1 graduated from primary school (Figure 2).

Figure 3

Father's Marital Status

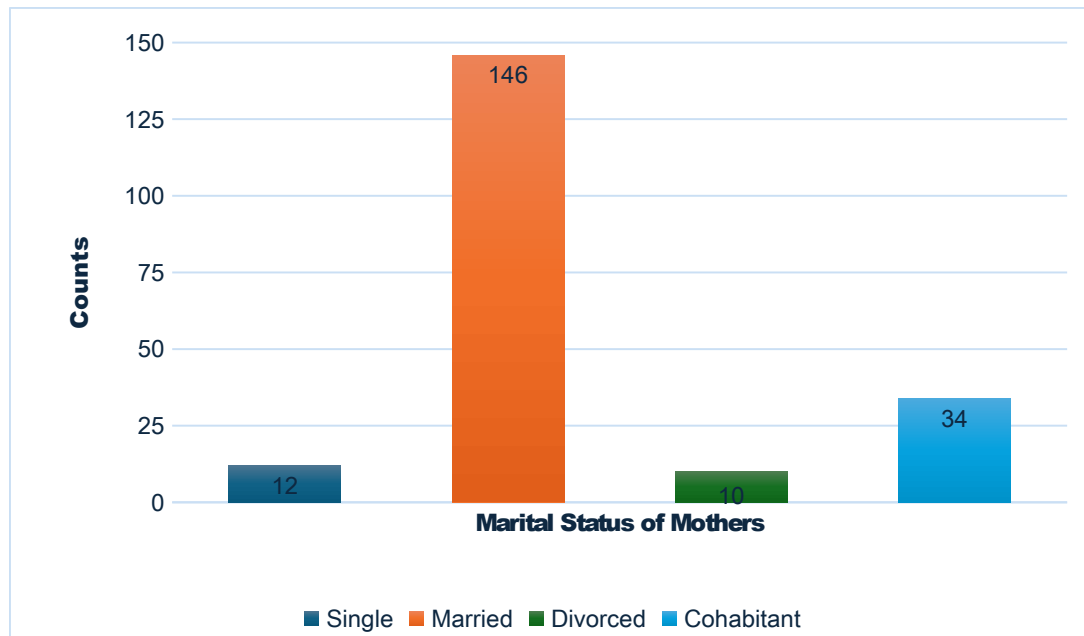


Note: The bar chart provides a visual representation of distribution of father's marital status. The numbers on top of the plots indicate the actual number of participants with the corresponding marital status.

According to the demographics, of the 202 fathers, 148 are married, 36 are cohabitants, 13 are divorced, and 5 are single (Figure 3).

Figure 4

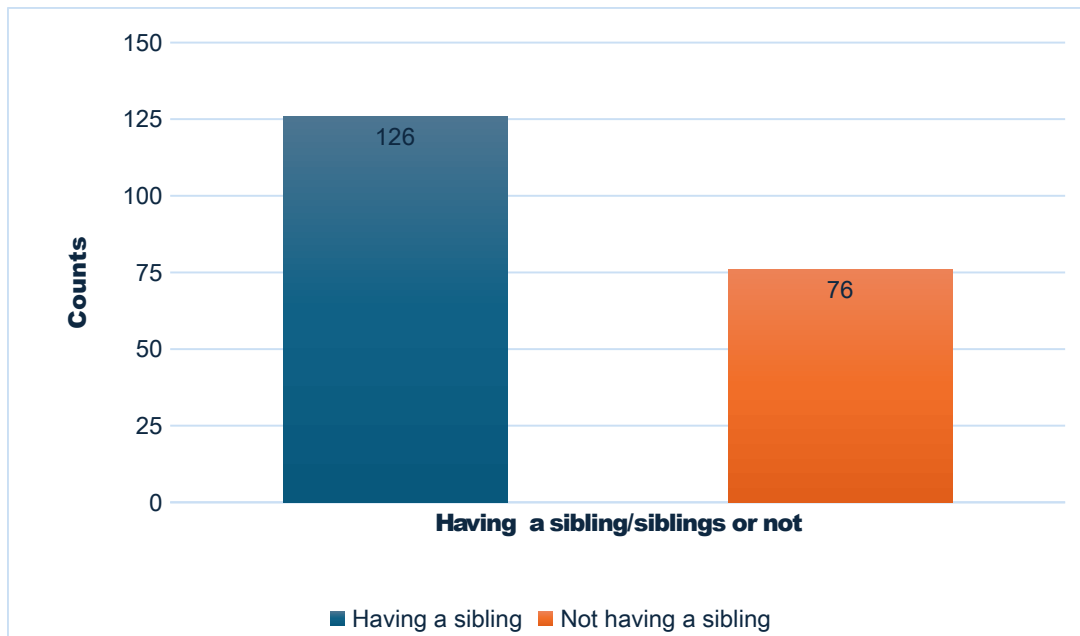
Mother's Marital Status



Note: The bar chart provides a visual representation of distribution of mother's marital status. The numbers on top of the plots show the actual number of participants with the corresponding marital status.

Figure 5

Having a sibling/siblings or not

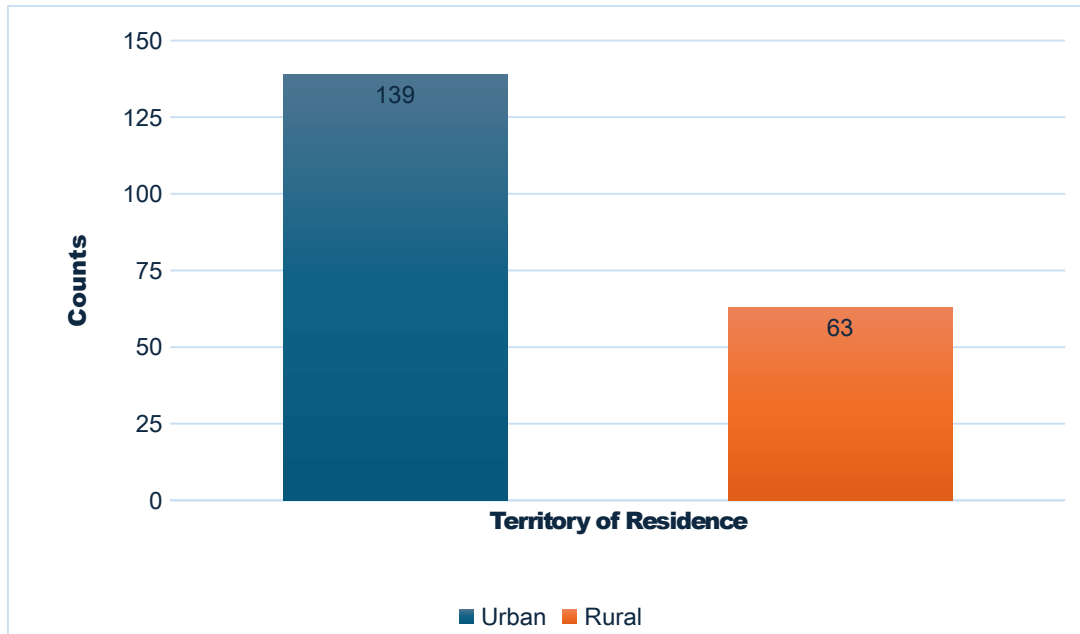


Note: The bar chart visually depicts the sibling status of participants' children. The numbers on top of the plots indicate the actual number of participants whose children either have a sibling or siblings, or do not have any siblings.

According to the demographics, of the 202 children, 126 have a sibling of siblings while 76 do not have sibling or siblings (Figure 5).

Figure 6

Territory of Residence



Note: The bar chart provides a visual representation of the distribution of parents' territories of residence. The numbers on top of the plots show the actual number of participants living either in urban or rural area.

According to the demographics, of the 202 family, 139 stated that they live in an urban setting while 63 live in rural area (Figure 6).

4.2 Testing the First Hypothesis

It was proposed that increased autistic traits were expected to show a positive association with heightened levels of social anxiety (Bellini, 2004; Gillott, Furniss, & Walter, 2001; Liew et al., 2014; Freeth et al., 2013). Pearson correlation analysis was conducted to examine the relationships among SRS (autistic traits), SPAI (social anxiety), and Familism variables. The findings validated the initial hypothesis. The results are summarized in Table 2.

Table 2

Pearson's correlations

Variable		SRS	SPAI	Familism
1.SRS	Pearson's r	---		
	p-value	---		
2. SPAI	Pearson's r	0.637	---	
	p value	<.001	---	
3. Familism	Pearson's r	0.180	0.150	---
	p value	0.010	0.033	---

A significant positive correlation was found between SRS and SPAI ($r = 0.637, p < 0.001$), indicating a strong association between autistic traits and social anxiety scores. This finding suggests that higher SRS scores are associated with higher SPAI scores. Additionally, a significant but weak positive correlation was observed between SRS and familism ($r = 0.180, p = 0.010$). This indicates that there is a small but significant relationship between autistic traits and familism, with higher autistic traits scores being associated with higher familism scores. Similarly, the correlation between SPAI and Familism was found to be significant ($r = 0.150, p = 0.033$). This also suggests a small positive relationship between SPAI and familism scores.

4.3 Hierarchical regression

A hierarchical regression was conducted to determine the effect of the predictor (autistic traits) on the outcome variable (social anxiety), and the impact of familism, on this relationship, by adding the other variables step by step. All the process highlighting the differences in each step is described below.

In the first step of the current research, age and gender were entered into the model as control variables. The results are summarized in the table 3.

Table 3

1st model: Age and Gender

Model 1		Estimate(β)	Sd error	t-value	p-value
	Intercept	32.6764	2.3590	13.852	<2e-16***
	Age	0.4131	0.1798	2.297	0.0226 *
	Gender	1.0606	1.2802	0.828	0.4084

*Note: The table shows the estimate, standard error (Sd error), t-value, p-value obtained for Model 1. Asterisks symbol is used to demonstrate significance in the results: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$*

The model was close to reaching statistical significance ($F(2,199) = 3.007, p = .052$). It explained 2.93% of the variance in SPAI scores ($R^2 = .0293$). This suggests that while the model was not quite significant at the conventional $p < .05$ level, it was very close and indicates a small amount of variance in SPAI scores. Age was a significant predictor ($\beta = .4131, t(199) = 2.297, p = .0226$) while gender was not ($\beta = 1.0606, t(199) = 0.828, p = .4084$).

In the second step, SRS was added as a predictor. The results can be shown in Table 4.

Table 4

2nd Model: Age, Gender, and SRS

Model 2		Estimate(β)	Sd error	t-value	p-value
	Intercept	28.90754	1.86476	15.502	<2e-16***

	Age	-0.09251	0.14675	-0.630	0.5291
	Gender	1.73941	0.99780	1.743	0.0828
	SRS	0.25683	0.02246	11.434	<2e-16***

*Note: The table shows the estimate, standard error (Sd error), t-value, p-value obtained for Model 1. Asterisks symbol is used to indicate statistically significant results: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$*

The overall model was statistically significant ($F(3,198) = 46.89, p < 2.2e-16$). It clearly shows that the predictors selected account for a substantial part of the variance in SPAI scores. The model's multiple R^2 was 0.4154, and the adjusted R^2 value was 0.4065. This suggests that approximately 41.5% of the variance in SPAI scores can be explained by the predictors. The linear regression model revealed that SRS is a significant predictor of SPAI scores ($\beta = .2568, t(198) = 11.434, p = <2e-16$). Higher SRS scores associated with higher SPAI scores. Age ($\beta = .0925, t(198) = -0.630, p = .5291$) and gender ($\beta = 1.7394, t(198) = 1.743, p = .0828$), however, did not significantly predict SPAI scores in this model.

In the third step, familism was added as an additional predictor. The results are shown in Table 5.

Table 5

3rd Model: Age, Gender, SRS, and Familism

Model 3		Estimate(β)	Sd error	t-value	p-value
	Intercept	26.71689	2.71718	9.833	<2e-16***
	Age	-0.14947	0.15541	-0.962	0.3374

Gender	1.85941	1.00309	1.854	0.0653
SRS	0.25498	0.02251	11.326	<2e-16***
Familism	0.05482	0.04948	1.108	0.2693

Note: The table shows the estimate, standard error (Sd error), t-value, p-value obtained for

*Model 1. Asterisks symbol is used to indicate statistically significant results: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$*

The overall model was statistically significant ($F(4,197) = 35.51, p < 2.2e-16$). This model explained 41.9% of the variance in SPAI scores ($R^2 = 0.419$, adjusted $R^2 = 0.4072$). The linear regression model reveals that SRS remains as a significant predictor of SPAI scores ($\beta = .2549$, $t(197) = 11.326, p < 2e-16$). Familism, however, did not have a significant predictive effect ($\beta = .0548$, $t(197) = 1.108, p = .2693$). Similarly, age ($\beta = -.1494$, $t(197) = -.962, p = .3374$) and gender ($\beta = 1.8594$, $t(197) = 1.854, p = .0653$) also did not significantly predict SPAI scores in this model.

4.4 Testing the Second Hypothesis

It was hypothesized that higher levels of familism are expected to buffer the negative impact of autistic traits on social anxiety symptoms, leading to lower levels of social anxiety among individuals with elevated autistic traits (Corona et al., 2016; Campos et al., 2014; Zamudio et al., 2020). To examine this hypothesis, a hierarchical regression approach was employed. In the fourth and last step, an interaction between SRS and Familism was added to the model. The results can be shown in Table 6.

Table 6

4th Model: Age, Gender, SRS, Familism, and the interaction of SRS and Familism on SPAI

Model 4		Estimate(β)	Sd error	t-value	p-value
	Intercept	35.8613673	4.4547562	8.050	7.82e-14***
	Age	-0.1085438	0.1540751	-0.704	0.4820
	Gender	1.7502724	0.9900347	1.768	0.0786
	SRS	0.00066379	0.1014448	0.006	0.9950
	Familism	-0.1223542	0.0844699	-1.448	0.1491
	SRS:Familism	0.0045921	0.0017872	2.569	0.0109*

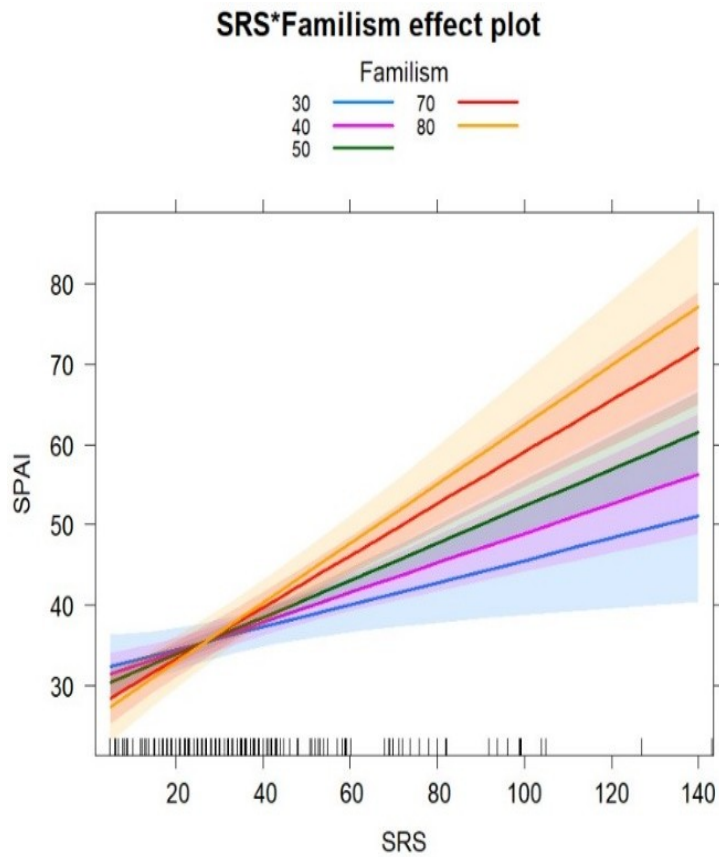
*Note: The table shows the estimate, standard error (Sd error), t-value, p-value obtained for Model 1. Asterisks symbol is used demonstrate significance in the results: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$*

The overall model was statistically significant ($F(5, 196) = 30.54, p < 2.2e-16$). This model explained 43.79% of the variance in SPAI scores ($R^2 = 0.4379$, adjusted $R^2 = 0.4236$). The interaction between SRS and familism was a significant predictor of SPAI scores ($\beta = .0045, t(196) = 2.569, p = .0109$). This result shows that the effect of SRS on SPAI scores is moderated by levels of familism. In other words, familism was found to be influencing the relationship between autistic traits and social anxiety positively. In this model, SRS itself was not significant ($\beta = -.0006, t(196) = .006, p = .9950$). Familism alone ($\beta = -.1223, t(196) = -1.448, p = .1491$) was not found to be significant either. Age ($\beta = -.1085, t(196) = -.704, p = .4820$) and gender ($\beta = 1.7502, t(196) = 1.768, p = .0786$) also did not significantly predict SPAI scores in this model.

To better understand the influence of familism regarding the connection between autistic traits and social anxiety, an interaction plot that can be observed in Figure 7 was generated.

Figure 7

The interaction of SRS and Familism on SPAI



Note: The graph shows how SPAI (social anxiety) scores are affected by SRS (autistic traits) over different levels of Familism)

Chapter 5

Discussion

ASD is a neurodevelopmental disorder with a multifactorial etiology affecting society, with its increasing prevalence rates. Recent studies suggest that autistic traits are sub-threshold difficulties in socialization, communication, and restricted interests that do not fulfil the formal criteria for ASD (Constantino and Todd, 2003). Autistic traits are highly important and beneficial to study since, rather than a disorder, ASD nowadays seems to be a neurodivergence that a noticeable proportion of the world population may possess. It has been observed that there is a comorbidity between autistic traits and social anxiety, a widespread psychological disorder marked by strong fear and avoidance of social interactions (Morrison & Heimberg, 2013). Those affected by social anxiety commonly feel extreme distress and anxiety in social settings, causing major disruptions in daily functioning in various aspects of their lives (Morrison & Heimberg, 2013). Social anxiety has become a topic of interest in the current study because social communication and interaction difficulties are fundamental aspects of autism. Additionally, contextual factors such as familism, which is defined as a cultural value that highlights interdependent family relationships that are warm, close, and supportive (Campos et al., 2014), can influence the relationship between these variables. Therefore, considering the above variables, the current study was designed to examine the link between autistic traits and social anxiety in children and adolescents aged 6–18 years old in Italy, also considering the moderating role of familism. The research involved 202 Italian parents who completed an online survey assessing their children's autistic traits using the Social Responsiveness Scale (SRS-2), social anxiety using the Social Phobia and Anxiety Inventory for Children (SPAI-C), and familism using the Mexican American Cultural Values Scale (MACVS). The research adopted a correlational, non-experimental design, and data were collected via social

media platforms like Facebook. Statistical analyses, including descriptive, correlation, and regression analyses were conducted using R Studio.

Participants in the present study were balanced in terms of gender, with a nearly equal number of male and female children and adolescents, and the mean ages for both genders are highly similar, minimizing potential age-related biases. The uniformity in parental ages across genders also reduces the likelihood of differences caused by parental ages as a confounding variable. Additionally, the education levels of both fathers and mothers show a significant proportion holding at least a high school diploma, indicating a relatively educated sample population. These characteristics provide a good foundation when interpreting the study's findings, ensuring that demographic factors are unlikely to skew the results significantly.

Pearson's r correlations were conducted to examine whether there were significant relationships between the variables. A significant but weak positive correlation was observed between the autistic traits and familism, with higher autistic traits being associated with higher familism scores. Similarly, a significant but weak positive correlation was also found between the social anxiety and familism, suggesting that higher levels of social anxiety are associated with higher levels of familism. Finally, a significant positive correlation was also observed between autistic traits and social anxiety.

5.1 First Hypothesis

It was hypothesized that increased autistic traits would be positively associated with heightened levels of social anxiety (Bellini, 2004; Gillott, Furniss, & Walter, 2001; Liew et al., 2014; Freeth et al., 2013). The results confirmed this hypothesis (See Table 2). The association between autistic traits and social anxiety was found to be positively strong. This outcome implies

that having autistic traits increases the likelihood of having social anxiety. Age and gender were controlled and showed no influence on this relationship. Consistent with previous research by Bellini (2004) and Gillott et al. (2001), results indicate that those exhibiting elevated autistic traits likewise report experiencing elevated anxiety levels. Moreover, the positive association between autistic traits and symptoms of social anxiety aligns with Liew et al. (2014), who found similar associations between autistic traits and various anxiety-related signs. Additionally, the observed association between early ASD traits and subsequent social anxiety resonates with the findings of Van Steensel et al. (2013), who noted that children with anxiety disorders often exhibited elevated ASD traits both in early development and later in life. This emphasizes the value of early detection and intervention in children exhibiting ASD traits, as it may help mitigate the development of anxiety disorders.

In the first step of the hierarchical regression, age and gender were added as control variables of social anxiety. Here, age was found to be a significant predictor ($p = 0.0226$), while gender was not ($p = 0.4084$). The model explained a small portion of the variance, with an R-squared value of 0.02933. In the second step, autistic traits (SRS) were added to the model, which significantly improved it. Autistic traits emerged as a highly significant predictor ($p < 2e-16$), while age and gender become non-significant ($p = 0.5291$ for age, $p = 0.0828$ for gender). The R-squared value increased to 0.4154, indicating that a substantial portion of the variance in social anxiety is explained by the inclusion of autistic traits. In the third step, familism was added to the model, but it did not reach significance ($p = 0.2693$), and it did not contribute much to the explained variance.

5.2 Second Hypothesis

It was hypothesized that higher levels of familism would buffer the negative impact of autistic traits on social anxiety symptoms, leading to lower levels of social anxiety among

individuals with elevated autistic traits (Corona et al., 2016; Campos et al., 2014; Zamudio et al., 2020). In other words, familism was expected to weaken the connection between autistic traits and social anxiety. To test this hypothesis, hierarchical regression was implemented to observe the effect of each variable step by step. The analysis resulted in contradictory findings. The interaction between autistic traits and familism was found to be a significant predictor of social anxiety scores. Contrary to expectations, familism was found to increase the effect of autistic traits on social anxiety in the current study.

These findings suggest that, rather than acting as a protective factor, familism may exacerbate social anxiety in individuals with elevated autistic traits. Even the association between familism and other two variables alone indicates that as familism increases both autistic traits and social anxiety tend to increase, although it is a weak statistical significance. In the same line, previous literature showed that familism is related to an increased degree of distress (Schwartz et al., 2010) and symptoms of inflammation (Fuligni et al., 2009). These responses were believed to stem from the tension of complying with family obligations and reactions to family conflicts (Fuligni et al., 2009). The underlying reasons for the contradictory results in the current study may be similar. For some people, rather than acting as a protective factor, being dependent on family and perceiving family obligations as a burden may cause to increased stress. From a child or adolescent perspective, trying to satisfy the expectations of parents might cause stress. These expectations may not always be realistic, which leave the child or adolescent under stress. Consequently, this may partly explain the positive effect of familism on the relationship between autistic traits and social anxiety.

Another important finding to discuss is that the level of the relationship between autistic traits and social anxiety was stronger at higher levels of familism compared to lower levels of

familism. This indicates that the impact of autistic traits on social anxiety is more evident when familism is higher. In other words, as familism is more, the association between autistic traits and social anxiety is stronger than the condition where familism is less. Furthermore, it was also observed that the influence of familism on the relationship between autistic traits and social anxiety becomes evident after a certain score in the SRS. The location of this score can be seen from the crossing point of lines in the bottom left of the graph (see figure 7).

5.3 Limitations

The limitation of the study includes its design, measures, and scope. Firstly, the study was conducted with a limited number of participants ($n=202$). Although there was a fair distribution in the age level, participants only consisted of Italians, which puts a barrier to the generalizability of the research across different cultures or countries. Secondly, most families live in urban areas, where children are exposed to diverse social experiences due to faster-paced lifestyles and cultural variety. Additionally, most children have siblings, who often serve as companions and role models, influencing their social development. Therefore, these two, the urban environment and sibling relationships may affect the social dynamics under study. Additionally, the surveys were completed by the parents, leaving room for biased observations across all three variables. In other words, whether the parents paid enough attention, understood the instructions and questions, or had biases in evaluating their children's behavior is in question.

5.4 Future Research

The first hypothesis regarding the positive association between autistic traits and social anxiety confirmed the previous findings. Future research could delve more into this relationship by trying to identify the underlying reasons of this association. The question of which autistic traits

affect which dimensions of social anxiety should be the principal area of interest, which could help reaching more particular associations, leading to valuable findings that could be used to develop individualized treatment plans for individuals with these conditions. The second hypothesis postulating that familism could be a protective factor and mitigate the negative effect of autistic traits on social anxiety was refuted by the current findings of the study. Conversely, familism amplified the relationship, especially more, over the high levels of familism. Future research should examine the underlying reasons for these complex phenomena by looking at which subcategories of familism could be more impactful on their influence and which subcategories of autistic traits and social anxiety specifically are more affected by the different subcategories and levels of familism. Moreover, further research can be conducted by incorporating different cultural groups, different countries, and different age groups to discover the differences between the associations. In the current study, majority of families live in urban settings, and most children have siblings. The homogeneity of these conditions may be paid attention in future research for better generalizability of their findings since they may influence the social dynamics of the families. Additionally, more research can be directed to the different dimensions of family cohesion, family functioning, or family dynamics. Lastly, more standardized tools that are valid and reliable across different cultures and countries for assessing family dynamics or functioning should be prioritized.

References

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.).

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.).

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). <https://doi.org/10.1176/appi.books.9780890425596>

Ann Katrin Sauer, M. S. C., Janelle Stanton, M. S. C., Sakshi Hans, M. S. C., & Andreas Grabrucker, P. H. D. (2021). Autism Spectrum Disorders: Etiology and Pathology. *Exon Publications*,
15. <https://doi.org/10.36255/exonpublications.autismspectrumdisorders.2021.etiology>

Argyle, M. (1986). Rules for social relationships in four cultures. *Australian Journal of Psychology*, 38(3), 309–318. <https://doi.org/10.1080/00049538608259017>

Ashwood, P., Wills, S., & Van de Water, J. (2006). The immune response in autism: A new frontier for autism research. *Journal of Leukocyte Biology*, 80(1), 1–15. <https://doi.org/10.1189/jlb.1205707>

Asperger, H. (1944). Die „Autistischen Psychopathen“ im Kindesalter. *Archiv für Psychiatrie und Nervenkrankheiten*, 117(1), 76–136. <https://doi.org/10.1007/BF01837709>

Baiano, C., Raimo, G., Zappullo, I., Cecere, R., Rauso, B., Positano, M., Conson, M., Aversana, L. D., Di Rosa, A., Esposito, G., Milo, R., Polito, F., Raimondo, C., Turco, A., & The

LabNPEE Group. (2022). Anxiety Sensitivity Domains are Differently Affected by Social and Non-social Autistic Traits. *Journal of Autism and Developmental Disorders*, 52(8), 3486–3495. <https://doi.org/10.1007/s10803-021-05228-w>

Baron-Cohen, S. (2008). Autism. *The British Journal of Psychiatry*, 193(4), 321–321. <https://doi.org/10.1192/bjp.193.4.321>

Baron-Cohen, S., Wheelwright, S., Cox, A., Baird, G., Charman, T., Swettenham, J., Drew, A., & Doehring, P. (2000). Early identification of autism by the CHecklist for Autism in Toddlers (CHAT). *Journal of the Royal Society of Medicine*, 93(10), 521–525. <https://doi.org/10.1177/014107680009301007>

Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001). The autism-spectrum quotient (AQ): Evidence from Asperger syndrome/high-functioning autism, males and females, scientists and mathematicians. *Journal of Autism and Developmental Disorders*, 31(1), 5–17. <https://doi.org/10.1023/a:1005653411471>

Basit, M., Sultan, A. N., Ahmed, S., Iftikhar, N., Ahmad, W., & Hafeez, M. (2022). Presentation of Restrictive and Repetitive Behaviors in Autism Spectrum Disorder. *Pakistan Journal of Medical & Health Sciences*, 16(04), Article 04. <https://doi.org/10.53350/pjmhs22164153>

Beck, A. T., Steer, R. A., & Brown, G. (1996). *Beck Depression Inventory–II (BDI-II)* [Database record]. APA PsycTests. <https://doi.org/10.1037/t00742-000>

Beidel, D. C. (1996). Assessment of childhood social phobia: Construct, convergent, and discriminative validity of the Social Phobia and Anxiety inventory for Children (SPAC). *Psychological Assessment*, 8(3), 235–240. <https://doi.org/10.1037/1040-3590.8.3.235>

Beidel, D. C., Turner, S. M., & Morris, T. L. (1995). A new inventory to assess childhood social anxiety and phobia: The Social Phobia and Anxiety Inventory for Children. *Psychological Assessment*, 7(1), 73–79. <https://doi.org/10.1037/1040-3590.7.1.73>

Bellini, S. (2004). Social Skill Deficits and Anxiety in High-Functioning Adolescents With Autism Spectrum Disorders. *Focus on Autism and Other Developmental Disabilities*, 19(2), 78–86. <https://doi.org/10.1177/10883576040190020201>

Bishop, D. V. M. (2002). The role of genes in the etiology of specific language impairment. *Journal of Communication Disorders*, 35(4), 311–328. [https://doi.org/10.1016/S0021-9924\(02\)00087-4](https://doi.org/10.1016/S0021-9924(02)00087-4)

Bögels, S. M., Alden, L., Beidel, D. C., Clark, L. A., Pine, D. S., Stein, M. B., & Voncken, M. (2010). Social anxiety disorder: Questions and answers for the DSM-V. *Depression and Anxiety*, 27(2), 168–189. <https://doi.org/10.1002/da.20670>

Bölte, S., Poustka, F., & Constantino, J. N. (2008). Assessing autistic traits: Cross-cultural validation of the social responsiveness scale (SRS). *Autism Research*, 1(6), 354–363. <https://doi.org/10.1002/aur.49>

Bolton, P. F., Golding, J., Emond, A., & Steer, C. D. (2012). Autism Spectrum Disorder and Autistic Traits in the Avon Longitudinal Study of Parents and Children: Precursors and Early Signs. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(3), 249–260.e25. <https://doi.org/10.1016/j.jaac.2011.12.009>

Brentani, H., Paula, C. S. de, Bordini, D., Rolim, D., Sato, F., Portolese, J., Pacifico, M. C., & McCracken, J. T. (2013). Autism spectrum disorders: An overview on diagnosis and

treatment. *Brazilian Journal of Psychiatry*, 35, S62–S72. <https://doi.org/10.1590/1516-4446-2013-S104>

Brugha, T. S., Spiers, N., Bankart, J., Cooper, S.-A., McManus, S., Scott, F. J., Smith, J., & Tyrer, F. (2016). Epidemiology of autism in adults across age groups and ability levels. *The British Journal of Psychiatry*, 209(6), 498–503. <https://doi.org/10.1192/bjp.bp.115.174649>

Campos, B., Perez, O. F. R., & Guardino, C. (2016). Familism: A cultural value with implications for romantic relationship quality in U.S. Latinos. *Journal of Social and Personal Relationships*, 33(1), 81–100. <https://doi.org/10.1177/0265407514562564>

Campos, B., Schetter, C. D., Abdou, C. M., Hobel, C. J., Glynn, L. M., & Sandman, C. A. (2008). Familialism, social support, and stress: positive implications for pregnant Latinas. *Cultural diversity & ethnic minority psychology*, 14(2), 155–162. <https://doi.org/10.1037/1099-9809.14.2.155>

Campos, B., Ullman, J. B., Aguilera, A., & Dunkel Schetter, C. (2014). Familism and psychological health: The intervening role of closeness and social support. *Cultural Diversity and Ethnic Minority Psychology*, 20(2), 191–201. <https://doi.org/10.1037/a0034094>

Cannell, J. J. (2008). Autism and vitamin D. *Medical Hypotheses*, 70(4), 750–759. <https://doi.org/10.1016/j.mehy.2007.08.016>

Cervantes, P. E., Matson, J. L., & Goldin, R. L. (2016). Diagnosing ASD in Very Early Childhood. In J. L. Matson (Ed.), *Handbook of Assessment and Diagnosis of Autism Spectrum Disorder* (pp. 157–173). Springer International Publishing. https://doi.org/10.1007/978-3-319-27171-2_9

Constantino, J. N., & Gruber, C. P. (2012). *Social responsiveness scale, Second Edition (SRS-2)*. Torrance, CA: Western Psychological Services.

Constantino, J. N., & Todd, R. D. (2003). Autistic Traits in the General Population: A Twin Study. *Archives of General Psychiatry*, 60(5), 524–530. <https://doi.org/10.1001/archpsyc.60.5.524>

Constantino, J. N., Abbacchi, A. M., LaVesser, P. D., Reed, H., Givens, L., Chiang, L., Gray, T., Gross, M., Zhang, Y., & Todd, R. D. (2009). Developmental Course of Autistic Social Impairment In Males. *Development and Psychopathology*, 21(1), 127–138. <https://doi.org/10.1017/S095457940900008X>

Constantino, J. N., Davis, S. A., Todd, R. D., Schindler, M. K., Gross, M. M., Brophy, S. L., Metzger, L. M., Shoushtari, C. S., Splinter, R., & Reich, W. (2003). Validation of a Brief Quantitative Measure of Autistic Traits: Comparison of the Social Responsiveness Scale with the Autism Diagnostic Interview-Revised. *Journal of Autism and Developmental Disorders*, 33(4), 427–433. <https://doi.org/10.1023/A:1025014929212>

Constantino, J. N., Zhang, Y., Frazier, T., Abbacchi, A. M., & Law, P. (2010). Sibling Recurrence and the Genetic Epidemiology of Autism. *American Journal of Psychiatry*, 167(11), 1349–1356. <https://doi.org/10.1176/appi.ajp.2010.09101470>

Corona, K., Campos, B., & Chen, C. (2017). Familism Is Associated With Psychological Well-Being and Physical Health: Main Effects and Stress-Buffering Effects. *Hispanic Journal of Behavioral Sciences*, 39(1), 46–65. <https://doi.org/10.1177/0739986316671297>

Crafa, D., & Warfa, N. (2015). Maternal migration and autism risk: Systematic analysis. *International Review of Psychiatry*. <https://www.tandfonline.com/doi/abs/10.3109/09540261.2014.995601>

Cuéllar, I., Arnold, B. and González, G. (1995), Cognitive referents of acculturation: Assessment of cultural constructs in Mexican Americans. *J. Community Psychol.*, 23: 339-356. [https://doi.org/10.1002/1520-6629\(199510\)23:4<339::AID-JCOP2290230406>3.0.CO;2-7](https://doi.org/10.1002/1520-6629(199510)23:4<339::AID-JCOP2290230406>3.0.CO;2-7)

Cui, X., McGrath, J. J., Burne, T. H. J., Mackay-Sim, A., & Eyles, D. W. (2007). Maternal vitamin D depletion alters neurogenesis in the developing rat brain. *International Journal of Developmental Neuroscience*, 25(4), 227–232. <https://doi.org/10.1016/j.ijdevneu.2007.03.006>

Currenti, S. A. (2009). Understanding and determining the etiology of autism. *Cellular and Molecular Neurobiology*, 30(2), 161–171. <https://doi.org/10.1007/s10571-009-9453-8>

D’Arcy, C., & Meng, X. (2014). Prevention of common mental disorders: Conceptual framework and effective interventions. *Current Opinion in Psychiatry*, 27(4), 294. <https://doi.org/10.1097/YCO.0000000000000076>

Dawson, G. (2008). Early behavioral intervention, brain plasticity, and the prevention of autism spectrum disorder. *Development and Psychopathology*, 20(3), 775–803. <https://doi.org/10.1017/S0954579408000370>

Dawson, G., & Bernier, R. (2013). A quarter century of progress on the early detection and treatment of autism spectrum disorder. *Development and Psychopathology*, 25(4pt2), 1455–1472. <https://doi.org/10.1017/S0954579413000710>

Durkin, M. S., Maenner, M. J., Baio, J., Christensen, D., Daniels, J., Fitzgerald, R., Imm, P., Lee, L.-C., Schieve, L. A., Van Naarden Braun, K., Wingate, M. S., & Yeargin-Allsopp, M. (2017). Autism Spectrum Disorder Among US Children (2002–2010): Socioeconomic, Racial, and Ethnic Disparities. *American Journal of Public Health*, 107(11), 1818–1826. <https://doi.org/10.2105/AJPH.2017.304032>

Factor, R. S., Ollendick, T. H., Cooper, L. D., Dunsmore, J. C., Rea, H. M., & Scarpa, A. (2019). All in the Family: A Systematic Review of the Effect of Caregiver-Administered Autism Spectrum Disorder Interventions on Family Functioning and Relationships. *Clinical Child and Family Psychology Review*, 22(4), 433–457. <https://doi.org/10.1007/s10567-019-00297-x>

Fedoroff, I. C., & Taylor, S. (2001). Psychological and Pharmacological Treatments of Social Phobia: A Meta-Analysis. *Journal of Clinical Psychopharmacology*, 21(3), 311.

Fombonne, E. (2003). Epidemiological surveys of autism and other pervasive developmental disorders: An update. *Journal of Autism and Developmental Disorders*, 33(4), 365–382. <https://doi.org/10.1023/a:1025054610557>

Francis, K., Karantanos, G., Al-Ozairi, A., & AlKhadhari, S. (2021). Prevention in Autism Spectrum Disorder: A Lifelong Focused Approach. *Brain Sciences*, 11(2), Article 2. <https://doi.org/10.3390/brainsci11020151>

Freeth, M., Bullock, T., & Milne, E. (2013). The distribution of and relationship between autistic traits and social anxiety in a UK student population. *Autism*, 17(5), 571–581. <https://doi.org/10.1177/1362361312445511>

Frith, U. (Ed.). (1991). *Autism and Asperger Syndrome*. Cambridge University Press. <https://doi.org/10.1017/CBO9780511526770>

Fuligni, A. J., Telzer, E. H., Bower, J., Irwin, M. R., Kiang, L., & Cole, S. W. (2009). Daily family assistance and inflammation among adolescents from Latin American and European backgrounds. *Brain, Behavior, and Immunity*, 23(6), 803–809. <https://doi.org/10.1016/j.bbi.2009.02.021>

Fuligni, A. J., Tseng, V., & Lam, M. (1999). Attitudes toward Family Obligations among American Adolescents with Asian, Latin American, and European Backgrounds. *Child Development*, 70(4), 1030–1044. <https://doi.org/10.1111/1467-8624.00075>

Gau, S. S.-F., Chou, M.-C., Chiang, H.-L., Lee, J.-C., Wong, C.-C., Chou, W.-J., & Wu, Y.-Y. (2012). Parental adjustment, marital relationship, and family function in families of children with autism. *Research in Autism Spectrum Disorders*, 6(1), 263–270. <https://doi.org/10.1016/j.rasd.2011.05.007>

Gaugler, T., Klei, L., Sanders, S. J., Bodea, C. A., Goldberg, A. P., Lee, A. B., Mahajan, M., Manaa, D., Pawitan, Y., Reichert, J., Ripke, S., Sandin, S., Sklar, P., Svantesson, O., Reichenberg, A., Hultman, C. M., Devlin, B., Roeder, K., & Buxbaum, J. D. (2014). Most genetic risk for autism resides with common variation. *Nature Genetics*, 46(8), 881–885. <https://doi.org/10.1038/ng.3039>

Georgiades, S., Szatmari, P., Zwaigenbaum, L., Bryson, S., Brian, J., Roberts, W., Smith, I., Vaillancourt, T., Roncadin, C., & Garon, N. (2013). A Prospective Study of Autistic-Like Traits in Unaffected Siblings of Probands With Autism Spectrum Disorder. *JAMA Psychiatry*, 70(1), 42. <https://doi.org/10.1001/2013.jamapsychiatry.1>

Gernsbacher, M. A., Stevenson, J. L., & Dern, S. (2017). Specificity, contexts, and reference groups matter when assessing autistic traits. *PLOS ONE*, 12(2), e0171931. <https://doi.org/10.1371/journal.pone.0171931>

Geschwind, D. H., & State, M. W. (2015). Gene hunting in autism spectrum disorder: On the path to precision medicine. *The Lancet. Neurology*, 14(11), 1109–1120. [https://doi.org/10.1016/S1474-4422\(15\)00044-7](https://doi.org/10.1016/S1474-4422(15)00044-7)

Geurts, H. M., Corbett, B., & Solomon, M. (2009). The paradox of cognitive flexibility in autism. *Trends in Cognitive Sciences*, 13(2), 74–82. <https://doi.org/10.1016/j.tics.2008.11.006>

Giacomo, A. D., Craig, F., Palermo, G., Coppola, A., Margari, M., Campanozzi, S., Margari, L., & Turi, M. (2021). <p>Differential Diagnosis in Children with Autistic Symptoms and Subthreshold ADOS Total Score: An Observational Study</p>. *Neuropsychiatric Disease and Treatment*, 17, 2163–2172. <https://doi.org/10.2147/NDT.S300452>

Gillberg, C. (1998). Chromosomal Disorders and Autism. *Journal of Autism and Developmental Disorders*, 28(5), 415–425. <https://doi.org/10.1023/A:1026004505764>

Gilliam, J. E. (2006). *Gilliam autism rating scale: GARS 2*. Pro-ed.

Gillott, A., Furniss, F., & Walter, A. (2001). Anxiety in high-functioning children with autism. *Autism*, 5(3), 277–286. <https://doi.org/10.1177/1362361301005003005>

Gould, R. A., Buckminster, S., Pollack, M. H., Otto, M. W., & Yap, L. (1997). Cognitive-behavioral and pharmacological treatment for social phobia: A meta-analysis. *Clinical Psychology: Science and Practice*, 4(4), 291–306. <https://doi.org/10.1111/j.1468-2850.1997.tb00123.x>

Grapel, J. N., Cicchetti, D. V., & Volkmar, F. R. (2015). Sensory Features as Diagnostic Criteria for Autism: Sensory Features in Autism. *The Yale Journal of Biology and Medicine*, 88(1), 69–71.

Gunby, K. V., Carr, J. E., & LeBlanc, L. A. (2010). TEACHING ABDUCTION-PREVENTION SKILLS TO CHILDREN WITH AUTISM. *Journal of Applied Behavior Analysis*, 43(1), 107–112. <https://doi.org/10.1901/jaba.2010.43-107>

Hazen, E. P., Stornelli, J. L., O'Rourke, J. A., Koesterer, K., & McDougale, C. J. (2014). Sensory symptoms in autism spectrum disorders. *Harvard Review of Psychiatry*, 22(2), 112–124. <https://doi.org/10.1097/01.HRP.0000445143.08773.58>

Heimberg, R. G., Hofmann, S. G., Liebowitz, M. R., Schneier, F. R., Smits, J. A. J., Stein, M. B., Hinton, D. E., & Craske, M. G. (2014). Social Anxiety Disorder in Dsm-5. *Depression and Anxiety*, 31(6), 472–479. <https://doi.org/10.1002/da.22231>

Hernández, M. M., & Bámaca-Colbert, M. Y. (2016). A Behavioral Process Model of Familism. *Journal of Family Theory & Review*, 8(4), 463–483. <https://doi.org/10.1111/jftr.12166>

Hertz-Picciotto, I., Schmidt, R. J., & Krakowiak, P. (2018). Understanding environmental contributions to autism: Causal concepts and the state of science. *Autism Research*, 11(4), 554–586. <https://doi.org/10.1002/aur.1938>

Hofmann, S. G., Anu Asnaani, M. A., & Hinton, D. E. (2010). Cultural aspects in social anxiety and social anxiety disorder. *Depression and Anxiety*, 27(12), 1117–1127. <https://doi.org/10.1002/da.20759>

Johnson, C. P., Myers, S. M., & and the Council on Children With Disabilities. (2007). Identification and Evaluation of Children With Autism Spectrum Disorders. *Pediatrics*, 120(5), 1183–1215. <https://doi.org/10.1542/peds.2007-2361>

Kangas, S., Uusiautti, S., & Maatta, K. (2011). Social interaction among children with Autism Spectrum Disorders (ASD). *International Journal of Early Childhood Special Education*, 3, 160–174.

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.

Karlsson, B., Sigström, R., Östling, S., Waern, M., Börjesson-Hanson, A., & Skoog, I. (2016). DSM-IV and DSM-5 Prevalence of Social Anxiety Disorder in a Population Sample of Older People. *The American Journal of Geriatric Psychiatry: Official Journal of the American Association for Geriatric Psychiatry*, 24(12), 1237–1245. <https://doi.org/10.1016/j.jagp.2016.07.023>

Keefe, S., Padilla, A., & Carlos, M. (1979). The Mexican-American Extended Family As An Emotional Support System. *Human Organization*, 38(2), 144–152. <https://doi.org/10.17730/humo.38.2.575482483n134553>

Kerns, C. E., Comer, J. S., Pincus, D. B., & Hofmann, S. G. (2013). Evaluation of the Proposed Social Anxiety Disorder Specifier Change for Dsm-5 in a Treatment-Seeking Sample of Anxious Youth. *Depression and Anxiety*, 30(8), 709–715. <https://doi.org/10.1002/da.22067>

Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., Wittchen, H.-U., & Kendler, K. S. (1994). Lifetime and 12-Month Prevalence of DSM-III-R Psychiatric Disorders in the United States: Results From the National Comorbidity Survey. *Archives of General Psychiatry*, 51(1), 8–19. <https://doi.org/10.1001/archpsyc.1994.03950010008002>

Klintwall, L., & Eikeseth, S. (2014). Early and Intensive Behavioral Intervention (EIBI) in Autism. In V. B. Patel, V. R. Preedy, & C. R. Martin (Eds.), *Comprehensive Guide to Autism* (pp. 117–137). Springer. https://doi.org/10.1007/978-1-4614-4788-7_129

Kobak, K. A., & Reynolds, W. M. (1999). Hamilton Depression Inventory. In *The use of psychological testing for treatment planning and outcomes assessment, 2nd ed* (pp. 935–969). Lawrence Erlbaum Associates Publishers.

La Greca, A. M., & Lopez, N. (1998). Social anxiety among adolescents: Linkages with peer relations and friendships. *Journal of Abnormal Child Psychology*, 26(2), 83–94. <https://doi.org/10.1023/a:1022684520514>

Lee, S. H., Ripke, S., Neale, B. M., Faraone, S. V., Purcell, S. M., Perlis, R. H., Mowry, B. J., Thapar, A., Goddard, M. E., Witte, J. S., Absher, D., Agartz, I., Akil, H., Amin, F., Andreassen, O. A., Anjorin, A., Anney, R., Anttila, V., Arking, D. E., ... Cross-Disorder Group of the Psychiatric Genomics Consortium. (2013). Genetic relationship between five psychiatric disorders estimated from genome-wide SNPs. *Nature Genetics*, 45(9), 984–994. <https://doi.org/10.1038/ng.2711>

Liew, S. M., Thevaraja, N., Hong, R. Y., & Magiati, I. (2015). The Relationship Between Autistic Traits and Social Anxiety, Worry, Obsessive–Compulsive, and Depressive Symptoms: Specific and Non-specific Mediators in a Student Sample. *Journal of Autism and Developmental Disorders*, 45(3), 858–872. <https://doi.org/10.1007/s10803-014-2238-z>

Loomes, R., Hull, L., & Mandy, W. P. L. (2017). What Is the Male-to-Female Ratio in Autism Spectrum Disorder? A Systematic Review and Meta-Analysis. *Journal of the American Academy of Child and Adolescent Psychiatry*, 56(6), 466–474. <https://doi.org/10.1016/j.jaac.2017.03.013>

Lord, C., Brugha, T. S., Charman, T., Cusack, J., Dumas, G., Frazier, T., Jones, E. J. H., Jones, R. M., Pickles, A., State, M. W., Taylor, J. L., & Veenstra-VanderWeele, J. (2020). Autism spectrum disorder. *Nature Reviews Disease Primers*, 6(1), 1–23. <https://doi.org/10.1038/s41572-019-0138-4>

Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Leventhal, B. L., DiLavore, P. C., Pickles, A., & Rutter, M. (2000). *The Autism Diagnostic Observation Schedule—Generic: A Standard Measure of Social and Communication Deficits Associated with the Spectrum of Autism*.

Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism Diagnostic Interview-Revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24(5), 659–685. <https://doi.org/10.1007/BF02172145>

Maenner, M. J. (2020). Prevalence of Autism Spectrum Disorder Among Children Aged 8 Years—Autism and Developmental Disabilities Monitoring Network, 11 Sites, United States, 2016. *MMWR. Surveillance Summaries*, 69. <https://doi.org/10.15585/mmwr.ss6904a1>

Maenner, M. J. (2021). Prevalence and Characteristics of Autism Spectrum Disorder Among Children Aged 8 Years—Autism and Developmental Disabilities Monitoring Network, 11 Sites, United States, 2018. *MMWR. Surveillance Summaries*, 70. <https://doi.org/10.15585/mmwr.ss7011a1>

Maenner, M. J. (2023). Prevalence and Characteristics of Autism Spectrum Disorder Among Children Aged 8 Years—Autism and Developmental Disabilities Monitoring Network, 11 Sites, United States, 2020. *MMWR. Surveillance Summaries*, 72. <https://doi.org/10.15585/mmwr.ss7202a1>

Magnusson, C., Rai, D., Goodman, A., Lundberg, M., Idring, S., Svensson, A., Koupil, I., Serlachius, E., & Dalman, C. (2012). Migration and autism spectrum disorder: Population-based study. *The British Journal of Psychiatry*, 201(2), 109–115. <https://doi.org/10.1192/bjp.bp.111.095125>

Magnússon, P., & Saemundsen, E. (2001). Prevalence of Autism in Iceland. *Journal of Autism and Developmental Disorders*, 31(2), 153–163. <https://doi.org/10.1023/A:1010795014548>

Martin, A. M., Marin, D. G., McIntyre, L. L., & Neece, C. (2022). Familism and Parenting Stress in Latinx Caregivers of Young Children with Developmental Delays. *The Family Journal*, 30(3), 411–418. <https://doi.org/10.1177/10664807211052480>

Matson, J. L., Nebel-Schwalm, M., & Matson, M. L. (2007). A review of methodological issues in the differential diagnosis of autism spectrum disorders in children. *Research in Autism Spectrum Disorders*, 1(1), 38–54. <https://doi.org/10.1016/j.rasd.2006.07.004>

Mayorga-Muñoz, C., Riquelme-Segura, L., Delvecchio, E., & Lee-Maturana, S. (2023). Association between Familism and Mental Health in College Adolescents during the COVID-19 Pandemic. *International Journal of Environmental Research and Public Health*, 20(5), 4149. <https://doi.org/10.3390/ijerph20054149>

McManus, S., Bebbington, P., Jenkins, R., & Brugha, T. (2016). *Adult Psychiatric Morbidity Survey: Mental health and wellbeing in England, 2014*. NHS. <https://digital.nhs.uk/data-and-information/publications/statistical/adult-psychiatric-morbidity-survey/adult-psychiatric-morbidity-survey-survey-of-mental-health-and-wellbeing-england-2014>

Memari, A. H., Ziaee, V., Shayestehfar, M., Ghanouni, P., Mansournia, M. A., & Moshayedi, P. (2013). Cognitive flexibility impairments in children with autism spectrum disorders: Links to age, gender and child outcomes. *Research in Developmental Disabilities*, 34(10), 3218–3225. <https://doi.org/10.1016/j.ridd.2013.06.033>

Microsoft Corporation. (2018). *Microsoft Excel* [Computer software]. Retrieved from <https://office.microsoft.com/excel>

Minami, K., & Horikawa, E. (2021). Social anxiety tendency and autism spectrum disorder in Japanese adolescence. *Pediatrics International: Official Journal of the Japan Pediatric Society*, 63(8), 903–909. <https://doi.org/10.1111/ped.14576>

Modabbernia, A., Velthorst, E., & Reichenberg, A. (2017). Environmental risk factors for autism: An evidence-based review of systematic reviews and meta-analyses. *Molecular Autism*, 8(1), 13. <https://doi.org/10.1186/s13229-017-0121-4>

Morrison, A. S., & Heimberg, R. G. (2013). Social Anxiety and Social Anxiety Disorder. *Annual Review of Clinical Psychology*, 9(Volume 9, 2013), 249–274. <https://doi.org/10.1146/annurev-clinpsy-050212-185631>

Moskowitz, A., & Heim, G. (2011). Eugen Bleuler's Dementia Praecox or the Group of Schizophrenias (1911): A Centenary Appreciation and Reconsideration. *Schizophrenia Bulletin*, 37(3), 471–479. <https://doi.org/10.1093/schbul/sbr016>

Olfson, M., Guardino, M., Struening, E., Schneier, F. R., Hellman, F., & Klein, D. F. (2000). Barriers to the Treatment of Social Anxiety. *American Journal of Psychiatry*, 157(4), 521–527. <https://doi.org/10.1176/appi.ajp.157.4.521>

Pabon E. (1998). Hispanic adolescent delinquency and the family: A discussion of sociocultural influences: Adolescence. *Adolescence*, 33(132), 941–941.

Palmer, C. J., Paton, B., Enticott, P. G., & Hohwy, J. (2015). 'Subtypes' in the Presentation of Autistic Traits in the General Adult Population. *Journal of Autism and Developmental Disorders*, 45(5), 1291–1301. <https://doi.org/10.1007/s10803-014-2289-1>

Pardo, C. A., & Eberhart, C. G. (2007). The Neurobiology of Autism. *Brain Pathology*, 17(4), 434–447. <https://doi.org/10.1111/j.1750-3639.2007.00102.x>

Posit team (2023). RStudio: Integrated Development Environment for R. Posit Software, PBC, Boston, MA. URL <http://www.posit.co/>.

Prelock, P. J., & Nelson, N. W. (2012). Language and communication in autism: An integrated view. *Pediatric Clinics of North America*, 59(1), 129–145, xi. <https://doi.org/10.1016/j.pcl.2011.10.008>

Priest, J. B., & Denton, W. (2012). Anxiety Disorders and Latinos: The Role of Family Cohesion and Family Discord. *Hispanic Journal of Behavioral Sciences*, 34(4), 557–575. <https://doi.org/10.1177/0739986312459258>

Randall, M., Egberts, K. J., Samtani, A., Scholten, R. J., Hooft, L., Livingstone, N., Sterling□Levis^a, K., Woolfenden, S., & Williams, K. (2018). Diagnostic tests for autism spectrum disorder (ASD) in preschool children. *Cochrane Database of Systematic Reviews*, 7. <https://doi.org/10.1002/14651858.CD009044.pub2>

Richler, J., Bishop, S. L., Kleinke, J. R., & Lord, C. (2007). Restricted and Repetitive Behaviors in Young Children with Autism Spectrum Disorders. *Journal of Autism and Developmental Disorders*, 37(1), 73–85. <https://doi.org/10.1007/s10803-006-0332-6>

Robinson, E. B., Munir, K., Munafò, M. R., Hughes, M., McCormick, M. C., & Koenen, K. C. (2011). Stability of Autistic Traits in the General Population: Further Evidence for a Continuum of Impairment. *Journal of the American Academy of Child & Adolescent Psychiatry*, 50(4), 376–384. <https://doi.org/10.1016/j.jaac.2011.01.005>

Rodebaugh, T. L., Holaway, R. M., & Heimberg, R. G. (2004). The treatment of social anxiety disorder. *Clinical Psychology Review*, 24(7), 883–908. <https://doi.org/10.1016/j.cpr.2004.07.007>

Rogers, E. J. (2008). Has enhanced folate status during pregnancy altered natural selection and possibly Autism prevalence? A closer look at a possible link. *Medical Hypotheses*, 71(3), 406–410. <https://doi.org/10.1016/j.mehy.2008.04.013>

Rogers, S. (2016). Early Start Denver Model. In R. G. Romanczyk & J. McEachin (Eds.), *Comprehensive Models of Autism Spectrum Disorder Treatment* (pp. 45–62). Springer International Publishing. https://doi.org/10.1007/978-3-319-40904-7_3

Ronald, A., & Hoekstra, R. A. (2011). Autism spectrum disorders and autistic traits: A decade of new twin studies. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 156(3), 255–274. <https://doi.org/10.1002/ajmg.b.31159>

Rosen, N. E., Lord, C., & Volkmar, F. R. (2021). The Diagnosis of Autism: From Kanner to DSM-III to DSM-5 and Beyond. *Journal of Autism and Developmental Disorders*, 51(12), 4253–4270. <https://doi.org/10.1007/s10803-021-04904-1>

Rossignol, D. A., Genuis, S. J., & Frye, R. E. (2014). Environmental toxicants and autism spectrum disorders: A systematic review. *Translational Psychiatry*, 4(2), e360–e360. <https://doi.org/10.1038/tp.2014.4>

Russell, A., & Brosnan, M. (2018). Habits and Autism: Restricted, Repetitive Patterns of Behaviour and Thinking in Autism. In B. Verplanken (Ed.), *The Psychology of Habit: Theory, Mechanisms, Change, and Contexts* (pp. 343–361). Springer International Publishing. https://doi.org/10.1007/978-3-319-97529-0_19

Sadeghi, S., & Pouretamad, H. R. (2022). Cognitive predictors of behavioral inflexibility in young children with autism spectrum disorder symptoms. *Advances in Cognitive Science*, 23(4), 117–129.

Sasson, N. J., & Bottema-Beutel, K. (2022). Studies of autistic traits in the general population are not studies of autism. *Autism*, 26(4), 1007–1008. <https://doi.org/10.1177/13623613211058515>

Schopler, E., Reichler, R. J., & Renner, B. R. (1986). *The childhood autism rating scale (CARS): For diagnostic screening and classification of autism*. Irvington.

Schwartz, S. J., Weisskirch, R. S., Hurley, E. A., Zamboanga, B. L., Park, I. J. K., Kim, S. Y., Umaña-Taylor, A., Castillo, L. G., Brown, E., & Greene, A. D. (2010). Communalism, familism, and filial piety: Are they birds of a collectivist feather? *Cultural Diversity and Ethnic Minority Psychology*, 16(4), 548–560. <https://doi.org/10.1037/a0021370>

Skuse, D., Warrington, R., Bishop, D., Chowdhury, U., Lau, J., Mandy, W., & Place, M. (2004). The Developmental, Dimensional and Diagnostic Interview (3di): A Novel Computerized Assessment for Autism Spectrum Disorders. *Journal of the American Academy of Child & Adolescent Psychiatry*, 43(5), 548–558. <https://doi.org/10.1097/00004583-200405000-00008>

Stein, G. L., Cupito, A. M., Mendez, J. L., Prandoni, J., Huq, N., & Westerberg, D. (2014). Familism through a developmental lens. *Journal of Latina/o Psychology*, 2(4), 224–250. <https://doi.org/10.1037/lat0000025>

Stewart, M. E., Barnard, L., Pearson, J., Hasan, R., & O'Brien, G. (2006). Presentation of depression in autism and Asperger syndrome: A review. *Autism: The International Journal of Research and Practice*, 10(1), 103–116. <https://doi.org/10.1177/1362361306062013>

Stone, W. L., Coonrod, E. E., & Ousley, O. Y. (2000). Brief Report: Screening Tool for Autism in Two-Year-Olds (STAT): Development and Preliminary Data. *Journal of Autism and Developmental Disorders*, 30(6), 607–612. <https://doi.org/10.1023/A:1005647629002>

Stromswold, K. (2001). The Heritability of Language: A Review and Metaanalysis of Twin, Adoption, and Linkage Studies. *Language*, 77(4), 647–723.

Suh, E., Diener, E., Oishi, S., & Triandis, H. C. (1998). The shifting basis of life satisfaction judgments across cultures: Emotions versus norms. *Journal of Personality and Social Psychology*, 74(2), 482–493. <https://doi.org/10.1037/0022-3514.74.2.482>

Takahashi, T. (1989). Social phobia syndrome in Japan. *Comprehensive Psychiatry*, 30(1), 45–52. [https://doi.org/10.1016/0010-440X\(89\)90117-X](https://doi.org/10.1016/0010-440X(89)90117-X)

Taylor, L. E., Swerdfeger, A. L., & Eslick, G. D. (2014). Vaccines are not associated with autism: An evidence-based meta-analysis of case-control and cohort studies. *Vaccine*, 32(29), 3623–3629. <https://doi.org/10.1016/j.vaccine.2014.04.085>

ten Hoopen, L. W., de Nijs, P. F., Slappendel, G., van der Ende, J., Bastiaansen, D., Greaves-Lord, K., Hakkaart-van Roijen, L., & Hillegers, M. H. (2023). Associations between autism traits and family functioning over time in autistic and non-autistic children. *Autism*, 27(7), 2035–2047. <https://doi.org/10.1177/13623613231151784>

Trammell, B., Wilczynski, S. M., Dale, B., & McIntosh, D. E. (2013). Assessment and Differential Diagnosis of Comorbid Conditions in Adolescents and Adults with Autism Spectrum Disorders. *Psychology in the Schools*, 50(9), 936–946. <https://doi.org/10.1002/pits.21720>

Triandis, H. C., Hui, C. H., Albert, R. D., Leung, S.-M., Lisansky, J., Diaz-Loving, R., Plascencia, L., Marin, G., Betancourt, H., & Loyola-Cintron, L. (1984). Individual models of social behavior. *Journal of Personality and Social Psychology*, 46(6), 1389–1404. <https://doi.org/10.1037/0022-3514.46.6.1389>

Triandis, H. C., Marin, G., Betancourt, H., Lisansky, J., & Chang, B.-H. (1982). *Dimensions of Familism among Hispanic and Mainstream Navy Recruits*: Defense Technical Information Center. <https://doi.org/10.21236/ADA114898>

Turner-Brown, L., & Frisch, M. (2020). Understanding and Addressing Restricted and Repetitive Behaviors in Children with Autism. In G. Vivanti, K. Bottema-Beutel, & L. Turner-Brown (Eds.), *Clinical Guide to Early Interventions for Children with Autism* (pp. 61–77). Springer International Publishing. https://doi.org/10.1007/978-3-030-41160-2_4

Uddin, L. Q. (2021). Brain Mechanisms Supporting Flexible Cognition and Behavior in Adolescents With Autism Spectrum Disorder. *Biological Psychiatry*, 89(2), 172–183. <https://doi.org/10.1016/j.biopsych.2020.05.010>

Valdivieso-Mora, E., Peet, C. L., Garnier-Villarreal, M., Salazar-Villanea, M., & Johnson, D. K. (2016). A Systematic Review of the Relationship between Familism and Mental Health Outcomes in Latino Population. *Frontiers in Psychology*, 7. <https://doi.org/10.3389/fpsyg.2016.01632>

van Steensel, F. J. A., Bögels, S. M., & Wood, J. J. (2013). Autism Spectrum Traits in Children with Anxiety Disorders. *Journal of Autism and Developmental Disorders*, 43(2), 361–370. <https://doi.org/10.1007/s10803-012-1575-z>

Van Wijngaarden-Cremers, P. J. M., van Eeten, E., Groen, W. B., Van Deurzen, P. A., Oosterling, I. J., & Van der Gaag, R. J. (2014). Gender and Age Differences in the Core Triad of Impairments in Autism Spectrum Disorders: A Systematic Review and Meta-analysis. *Journal of Autism and Developmental Disorders*, 44(3), 627–635. <https://doi.org/10.1007/s10803-013-1913-9>

Whitehouse, A. J. O., Hickey, M., & Ronald, A. (2011). Are Autistic Traits in the General Population Stable across Development? *PLOS ONE*, 6(8), e23029. <https://doi.org/10.1371/journal.pone.0023029>

Wing, L., Leekam, S. R., Libby, S. J., Gould, J., & Larcombe, M. (2002). The Diagnostic Interview for Social and Communication Disorders: Background, inter-rater reliability and clinical use. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 43(3), 307–325. <https://doi.org/10.1111/1469-7610.00023>

Wu, S., Wu, F., Ding, Y., Hou, J., Bi, J., & Zhang, Z. (2017). Advanced parental age and autism risk in children: A systematic review and meta-analysis. *Acta Psychiatrica Scandinavica*, 135(1), 29–41. <https://doi.org/10.1111/acps.12666>

Yang, T., Liu, J., Zhang, Y., Zhang, Q., Shangguan, L., Li, Z., Luo, X., & Gong, J. (2021). Coping style predicts sense of security and mediates the relationship between autistic traits and social anxiety: Moderation by a polymorphism of the FKBP5 gene. *Behavioural Brain Research*, 404, 113142. <https://doi.org/10.1016/j.bbr.2021.113142>

Zamudio, G., Wang, C. D., & Jin, L. (2020). Adult Attachment, Social Self-Efficacy, Familismo, and Psychological Wellbeing: A Cross-Cultural Comparison. *The Counseling Psychologist*, 48(7), 922–952. <https://doi.org/10.1177/0011000020930637>

Zwaigenbaum, L., Bauman, M. L., Stone, W. L., Yirmiya, N., Estes, A., Hansen, R. L., McPartland, J. C., Natowicz, M. R., Choueiri, R., Fein, D., Kasari, C., Pierce, K., Buie, T., Carter, A., Davis, P. A., Granpeesheh, D., Mailloux, Z., Newschaffer, C., Robins, D., ... Wetherby, A. (2015). Early Identification of Autism Spectrum Disorder: Recommendations for Practice and Research. *Pediatrics*, 136(Supplement_1), S10–S40. <https://doi.org/10.1542/peds.2014-3667C>

Zwaigenbaum, L., Bryson, S., & Garon, N. (2013). Early identification of autism spectrum disorders. *Behavioural Brain Research*, 251, 133–146. <https://doi.org/10.1016/j.bbr.2013.04.004>