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Theoretical Multi-Level Mapping of the Contribution of Emotional Regulation to Sensory Difficulties in Autism Spectrum Disorders; from Brain to Behaviour

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Abstract

Introduction: Autism spectrum disorder (ASD) is characterised by pervasive difficulties in communication, socialisation and sensory integration often compounded by emotional dysregulation. These overlapping challenges significantly impair daily functioning and quality of life. This study aimed to map the multidimensional relationship between emotional regulation, sensory processing and behavioural outcomes in children with ASD.

Methods: A mixed-methods design was employed combining quantitative assessments (SRS-2, SP-2, BRIEF, EEG, HRV, cortisol, and electrodermal measures) with qualitative parental interviews. Participants included children with ASD and controls. Quantitative analyses used correlation, regression, and group comparison techniques while thematic analysis contextualised parental narratives.

Results: The results indicated that sensory hypersensitivity and emotional regulation difficulties were the strongest predictors of behavioural outcomes, explaining nearly 70% of the variance. Correlation analyses demonstrated robust links between social responsiveness, sensory sensitivity and executive dysfunction ($r > 0.60$). Neurophysiological markers confirmed chronic hyperarousal and stress vulnerability. The respondents consistently reported meltdowns, shutdowns and aggression as predictable consequences of sensory–emotional overload rather than isolated behaviours.

Conclusion: The study concludes that emotional dysregulation and sensory processing challenges are central to ASD behaviours. It recommends integrated, multidisciplinary interventions, parent-inclusive strategies and longitudinal multimodal research to refine interventions and improve holistic care for ASD-affected children.

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1 Chapter 1: Introduction

1.1 Research background

Neurodevelopment in children is based on the systematic coordination between brain systems leading to brain maturation, which simultaneously induces changes in behaviours (Wakschlag, Perlman, Blair, Leibenluft, Briggs-Gowan et al., 2018). The peripheral and central nervous systems undergo slow and gradual maturation over time and the process continues from childhood until adolescence (Sharma, Arain, Mathur, Rais, Nel *et al*., 2013). The growth and developmental patterns in the brain of a child reciprocate with the consistent enhancement in the structure and function of the cerebral cortex, myelin sheaths, and synaptic framework (Khelfaoui, Ibaceta-Gonzalez & Angulo, 2024). The executive functions of children improve with the development of the nervous system and they eventually learn to regulate emotions, socialise with peers, and get acquainted with language skills. However, neurodevelopmental disorders are associated with brain dysfunction that not only impacts sensory processing but also reduces the emotional management skills of the affected children (Rodríguez-Armendariz, Vela-Romero & Galiana, 2024). As a result, the children can develop negative behaviours and attentional problems and experience difficulties in emotional reactivity and expressive/receptive language (Bergman, Vrijsen, Rinck, van Oostrom, Kan *et al*., 2021). A significant reduction in gross and fine motor skills is also considered a neurodevelopmental manifestation (Villar, Fernandes, Purwar, Staines-Urias, Di Nicola *et al*., 2019). The neurodevelopmental complications in children can vary in accordance with the age of the child, sociodemographic variables, and parental risk factors such as maternal/paternal age at delivery, chorionic conditions, or gestational weight gain

(Segre, Clavenna, Roberti, Scarpellini, Cartabia *et al*., 2024). Genetics also play a pivotal role in the causation of neurodevelopmental abnormalities in paediatric populations (Ropers, 2008).

1.2 Autism spectrum disorder – Definition and an overview

The predominant manifestations of neurodevelopmental disease, such as autism spectrum disorder (ASD) include loss of interest, repetitive/restricted behaviours, difficulties in socialisation, and communication challenges (Hodis, Mughal & Saadabadi *et al*., 2025). While the clinical presentation of the symptoms of ASD is highly heterogeneous, they mostly emanate during early childhood. The manifestations of ASD adversely impact the health-related quality of life of the affected children in the long term (Tedla, Asiri, Reddy, Sangadala, Gular, *et al*., 2024). The multifaceted complications degrade the social interactions of the children and they face difficulty in showing their interests and sharing their thoughts in the form of verbal messages. Additionally, ASD impacts the non-verbal communication skills of the patients (Brignell, Chenausky, Song, Zhu, Suo *et al*., 2018). The absence of facial expressions and deficits in body language and eye contact in paediatric patients increasingly challenge their communication patterns. Patients with ASD experience problems in adjusting their behaviours (Hastings, Hastings, Swales & Hughes *et al*., 2021). The pervasive communicable challenges also impact their ability to bond with peers and establish friendships.

The repetitive interests and behavioural patterns in ASD-affected children are reflected in their monotonous activities (Wolff, Botteron, Dager, Elison, Estes, et al., 2014). Research demonstrates the occurrence of ≥2 repetitive activities in children with ASD. For example, patients could engage in idiosyncratic phrases, echolalia, flipping

objects, and patterning of toys, indicating simple motor stereotypies (Giliberti, Frisina, Giustiniano, Carbonaro, Roccella *et al*., 2025). They may also show highly conservative non-verbal or verbal attitudes, insistence, and inflexible compliance with their schedules. These behavioural patterns are reflected in several activities, such as consumption of similar food items per day, travelling with the same path, rigidity in thoughts, transition difficulty, and incapacity to accommodate changes (Sethi, Harrop, Zhang, Pritchett, Whitten *et al*., 2018). Patients with ASD can show highly unusual focus/hyperfocus in abnormal or restricted activities (Dupuis, Mudiyanselage, Burton, Arnold, Crosbie *et al*., 2022). They can have the tendency to show perseverative interests and remain preoccupied with unconventional objects. Children with ASD react abnormally to sensory inputs (Posar & Visconti, 2018). For example, they tend to close their ears with their hands after encountering flashing lights, spinning wheels, car machines, or vacuum cleaners. Sometimes they become hyperreactive, while some other times they exhibit hypo-reactivity. They also have a keen interest in movements or lights. They often explore objects with an urge to touch and smell them excessively and with high fascination (Alrehaili, ElKady, Alrehaili & Alreefi, 2023). They provide unusual responsiveness to textures or sounds and have variable reactions to temperature and pain. The early onset phase of ASD is associated with a high frequency of symptoms that aggravate the increasing social requirements in environments devoid of appropriate learning support (Gonçalves & Monteiro, 2023).

Paediatric patients with ASD often face challenges in accessing healthcare environments due to high sensory overload and sensory sensitivities (Al-Jabri, Alnuwaiser, Abdulghaffar, Almuhanna, Salaam et al., 2023). Their meltdowns or shutdowns are often involuntary and are the outcomes of their hypersensitivities. Children with ASD have an extremely low tolerance for new experiences and they

increase their sensory burden, which is why such children develop intense anxiety in changed circumstances (Williams, Suzman & Woynaroski., 2021). Due to the overwhelming emotional or sensory input, shutdowns could be experienced by ASD-affected children due to verbal communication challenges, anxiety, and sensory overload (Green & Ben-Sasson, 2010). In addition, hyper-empathy could be experienced by high masking trait children (Shalev, Warrier, Greenberg, Smith, Allison *et al*., 2022). Furthermore, the absence of alternative or augmentative communication support and ongoing stereotypes and stigma challenge their verbal communication and increase their sensory difficulties (Yau, Choo,Tan, Monson & Bovel., 2024).

The environmental and genetic attributes variably trigger ASD in predisposed children (Almandil, Alkuroud, AbdulAzeez, Al-Sulaiman, Elaissari et al, 2019). Importantly, brain anomalies, including cortical changes in temporal and frontal lobes and defects in the limbic system are observed in those with ASD. Furthermore, ASD progresses with significant abnormalities in the connectivity and anatomy of the cerebellum (Wang, Cao, Wang, Bai, Liu et al., 2025). Clinical research has revealed highly deteriorated neuronal differentiation and defects in the formation of cortical layers, indicating cortical laminar framework disruption in ASD patients (Liu, Bautista, Liu & Zikopoulos *et al*., 2020). Contemporary studies have also demonstrated elevated extra-axial fluid and cortical size overgrowth in ASD (Shen, Nordahl, Young, Wootton-Gorges, Lee *et al*., 2013). Notably, genetic factors also play a pivotal role in the causation of ASD in paediatric patients. For example, monozygotic twins and those whose siblings are affected with ASD have a high risk of developing the disease (Sandin, Lichtenstein, Kuja-Halkola, Larsson, Hultman *et al*., 2014). Recent studies have revealed the possible role of neuroinflammation, neurotransmission pathways, and transcription factors in ASD development (Gevezova, Sbirkov, Sarafian, Plaimas, Suratanee *et al*.,

2023; Jiang, Lin, Long, Ke, Fukunaga *et al*., 2022). Other significant genetic causes include changes in histone acetylation/DNA methylation, epigenetic mechanisms, and dysregulation in splicing and transcription.

The progression of ASD is impacted by postnatal environmental perinatal and prenatal attributes (Hodges, Fealko & Soares *et al*., 2020). The infants of mothers who were treated with antiepileptic medications and received folic acid prenatal supplements in the past are at high risk of ASD (Bjørk, Riedel, Spigset, Veiby, Kolstad *et al*., 2018). Furthermore, valproic acid and thalidomide prenatal exposure also increase ASD predisposition in some children (Love, Sominsky, O'Hely, Berk, Vuillermin *et al*., 2024). Medical research has revealed the role of pregnancy-related immune activation/maternal infection, maternal psoriasis, thyroid disease, diabetes, and other autoimmune diseases in triggering the ASD onset in children (Jain, Baer, McCulloch, Rogers, Rand *et al*., 2021). The ASD risk also reciprocates with premature birth or prolonged or reduced inter-pregnancy intervals. Other maternal factors associated with ASD occurrences include reduced Apgar scores, preterm delivery, low birth weight, caesarean delivery, and uterine bleeding (Modabbernia, Sandin, Gross, Leonard, Gissler *et al*., 2018).

The ASD occurrence and associated tactile hypersensitivity are significantly impacted by these postnatal, perinatal, and prenatal risk factors (Hisle-Gorman, Susi, Stokes, Gorman, Erdie-Lalena *et al*., 2018). Additionally, the sensory pathways' maturation processes are influenced by the neurophysiological complications of birth, exposure to medication, and early activation of the immune system (Cainelli, Vedovelli & Bisiacchi, 2024). The tactile stimuli responses are eventually affected in ASD-affected

children, which also deteriorates their behaviour and emotional regulation (Thye, Bednarz, Herringshaw, Sartin, & Kana *et al*., 2018).

1.3 The prevalence of ASD

Research studies have shown at least one occurrence of ASD in every 100 children on a global scale (Zeidan, Fombonne, Scorah, Ibrahim, Durkin, et al., 2022). Literature also reveals a 33% incidence rate of intellectual disability in children with ASD. Notably, a 3:1 male-female ratio of ASD is also reported and associated with sociodemographic attributes, indicating a higher prevalence in male children compared to female children (Loomes, Hull & Mandy *et al*., 2017). Additionally, the currently reported rates of ASD in children vary in accordance with the economic status of their families (Li, Dodd-Butera, Beaman & Burtea, 2022). Children belonging to lower socioeconomic groups have a lower risk of ASD compared to those from the higher socioeconomic class (Raina, Chander, Bhardwaj, Kumar, Sharma *et al*., 2019). The current epidemiology of ASD indicates dynamic and complex interactions between socioemotional, help-seeking, service capacity, and community awareness factors. A cross-sectional analysis of adolescents and children in the United States indicated a 2.3-2.5% prevalence of ASD in children of eight years of age (Baio, Wiggins, Christensen, Maenner, Daniels, et al., 2018). However, the ASD prevalence in Australia, the Middle East, and Europe was reported to be 1.41-2.52%, 0.11-1.53%, and 0.42-3.13%, respectively (Bougeard, Picarel-Blanchot, Schmid, Campbell & Buitelaar, 2021). A meta-analysis of 74 studies revealed 1.7%, 1%, 0.5%, 1%, and 0.4% incidence rates of ASD in Australia, Africa, Europe, America, and Asia, respectively (Salari, Rasoulpoor, Rasoulpoor, Shohaimi, Jafarpour *et al*., 2022). A two-phase cross-sectional analysis revealed two two-times elevated occurrences of ASD among children residing in rural locations in comparison to those in urban areas (Raina *et al*.,

2019). Studies also affirm a high health burden on a global scale, under the impact of ASD, in the paediatric population (Salari *et al*., 2022). A population-based study revealed a high risk of intellectual disability in Black children with ASD versus White children with the same diagnosis (Maenner, Warren, Williams, Amoakohene, Bakian *et al*., 2023). Notably, ASD prevalence was found to be greater in Black children in comparison to White children. Another recent meta-analysis revealed that Western nations have a high prevalence of ASD; however, six ASD diagnoses were confirmed in every 1000 children in the South Asian regions (Shrestha, Basukala, Thapa, Shrestha, Basnet *et al*., 2024). The increased occurrences of ASD across the globe not only increase the healthcare burden but also require additional healthcare resources for disease prevention and treatment.

The diagnostic parameter for ASD includes sensory challenges leading to tactile hypersensitivity. The nervous system of the patients with ASD generates atypical responses to tactile, proprioceptive, vestibular, gustatory, olfactory, and visual stimuli. The sensory complications in ASD can result in hypo-responsiveness or hyperresponsiveness under a range of situations or circumstances. Children with ASD often experience great difficulty in undertaking their activities of daily living and emotional regulation. They also cannot adjust or acclimatise with their environment and experience social and behavioural issues in relation to their tactile hypersensitivity. Overall, these complications in ASD-affected children increase their stress and functional defects.

1.4 Tactile hypersensitivity in autism

The difficulties in sensory processing in ASD patients are the outcomes of emotional dysregulation (Sung, Glazer, O'Grady, McEntee, Bosley *et al*., 2022). The tactile

stimuli produce unwarranted responses that alter the sensory pathways, resulting in tactile hypersensitivity. The normal physiological development in children progresses with their emotional regulation and socialisation abilities, which are made possible by the ability to perceive stimuli through touch (Malik & Marwaha, 2025). However, in ASD, the disturbed emotions are associated with defects in tactile processing, resulting in reduced self-care ability in the affected children (Puts, Wodka, Tommerdahl, Mostofsky & Edden, 2014). This is why the independent execution of activities of daily living appears highly challenging in ASD patients. Social withdrawal, depression, and elevated stress responses in ASD patients are also the outcomes of tactile processing abnormalities (Thye *et al*., 2018). The scientific literature reveals high incidences of pain insensitivity, irritability, stress, and panic in ASD patients with tactile hypersensitivity (Allely, Gillberg & Minnis, 2013; Orefice, 2020). These manifestations interfere with the learning abilities of the affected children and severely impact their physical and mental health outcomes.

Literature reveals a clinical correlation between social attributes and tactile abnormalities in children with ASD. Some studies have correlated repetitive behaviours with over-responsiveness and social impairments with under-responsiveness in ASD (Istvan, Nevill & Mazurek, 2020). Conversely, the contemporary literature also provides a direct link between social features and over-responsiveness (Green, Hernandez, Bowman, Bookheimer & Dapretto *et al*., 2018). The social impairments in ASD are specifically associated with discrimination and detection ability or touch processing. ASD patients have changes/impairments in their olfactory detection threshold (Kumazaki, Muramatsu, Fujisawa, Miyao, Matsuura *et al*., 2016); however, no study has established a direct linkage between the clinical/ psychological attributes and the discrimination and amplitude threshold. Of note, the

information processing defects in ASD are due to the sensory and emotional manifestations, which are marginally contributed by touch processing (Baum, Stevenson & Wallace, 2015). The behavioural regulation in ASD children is indicated by sensory dysfunction (Mikkelsen, Wodka, Mostofsky & Puts 2018).

The distinctive behaviours exhibited by ASD-affected children include the avoidance of head and body touching during grooming and dressing, which are indicative of their tactile hypersensitivity (Marco, Hinkley, Hill & Nagarajan, 2011). The receptor pathway of the Pacinian corpuscles in adults with ASD develops hypersensitivity indicated by vibrotactile stimuli, thermal stimuli, and reduced tactile perceptual thresholds (Marco *et al*., 2011). The subcutaneous and dermis regions across the upper and lower extremity joints, soles, and palms of the body contain the unique mechanoreceptors, called Pacinian corpuscles, with the ability to sense intense pressure and vibration of an elevated frequency (Roudaut, Lonigro, Coste, Hao, Delmas *et al*., 2014). However, the extra sensitivity of Pacinian corpuscles in ASD patients results in the development of avoidance attitudes against the tactile stimuli irrespective of their intensity and despite them being non-harmful (Mikkelsen *et al*., 2018).

Contrarily, in paediatric patients, no vibrotactile-related variations in the tactile perceptual threshold have been observed in scientific studies (Espenhahn, Godfrey, Kaur, McMorris, Murias *et al*., 2022). However, some studies have recommended a relationship between social-emotional reactions and phenotype for behavioural tactile sensitivity in ASD patients (Mikkelsen *et al*., 2018). These findings indicate that the neurological or developmental attributes or age of ASD patients can possibly alter/modify their tactile hypersensitivity. Nevertheless, the sensory phenotype and affective traits can modulate the behavioural tactile responses, which forms the basis

of a relationship between social-emotional processing and tactile hypersensitivity (Schaffler, Middleton & Abdus-Saboor, 2019). Furthermore, the central neural integration pathways and peripheral sensory mechanisms are substantially impacted and their alterations are responsible for variations in sensory processing mechanisms in ASD patients (Gonçalves & Monteiro, 2023).

1.5 Emotional patterns in ASD in the context of sensory issues

The significant changes in the sensory processing mechanisms in ASD-affected children reduce their environmental adaptation ability, social interaction skills, and learning capacity (Jones, Hanley & Riby *et al*., 2020). The sensory experiences trigger abnormal responses in children with ASD. For example, the sensory stimuli in ASD patients, such as changes in body positions, body movements, touch, different tastes, smells, sounds, and sights induce under-responsiveness (hypo-reactivity) or over-responsiveness (hyperreactivity) (Marco *et al*., 2011). As per the Dunn's sensory processing model, the sensory issues in ASD-affected children can be classified into low registration, sensory avoiding, sensory seeking, and sensory sensitivity. This classification relies on the sensory stimuli responses and their neurological threshold that significantly vary among ASD patients (He, Williams, Harris, Powell, Schaaf *et al*., 2023). Those with a reduced threshold against a particular sensory input exhibit both sensory avoidance and sensory sensitivity. The sensory inputs are frequently circumvented by those children affected by sensory avoidance (He *et al*., 2023). These children tend to distance themselves from the stimuli source by covering their ears or eyes (Balasco, Provenzano & Bozzi, 2020). Alternatively, children adopt other attitudes/behaviours to show their distress and do not keep themselves away from the stimuli. An elevated sensory stimuli threshold is observed in sensory-seeking and low-registration children (Passarello, Tarantino, Chirico, Menghini, Costanzo *et al*., 2022).

Of note, unlike the low registration children, additional sensory input, due to the elevated neurological threshold, is pursued by sensory-seeking children who are affected with ASD. ASD children, who are highly sensory seeking, find it increasingly difficult to habituate due to their central nervous system's reduced ability to process the sensory signals at an optimum threshold (Marco *et al*. 2011). This is why they require high-intensity sensory input to habituate and acclimatise to sensory stimuli. Conversely, ASD children who exhibit low sensory thresholds have greater sensory input processing ability. As a result, they tend to react to the sensory stimuli at a rapid rate in comparison to those with high sensory thresholds. Such children, however, attempt to escape the sensory stimuli and experience increased sensory load as soon as they encounter the corresponding stimuli (Marco *et al*. 2011). These mechanisms underscore the fact that the ASD-related sensory subtypes show high variations in their behaviours, which are reflected by their ability to habituate.

Importantly, the sensory difficulties in ASD children lead to their confusion and altered perceptions that substantially change their worldview in comparison to typically developing (TD) children (Balasco, Provenzano & Bozzi, 2020). This is why ASD-affected children prevalently develop breakdowns and get frightened and muddled under the impact of their sensory issues. They also experience challenges in undertaking their activities of daily living and develop problems in attention and emotional regulation and behaviours due to poor sensory regulation (Gentil-Gutiérrez, Cuesta-Gómez, Rodríguez-Fernández & González-Bernal *et al*., 2021). The adaptive social behaviours in ASD-affected children are poorly developed and exhibit early behavioural issues that correlate with their sensory over-responsivity. Their behavioural issues are further aggravated when the parents of such children misinterpret their sensory overload as difficult attitudes and ignore their sensory

requirements (McCormick, Hepburn, Young & Rogers, 2015). The emotional recognition difficulty and language delay in ASD-affected children are triggered by their sensory processing issues. Notably, the sensory difficulties in ASD independently predict a variety of behavioural complications that reduce self-regulation, social communication, attentional responses, and conduct and increase repetitive behaviours (Boyd, Baranek, Sideris, Poe, Watson *et al*., 2010). The communication and language-related challenges in ASD children are associated with their auditory processing challenges. Additionally, social communication, attentional response, and conduct issues in ASD children correlate with visual processing challenges (Chung & Son, 2020). Literature also reveals that abnormal sensory information responses in children with ASD occur due to attention mediation by the defective cortical circuitry (Sivapalan, Sivayokan, Raveenthiran & Sivayokan, 2024).

The Reizschutz concept of the protective barrier to the stimulus reveals the tendency of autism-affected children to challenge their sensory overload by constructing defending approaches leading to developmental challenges (Balasco *et al*., 2020). The behavioural and emotional difficulties in ASD were also elaborated by Eveloff who considered them as the outcome of disrupted self-representations due to the absence of environmental stimuli exposure at an early stage (Balasco, Provenzano & Bozzi, 2020). Notably, the social perception theory emphasised social cognition deficits as the independent predictor of sensory symptoms in ASD. This theory emphasises that ASD patients particularly experience difficulty in responding to emotional stimuli due to their sensory processing issues (Patil & Kaple, 2023). Literature reveals that the negative emotions and avoidance behaviours among ASD children is due to their excessive responsiveness to sensory stimulation (Balasco, Provenzano & Bozzi, 2020). Furthermore, ASD-affected children have low adaptive skills due to their

sensory issues, which trigger their sensory-seeking behaviours and hyper responsiveness. The deep pressure simulation is a therapeutic approach which utilises tactile inputs/weighted blankets to enhance sensory responses in the ASD-affected children. These children also lack autonomy, have low social cognition and have a reduced health-related quality of life (Kojovic, Ben Hadid, Franchini & Schaer, 2019).

The cognitive and sensory complications in ASD-affected children make them emotionally vulnerable and impact their daily activities (Mazefsky & White, 2014). Furthermore, as these children they try to cope, manage and process sensory information they may experience psychological distress as a result. Eventually, they develop anxiety and depression that correlate with their reduced tolerance to uncertain environment and atypical sensory processing (Mazefsky & White, 2014). Children with ASD subsequently develop social withdrawal under the sustained impact of their emotional and sensory overload, which further aggravates their anxiety and depression. Additionally, ASD patients are also affected by rigidity of thoughts that continues to increase their anxiety, confusion, and restrictive/repetitive behaviours (South & Rodgers, 2017).

1.6 Emotional dysregulation signs/symptoms in ASD

The Child Behaviour Checklist recognises aggressive attitude, depression/withdrawal, and depression/anxiety as the predominant manifestations of ASD (Mazefsky, Anderson, Conner & Minshew, 2010). However, improper speech, hyperactivity, stereotypic attitude, social withdrawal, lethargy, and irritability are identified as ASD signs in the Aberrant Behaviour Checklist (Chua, Abd Rahman & Ratnasingam, 2023). Other potential manifestations include frustration, anger, and negative emotional

event-related consistent and recurrent thinking patterns (Dell'Osso, Massoni, Battaglini, De Felice, Nardi *et al*., 2023). The difficulties in emotional regulation in ASD challenge response inhibition, interpretation of social situations, perspective taking, and emotional identification in ASD-affected children. Emotional dysregulation is majorly associated with repetitive behaviours and social difficulties in children with ASD (Northrup, Hartman, MacKenzie, Sivathasan, Eldeeb *et al*., 2024). They often experience uneasiness, sadness, and reduced positive affect. The emotional reactions of ASD-affected children are often sustained, rapidly escalating, and intense in character (Conner, Golt, Shaffer, Righi, Siegel *et al*., 2020). The specific signs identified by various studies include hitting/kicking a toy, yelling, and avoidance, indicating sustained passive emotional reactions with structured assignments. The ASD-affected children often cannot tolerate loud noise and in response exhibit physical outbursts, crying, or screaming (Williams, He, Cascio & Woynaroski., 2021). The sustained state of distress among ASD patients is difficult to manage in the absence of external support (Tathgur & Kang, 2021). Since ASD patient cannot effectively control their emotional stimuli, they develop mood swings, withdrawal, and fear responses to unmet social requirements or difficult situations (Morie, Jackson, Zhai, Potenza & Dritschel, 2019).

1.7 Emotional regulation models concerning ASD

The stress responses, social behaviour, and emotional regulation are governed by the autonomic nervous system, which is directly impacted by the 10^{th} cranial nerve (i.e., vagus nerve) (Breit, Kupferberg, Rogler & Hasler, 2018). The vagus nerve actively communicates messages from digestive system, lungs, and heart to the brain and vice versa. The alternative state of relaxion and stress and optimisation of social engagement and calm states is governed by the myelinated vagus nerve. The

appropriate function of the vagus nerve is critical to the maintenance of social responsiveness and emotional self-control in individuals (Breit *et al*., 2018). This is why a thorough understanding of the vagus nerve function is necessary in ASD patients to understand its possible influence on the patient's behaviour, sensory processing, and stress management ability (Engineer, Hays, & Kilgard, 2017).

The rapid disinhibition and inhibition of the tone of the myelinated vagus not only impacts cardiac function but also helps to comfort or mobilise a person (Porges, 2007). It also downgrades the activity of the hypothalamic-pituitary-adrenal axis and hinders the impact of the sympathetic nervous system on cardiac function. The visceral state modulation by the myelinated vagus is known as a vagal break, which is a mechanism to help the persons to quickly disengage or engage among themselves or with things and acquire satisfaction or self-soothing attitudes. The polyvagal theory emphasises the role of a vagal break in minimising stress vulnerability and improving flexibility in the behaviours of normal individuals (Barbier, Chen, & Huizinga, 2022). This vagal break is actionized when the environmental factors impact the myelinated vagus and trigger its situational disengagement and engagement. The stimuli-based autonomic flexibility is impacted in patients with ASD due to their incapacity to utilise the vagal break (Muscatello, Kim, Vandekar & Corbett, 2021). This abnormality is the outcome of threat neuroception dysfunction in ASD-affected children that reduces parasympathetic activity and results in chronic vagal withdrawal against unperceived social stimulus (Owens, Mathias & Iodice, 2021).

A study utilised a predictive coding approach to unravel the neurological complications of ASD among children (Gonzalez-Gadea, Chennu, Bekinschtein, Rattazzi, Beraudi *et al*., 2015). The findings from this approach indicated that ASD-affected children had

abnormal emotional responses to unfamiliar events due to elevated activation of the dorsolateral prefrontal cortex and lowered activity of the superior frontal cortex (Richey, Damiano, Sabatino, Rittenberg, Petty *et al*., 2015). Additionally, patients with ASD develop defects in their inhibitory control, leading to neural abnormalities and associated abnormal behaviours such as striking an object, shouting, or repeating a task (Mosconi, Kay, D'Cruz, Seidenfeld, Guter, 2009).

The interoception-alexithymia model advocates the association between interoceptive processing and the development of alexithymia in children with ASD (Butera, Kaplan, Kilroy, Harrison, Jayashankar *et al*., 2023). Additionally, a greater functional association is observed between the left lateral prefrontal cortex and left ventral anterior insula in ASD patients (Uddin & Menon, 2009). Scientific evidence reveals a significant decline in the left ventral anterior insula, right dorsal anterior insula, right precuneus, and left anterior insula connectivity, leading to alexithymia in ASD (Butera *et al*., 2023). Overall, this model affirms an association between the lateral prefrontal cortex and insula and significant variations across the left hemisphere and interhemispheric regions that impact the emotional responsiveness in ASD (Leisman, Melillo & Melillo, 2023).

1.8 Problem statement

This study explores the cause-and-effect relationship between emotional dysregulation and sensory difficulties (and resultant emotional disturbances) and associated dysfunctional behaviours and abnormal brain activity, in ASD patients. The effective mapping between these ASD attributes will help understand the cause-and-effect relationship between emotional dysregulation, tactile hypersensitivity, and other clinical manifestations in children with ASD.

This study addresses the current knowledge gaps regarding ASD mechanisms and aims at mapping brain physiology to sensory symptoms and behaviours in paediatric patients with ASD.

1.9 Research objectives

1. Assessing the impact of parent-child attributes and child's emotional regulation indices on the behavioural patterns and sensory difficulties (observational behaviour and parent report regarding tactile hypersensitivity) in ASD-affected children
2. To examine the emotional dysregulation patterns among children with ASD
3. Evaluating the interactions of emotional regulation, interoception, and alexithymia across the ASD and control groups
4. To classify ASD children in accordance with their sensory complications and dysregulated emotions

1.10 Research question

Compared to the TD children, how does emotional dysregulation in children with ASD impact their sensory issues, emotional problems, and associated troublesome behaviours?

Population	Paediatric patients with ASD
Exposure	Emotional dysregulation
Comparison	TD (normal) children
Outcomes	Sensory issues Emotional problems

	Problematic behaviours Atypical brain activity

1.11 Study hypothesis

1. A higher burden of sensory difficulties (leading to aberrant responsiveness or tactile hypersensitivity) will be observed in children with an increased frequency of emotional dysregulation in ASD, which will further correlate with abnormal brain activity and behavioural complications.
2. Parental observations and reporting will be pivotal in the emotional and sensory complications in children with ASD.
3. Behavioural issues, communication gaps, and social complications in ASD children will correlate with their sensory stimuli and their atypical responses.

2 Chapter 2: Literature Review

2.1 ASD and its impact on emotional regulation in children

2.1.1 ASD mechanisms

Children with ASD develop abnormalities in their neuronal connectivity and macrocephaly (or brain overgrowth) (Won, Mah & Kim, 2013). Synaptopathy in ASD is the outcome of sustained disruption of the neural circuitry and the function and structure of synapses due to the disfigurement of synaptic proteins. Microcephaly is the direct outcome of the defects in the cytoarchitecture of the brains of ASD-affected children. The structural and functional defects in ASD patients are more prominent in the subcortical limbic system, cerebellum, parietal-temporal lobe, and frontal lobe,

particularly in the initial stages of their development (Hashem, Nisar, Bhat, Yadav, Azeem *et al*., 2020). Autopsy and magnetic resonance imaging studies reveal a significant decline in the Purkinje cells in the cerebellum as well as the cerebellar hemisphere and cerebellar vermis hypoplasia. When ASD patients attempt to engage themselves in various attention-related tasks, their cerebellum cannot gain the required threshold for its normal activity. Alternatively, during their engagement in various motor tasks, their cerebellar activity acquires a normal pattern. Young children with ASD are often affected by cerebellar cortex hyperplasia (van der Heijden, Gill & Sillitoe, 2021). Eventually, the enlargement of the temporal and frontal lobes adversely impacts the language and social function of the ASD-affected children.

Research evidence also correlates ASD development with defects in subcortical brain locations, such as the hippocampus and amygdala (Won *et al*., 2013). Contrary evidence from several studies has revealed a possible decline in the volume of the amygdala and overall number of neurons or an early increase in the size of the amygdala in patients with ASD. Similar evidence indicates the possibility of hippocampus volume elevation or decline in ASD scenarios. The assessments through functional magnetic resonance imaging and diffusion tensor imaging indicate a loss of integrity in the white matter tracts in the superior temporal, anterior cingulate cortex, and prefrontal cortex locations in the brains of ASD-affected children, which eventually impacts their social cognition (Im, Ha, Kim, Cheon,Cho *et al*., 2018). ASD is also associated with defects in brain structures responsible for working memory. Studies also suggest inter-regional under-connectivity or local over-connectivity in children with ASD.

Notably, a range of scientific studies reveal the possible role of striatal dysfunctions in triggering autistic-like behaviours (Won *et al*., 2013). The sociability impairments in ASD also correlate with optogenetic stimulation-triggered medial prefrontal cortex excitation that gradually disrupts the excitation-inhibition balance by impacting the neural circuitry. Of note, establishing a direct relationship between ASD-related behavioural complications and neuroanatomical anomalies warrants further investigation. However, contemporary studies advocate ASD development in children in the context of abnormal activities of neurotrophic factors and growth factors (Spoto, Butera, Albertini, Consoli, Ceraolo *et al*., 2025). The ASD development also correlates with tyrosine kinase activity-based hepatocyte growth factor's transmembrane receptor "Mesenchymal-Epithelial Transition (MET)". The ASD-related MET transcription is disintegrated by the promoter location's genetic variations. In ASD-affected children, the cerebral cortex has a reduced concentration of protein and associated MET mRNA. The ASD pathology reveals defects in the structure of neurons that correlate with dendritic morphology and neurite outgrowth occurring under the influence of neuronal neurotrophic factors, such as hepatocyte growth factor, which integrates with MET to induce ASD changes (Goikolea-Vives & Stolp, 2021).

2.1.2 ASD manifestations

The predominant signs and characteristics of ASD include social interaction/communication deficit and repetitive activities/interests/behaviours (Hodges *et al*., 2020). Additionally, children with ASD cannot interpret social and emotional cues appropriately, which is why they fail to reciprocate social communication. They are unable to respond to the interactions and cannot share their feelings and likes or interests with peers and family members. Notably, the non-verbal communicative behaviours of ASD-affected children are due to the fact that they

cannot exhibit body language properly and maintain eye contact during interactions. Furthermore, ASD patients experience substantial difficulty in understanding, maintaining, and developing relationships. They fail to generate interest in communicating with peers, show reluctance in making friends, often cannot construct/show imaginative plays, and avoid behavioural adjustments based on the changing circumstances (Hodges *et al*., 2020). They repetitively use the same words and objects, which reveals their stereotyped motor activities. Their nonverbal/verbal behaviours are often ritualised, they show resistance in complying with their routines, remain inflexible, and exhibit highly insisting attitudes. The focus and intensity of their interests are very fixated/restricted. They sometimes immensely engage with their environment's sensory aspects, and the sensory inputs trigger their hypo-reactivity or hyperreactivity in varying scenarios. At times, the learning approaches of ASD patients may mask their early symptoms, which are more apparent in situations wherein the social requirements of the patients surpass their current abilities. Of note, the DSM-5 criteria recognise sensory symptoms as one of the diagnostic parameters of ASD in children, in addition to their repetitive interventions, interests, attitudes, and restricted behaviours (Wiggins, Rice, Barger, Soke, Lee, et al., 2019). Patients with ASD are also prone to other clinical complications, such as intellectual disability, epilepsy, sleeping problems, anxiety, and depression (Hirota & King, 2023). The cooccurrence of hyperactivity, attention deficit, and emotional dysregulation in ASD increases sensory complications and adds to the incidence of tactile hypersensitivity.

2.1.3 Neurobiological context – Amygdala, Prefrontal cortex, and HPA axis

In children with ASD, the responses due to brain's behavioural stress (for example: withdrawal, agitation, or anxiety) and neuroendocrine stress (due to hormonal changes) are induced by the limbic circuitry, comprising the prefrontal cortex,

amygdala, and hippocampus (Makris, Agorastos, Chrousos & Pervanidou, 2022). The stress management system in ASD-affected children is repetitively triggered for an extended duration due to defects in feedback-control procedures, caused by chronic stress in the hypothalamic-pituitary-adrenal (HPA) axis. Over time the hippocampus and amygdala fail to downregulate the activity of the hypothalamus under continued stress. Atypical development of the hippocampus and amygdala is often observed in ASD-affected children which further disrupts the stress responses. There is a possibility of typical brain development in the ASD-affected children during the initial phase of the disease (Leisman *et al*., 2023). However, the development of chronic stress in ASD impacts the brain's physiology and the neurobiological function, which triggers the atypical brain development (Hashem *et al*., 2020). Eventually, the ASD patients are impacted with stress-induced dysregulated social-emotional processing. These alterations have epigenetic causes, which under the impact of early life stress minimise the volumes of the caudate nucleus, anterior cingulate gyrus, orbitofrontal cortex, dorsolateral prefrontal cortex, insula, corpus callosum, and hippocampus. Recent evidence also indicates a possible association between amygdala hyperresponsiveness and early life stress in children with ASD. Early life stress is also known to reduce the alliance between the amygdala and prefrontal cortex and trigger the stimulus for negative emotions in ASD patients. The chronic stress influences on children with ASD impact their amygdala, which eventually disrupts their emotional mechanisms, thereby deteriorating their social behaviours, perceptions, attention, memory, emotional (implicit) learning, and emotional regulation/inhibition (Makris *et al*., 2022). When the prefrontal cortex is engaged in any stressor's cognitive and social appraisal in an ASD patient, the amygdala gets downregulated, which triggers a sustained response to the social stimulus-based stress and induces emotional

dysregulation. Furthermore, as and when ASD-affected children try to perceive emotions by interpreting facial expressions, this intervention lowers the coupling or functional association between the amygdala, ventrolateral prefrontal cortex, and ventromedial prefrontal cortex (Ibrahim, Eilbott, Ventola, He, Pelphrey *et al*., 2019). This impact is significant since the interpretation of social cues and emotional response optimisation are controlled by the ventromedial prefrontal cortex (Alexander, Wood, & Roberts, 2022); alternatively, the reduction in the improper emotional reactions and cognitive function are managed by the ventrolateral prefrontal cortex (Mitchell, 2011). Importantly, the emotional states and threats are processed by amygdala, whose activity is moderated by the prefrontal regions (Alexandra Kredlow, Fenster, Laurent, Ressler & Phelps, 2021). A significant decline in the ventrolateral prefrontal cortex and ventromedial prefrontal cortex coupling disrupts the emotional regulation and socialisation in ASD patients by lowering their ability to process stimuli and optimise their emotional reactions (Ibrahim *et al*., 2019). Additionally, under uncontrolled stress, lowered activity of the medial prefrontal cortex and overstimulated amygdala potentiate its adverse mnemonic influence, which is further aggravated under the impact of increased levels of corticosterone (Makris *et al*., 2022). Additionally, the chronic stress in ASD and eventual hyperactivity of the amygdala on the hippocampus not only adds to the cognitive decline but also increases the emotional manifestations.

2.1.4 Genetic factors in ASD

Mutations in genes, such as SHANK2, GABRB3, ATP10A, and SNRPN have a predominant role in the aetiology of ASD (Mkhitaryan, Avetisyan, Yeritsyan, Harutyunyan & Yenkoyan, 2025). The mutated sequences in the 11q13, 16p11, and 15q11−q13 locations of these genes induce the onset and development of

ASD. Recent studies demonstrate that SNRPN dysregulation adversely impacts the mRNA splicing that disrupts the ASD phenotypes (observable characteristics) and also triggers the behavioural defects and intellectual disability in ASD-affected children. Notably, the disturbance in neuronal communication and synaptic function due to SNRPN mutation induces emotional dysregulation in ASD patients. Furthermore, a sustained mutation in ATP10A disrupts the membrane integrity, transport of phospholipids, synaptic connectivity/plasticity, and neuronal membrane signalling and consistency (Jiang, Yuen, Ryan, Jin, Wang, Chen *et al*., 2013). The inhibitory neurotransmission in ASD is impacted by a mutation in GABRB3, which eventually deteriorates the inhibitory-excitatory balance. Research findings also reveal a direct association of ASD with the protein 'SHANK3' mutation that potentially disrupts excitatory synapses in terms of their signalling, organisation, and functional and structural integrity. These long-term defects result in a sustained decline in social communication and neuronal connectivity impairment in children with ASD. Similarly, mutations in SEZ6L2 in ASD trigger behavioural and synaptic defects and cease the development of neurons (Mkhitaryan *et al*., 2025). Nevertheless, the literature results reveal elevated rates of mutations in ASD-related genes in females versus males. However, scientific studies provide credible evidence concerning the higher level of resilience in females against ASD-related genes (Zhang, Li, Li, Zhang, Teng, et al., 2020). They have a greater ability to counter ASD-based genetic mutations with improved neural plasticity and DNA repair processes.

2.1.5 Environmental factors in ASD

The literature documents a variety of environmental factors impacting the occurrence of ASD in paediatric populations (Karahmadi, Karimi, Kamali, & Mousavi *et al*., 2017). The risk of ASD occurrence in the offspring is increased with ≥34 years of prenatal age

in mothers. The possible causes of the prenatal age attribution of ASD include DNA methylation alteration and germline cell mutations, which lead to sperm genomic imprinting disorder by inducing epigenetic changes in the genes responsible for the development of neurons. Furthermore, age advancements in parents deteriorate the function of their nervous and immune systems, which adds to the risk of ASD in their offspring (Lyall, Song, Botteron, Croen, Dager *et al*., 2020). The physical health of the mother is another significant factor influencing the occurrence of ASD in children. Maternal conditions, such as obesity, hypertension, diabetes (or metabolic syndrome) and maternal bleeding increase the risk of in-utero hypoxia, which results in an abnormality in the neurons of the hippocampus, membrane adhesion, and changes in myelination, thereby impacting the brain development of the foetus. The risk of ASD also increases in scenarios wherein the mother develops bacterial or viral infections during the first/second trimester of the pregnancy (Zerbo, Qian, Yoshida, Grether,Van de Water et al., 2013). Moreover, mental health complications in the mother, prenatal medication utilisation, and low socioeconomic status of the family also influence the incidence and prevalence of ASD in children. The postnatal risk factors for ASD include birth defects, low birth weight, infections in infants, anaemia, feeding problems, encephalopathy, hyperbilirubinemia/jaundice, meningitis, and breathing difficulty in the infant (Yong, Dou, Gao, Xu, Xiao, et al., 2021).

2.1.6 Emotional reactivity

Children with ASD experience substantial difficulty in modulating their emotions (Day, Mazefsky & Wetherby *et al*., 2022). Of note, older children with ASD attempt to manage their emotions with the limited use of problem-solving (or adaptive) approach and the increased utilisation of avoidance (or maladaptive) strategy (Restoy, Oriol-Escudé, Alonzo-Castillo, Magán-Maganto, Canal-Bedia *et al*., 2024). The emotional

dysregulation in ASD results in a consistent decline in positive joyful expressions and an elevation in negative affectivity (Day *et al*., 2022). Furthermore, compared to those without ASD, children with ASD appear less fearful due to their dysregulated emotions (Dell'Osso *et al*., 2023). Studies have also revealed that ASD-affected children often utilise fewer effective strategies to minimise stress and seek lesser maternal support in comparison to those without ASD. These effective strategies include social support-seeking behaviours, an inclination towards positive emotions and reluctance towards adopting negative emotions, deep breathing, distraction, and cognitive reappraisal (Cai & Samson, 2025). Furthermore, ASD-affected children exhibit lesser joy via body movements and facial expressions as and when they get engaged in joy-provoking assignments (Atkinson, 2009). The end-of-the-line tasks often trigger vocal stress, negative facial expressions, and tension in ASD-affected children. Studies suggest a possible association between emotional reactivity and verbal cognitive functioning in ASD patients. Scientific evidence further reveals that emotional dysregulation in ASD-affected children triggers their irritability, which manifests in the form of affective and behavioural complications, such as angry mood and physical aggression, respectively (Kalvin, Gladstone, Jordan, Rowley, Marsh *et al*., 2020).

2.1.7 Defects in emotional regulation

The overall findings from several studies reveal a direct association of ASD with negative emotionality, resulting in long-term negative responses, frequent outbursts, and tantrums. These difficulties emanate due to emotional dysregulation and result in behavioural, psychiatric, and social problems (Dell'Osso *et al*., 2023; Mazefsky, 2015). Research evidence further reveals age of ASD-affected children is an important factor that impacts their emotional reactivity (Day *et al*., 2022). Increased negative affect duration, intensity, and frequency are observed in younger children with ASD

compared to older children with the same diagnosis. Although the age of the ASD-affected children does not determine the level of their recovery or frustration, younger age results in more negative responses. Importantly, the emotional reactivity in children with ASD is inversely proportional to their emotional regulation. Studies also reveal the verbal ability and adaptive behaviours of children with ASD as predictors of their recovery and reactivity (Miranda, Berenguer, Baixauli & Roselló, 2023). Accordingly, elevated negative affect, indicating higher reactivity, is observed in patients with reduced adaptive behaviours. A real-world study provided a video game to the ASD-affected children. The video game was designed to challenge the problem-solving ability, cognitive skills, and patience of the participants. However, the children with ASD could not complete that assignment and appeared frustrated (Carter Leno, Forth, Chandler, White, Yorke *et al*., 2021). The literature results revealed that the ASD-affected individuals also experience substantial difficulty in recovering from these frustration-based assignments. Of note, low adaptive behaviours are observed in ASD-affected children with elevated emotional reactivity (Mazefsky, Herrington, Siegel, Scarpa, Maddox *et al*., 2013). This is why these children quit the frustration-based assignments more swiftly in comparison to those without ASD. Emotional regulation in ASD is also predicted by the executive functioning skills of the affected children (Conner *et al*., 2020). Furthermore, elevated negative reactivity is observed in ASD patients with underdeveloped adaptive behaviours (Northrup, Goodwin, Montrenes, Vezzoli, Golt *et al*., 2020). Such children find it increasingly difficult to cope with their irritation and emotional burden. These findings reveal that the severity of irritability and stereotyped and self-injurious (or problem) behaviours in ASD patients has a direct association with their adaptive coping skills.

2.2 Sensory Processing Issues in ASD

Research has shown that the increased frequency of impaired communication and low socialisation among ASD patients correlates with their hypo-responsiveness in the context of tactile modality (Foss-Feig, Heacock, & Cascio *et al*., 2012). These defects induce repetitive behaviours in children with ASD. Although the core ASD characteristics variably correlate with tactile processing, leading to sensory manifestations and tactile hyperresponsiveness. The early ASD detection depends on the appropriate differential evaluation of the variable sensory symptoms, which are the outcomes of differential tactile processing pathways. The DSM-5 parameter defines ASD in terms of sensory processing issues/differences (Patil & Kaple, 2023). In at least 90% of patients with ASD, hypo-responsiveness/hyper-responsiveness is the direct outcome of atypical sensory complications. The following significant modalities associated with ASD unravel the complex mechanisms directing the interactions between sensory and emotional processing.

2.2.1 Tactile

The children affected with ASD react abnormally to variations in texture, temperature, pressure, or touch (Riquelme, Hatem & Montoya, 2016). Since they become hypersensitive to their environments, they tend to behave inappropriately while undertaking routine activities such as dressing, grooming, and clothing. Alternatively, ASD can also trigger hyposensitivity/hypo-responsivity in patients, due to which they thrive for sensation via intense rubbing on their skin surface and via the application of profound pressure (Bogdanova, Bogdanov, Pizano, Bouvard, Cazalets *et al*., 2022). Since the vibrations and pressure sensing in individuals are governed by the mechanoreceptors, such as Pacinian corpuscles and similar neurosensory processes, their abnormal processing results in significant variations in the tactile perception in

ASD-affected children (Jenkins & Lumpkin, 2017). The changes in the mechanisms of their neurosensory pathways adversely impact their attitudes and disrupt their socialisation. The children with ASD eventually perceive any normal stimulus as inundate induction and exhibit aggressiveness in their conduct and responses. A high variation in the tactile stimuli's responsiveness and threshold in ASD patients warrants the personalisation of their diagnostic assessment and treatment strategies (Ide, Yaguchi, Sano, Fukatsu & Wada, 2018).

2.2.2 Auditory

In the ASD-affected children, the variations in the auditory processing pathways make them different from the TD children since they develop hypersensitivity to the background noise and have low tolerance to sounds of normal frequency (Poulsen, Williams, Dwyer, Pellicano, Sowman *et al*., 2024). In addition, ASD-affected children become hypersensitive to loud sounds and cannot filter the unwanted or interfering noises in their auditory apparatus (Danesh, Howery, Aazh, Kaf, & Eshraghi *et al*., 2021). The noisy environment adds to their irritation and impulsivity. The typical responses in the ASD patients to high-frequency noise include crying, shouting, and closing their ears with their hands (Williams *et al*., 2021). The sound emanating from the engines of the cars or vacuum cleaners appears highly challenging and difficult to resist for the ASD patients (Torres & Denisova, 2016). Alternatively, a few of the ASD patients cannot process low-frequency evoked sounds and have low auditory processing ability (Mamashli, Khan, Bharadwaj, Michmizos, Ganesan *et al*., 2016). They require high-volume auditory inputs due to their decreased sensitivity to normal pitch sounds. Due to the defective processing of auditory inputs in ASD-affected children, they fail to communicate with their peers and family members. This also impacts their overall learning trajectory and language processing capacity (Gonçalves

& Monteiro, 2023). Furthermore, the poor social and emotional regulation and overwhelming or unpleasant responses to sounds due to the sensory overload impact the function of the brain and deteriorate their high-level skills (Green & Ben-Sasson, 2010). Additionally, there is a high risk of misdiagnosis due to the defective auditory sensory processing and neurocognitive decline in ASD (Balasco, Provenzano & Bozzi, 2020; Blumberg, Zablotsky, Avila, Colpe, Pringle *et al*., 2016).

2.2.3 Visual

Patients with ASD have low tolerance for environments with a high visual appeal (Lawson, Mathys & Rees, 2017). In addition, the fluorescent lights/light-emitting diode bulbs induce an unpleasant effect in ASD patients. They also cannot tolerate bright lights and tend to close their eyes to avoid the gaze. On the other hand, ASD patients with low sensitivity to light aspire for a high visual stimulus (Chung & Son, 2020). They aspire for bright light and feel comforted with exposure to spinning things. The high variations in visual processes across the ASD-affected children influence their ability to interact with alternating environments, overall learning capacity, and attention span.

2.2.4 Sensory profile 2

The sensory processing skill/ability of children affected with ASD sensory profile 2 (SP-2) is appropriately evaluated by the SP-2 diagnostic tool (Marcilla-Jorda, Grande, Coelho, Rubio-Belmonte & Moro-Ipola *et al*., 2024). The caretakers or patients of the children with ASD need to address and provide responses to a uniquely crafted questionnaire to provide significant cues indicating the behavioural reactions of the patients. The scores obtained from SP-2 help to investigate and identify proprioceptive, vestibular, tactile, visual, and auditory processing in ASD patients. It

also facilitates the assessment of poor registration, sensory sensitivity, avoidance, and sensory seeking interventions of the ASD patients (Marozza *et al*., 2025). The SP-2 scores are indicative of the ASD patients' socialisation, participation in a range of interventions, and associated sensory processing difficulties.

2.2.5 Sensory experiences questionnaire

The sensory experiences questionnaire (SEQ) is a diagnostic approach to evaluate the ASD-related sensory attributes (Ausderau, Sideris, Furlong, Little, Bulluck et al., 2013). The sensory stimuli and the associated abnormal reactions in ASD patients are evaluated via SEQ in terms of their intensity and frequency (Zetler, Cermak, Engel-Yeger, Baranek et al., 2022). The caretakers of the ASD-affected children are required to provide insights into the sensory experiences and associated preferences of the children, which subsequently help to individualise the treatment interventions. The systematic assessment of the sensory context, modalities, and patterns also assists in evaluating their sensory hypo-responsiveness or hyper-responsiveness.

2.2.6 Sensory processing disorder

Although the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM–5; American Psychiatric Association, 2013) parameter does not list sensory processing disorder in ASD as a clinical diagnosis, it recognises atypical sensory processing as an associated ASD feature (Patil & Kaple, 2023). The DSM-5 manual also provides evidence concerning a direct association of ASD manifestations with sensory inputs and their abnormal reactions in the affected patients. The sensory processing defects identified in patients with sensory processing disorder correlate with the neurodevelopmental abnormalities that may or may not be related to ASD. However, it's important to recognise the early phenotypic markers of the sensory processing

disorder to evaluate its possible association with ASD symptomatology in a variety of patient scenarios (Passarello *et al*., 2022). It is therefore highly recommended to perform neuroimaging studies and practice psychosocial measures and utilise the findings from the clinical observations and caregiver reports for developing the optimised neuromodulation interventions for the ASD affected children.

2.2.7 The interactions between sensory processing and emotional regulation

The bidirectional and complex associations between sensory processing and emotional regulation in children with ASD are underscored in scientific literature (Brandes-Aitken, Powers, Wren, Chu, Shapiro *et al*., 2024). It is important to note that the high level of distress and anxiety in ASD-affected children are the direct outcomes of their elevated sensory load. The sensory sensitivity in ASD patients is often associated with emotional dysregulation. This is why children with ASD find it increasingly difficult to resist their sensory inputs and self-manage their sensory overload.

2.2.8 The contemporary findings and theories

The emotional distress and developmental delay in ASD-affected children are directly connected with their inability to handle high sensory stimuli. They try to avoid these overwhelming sensory stimuli by using protective responses, such as hand flapping, rocking, and avoiding eye contact or any contact with ~~sensory~~ stimuli. These coping interventions have not been taught to the ASD-affected children but are rather triggered by their hyperactive sensory system that helps to minimise the sensory input. This mechanism is well elaborated by Reizschutz or the protective shield theory (Brown, 2020). Since the ASD-affected children have limited or no exposure to environmental stimuli due to their sensory processing defects, they lack the self-

representation skills that correlate with their avoidance attitudes and language deficits, which eventually challenge their social interactions (Balasco, Provenzano & Bozzi, 2020; Brown, 2020). This sensory processing deficit-induced sensory avoidance is possibly responsible for the emotional turmoil and restricted physiological development in ASD-affected children. A contrary concept reveals that the early environmental stimulus exposure in children with ASD is often inappropriate (or below the required threshold), which subsequently deteriorates their self-representations (i.e., self-consciousness about their emotions and identity). This is why children with ASD develop behavioural restrains, attitudinal problems, and emotional overburdening. This alternative contention is recognised by the psychodynamic theory.

Another contemporary concept reveals that the inappropriate management of the emotional overload in ASD patients is due to their sensory processing challenges, which results in the development of social cognition difficulties (Thye *et al*., 2018). This alternative concept is recognised by the theory of social perception. According to this theory, the high responsiveness to sensory stimuli is the predominant cause of the avoidance of the stimulus and the associated passive emotions in the ASD patients (Mikkelsen *et al*., 2018). Alternatively, the broken mirror neuron hypothesis contends that the social challenges in ASD patients are the outcomes of sustained abnormalities in the mirror neuron system and its regulatory systems (Yates & Hobson, 2020). The hypothesis concerning synaptogenesis impairment indicates that serious abnormalities in protein synthesis and synaptic transmission induce multifactorial neurodevelopmental conditions leading to repetitive behaviours, social isolation, and defective communication in ASD patients (Lepeta, Lourenco, Schweitzer, Martino Adami, Banerjee *et al*., 2016). The sustained mutations in the predominant genes (i.e.,

MECP2, FMR1, TSC1/2, SHANK, NLGN, and NRXN) governing these functions trigger ASD-related synaptopathology (Guang, Pang, Deng, Yang, He *et al*., 2023). In addition, the sensory defects in ASD induce alexithymia and anxiety that continue to deteriorate the social adaptability and emotional self-regulation in ASD-affected children (Poquérusse, Pastore, Dellantonio & Esposito, 2018).

2.2.9 The regions of the brain and associated processes – the neuroanatomy

The anterior-dorsal hindbrain includes a neuron-dense and foliated structure known as the cerebellum, which is located above the brainstem and pons and beneath the cerebral cortex's occipital lobe (Donovan & Basson, 2016). The cerebellum typically controls affective regulation, cognitive processing, language, and other higher-order cognitive functions of higher order. Children with ASD develop a neuroanatomical change in the cerebellum impacting the regulation of their intellect and emotions. This change is recognised as cerebellar hypoplasia in medical parlance. Scientific studies reveal a gradual and constant decline in the left lobule 7B, right crus I, and lobule 9 (cerebellar vermis) in ASD patients (Lu, Chen, Wang, Huang, Jiang *et al*., 2023). The contemporary evidence depicts a reduction in the volume of the grey matter in crus 2/1 and lobule 7 of the cerebellum in ASD (D'Mello, Crocetti, Mostofsky & Stoodley, 2015). These abnormalities in the cerebellum are responsible for a range of cognitive deterioration and behavioural problems in patients with ASD. In addition, the morphological changes in the archicerebellar and posterolateral neocerebellar cortices in the cerebellar hemisphere in ASD trigger the associated symptoms. The unique behavioural phenotypes in ASD are associated with structural and functional abnormalities in the cerebellum of the affected patients and are responsible for their abnormal attitudes (Sydnor & Aldinger, 2022).

The anterior medial temporal lobe incorporates a bunch of nuclei, which is of the shaped like an almond, and is known as the amygdala (Donovan & Basson, 2016). This structure is directly responsible for processing the mechanisms and pathways associated with aggression, pleasure, fear, and emotional regulation. The structural/functional defects or abnormal growth of the amygdala in those with ASD can impact/deteriorate their ability to recognise emotions and facial expressions, emotional memory, reward prediction, and social interactions (Donovan & Basson, 2016). According to the medical studies, the asocial behaviours in ASD correlate with the functional alterations in the amygdala, and are prevalent in the glutamatergic subpopulation (Hennessey, Andari & Rainnie, 2018). Alternatively, those with posterior dorsal medial amygdala neurons (i.e., GABAergic subpopulation) exhibit social grooming and aggression. Notably, the higher-order functions, such as communication, learning, social behaviours, emotions, working memory, planning, and decision-making are governed by the frontal cortex (Friedman & Robbins, 2021). A significant decline in the size of the brain and the early abnormal growth of the frontal cortex in ASD-affected children impairs their cognition and stress self-management capacity. Another hypothesis reveals that the ASD cortex undergoes changes in the distribution of the mini-column and focal deterioration, resulting in epigenetic alterations in the radial glial neuronal stem cells (Friedman & Robbins, 2021). A contemporary theory reveals potential disruptions in the Reelin and WNT signalling pathways leading to the ASD symptoms (Jiang *et al*., 2022). Furthermore, the neuronal mispositioning in ASD occurs due to abnormalities in the migration, differentiation, and proliferation of neurons. A recent hypothesis indicates that the disturbances in the inhibitory-excitatory balance in neurons and deterioration in the connectivity across the frontal, thalamic, and cerebellar regions of the brain trigger ASD in children

(Nelson, Sacha & Valakh, 2015). Studies have also revealed that abnormalities in the locus coeruleus-derived fibre tracts and the forebrain function, such as inappropriate monoamine transfer impact the mini-columnar circuitry in ASD (London, 2018). The neocortices in ASD develop defects in their microfoci that result in radial migration defects, triggering neuroanatomical abnormalities in the affected patients. However, prospective studies are warranted to reconfirm and further explore the defects in several brain regions in the ASD-affected children.

2.3 Tactile hypersensitivity

The defects in the inhibitory processes in ASD-affected children impact their ability to discriminate or detect the stimuli (Fukuyama, Kumagaya, Asada, Ayaya & Kato *et al*., 2017). This defect correlates with tactile stimuli-related hypersensitivity in children with ASD. The atypical tactile responses impact the communication skills of ASD patients in a manner that makes it challenging to share their experiences, concentrate on daily activities, or relax in normal scenarios. A few research studies correlate thermal pain in ASD patients with their tactile hypersensitivity (Bogdanova, *et al*., 2022). Other studies have revealed a low threshold and high reactivity or responsiveness for skin conductance in ASD patients (Dijkhuis, Gurbuz, Ziermans, Staal, & Swaab, 2019). The contemporary literature also suggests that irrespective of the non-social or social context/cognitive assignments, the sensory stimuli in ASD patients trigger high autonomic/sudomotor responses (Bharath, Moodithaya, Halahalli, Undaru, Nallilu *et al*., 2020). The correlation between ASD symptoms and elevated activity of the sympathetic nervous system is also revealed by the scientific literature. Studies have demonstrated marked variations in vibrotactile stimulation-related thresholds in ASD patients, which are possibly responsible for their perceptual processing variations. A reduced detection task-based threshold in ASD also correlates with tactile stimuli-

related hypersensitivity in ASD-affected children (Puts *et al*., 2014). The sustained defects in the intricate neurocognitive and neurosensory pathways aggravate sensory difficulties that eventually trigger tactile hypersensitivity and associated emotional issues in ASD patients. However, the complete pathophysiology concerning these defects is yet unknown and warrants further investigation through clinical studies.

2.4 Interoception/Alexithymia

Interoception is the innate ability of individuals to feel and know the physiological/pathological conditions, including thirst, hunger, toileting, trauma, palpitations, heart beating etc (Schmitt & Schoen, 2022). However, alexithymia is a contrary condition wherein individuals fail to elaborate on and define their feelings and emotions. It results in impaired expression in ASD-affected children and increases their risk and incidence of depression, anxiety, and the development of passive feelings (Hogeveen & Grafman, 2021). Literature findings reveal that increased alexithymia in ASD is associated with elevated mental health complications and contributes to problems in emotional processing in nearly half of the ASD-affected individuals (Kinnaird, Stewart & Tchanturia, 2020).

2.4.1 Association between alexithymia and subjective interoception

A hypothesis concerning alexithymia correlates this condition with defective interoception (Brewer, Cook & Bird, 2016). The children affected with ASD experience alexithymia-related emotional complications. A few studies revealed that following the statistical adjustment in alexithymia outcomes in ASD patients, the corresponding emotional complications had no noticeable association with ASD. This outcome reveals that alexithymia profoundly impacts the clinical manifestations in ASD patients.

Clinical studies have underscored that alexithymia in ASD patients reduces their interoceptive ability (Shah, Hall, Catmur & Bird, 2016). Eventually, the affected children experience and develop inattention and confusion. In addition, they develop abnormal responses to stimuli due to defects in their autonomic nervous system. The ASD-affected children, due to alexithymia, fail to understand and appropriately respond to their bodily inputs which severely impacts their self-regulation and emotional self-management.

2.4.2 Emotional facial expressions in the context of neural processing alterations

The abnormal neural processing in ASD patients is reflected through their inappropriate socialisation, reduced executive functions, and emotional turbulence (indicated by their facial expressions) (Crowell, Keluskar & Gorecki *et al*., 2019; De Stefano, Schmitt, White, Mosconi, Sweeney *et al*., 2019). The functional defects in the temporal parietal junction and insula impact the emotional processing and conspicuousness in ASD patients. These defects in ASD are mediated by abnormal interoception and comorbid conditions. In comparison to TD children, there is a greater functional association between the left lateral prefrontal cortex and left ventral anterior insula in ASD patients. Additionally, the development of alexithymia in ASD-affected children impacts their ability to process and interpret emotions and results in atypical facial expressions by disrupting the interactions across left ventral anterior insula, right dorsal anterior insula, right precuneus, and left anterior insula (Butera *et al*., 2023).

2.5 Brain physiology and ASD behaviours

The behaviours of the ASD-affected children are impacted by alterations in their physiological processes governed by neural networks, hormones (and their receptors), and the autonomic nervous system (Wang, Wang, Wu, Wang & Sun, 2023).

2.5.1 Electroencephalography

The electroencephalography assessment indicates substantial alterations in the neural inhibition and excitatory mechanisms in ASD patients (Milovanovic & Grujicic, 2021). They are revealed by significant reductions in the gamma power and elevations in alpha/theta power. These changes are indicative of disrupted connectivity across the brain regions and atypical oscillatory activity of the brain.

2.5.2 Skin conductance

The electrodermal activity or skin conductance is substantially impacted in ASD-affected children (Ferguson, Hamlin, Lantz, Villavicencio, Coles *et al*., 2019). Eventually, any sensory stimulus is associated with delayed arousal and reduced or elevated responsiveness in ASD patients. The inappropriate functioning of the autonomic nervous system is possibly responsible for abnormal skin conductance, which adds to the behavioural complications and emotional overload in children with ASD.

2.5.3 Cortisol

The abnormal concentration of peripheral cortisol in ASD patients increases their perception of dangers or threats and increases their physiological stress (Gao, Zou, Yang, Zhao, Wang *et al*., 2022). Eventually, the ASD patients have increased social requirements, behavioural challenges, and a high risk for withdrawal. These complications are potentiated in patients with a dysregulated HPA axis.

2.5.4 Neural synchrony

The neural activity coordination or neural synchrony/interpersonal neural synchronisation is significantly influenced in patients with ASD (Quiñones-Camacho, Fishburn, Belardi, Williams, Huppert *et al*., 2021). A decline in neural synchrony in ASD elevates sensory hyposensitivity and challenges social cognition. The cognitive control, emotional responses, and socialisation in ASD patients correlate with improper coordination of the neural activities in the central, salience, and default networks in various locations of the brain. Subsequently, ASD-affected children experience repetitive behaviours and refrain from interacting in their social networks.

2.6 Parent-child interactions

The social environments of the ASD-affected children are influenced by their interactions with parents/caregivers. The children's perception of their parents significantly impacts their emotional regulation (Li, Li, Liu, Chen, Xing *et al*., 2022). The following evidence-based tools are available to observe and report the parent-child interactions and improve and personalise the health support measures for ASD management.

2.6.1 Social Responsiveness Scale

The Social Responsiveness Scale (SRS-2) tool utilises a questionnaire to evaluate the physiological attributes and social interaction patterns in children with ASD (Channell, 2020). It also helps to evaluate their problem behaviours, social skills, expressive language, and non-verbal communication.

2.6.2 Behavioural Assessment of Executive Function

The Behavioural Assessment of Executive Function (BRIEF)-2 approach is used to evaluate the cognition/executive function in ASD patients (Gentil-Gutiérrez,

Santamaría-Peláez, Mínguez-Mínguez, González-Santos, Fernández-Solana *et al*., 2022). It helps to assess their material organisation, task supervision, organisation and planning skills, working memory, initiative taking ability, emotional control, flexibility, self-supervision, and inhibition characteristic.

2.6.3 Anxiety Disorders Interview Schedule

The Anxiety Disorders Interview Schedule, Child/Parent Version (ADIS/ASA) helps to identify ASD-related behaviours and segregate them from comorbid anxiety conditions (Kerns, Renno, Kendall, Wood, & Storch *et al*., 2016). It further assists in monitoring the characteristics of ASD patients, such as compliance with the schedule, sensory sensitivity, apprehensions, responses to stimuli, and abnormal behaviours.

2.6.4 Ecological validity

The ecological validity approach helps to identify the behavioural, emotional, social, and sensory challenges of ASD-affected children by analysing the parental observations (Charman, Hepburn, Lewis, Lewis, Steiner *et al*., 2013). This model helps to formulate personalised interventions, for children with ASD, based on therapeutic alliance, family's disability awareness levels, community support, family configuration, and communication language. These interventions also assist in improving the health-related quality of life and increasing the quality-adjusted life years of the ASD patients.

3 Chapter 3: Research Methodology

3.1 Research Design

The complex association between sensory challenges and emotional regulation in ASD-affected children was evaluated in this study through a mixed-method approach (Smajic, Avdic, Pasic, Prcic & Stancic*,* 2022). An exclusively qualitative or quantitative approach could not appropriately unravel the interactions between the neurophysiological, sensory, and behavioural markers and their influence on the intricate ASD phenotype. Accordingly, a mixed-method intervention was proposed for analysing the study outcomes (DeBoth, Reynolds, Lane, Carretta, Lane *et al*., 2021). Additionally, the mixed method approach offered both qualitative and quantitative options to analyse the behavioural profiles of the children with ASD.

The use of quantitative interventions in this study was based on the need to statistically assess the ASD outcome metrics with the systematic use of physiological interventions and psychometric parameters (Bishop-Fitzpatrick, Minshew, & Eack, 2012; Cherewick, Daniel, Shrestha, Giri, Dukpa *et al*., 2023). The use of these evidence-based tools facilitated the quantification of the ASD-related data and categorically compared and contrasted the statistical outcomes across the ASD-affected and TD children. Alternatively, the use of qualitative approaches assisted in evaluating those specific ASD parameters that could not be analysed through quantitative measures. These parameters include behavioural outcomes/responses, child-parent communication dynamics, and subjective experiences of the ASD patients (Franz & Kelly, 2021).

The quantitative and qualitative data collection was guided by an evidence-based data triangulation approach (Arias Valencia, 2022). This triangulation strategy facilitates a

detailed understanding of a process while utilising a variety of data sources and multiple interventions. Accordingly, the basis for using the triangulation strategy was its potential to utilise a range of perspectives, strategies, and sources to enhance the validity and reliability of the overall outcomes. It also helped to thoroughly understand and interpret the study outcomes by allowing an in-depth analysis of ASD data. Triangulation also facilitated a robust alliance of physiological parameters and the interview data.

The physiological parameters were objectively aligned with the parent-based interview outcomes (Cheng, Smith, Butler, Taylor, & Clayton *et al*., 2022). Subsequently, a deeper analysis of the psychometric scales and electroencephalogram (EEG) findings was undertaken (Milovanovic & Grujicic, 2021). The assessments in ASD-affected children facilitated the interpretation of their behavioural, emotional, and sensory interactions. Notably, the latest research in the neuromedicine domain has adopted similar mixed-method strategies to assess the neurodevelopmental conditions and associated clinical complications in paediatric populations (Wu, Deatrick, McQuaid & Thompson, 2019).

3.2 Participants

The study population was classified into TD children (n = 25) and ASD-affected children (n = 25). The homogeneity across the characteristics of the participants was ascertained with the diligent implementation of the inclusion parameters.

3.2.1 Inclusion parameters

The selected paediatric patients with ASD and those without ASD were at least six years of age. However, the maximum age defined for the selection was 12 years. The

ASD-affected patients had a definitive diagnosis of ASD in accordance with the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM–5; American Psychiatric Association, 2013). The ASD diagnosis in each of the selected patients was verified with a registered physician. The ADOS (Autism Diagnostic Observation Schedule)-2 diagnostic tool was used to reaffirm the ASD diagnosis (Maddox, Brodkin, Calkins, Shea, Mullan *et al*., 2017). Additional information from the parents of the ASD-affected children, regarding their signs and symptoms, was obtained to validate the ADOS-2 scores. The inclusion criteria for this mixed-method study mandated the exclusive selection of those ASD-affected patients who were able to interact through verbal communication. This is because of the fact that ASD-affected children were required to verbally interact with the instructor and comply with the guidelines for engaging themselves with the neurophysiological recording intervention and the psychometric assignments. The experimental protocol had to be accomplished with these predefined inclusions. Additionally, the self-support interventions facilitated the acquisition of reliable information in concordance with the selection parameters. The propensity score matching was performed on the initial set of the total selected patients to balance their numbers in each cohort (Kane, Fang, Galetta, Goyal, Nicholson *et al*., 2020). The demographic characteristics of both study groups were appropriately matched to minimise the undue impact of biological or developmental attributes on the overall results.

3.2.2 Exclusion criteria

Patients with differential genetic conditions such as Down syndrome and fragile X syndrome were not included in this mixed-method study. Additionally, those with neurophysiological/autonomic dysfunction due to ongoing treatments, concomitant neurological complications, and sensory manifestations (unrelated to ASD) were also

excluded from the analysis. Children with high severity behavioural/emotional dysregulation or physical abnormalities (that could hinder their engagement in the study) were further excluded to reduce the risk of biased results. The appropriate execution of the study assignments based on the sensory processing evaluation warranted the exclusion of the children with vision/hearing loss. Patients with a cognitive deficit based on a <70 total intelligence quotient (IQ) score were also excluded from the study to reduce the impact of significant cognitive impairment on the study outcomes. Those on antipsychotic medications were excluded from this study due to their possible impact on the behavioural, emotional, or physiological outcomes. These significant exclusions ascertained that the underlying neurobiological attributes or physiological/behavioural markers had minimal or no impact on the final results.

3.2.3 Sampling approaches

Purposive sampling was utilised to select the participants in concordance with the inclusion criteria (Campbell, Greenwood, Prior, Shearer, Walkem *et al*., 2020). The special education schools, rehabilitation centres, hospitals, and neurodevelopmental institutes were consulted for recruiting the children with ASD. The diagnostic parameters for ASD were cross-checked before shortlisting the patients with a definitive diagnosis of ASD. This verification ascertained that the selected patients appropriately depicted the broader patient populations with ASD Alternatively, mainstream educational institutes, including schools, were approached to select the TD children for the control group. Importantly, the geographical placement, age, race, socialisation, and economic status of the participants in both study groups were statistically matched to minimise the risk of confounding and enhance the transferability of the outcomes. Prior to initiating the study selection procedure, the

caregiver networks, school administrators, and parents were contacted for initial screening. The G-power assessment tool was performed to estimate the statistical power before the recruitment procedure (Kang, 2021). The sample size range of 50-100 participants was subsequently calculated. Additionally, the alpha level, statistical power, and effect size were recorded as 0.05, 0.8, and 0.5 (Cohen's D value), respectively (Gülkesen, Bora, Ilhanli, Avsar, & Zayim *et al*., 2022). These preliminary measures facilitated the accomplishment of sampling objectives and ascertained the systematic evaluation of the similarities and differences between the ASD and TD study groups.

3.2.4 Demographics

This study initially deployed a total of 76 participants, including 35 in the study group and 41 in the control group. The propensity score matching of the baseline characteristics resulted in the segregation of 25 patients in each of the ASD and control groups, respectively. The mean age (standard deviation: SD) of the participants was 9.1±1.5 years (age range: 6-12 years). The male-to-female ratio of 4:1 in the selected participants concorded with the literature findings (Loomes *et al*., 2017). Most of the study participants, in study and control groups, were recruited from both suburban and urban locations. Nearly 26% of the patients were Caucasians, 70% were Blacks, and 4% were Whites. Approximately 70% of the participants were from the upper-middle class, 25% were from the middle class, and 5% belonged to the impoverished sections. Excluding the 5% underprivileged patients, 95% of them pertained to financially stable families, and either of their parents was at least a graduate and employed. The consistency in the baseline characteristics of the study participants helped to minimise their impact on the study results.

3.3 Measures (interventions) and instruments

The admix of quantitative and qualitative approaches was used to perform the multifactorial evaluation of sensory processing and emotional regulation in ASD-affected children versus the TD children (Abdel Ghafar, Abdelraouf, Abdelgalil, Seyam, Radwan et al., 2022; Mahdi, Viljoen, Yee, Selb, Singhal, et al., 2017). These assessments were undertaken by measuring the corresponding neurophysiological and behavioural attributes.

3.3.1 Qualitative Measures

3.3.1.1 ADIS-P (Parent Version of the Anxiety Disorders Interview Schedule)

The ADIS-P intervention was utilised to gather practical information about the emotional regulation approaches and anxiety levels concerning their ASD-affected/TD children (Kerns *et al*., 2016). The data were gathered via semi-structured interview sessions that allowed the parents/caretakers of the children to subjectively explain the daily activities, emotions, and behaviours of their children. The utilisation of the ADIS-P tool assisted in the collection of potential data to analyse the experiences of the children in both study groups with respect to their environments. This tool also facilitated the analysis of the possible associations between behavioural outcomes of the children in relation to their sensory difficulties and emotional regulation. The parents had the opportunity to share their individual perspectives and observations about the anxiety levels of their children.

3.3.1.2 FSSC-R (Revised Edition of Fear Survey Schedule for Children)

The FSSC-R tool was utilised to collect and analyse data concerning the emotional dysregulation and apprehensions/fears among children in the ASD and TD groups

(Muris, Ollendick, Roelofs & Austin, 2014). The structured interview sessions were conducted with the caretakers/parents to identify the distress/fear or insecurities of children in relation to the healthcare facilities, danger/risk factors, death, animals, and unknown/lesser-known things. The follow-up questions for the parents were framed to facilitate the comprehensive description of the emotional dysregulation, including its complications and inducers, in the studied children.

3.3.2 Quantitative interventions

3.3.2.1 SRS-2 (2nd Edition of the Social Responsiveness Scale)

The social impairment/socialisation levels in the children across the ASD and TD groups were examined by the SRS-2 tool (Channell, 2020). A questionnaire was distributed across the parents/caretakers to gather their inputs regarding the behaviours and social interaction patterns, and overall communication skills of the studied children. The SRS-2 scores for each of the ASD versus TD child were generated by analysing the collected data regarding the following parameters.

1. Repetitive behaviours
2. Restricted/hindered interests
3. Socialisation (including the motivation levels)
4. Social interaction
5. Executive function from the social perspective/social cognition
6. Self-consciousness/social awareness

The social responsiveness level was interpreted and compared between the study groups in accordance with the total SRS-2 scores.

3.3.2.2 SP (Sensory Profile)-2

The daily sensory processing levels of the studied children were further assessed by the SP-2 intervention (Licciardi & Brown, 2021). The respective inputs from the parents were translated into sensory scores concerning the position of the body, movements, touch, vision, and auditory perceptions. The processing of the sensory information in the children was analysed by evaluating their registration level, sensitivity, avoidance, and attention-seeking attitudes. The sensory manifestations in children of both groups were classified as per the SP-2 scores. Subsequently, the emotional regulation in each participant was interpreted in accordance with the identified sensory processing patterns.

3.3.2.3 ADOS-2 (Second Edition of Autism Diagnostic Observation Schedule)

The intensity of the ASD manifestations was interpreted by the severity scores, calculated with ADOS-2 intervention (Greene, Vasile, Bradbury, Olsen & Duvall, 2021). The ASD diagnosis versus the TD status was reaffirmed by analysing the severity of repetitive activities, restricted acts, play patterns, and interactions in the studied children. The data used to generate the ADOS-2 severity scores included the behavioural observations and developmental history reported by the key informants, including the parents or caretakers.

3.3.2.4 EEG – Electroencephalography

The neurophysiological markers in children from the ASD and TD groups were confirmed by the systematic analysis of neural synchrony and cerebral function via the

EEG intervention (Froese & Valencia, 2020). The data were quantified for somatosensory defects and abnormalities in the frontal region of the brain. The scalp of each participant was exposed to the electrodes, and coherence intervention was initiated to evaluate the electrical activity of the brain. In addition, the theta, beta, and alpha frequencies were processed to record the respective power spectral density. These observations facilitated a comprehensive assessment of the physiology of the brain and its sensory processing and regulation pathways.

3.3.2.5 Cardiovascular reactivity

The cardiovascular responsiveness to the sensory and emotional stimulations was interpreted in terms of variations in the heart rates in ASD versus TD children (Amoss, Leong, Evans, Ousley, Herrington *et al*., 2018). The reduced or increased variations in the heart rates provided insights into the physiological stress and its association with the emotional responsiveness and function of the autonomic nervous system in the studied children.

3.3.2.6 Electrodermal activity/Skin conductance

The function of the sympathetic nervous system and emotional arousal among the ASD children versus TD children was correlated with their skin conductance responses (McCormick *et al*., 2014). The responsiveness of the sweet gland was assessed by the placement of electrodes on the surface of the skin. The emotional conditions of the participants were depicted with the sweet gland function. Emotional arousal was indicated by an increased frequency of skin conductance responses. The overall skin conductance results provided insights into the severity of sensory hypersensitivity and emotional regulation in the studied children.

3.3.2.7 Cortisol level quantification

The stress response system of the ASD versus TD patients was examined by evaluating their HPA axis function, which was measured by level of their cortisol in saliva (Edmiston, Blain & Corbett, 2016). The acute or chronic stress responses, indicating emotional dysregulation, in the participants were revealed by the elevated level of their cortisol.

3.3.2.8 SEQ – Sensory Experiences Questionnaire

The sensory experiences of the participants were objectively recorded by a questionnaire, which was disseminated across the parents of the ASD-affected and TD children (Suter, Pehlivan, Ak, Harris, & Lyons-Warren, 2024). The data regarding the sensory domains corresponded to the responses of the participants against the sensory stimuli. The SEQ outcomes also depicted the impact of sensory processing challenges on the activities of daily living of the studied children.

3.3.2.9 BRIEF – Behaviour Rating Inventory of Executive Functions

The parents of the study participants were interviewed, and the data were collected as per BRIEF parameters to identify the behavioural regulation, organisation, planning, and emotional control abilities of the participants (Lace, Seitz, Austin, Kennedy, Ferguson *et al*., 2021). The behavioural patterns, emotional regulation level, and associated cognitive function of the studied children were indicated by their individual scale scores and composite scores. The inhibition and emotional control were identified by the individual scale scores; however, the metacognition and behavioural regulation indices were determined to calculate the composite scores.

3.3.2.10 Emotional reactivity

The quantitative assessment of the emotional response frequency, duration, and intensity in the study participants was performed to identify their emotional reactivity (Dell'Osso *et al*., 2023). The structured assignments were assigned to the participants, and the respective observations were coded for their quantitative assessment. In addition, the parents were requested to provide their inputs about the emotional responsiveness of their children on standard rating scales. The emotional dysregulation levels in the ASD and TD children were directly proportional to their emotional reactivity scores.

3.3.2.11 Reaction Time

The attentional control and cognitive processing were identified in the participants after assigning them a range of activities (Ridderinkhof, de Bruin, van den Driesschen & Bögels, 2018). The information processing time was calculated by the statistical analysis of the observations, indicating how rapidly the children were able to respond to the sensory stimuli provided in different assignments. The information processing ability of the children was revealed by this reaction time. In addition, the emotional conditions and sensory hypersensitivity appeared to be the predominant factors impacting the reaction time in the ASD-affected and TD children.

3.3.2.12 Detection threshold

The detection threshold intervention evaluated the ability of the ASD and TD children to respond to the tactile stimulation (Mikkelsen *et al*., 2018). The tactile hypersensitivity was indicative of reduced thresholds. Contrarily, increased thresholds revealed

hyposensitivity. This intervention helped to identify tactile processing differences among the studied children.

3.3.2.13 Amplitude and frequency discrimination

This test helped to identify the exact amplitude and frequency of the tactile stimuli and the corresponding responses in the participants (Rotschafer, 2021). The ASD and TD children were directed to respond to the stronger tactile stimulus after being presented with two distinct tactile stimuli of different intensities. The tactile sensory discrimination capacity was evaluated by quantifying the shortest identifiable variation in the amplitude of the tested stimuli.

3.3.2.14 Temporal order judgment

Temporal order judgment is a test that is used to evaluate information processing speed of participants (Chan *et al*., 2016). Two unique consecutive stimuli are provided to the participants and they need to recognise the initial stimulus (Chan *et al*., 2016). The temporal processing potential of the participants is statistically examined by identifying the first timepoint when they interpreted the appropriate order of the stimuli. Accordingly, the shortest duration of stimulus identification was quantified to statistically evaluate the temporal order judgment.

3.4 Data collection

3.4.1 Qualitative surveys

The video conferencing platforms were established to conduct the interviews with the parents of the participating children (Geoffray, Bourgeois-Mollier, Maleysson-Baste, Gallifet, Dochez *et al*., 2025). All data were encrypted to safeguard the health-related confidential information. The clinical psychologists, with expertise in managing ASD

complications, were deployed to initiate the interview sessions. The audio-video recordings of the parental interviews were thoroughly converted into verbatim transcripts. The contextual attributes and non-verbal cues were identified and reported via the field notes (Bianco, Finisguerra, Betti, D'Argenio & Urgesi, 2020). In addition, offline surveys were administered to the parents to capture additional information. Any doubts among parents regarding the interview and survey sessions were clarified through one-on-one calls with the psychologists.

3.4.2 Physiological evaluation

Rooms with dim lights were assigned to capture the physiological observations, including the EEG findings (Sung, Lin, Chu, & Lin, 2022). The resistance across the EEG caps was set to less than 5 kilohms. The children were asked to rest for at least five minutes before being exposed to auditory, visual, and tactile stimuli. BIOPAC software was used to synchronise the information concerning the electrodermal activity and heart rate variability in participating children (Tschacher, Ribeiro, Gonçalves, Sampaio, Moreira *et al*., 2025). The hormonal integrity of the samples of saliva was preserved by storing them in cold temperatures (<0°C).

3.5 Data assessment

3.5.1 Quantitative assessment

The statistical analyses of the data were undertaken via SPSS (version 31) (Gupta, Mishra, Pandey, Singh, Sahu *et al*., 2019). The normally distributed data were assessed via Analysis of Variance (ANOVA) and independent sample t-test (or equivalent parametric assessments) (Mishra, Singh, Pandey, Mishra & Pandey, 2019). The Shapiro-Wilk test was used for normality evaluation (Habibzadeh, 2024). The non-parametric data were statistically evaluated via the Mann-Whitney U test (Sundjaja,

Shrestha & Krishan, 2023). The associations between the neurophysiological indices and emotional regulation scores were determined by Spearman/Pearson correlation coefficients (Rovetta, 2020). The variables concerning the cognitive and sensory functions were statistically evaluated to identify emotional dysregulation. A multivariate regression approach was used for this statistical assessment (Grant, Hickey & Head, 2019).

Odds ratios, Eta-squared, and Cohen's D approaches were used for calculating the effect sizes (Serdar, Cihan, Yücel & Serdar, 2021). The statistical significance of all outcomes was determined by the probability value ($p \leq 0.05$) (Kwak, 2023). In addition, the predefined variables were compared and contrasted via the Bonferroni correction. The Swartz Centre for Computational Neuroscience (SCCN)/University of California, San Diego (UCSD)-based MATLAB's high-performing EEGLAB tool was used to process the EEG data (Martínez-Cancino, Delorme, Truong, Artoni, Kreutz-Delgado *et al.*, 2021). The next steps included the extraction of brand-related power, independent component assessment, and exclusion of the artefact. The normal data distribution was obtained by log transforming the cortisol outcomes. Additionally, the Kubios HRV tool was used to collect and analyse data concerning the health rate variability (Rogers, Schaffarczyk & Gronwald, 2022).

3.5.2 Qualitative assessment

The non-quantifiable parameters in this study were analysed in terms of potential themes. The deductive and inductive approaches were used for the conversion of core concepts based on sensory processing and emotional regulation into meaningful codes. The iterative review and assessment of the thematic maps was followed by the debriefing process to improve the outcomes (Eells, 2016). The predominant themes

constructed for qualitative evaluation included regulatory coping processes, sensory accommodation, emotional responsiveness, tactile hypersensitivity, sensory difficulties, and emotional regulation. Nearly one fourth of the samples were processed to calculate the inter-rated reliability (Zhao, Feng, Ao & Liu, 2022). The agreement among the data interpreters was confirmed by the Kappa statistic (reference range: 0.81-1.0) (Dettori & Norvell, 2020). The analysis of transcripts was continued until the cessation of new codes, indicating the saturation of the observed themes.

3.6 Ethical perspectives

The local Ethics Committee granted permission for this mixed-method study. The conventions of the Declaration of Helsinki guided the study measures ("World Medical Association Declaration of Helsinki," 2013). The caretakers of all participants were thoroughly informed about the objectives, risks, benefits, and outcomes of this study. The parents provided written informed consent for the study (Manti & Licari, 2018). However, verbal consent was obtained from the ASD-affected and TD children. The confidentiality of the health information was maintained with the use of encryption and generation of anonymous codes to conceal the identity of the participants. The safety and comfort of all children was ascertained, and the presence of caretakers was mandatory throughout the physiological assessments and EEG intervention. Patients had the right to exit from the study at any point in time without specifying the reason.

4 Chapter 4: Results

4.1 Descriptive Statistics

This chapter presents the results obtained from the participants included in this study. The chapter describes numerical findings obtained through regression and correlational analysis and the descriptive findings obtained through questionnaires. This approach provides a broader view of the findings obtained from the study.

4.2 Child Age and Gender Distribution

The average age of children in the ASD group was 9.0 years (SD = 1.95), while the control group had a slightly lower average of 8.87 years (SD = 2.17). This similarity in age distribution between the groups indicates a well-matched sample in terms of developmental, mental age and chronological stage. Regarding gender distribution, the ASD group had a higher proportion of male children (Figure 1) (n = 41, 77.4%) compared to females (n = 12, 22.6%), as shown in the figure below. In contrast, the control group had a relatively balanced gender split: 24 females (51.1%) and 23 males (48.9%). This gender disparity aligns with previous findings that suggest a higher incidence of ASD diagnoses in male children.

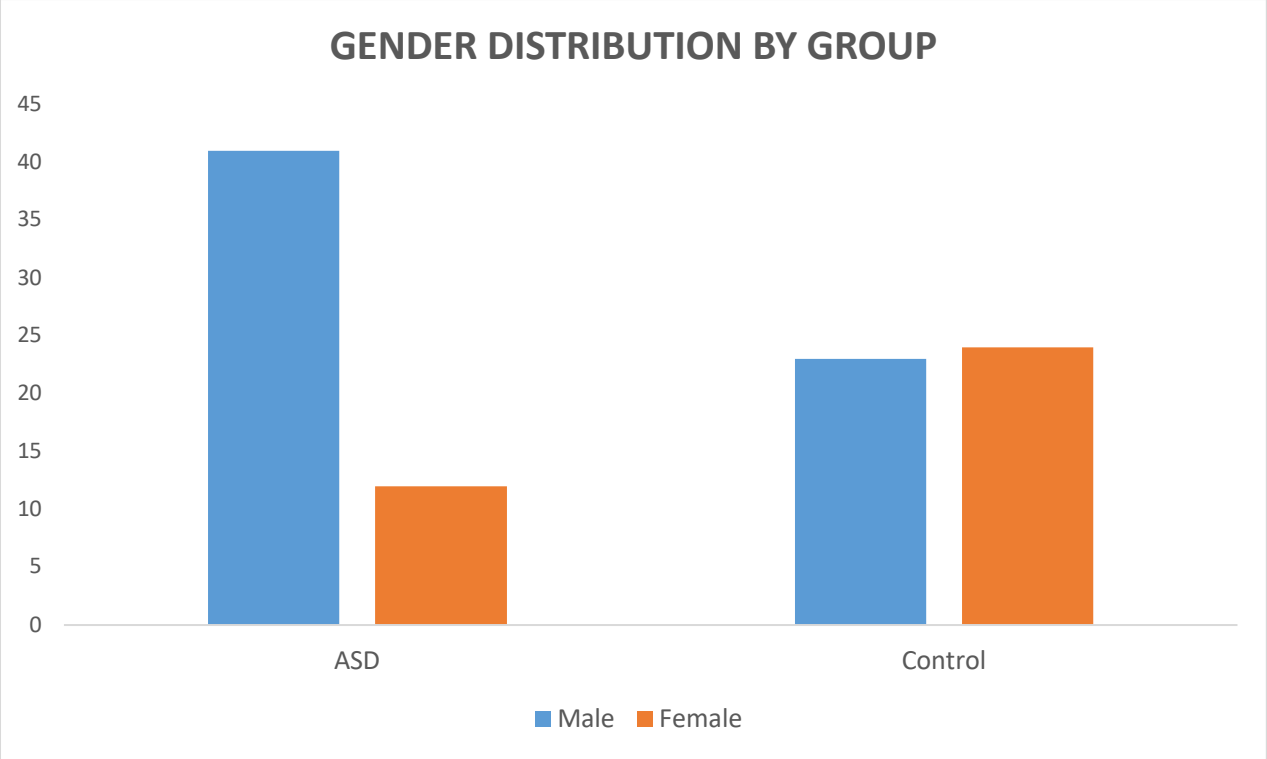


***Figure 1**: Gender distribution in ASD and Control groups as shown in a stacked bar chart. The X axis represents the study groups (ASD and Control), while the Y axis shows the number of participants. Bars are divided by gender, with female and male counts stacked within each group.*

4.3 Ethnicity composition

Ethnic distribution in both groups showed that the majority of participants identified as Caucasian (Figure 2). In the ASD group, 69.8% were Caucasian (n = 37), followed by Hispanic/Latino (24.5%, n = 13) and Black/African American (5.7%, n = 3). The control group had a similar composition, with 80.9% Caucasian (n = 38), 17.0% Hispanic/Latino (n = 8), and 2.1% Black/African American (n = 1). The ethnic representation across groups reflects a predominantly White sample, which may influence the generalisability of the findings.

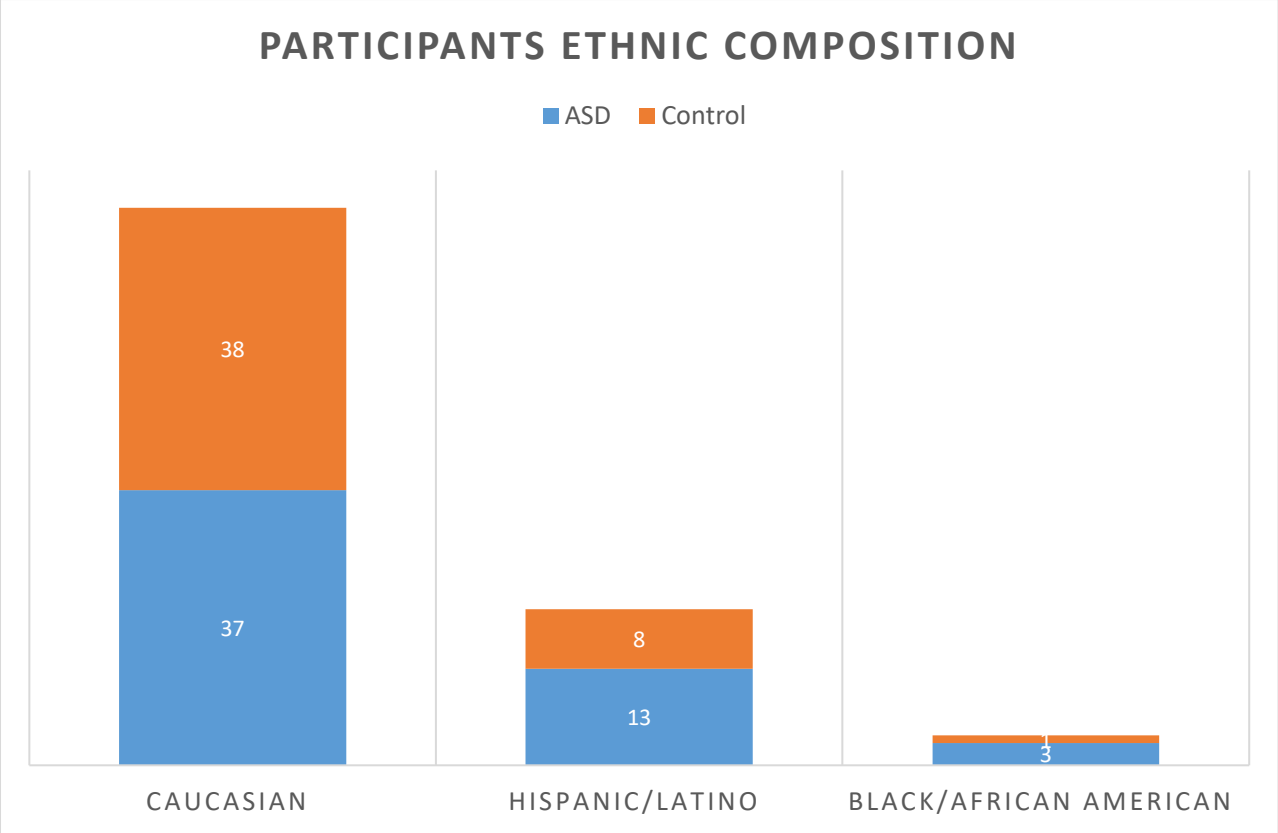


***Figure 2**: Ethnicity comparison among the participants in both ASD and control groups. The X axis shows the number of participants while the Y-axis shows their ethnicity*

4.4 Caregiver Age and Education

The average caregiver age category for both groups was approximately similar (Figure 3). Most caregivers fell within the 36–45-year age range. The mean age rank (ordinal) for the ASD group was 2.72 (SD = 0.77), and for the control group, it was 2.74 (SD = 0.74), indicating nearly identical distributions.

Educational attainment was notably high across both groups. In the ASD group, 45.3% (n = 24) held a Master's degree, 34.0% (n = 18) had a Bachelor's degree, and 20.8% (n = 11) possessed a Doctoral degree. The control group mirrored these trends with 55.3% (n = 26) holding a Master's, 27.7% (n = 13) a Bachelor's, and 17.0% (n = 8) a

Doctoral qualification. These high education levels suggest a well-educated participant sample, possibly indicating increased awareness and access to ASD-related resources.

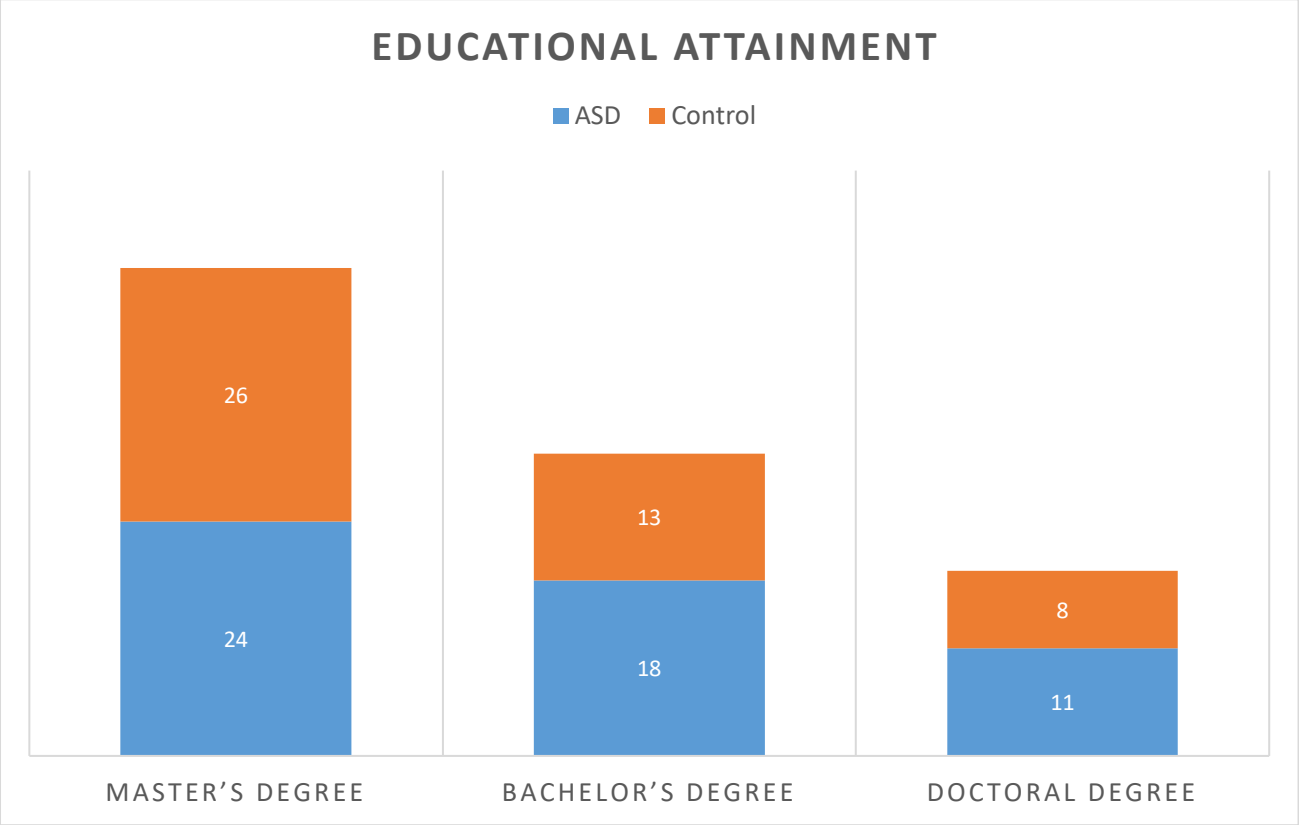
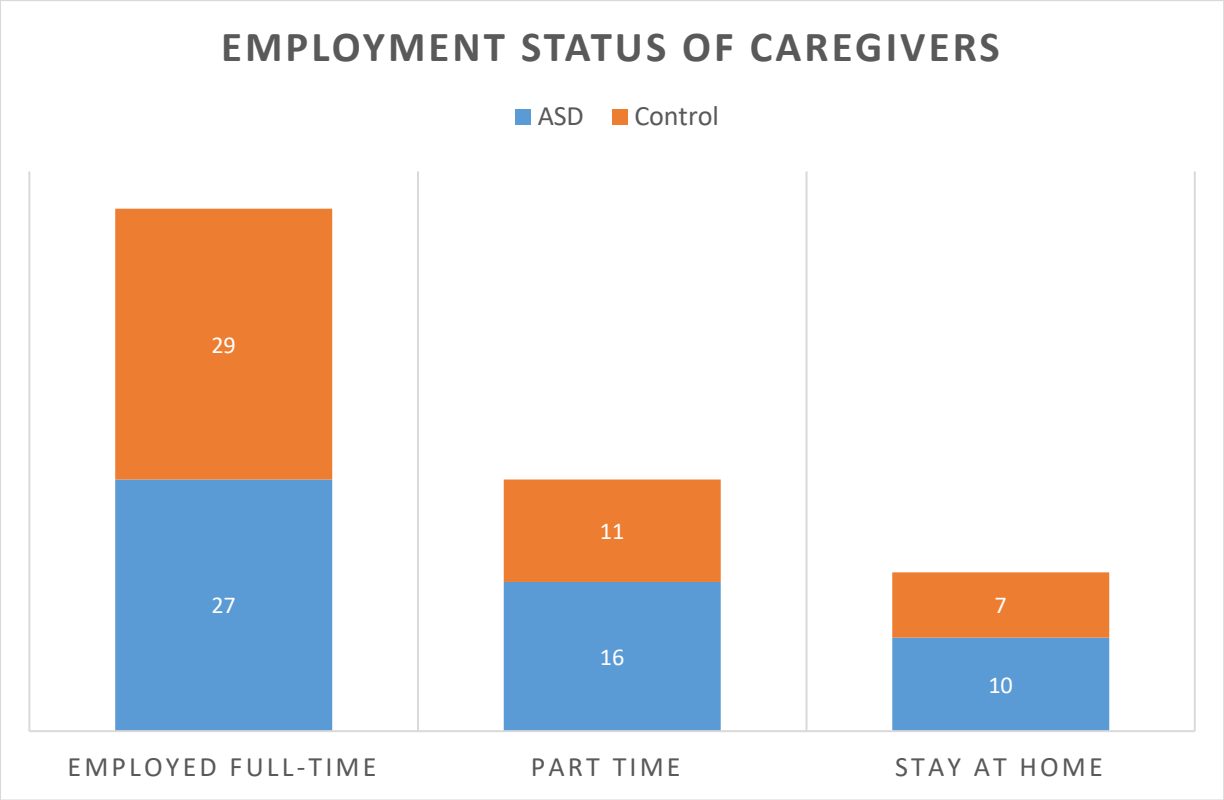


***Figure 3*: This figure shows the educational status of the caregiver by group. The X-axis shows the frequency while the Y-axis shows the educational level.**

Employment and Income Characteristics

The majority of caregivers in both groups reported being employed full-time. In the ASD group, 50.9% (n = 27) were employed full-time, 30.2% (n = 16) part-time, and 18.9% (n = 10) were stay-at-home parents. Similarly, in the control group, 61.7% (n = 29) were full-time employed, 23.4% (n = 11) part-time, and 14.9% (n = 7) stay-at-home (Figure 4). Household income levels varied across groups. The most common income bracket in the ASD group was £40,000–£60,000 (n = 27, 50.9%), followed by £60,000–

£80,000 (n = 11, 20.8%). Only a small number of ASD households reported income above £80,000 (n = 4, 7.5%) or below £20,000 (n = 1, 1.9%). In the control group, 31.9% (n = 15) reported income in the £20,000–£40,000 range, 27.7% (n = 13) in £60,000–£80,000, and 23.4% (n = 11) in £40,000–£60,000. Thus, the control group showed more income variability, with a slightly greater proportion of participants in the lower-income bracket.



<u>Figure 4:</u> This figure shows the employment status of the caregiver in both ASD and control groups. The X-axis shows the numerical value while the Y-axis shows the type of employment.

4.5 Household Size and Location

Household size in both groups was predominantly mid-sized, with 5–6 members being the most common configuration. The ASD group had 64.2% (n = 34) of participants in 5–6 member households, while the control group had 61.7% (n = 29) in the same

category. Smaller households (3–4 members) were slightly more frequent in the control group.

Concerning geographical location, the ASD group had more participants residing in urban areas (n = 25, 47.2%), followed by suburban (n = 19, 35.8%) and rural areas (n = 9, 17.0%). The control group showed a similar trend with 22 participants (46.8%) from urban settings, 14 (29.8%) from suburban, and 11 (23.4%) from rural locations as illustrated in the chart below.

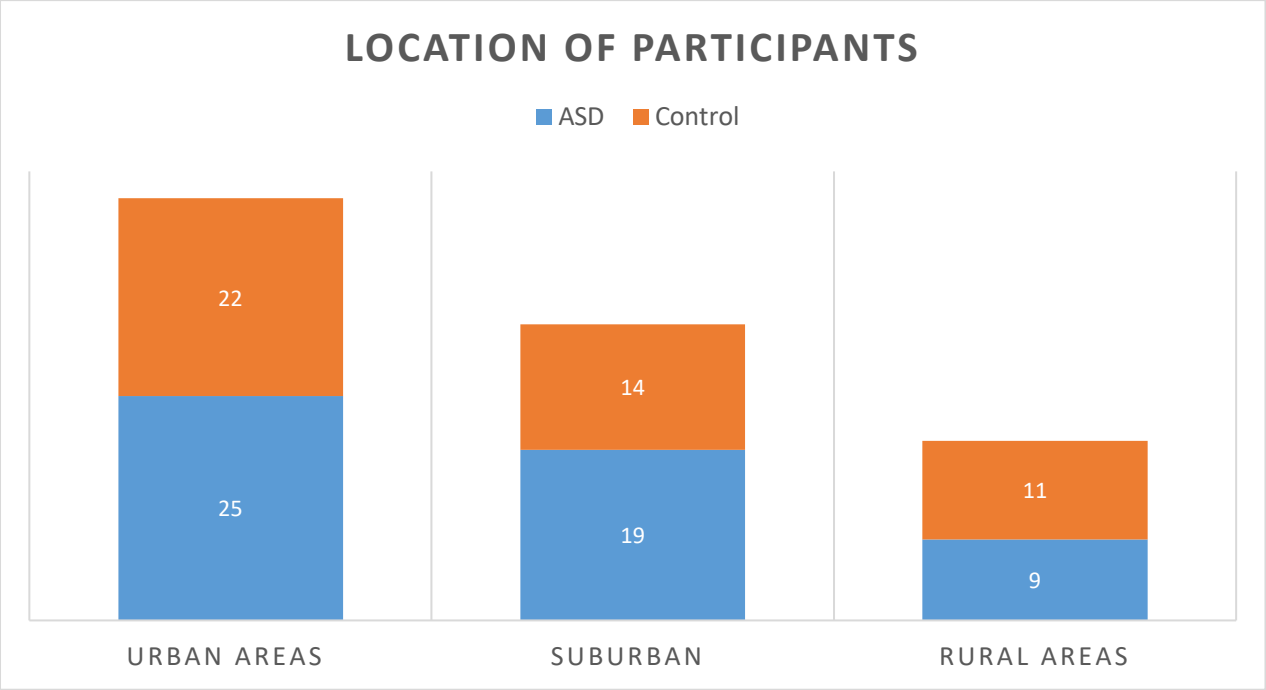


***Figure 5**: This figure shows the Geographical location of participants in both ASD and Control group. The X-axis shows the number of participants in ASD and control group while the Y-axis shows their geographical location.*

4.6 Comparison of Baseline Data between ASD and Control Groups

To understand potential differences in emotional regulation, sensory experiences, and behavioural expressions, the mean scores and standard deviations of 18 questionnaire items were calculated for both the ASD and control groups. These were

grouped into three primary domains: Emotional Regulation (ER1–ER6), Sensory Difficulties (SD1–SD6), and Behavioural Outcomes (BO1–BO6). The findings indicated consistently higher mean scores in the ASD group across all dimensions, suggesting heightened difficulties in comparison to their typically developing (TD) counterparts.

4.6.1 Emotional Regulation (ER1–ER6)

Children in the ASD group reported substantially higher mean scores across all emotional regulation items. For instance, on ER1, which reflect difficulty calming after emotional arousal, the ASD group had a mean of 3.85 (SD = 0.79) compared to 2.87 (SD = 0.88) in the control group. Similarly, on ER2 and ER3— capturing frequency of emotional outbursts or challenges with emotional identification—ASD participants scored around 3.98 and 3.92, while control children scored significantly lower (2.93 and 2.83, respectively) (Figure 6).

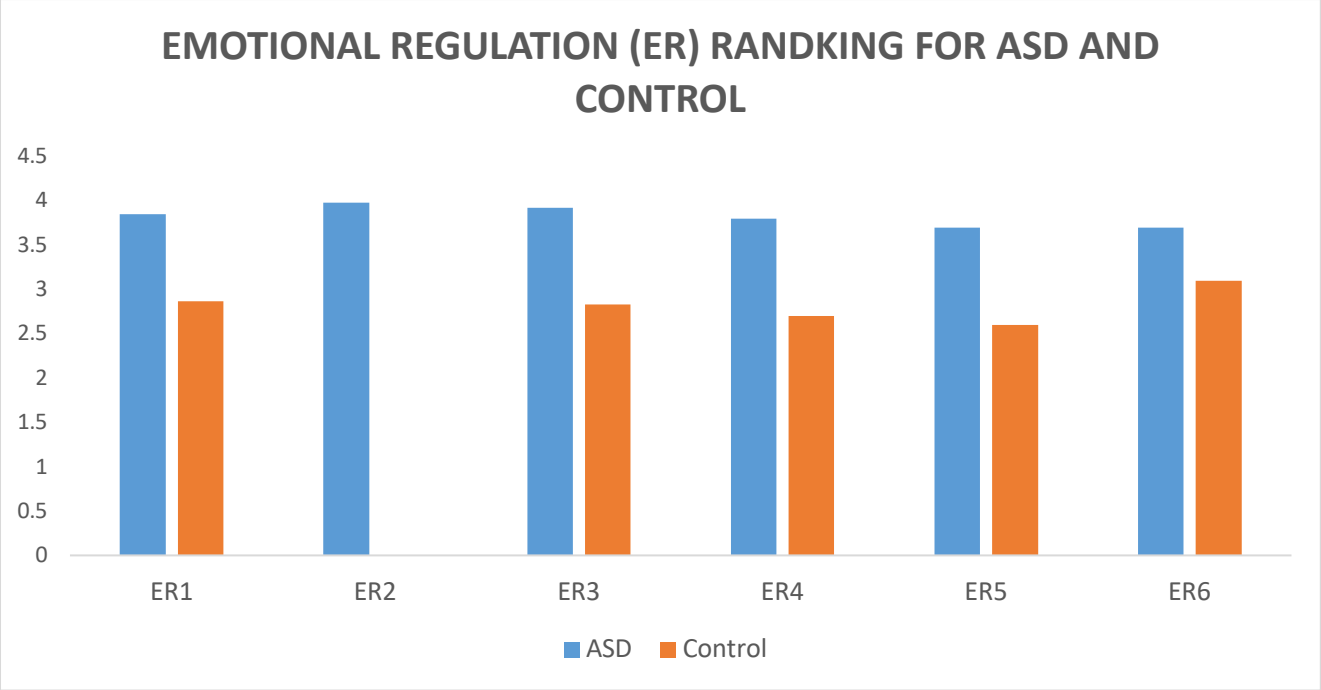


***Figure 6**: This figure compares ASD and control group for emotional regulation sub-scale. The X axis shows the mean score while the Y-axis shows the ER sub-scale across the two groups.*

These consistently elevated means suggest more frequent and severe challenges in emotion regulation among children with ASD, validating previous observations of emotional dysregulation as a central characteristic of the disorder.

4.6.2 Sensory Difficulties (SD1–SD6)

Sensory difficulty items also showed notable disparities. ASD group means ranged from 3.65 to 3.84 across the SD items, compared to control group means ranging between 2.60 and 2.83. For example, SD3 (possibly related to discomfort with tactile sensations) showed a mean score of 3.84 (SD = 0.89) in ASD children, indicating significant hypersensitivity. Control group responses on this item were notably lower (M = 2.70, SD = 0.90). These results underscore that sensory hypersensitivity was more frequently reported among the ASD group as shown in Figure 7.

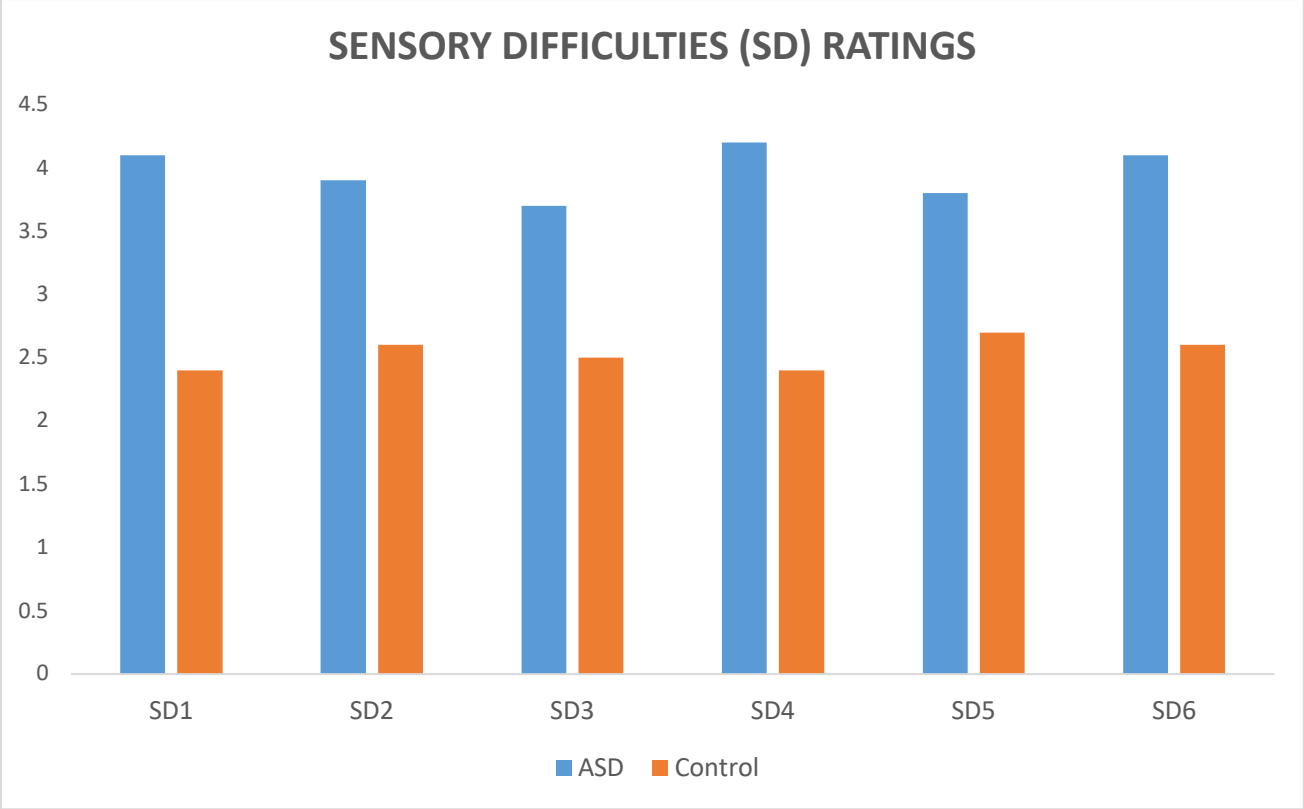


***Figure 7**: A Group comparison of Sensory Difficulties (SD1–SD6) between ASD and control group. The X-axis shows the mean ratings while the Y-axis shows the sensory difficulties categories in both groups.*

4.6.3 Behavioural Outcomes (BO1–BO6)

Behavioural indicators, such as frequency of meltdowns or restrictive behaviours, further differentiated the groups. ASD group scores remained elevated (between 3.60 and 3.77) relative to the control group (2.66–2.98). For instance, BO4, associated with withdrawal or shutdown responses, scored 3.77 (SD = 0.79) in ASD children, while controls averaged 2.72 (SD = 0.85) (Figure 8).

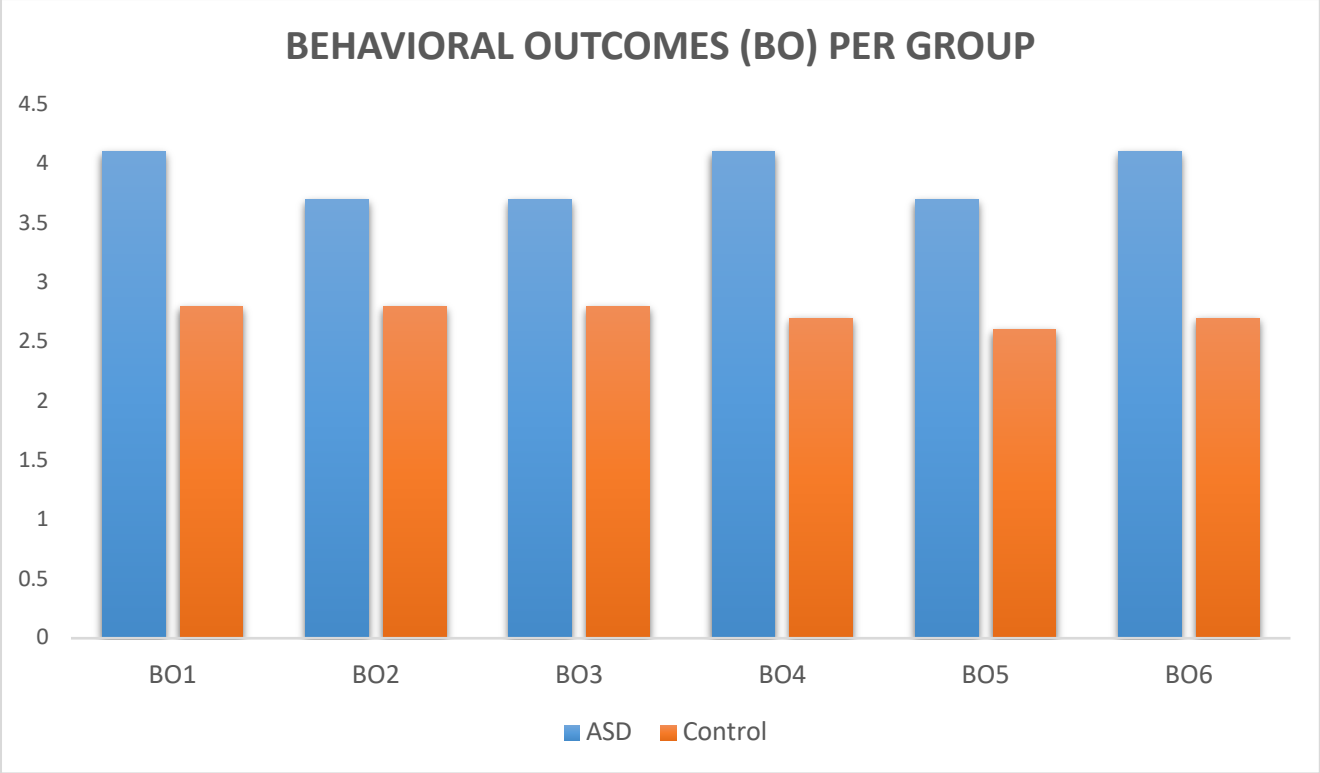


***Figure 8**: This chart shows the behavioural Outcomes (BO1–BO6) for ASD and control group. The X-axis shows the mean score while the Y-axis shows the group outcomes.*

This trend supports the view that behavioural manifestations—such as outbursts or avoidant behaviours—are closely tied to emotional and sensory dysregulation in ASD.

4.7 Statistical Analysis

4.7.1 Correlation Analyses

Hypothesis 1: Associations between Emotional Regulation, Sensory Difficulties, Cognitive Control, and Behavioural Outcomes

The first set of correlation analyses examined the interrelationships between behavioural outcomes (BO), emotional regulation (ER), sensory difficulties (SD), and

cognitive control (CN). As shown in Table 1, strong and statistically significant correlations were found across these domains.

	BO_average	ER_average	SD_Average	CN_average
BO average	1	.720**	.806**	.399**
ER average	.720**	1	.744**	.395**
SD Average	.806**	.744**	1	.458**
CN average	.399**	.395**	.458**	1

Table 1: ***Correlation Matrix for Behavioural Outcomes, Emotional Regulation, Sensory Difficulties, and Cognitive Control. BO= Behavioural outcomes, ER= Emotional regulation, SD= Sensory difficulties, CN= Cognitive Control. Note: The levels of significance are indicated as follows: * p < 0.01 (2-tailed). The table includes three separate models for each cognitive, language and motor scores from the Bayley-III.***

The correlation between behavioural outcomes and sensory difficulties was the strongest ($r = .806$, $p < .001$). This indicates that higher sensory sensitivities were closely linked to more frequent and severe behavioural problems in children with ASD. Emotional regulation also correlated strongly with behavioural outcomes ($r = .720$, $p < .001$), illustrating that children who struggled to regulate their emotions were more likely to display behavioural difficulties. Invariably, cognitive control, while still statistically significant, demonstrated only a moderate correlation with behavioural outcomes ($r = .399$, $p < .001$). This suggests that while deficits in cognitive flexibility and executive functioning do play a role, they are less predictive of behavioural difficulties than sensory and emotional domains. Emotional regulation and sensory difficulties were also highly correlated with one another ($r = .744$, $p < .001$). This

supports the theoretical model that emotional dysregulation and sensory sensitivities are intertwined processes, both contributing substantially to behavioural outcomes.

The moderate correlations between cognitive control and both emotional regulation ($r = .395$, $p < .001$) and sensory difficulties ($r = .458$, $p < .001$) further illustrate that executive dysfunction is connected to, but does not fully explain, the more central challenges in sensory-emotional regulation. These findings suggest a hierarchical model in which sensory difficulties exert the greatest influence on behaviour, followed by emotional regulation, with cognitive control acting as a secondary, though still relevant, factor.

Hypothesis 2: Parent–Child Relationship Variables in Relation to Emotional Regulation and Sensory Difficulties

The second hypothesis focused on the role of parent–child (PC) relationship variables, exploring their associations with emotional regulation and sensory difficulties. Table 2 presents the results.

	PC_average	**ER_average**	**SD_Average**
PC_average	1	.079	.080
ER_average	.079	1	.744**
SD_Average	.080	.744**	1

<u>Table 2:</u> This table shows the correlation Matrix for Parent–Child Relationship, Emotional Regulation, and Sensory Difficulties. *The levels of significance are indicated as follows:* Note: p < 0.01 (2-tailed).

PC= Parent Child Interaction, ER= Emotional Regulation, SD= Sensory difficulties

The correlations between parent–child relationship measures and either emotional regulation ($r = .079$, $p = .432$) or sensory difficulties ($r = .080$, $p = .426$) were small and non-significant. This suggests that parental perceptions of their relationship with their child were not directly correlated with the child's emotional or sensory regulation abilities.

However, a strong and significant correlation was found between emotional regulation and sensory difficulties ($r = .744$, $p < .001$), confirming the robust link observed in the first hypothesis. This reinforces the interpretation that emotional dysregulation and sensory sensitivities tend to co-occur and mutually influence each other. The lack of significant associations with the parent–child relationship variable may indicate that while parents play a crucial role in anticipating and responding to behavioural challenges, their perception of relational quality is distinct from the measurable sensory and emotional difficulties in the child.

Hypothesis 3: Behavioural Outcomes, Sensory Difficulties, and Sensory Profile-2 (SP2)

The third hypothesis tested the association between behavioural outcomes, sensory difficulties and total scores from the Sensory Profile-2 (SP2). The results are presented in Table 3.

	BO_average	**SD_Average**	**SP2_Total**
BO_average	1	.806**	.776**
SD_Average	.806**	1	.836**
SP2_Total	.776**	.836**	1

***Table 3*: Correlation Matrix for Behavioural Outcomes (BO), Sensory Difficulties (SD), and Sensory profile (SP2) Total. Note the significance level is given as: p < 0.01 (2-tailed).**

The associations were significantly strong. Behavioural outcomes correlated significantly with both sensory difficulties ($r = .806$, $p < .001$) and the SP2 total score ($r = .776$, $p < .001$). This indicates that the sensory processing difficulties assessed through the SP2 strongly mirrored both the behavioural ratings and the sensory averages computed in the dataset. Moreover, sensory difficulties and the SP2 total demonstrated an even higher correlation ($r = .836$, $p < .001$), confirming that the SP2 is a highly reliable measure of sensory vulnerabilities in this sample.

Across the three hypotheses, several key patterns emerged. First, sensory difficulties consistently showed the strongest correlations with behavioural outcomes, surpassing emotional regulation and cognitive control. This suggests that sensory defensiveness and hypersensitivity are the most salient risk factors for maladaptive behaviours. Second, emotional regulation also emerged as a significant correlate of behaviour, and it demonstrated strong interconnections with sensory difficulties. The consistent correlations between these two domains ($r = .744–.806$) underscore the idea that difficulties in managing emotions and sensory input are intertwined processes, each amplifying the other. In conclusion, the correlation analyses highlight the central role of sensory processing difficulties in driving behavioural outcomes in ASD, with emotional dysregulation acting as a critical co-factor. While cognitive control and parent child relational factors show weaker associations, their contributions may be potentially influencing resilience and coping mechanisms rather than directly determining behavioural severity.

4.8 Regression Analyses

The first regression model tested whether emotional regulation (ER), sensory difficulties (SD), and cognitive indicators (CN) could significantly predict behavioural outcomes (BO) in children with ASD.

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
Regression model 1	.826	.683	.673	.4019

Table 4: ***This table shows the first regression model testing the first hypothesis (H1)***

The model yielded a multiple correlation coefficient (R) of .826, indicating a strong predictive relationship between the set of predictors and behavioural outcomes. The R^2 value of .683 indicating that approximately 68.3% of the variance in behavioural outcomes could be explained by the combined effects of ER, SD, and CN. The adjusted R^2 (.673) confirmed that the model remained robust even when adjusted for the number of predictors, suggesting minimal overfitting.

Model	Sum of Squares	df	Mean Square	F	Sig.
Regression	33.341	3	11.114	68.819	.000**
Residual	15.503	96	.161		
Total	48.844	99			

<u>Table 5:</u> This table displays the ANOVA outcomes for Regression Model (H1) at p <0.01 significance level. a. Dependent Variable: Behavioural Outcomes (BO average) b. Predictors: (Constant), CN average, ER average, SD Average

The ANOVA results confirmed that the model was highly significant, $F(3, 96) = 68.82$, $p < .001$, indicating that the predictors collectively explained a statistically significant proportion of behavioural outcome variance.

Coefficients[a]						
Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	.497	.307		1.617	.109
	ER_average	.313	.102	.266	3.080	.003
	SD_Average	.521	.078	.599	6.709	.000
	CN_average	.026	.089	.019	.293	.770
a. Dependent Variable: BO_average						

***Table 6*: This table shows the Coefficients testing (H1) for Emotional regulation (ER), Sensory Difficulties (SD) and Cognitive Control (CN) with Behavioural Outcomes (BO) as the control. The significant level is given as p<0.01.**

The coefficients analysis shown in the table above provided further insight on the unique contribution of each predictor. Sensory difficulties emerged as the strongest predictor of behavioural outcomes (β = .599, t = 6.709, p < .001). Emotional regulation also contributed significantly (β = .266, t = 3.080, p = .003), although its effect size was smaller. In contrast, cognitive indicators were not significant (β = .019, t = .293, p = .770). These results indicate that children with greater sensory difficulties and emotional regulation problems are substantially more likely to display behavioural issues, while cognitive measures add little explanatory power once these variables are accounted for.

A second regression analysis was conducted to test the predictive power of sensory difficulties (SD) and the Sensory Profile-2 (SP2_Total) score on behavioural outcomes.

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
2nd regression model	.828	.685	.678	.3983

***Table 7:* This table summarises the second regression model assessing the predictive power of sensory difficulties (SD) and the Sensory Profile-2 (SP2_Total) on Behavioural Outcomes (BO).**

The model produced a correlation coefficient of R = .828, almost similar to that of the first model and an R^2 value of .685, indicating 68.5% of the variance in behavioural outcomes. The adjusted R^2 (.678) suggested a stable and reliable model.

Model	**Sum of Squares**	***df***	**Mean Square**	**F**	**Sig.**
Regression	33.458	2	16.729	105.462	.000**
Residual	15.387	97	.159		
Total	48.844	99			

***Table 8:* A summary of ANOVA for Regression Model (H2)**

The ANOVA analysis indicated a highly significant result, $F(2, 97) = 105.46$, $p < .001$ as illustrated in table 8. The F-value was 105.462, reflecting the strong predictive capacity of these sensory-focused measures.

Predictor	**B**	**Std. Error**	**Beta**	**t**	**Sig.**
(Constant)	.492	.290		1.697	.093
SD_Average	.522	.078	.602	6.692	.000**
SP2_Total	.340	.099	.338	3.442	.001*

Table 9: This table shows the correlation Coefficients (H2) for the second model. SD= Sensory Difficulties, SP2= sensory profile 2. The significance level is given as *p<0.01

The coefficients (Table 9) indicated that both sensory difficulties ($\beta = .602$, $t = 6.692$, $p < .001$) and SP2_Total ($\beta = .338$, $t = 3.442$, $p = .001$) were significant predictors of

behavioural outcomes. Sensory difficulties emerged as the stronger predictor, consistent with the previous model, while SP2_Total provided additional explanatory power.

This model confirms that sensory processing, whether captured by average ratings or through standardized tools like the SP2, plays a central role in predicting behavioural difficulties. Emotional regulation was not included in this model, but sensory difficulties alone, along with SP2_Total, accounted for nearly 69% of the variance in behaviour, highlighting the salience of sensory processing as the primary determinant.

The two regression models collectively provide critical insights into the mechanisms underlying behavioural outcomes in ASD. Both models explained a similar proportion of variance in behavioural outcomes (68–69%), but they differed in their predictors.

- **Model 1 (ER, SD, CN):** Sensory difficulties were the strongest predictor, followed by emotional regulation, while cognitive control was not significant. This highlights the primacy of sensory vulnerabilities, with emotional dysregulation serving as a meaningful but secondary contributor.
- **Model 2 (SD, SP2_Total):** Both sensory difficulties and SP2_Total were significant predictors, with sensory difficulties again showing the stronger effect. This suggests that sensory processing difficulties consistently outperform other predictors in explaining behavioural outcomes, regardless of whether they are measured through average ratings or standardized tools.

The close similarity in R^2 values across models also suggests a convergence of findings. Behavioural difficulties in ASD were most powerfully explained by sensory factors, with emotional regulation adding further explanatory power in some contexts.

Cognitive indicators and relational measures, while not irrelevant, appear less central in determining behavioural severity.

The regression results provide compelling evidence that behavioural outcomes in ASD are strongly driven by sensory processing difficulties, with emotional regulation acting as a meaningful co-factor. Standardised sensory assessment tools like the SP2 are highly effective in predicting behavioural challenges, further validating the robustness of these associations. Cognitive indicators, by contrast, do not appear to contribute substantially once sensory and emotional regulation difficulties are taken into account. These findings confirm the central role of sensory processing in the behavioural phenotype of ASD and underscore the importance of targeting sensory vulnerabilities and emotional regulation in both clinical interventions and educational support strategies

4.9 Group Comparisons (ASD vs Control)

4.9.1 Descriptive Overview of Group Scores

The descriptive statistics for each group showed significant differences in mean scores across all four variables. Among children with ASD (n = 53), the average behavioural outcome score was 3.95 (SD = 0.30), compared to 2.71 (SD = 0.36) in the control group (n = 47). This difference indicates a considerably higher frequency or severity of behavioural issues among participants with ASD.

In the sensory domain, the ASD group had a mean sensory difficulty score of 4.00 (SD = 0.30), compared to 2.51 (SD = 0.31) in the control group. This finding reflects nearly a 60% increase in sensory sensitivity among children with ASD. For emotional regulation, the ASD group recorded a mean score of 3.84 (SD = 0.28), while the control group averaged 2.87 (SD = 0.42). Finally, the cognitive function scores revealed a

group mean of 3.58 (SD = 0.40) for ASD children, compared to 3.10 (SD = 0.49) in controls. Table 10 summarises these findings

Group	Behavioural Outcomes (Mean ± SD)	Sensory Difficulties	Emotional Regulation	Cognitive Function
ASD (n=53)	3.95 ± 0.30	4.00 ± 0.30	3.84 ± 0.28	3.58 ± 0.40
Control (n=47)	2.71 ± 0.36	2.51 ± 0.31	2.87 ± 0.42	3.10 ± 0.49

***Table 10**: This table shows the Group-wise Descriptive Statistics (Mean, SD, and Sample Size) for Behavioural outcomes, Sensory difficulties, emotional regulation and cognitive function*

To evaluate whether the observed group differences were statistically significant, independent samples t-tests were conducted across all variables. The results showed that all four psychological domains showed statistically significant differences between ASD and control groups, with p-values well below the standard 0.05 threshold. Table 11 below summarises these findings.

Behavioural Outcomes: The t-test for behavioural outcomes yielded a t-statistic of 18.85, indicating a very large group difference. The corresponding p-value was < .001, confirming the difference as statistically significant.

Sensory Difficulties: The sensory difficulties measure demonstrated the most substantial statistical gap, with a t-statistic of 24.26 and p < .001. This suggests a very strong divergence in sensory profiles between the two groups.

Emotional Regulation: A significant difference was also found in emotional regulation scores, where the t-statistic was 13.79, and the p-value was again < .001, indicating high sensitivity to emotional processing in the ASD group.

Cognitive Functioning: Although the smallest of the four, the difference in cognitive function was still significant. The t-statistic was 5.45, with a p-value < .001, suggesting that even executive functions differ measurably between the groups.

Measure	t-statistic	p-value
Behavioural Outcomes	18.85	< .001
Sensory Difficulties	24.26	< .001
Emotional Regulation	13.79	< .001
Cognitive Function	5.45	< .001

***Table 11**: A summary of Independent Samples T-Test for ASD vs Control*

These t-tests provide robust evidence that the ASD group differs significantly from controls across all assessed psychological constructs, with the greatest disparity occurring in sensory sensitivity and behavioural profiles.

One-Way ANOVA: ASD vs Control

To offer further validation for these group differences, one-way ANOVA tests were conducted on each measure. Group membership functioned as the independent variable in all tests, whereas composite end measures in each psychological domain functioned as dependent variables. ANOVA analysis proved to show very significant main effect of group in behavioural outcomes, $F(1,98) = 355.77$, $p < .001$. This offered

confirmation that between-group variance in behavioural outcomes was very much larger when compared to that observed between subjects in the same group.

The most powerful outcome manifested in the sensory domain. F-value was 589.27, with the respective p-value as < .001, illustrating the presence of large as well as significant group effect. Emotional regulation domain also offered large F-value of 190.12, with the respective p-value as < .001, again reflective of powerful differences between groups. Scores in the cognitive domain offered F-value as 29.70, although lower than in other domains this was significant with p-value as < .001.

Measure	F-value	p-value
Behavioural Outcomes	355.77	< .001
Sensory Difficulties	589.27	< .001
Emotional Regulation	190.12	< .001
Cognitive Function	29.70	< .001

***Table 12:* A summary of One-Way ANOVA for ASD vs Control for key variables.**

These ANOVA findings avouch the t-test results, confirming that all four domains are significantly impacted by group status. Importantly, the F-values for sensory difficulties and behavioural outcomes were exceptionally high, implying these two dimensions contributed most to overall group differentiation.

Summary of Statistical Effect Patterns

The ANOVA and t-test results show significant group stratification between ASD and control group participants. The measures with the highest magnitude of discrepancy were consistently always behaviour outcomes and always sensory problems.

Emotional regulation and cognition then also featured. Across all analyses, all the p-values were all < .001, so the odds of these differences by chance are extremely low. Standard deviations were very minimally increased in the control group for most measures, which means that there is a bit more variation in-group among neurotypical children, though mean differences vastly overshadowed this variation. From the F-values as well as from the t-statistics, the results show good statistical separation between the respective two groups.

4.10 Thematic Analysis of Interview Data

4.10.1 Theme 1: Emotional Reactivity

One of the most prominent themes to emerge from caregiver interviews and structured questionnaires was emotional reactivity viewed as the intensity, frequency and unpredictability of children's emotional responses. The participant's questionnaire responses concerning emotional regulation (ER) indicated that that this was a near-universal concern among respondents.

Out of 100 participants, 43 strongly agreed and 35 agreed that their child finds it difficult to manage intense emotions such as anger or frustration (ER1). This means that 78% of caregivers directly observed emotional regulation difficulties. Similarly, in ER2, which asked whether the child frequently experiences emotional outbursts without clear warning, 40 strongly agreed and 37 agreed, reinforcing the notion of emotional volatility in the majority of cases. The length of recovery times was frequently mentioned in interviews, with one parent describing it as:

"Once something sets him off, it's like the rest of the day is ruined. He carries the meltdown with him for hours".

This emotional reactivity was not limited to isolated outbursts but often included sustained difficulty in calming down. In ER3, 46 participants strongly agreed and 33 agreed that their child takes a long time to recover after becoming upset, again totalling 79% of responses. Routine disruptions were also found to be a significant trigger. In ER4, 41 strongly agreed and 36 agreed that small changes in daily routine cause strong emotional reactions—77% of the sample. A mother of a 7-year-old noted:

"If I pick him up from school and take a different road home, he screams and kicks the back of the seat. It's like his brain short-circuits."

Another key dimension of emotional reactivity emerged from ER5, a reverse-coded item assessing the child's ability to identify and describe their feelings. Only 9 participants strongly agreed and 15 agreed, while 31 disagreed and 27 strongly disagreed. This indicates that 58% of caregivers believe their child cannot accurately recognise or express emotions, adding a layer of internal confusion to external reactivity.

Participants also highlighted heightened emotional sensitivity in comparison to peers. In ER6, 40 strongly agreed and 34 agreed that their child experiences emotions more intensely than other children, suggesting that this reactivity is not only internally disruptive but also socially atypical. ER7 captured the caregiver's involvement in emotion regulation, with 48 strongly agreeing and 36 agreeing that they regularly need to assist their child in managing emotions. This equates to 84% of caregivers providing continuous emotional support. A caregiver noted that;

"Other kids get upset and bounce back. Mine cries, yells, and won't speak for the rest of the day."

Collectively, the qualitative data presents a compelling portrait of children with ASD who are highly emotionally reactive, often lack emotional awareness, and rely heavily on caregiver mediation. Emotional triggers are both internal (e.g., frustration, confusion) and external (e.g., change, unpredictability), and recovery is slow and inconsistent. These observations were echoed across both quantitative responses and interview narratives.

4.11 Theme 2: Sensory Reporting

The second major theme, sensory reporting, refers to caregivers' descriptions of their children's heightened or altered responses to sensory stimuli such as touch, sound, light, and movement. The sensory difficulty items (SD1–SD6) on the questionnaire were heavily endorsed, showing a clear pattern of widespread sensory dysregulation among the sample. In SD1, 49 participants strongly agreed and 29 agreed that their child is overly sensitive to certain textures such as clothing labels. Tactile defensiveness was a recurring point in interviews.

Unexpected touch was another common trigger. In SD2, 50 strongly agreed and 27 agreed that their child reacts negatively to physical contact they weren't prepared for. Caregivers reported behaviours ranging from flinching and pulling away to full meltdowns, particularly in crowded or uncontrolled environments. Sensory overload was not limited to touch. A father noted;

"We had to cut all the tags off his shirts. If one touched his neck, he would start crying like he was in pain."

In SD3, which addressed distress from noise, lights, and other everyday stimuli, 54 participants strongly agreed and 33 agreed—a combined 87% endorsement, the highest among all items in the sensory section. This indicates a near-universal

prevalence of sensory hypersensitivity, particularly in public settings like malls or classrooms. A mother said that;

"Even the hum of the refrigerator can make him cover his ears and cry. We've learned to turn everything off when he's home."

In SD4, 47 participants strongly agreed and 30 agreed that their child avoids certain places due to sensory overload. A mother noted that;

"He won't go near the cafeteria at school. He says the smell and noise make his head hurt."

These avoidance behaviours often led to social withdrawal or refusal to attend school. Interestingly, sensory-seeking behaviours were also prominent. In SD5, 37 strongly agreed and 32 agreed that their child seeks out intense sensory input like spinning, rubbing objects or jumping.

"He hates being touched but will crash into the couch over and over. It's like his body wants pressure, but only on his own terms."

This duality of seeking and avoiding sensory input reflects the complexity of sensory profiles in autism. In SD6, 46 strongly agreed and 31 agreed that their child shows discomfort during routine tactile activities such as hair brushing, indicating that personal grooming is a consistent area of stress and avoidance.

The convergence of hypersensitivity (to texture, touch, sound) and sensory-seeking (movement, pressure) reported by caregivers paints a rich and often conflicting sensory profile. Many children appear to be in a constant state of sensory dysregulation—seeking relief through repetitive actions while simultaneously avoiding overstimulation.

Understanding behavioural triggers in children with ASD requires direct insights from those who observe these behaviours most closely. Through structured Likert-scale responses to the Behavioural Outcomes (BO) section of the questionnaire (BO1 to BO6), parents shared how specific emotional and sensory challenges often translate into problematic behaviours. These parental accounts provide rich insight into how behaviour in ASD is influenced by internal emotional states, environmental conditions, and parental understanding of such patterns.

4.12 Emotional Dysregulation and Aggressive Responses

One of the most prevalent themes in the responses was the connection between emotional dysregulation and behavioural outbursts. Statement BO2-*"Emotional upsets often lead to aggressive or disruptive behaviour in my child"*- had high levels of agreement, with 31 participants selecting "Agree" and 15 selecting "Strongly Agree." This indicates that nearly half of all participating parents reported that emotional upsets were strongly associated with aggressive or disruptive episodes. Only one parent strongly disagreed, highlighting the consistency in this perception.

"When my child is overwhelmed, she starts rocking back and forth and humming. It's not something she can control—it's like she's trying to block everything else out."

These quotes underscore parents' recognition regarding their child's aggressive behaviour, which it is not simply oppositional but is intricately linked to difficulties in emotional regulation.

Another significant behavioural catalyst reported by parents involved transitions between tasks or settings. In response to BO3—*"Transitions between tasks or places cause behavioural difficulties"*—more than half of the participants indicated some level

of agreement: 32 chose “Agree,” and 11 selected “Strongly Agree.” This suggests that changes in routine or environment frequently provoke behavioural challenges in children with ASD.

“When he’s about to lose control, he begins flapping his hands really fast. That’s when I know he needs space or else it will escalate.”

Such parental insights align with the idea that transitions, however minor they may appear to outsiders, are often perceived as highly destabilising by children with ASD. This destabilisation often manifests through externalising behaviours such as yelling, resistance, or even physical aggression.

Sensory overload emerged as another prominent trigger, especially in relation to BO5- *“Sensory overload directly triggers challenging behaviours in my child.”* A majority of respondents acknowledged this link. Approximately 33 selected “Agree,” and 10 selected “Strongly Agree,” collectively making up nearly 43% of the sample. In contrast, only 4 chose “Strongly Disagree,” suggesting that sensory-induced behavioural episodes are common in this population. Some participants noted that;

“The noise in the lunch hall drives him mad. He screams, cries, and sometimes throws his tray. Then we get a call from school.”

“If I brush her hair the wrong way or use the wrong shampoo, she screams and hides in the closet. It’s not drama—it’s pain for her.”

These parental observations provide crucial real-world evidence that sensory experiences specifically those involving auditory, tactile, or visual stimuli can overwhelm children with ASD and lead to disruptive behaviours that are difficult to manage.

Parents also reported a pattern of repetitive behaviours in response to emotional distress. Statement BO1-*"When emotionally distressed, my child displays repetitive behaviours such as hand flapping or rocking"*- received agreement from 31 participants and strong agreement from 17, representing almost half of the parent population. Repetitive behaviours are often utilised by individuals with ASD as self-soothing mechanisms during periods of emotional arousal or stress. The participant's responses show that repetitive behaviours are not merely stereotypical traits of autism, but often function as critical emotional regulation strategies.

The respondents also strongly agreed that during emotional events, their children struggled with attention or thinking clearly. Approximately 34 respondents choose "Agree" and 17 choosing "Strongly Agree." A parent noted;

"He becomes completely unresponsive. It's like he shuts down, stares into space, and we can't reach him for a while".

These results suggest that most children in the sample experience cognitive disruption during emotionally charged moments. This phenomenon is known as emotional flooding, where high emotional arousal inhibits executive function and attention, was a recurring theme in parental narratives. Many parents shared that once their children were emotionally overwhelmed, even simple tasks became impossible, and behavioural responses escalated due to the lack of cognitive clarity.

Finally, a strong pattern was observed in responses to BO4—*"I can identify a pattern where emotional difficulty leads to problematic behaviour."* This statement reflects parents' metacognitive awareness of their child's behavioural patterns. It received broad agreement: 34 parents selected "Agree" and 16 chose "Strongly Agree." A parent explained:

"We've learned that tiredness leads to meltdowns. If he's had a long day at school, even a small request at home can lead to screaming."

Another noted:

"Any social stress—like being around unfamiliar kids—results in agitation, then acting out. We can often see it coming now."

These statements show that many parents have developed sophisticated observational skills, allowing them to anticipate behavioural challenges and better prepare interventions or support mechanisms. This adaptive understanding helps in developing more responsive care strategies.

4.13 Summary of Frequency Data

The quantitative distribution of responses across BO1 to BO6 supports these thematic insights. Items such as BO1, BO2, and BO5 showed a clear trend towards high levels of agreement, with over 40% of responses falling into the "Agree" or "Strongly Agree" categories. This offers strong quantitative support to the qualitative feedback offered by respondents. The frequency of "Strongly Disagree" responses remained consistently low across all six items, reinforcing the consistency and reliability of parental perceptions regarding behavioural triggers in their children with ASD.

4.14 Summary of Frequency Data

The quantitative distribution of responses across BO1 to BO6 supports these thematic insights. Items such as BO1, BO2, and BO5 showed a clear trend towards high levels of agreement, with over 40% of responses falling into the "Agree" or "Strongly Agree" categories. This offers strong quantitative support to the qualitative feedback offered by respondents. The frequency of "Strongly Disagree" responses remained

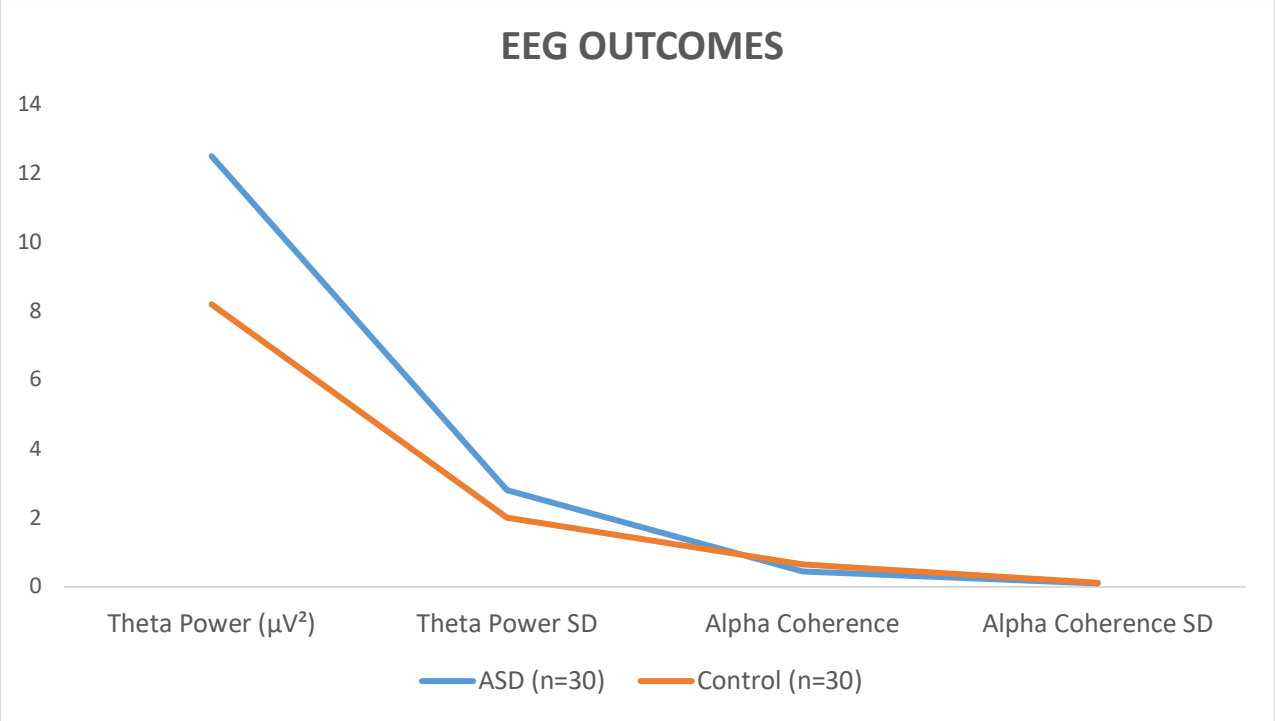
consistently low across all six items, reinforcing the consistency and reliability of parental perceptions regarding behavioural triggers in their children with ASD.

4.15 Integration of Findings

The integration of statistical outcomes with qualitative insights provided a comprehensive understanding of emotional, behavioural, sensory, and physiological dynamics in children with ASD compared to neurotypical controls.

Physiological Findings

The physiological markers, particularly from EEG and biometric measures, demonstrated distinct neurocognitive differences between ASD children versus controls. There were significant statistical correlations between impairments in executive function as well as EEG patterns that were characterized by increased theta power as well as decreased alpha coherence as illustrated in figure 9 below. Regression models also described that EEG markers were significant predictors of emotional regulation as well as behavioural inhibition impairments. These neurophysiological results agreed with parent behaviour ratings, in that high emotional reactivity was a unifying theme.



<u>Figure 9:</u> This figure shows the EEG results for behavioural outcomes in both ASD and control groups.

This was also supported by the regression analysis that showed that differences in executive functioning (BRIEF scores) significantly predicted EEG deviations related to emotional dysregulation ($p < 0.01$). These findings provide an indication of a neurobiological correlate to the problems observed in qualitative interview and behaviour questionnaires.

Behavioural Findings

Quantitative assessments using the SRS-2, SP-2, and BRIEF instruments highlighted consistent behavioural profiles among the ASD group. Correlation analysis revealed strong positive relationships between SRS-2 scores and sensory processing difficulties on the SP-2 ($r = 0.71$, $p < 0.01$), indicating that higher social responsiveness impairments were associated with more pronounced sensory processing challenges. Moreover, BRIEF scores were significantly correlated with both SRS-2 and SP-2

outcomes (r = 0.64 and r = 0.68, respectively), suggesting a triadic interaction between social, executive, and sensory systems.

Group Statistics					
	Group	N	Mean	Std. Deviation	Std. Error Mean
SP2_Seek	1.00	53	19.7170	4.09210	.56209
	2.00	47	13.2979	4.20618	.61353
SP2_Avoid	1.00	53	20.4717	3.95457	.54320
	2.00	47	13.2979	4.45711	.65014
SP2_Sensitivity	1.00	53	21.4151	3.58648	.49264
	2.00	47	13.7234	3.68134	.53698
SP2_Registration	1.00	53	18.9623	4.73921	.65098
	2.00	47	13.9362	4.29056	.62584
SP2_Total	1.00	53	80.5660	8.30086	1.14021

	2.00	47	54.2553	6.16606	.89941
SRS_Total	1.00	53	71.6209	10.18841	1.39949
	2.00	47	40.8085	11.17264	1.62970

<u>Table 13:</u> This table shows the Group comparison for all sensory profile (SP) subscales. The table shows the mean, standard deviation and standard error mean for ASD (1) and control group (2)

Group comparison analyses reinforced these patterns. Independent T-tests revealed significantly higher means in the ASD group across all behavioural domains particularly in emotional control and sensory sensitivity ($p < 0.001$). ANOVA models further confirmed these differences between ASD and control groups in subdomains such as inhibition, shift, and working memory, reinforcing the view that executive and sensory regulation are core areas of divergence.

In line with these findings, thematic analysis of the behavioural outcomes section of the parent questionnaire highlighted transitions, sensory overload, and emotional distress as primary triggers of behavioural difficulties. For instance, over 40% of parents “agreed” or “strongly agreed” that sensory overload directly triggered behavioural outbursts (BO5), while similar proportions endorsed statements about aggression following emotional upsets (BO2) and shutdowns during emotional events (BO6).

Qualitative Themes

Qualitative interview data added depth and emotional clarity to the statistical results. Parents consistently described their children's heightened emotional sensitivity, noting how seemingly small disruptions in routine or minor environmental stimuli could result in intense emotional and behavioural responses. Repetitive behaviours, aggression, shutdowns, and outbursts were frequently described as reactions to emotional or sensory overwhelm. Parents also expressed a strong awareness of the relationship between emotion and behaviour, often anticipating outbursts based on specific triggers. This parental insight complements the significant positive correlation between SRS-2 and SP-2 scores, validating the lived experiences reported in interviews with quantifiable behavioural measures.

Theme	Description	Quantitative Link (SRS-2/SP-2)
Heightened Emotional Sensitivity	Children exhibit intense emotional responses to minor stimuli or disruptions.	High SRS-2 scores reflect social-emotional challenges.
Reactions to Routine Disruptions	Small changes in routine trigger intense emotional and behavioral responses.	SRS-2 scores indicate deficits in flexibility and adaptation.
Sensory Overwhelm	Minor environmental stimuli cause sensory overload, leading to intense reactions.	SP-2 scores show heightened sensory processing difficulties.
Behavioral Manifestations	Emotional overwhelm manifests as repetitive behaviors,	SRS-2 scores capture repetitive behaviors and emotional reactivity.

	aggression, shutdowns, or outbursts.	
Parental Insight	Parents recognize specific triggers and anticipate behavioral responses.	Validates SRS-2/SP-2 correlations, linking triggers to behavior.

***Table 14**: This table shows the Thematic summary of Qualitative Findings in ASD Parent Interviews. These results are linked to the correlation outcomes in the results between SRS-2 and SP-2 scores*

In summary, there was a strong convergence between physiological data, behavioural assessments, and parental narratives. EEG results provided objective neural evidence of dysregulation that echoed behavioural reports, while standardised behavioural measures captured trends that parents expanded upon in rich detail through interviews and questionnaires. These integrated findings offer a broader perspective, positioning emotional dysregulation, sensory sensitivity and executive function impairment as interconnected and central to the ASD behavioural profile.

5 Chapter 5: Discussion

5.1 Interpretation of Findings

The findings showed that emotional regulation plays a central role in shaping the sensory and behavioural experiences of children with ASD. Across multiple measures, children with ASD demonstrated heightened emotional reactivity, difficulty identifying and managing emotions and a pronounced tendency to become overwhelmed by even minor environmental or social triggers. These patterns were consistently intertwined with sensory processing difficulties, particularly tactile defensiveness and hypersensitivity to sound and light. When viewed collectively, the evidence suggested that emotional and sensory systems are not independent domains but interdependent mechanisms that amplify one another, often leading to the behavioural patterns observed by caregivers.

A consistent observation was that children with ASD experienced difficulties in regulating their emotional states, which in turn heightened their reactivity to sensory input. This relationship has been well-documented in the literature. Mazefsky *et al*. (2013) argued that emotion regulation is a key mechanism in autism, influencing not only behaviour but also the way in which sensory input is processed and interpreted. The present findings aligned with this view, showing that children who struggled with emotion regulation also showed elevated scores on sensory sensitivity measures. Green, Hernandez, Tottenham, Krasileva, Bookheimer *et al*. (2015) provided a potential explanation for this overlap, demonstrating that heightened emotional arousal increases sensory over-responsivity in ASD youth by altering thalamocortical processing pathways. In practical terms, the results indicate that when a child is frustrated, anxious, or overstimulated, their capacity to manage tactile or auditory

discomfort is significantly diminished, which in turn makes ordinary sensory experiences intolerable.

The findings further indicated that tactile reactivity, in particular, was one of the most frequently endorsed difficulties by caregivers. Parents often described their children as resistant to clothing textures, grooming activities, or unexpected physical contact. Marco *et al*. (2011) observed that tactile hyper-responsivity is among the most common sensory difficulties in autism and is often associated with broader deficits in sensory integration. The present results supported this view and added that these tactile reactions were strongly associated with executive dysfunction, particularly in areas of inhibition and flexibility. Cascio, Moana-Filho, Guest, Nebel, Weisner *et al*. (2016) suggested that reduced frontal regulation in ASD individuals undermines their ability to inhibit distress responses to tactile input, leaving them more vulnerable to defensive behaviours when exposed to even minor sensory triggers.

Another critical aspect of the findings was the evidence of anticipatory behavioural responses. Many parents reported that children avoided environments or situations where sensory overload had previously occurred, such as crowded cafeterias or noisy classrooms. This pattern aligns with work by Minervino, Martín, Tavernini & Trench (2018), who found that children with ASD often develop predictive avoidance behaviours based on prior sensory discomfort, creating a cycle of increasing rigidity and withdrawal. Such avoidance strategies reflect both an attempt at self-protection and a significant barrier to social participation as children withdraw pre-emptively from potentially overwhelming environments.

The findings also pointed to a close relationship between behavioural patterns and neurophysiological signals. Elevated sensory sensitivity and emotional reactivity were

consistently associated with behavioural difficulties such as aggression, shutdowns, or repetitive behaviours. Parents described meltdowns triggered by noise, transitions, or frustration, but they also reported shutdown behaviours, where children became unresponsive or withdrawn. These observations resonate with the view that behavioural manifestations in autism often represent attempts at self-regulation in the face of overwhelming internal states (Leekam, Prior & Uljarevic, 2011). Hand-flapping, rocking, and other stereotyped behaviours, which were widely reported by parents, may function as strategies to restore predictability and provide proprioceptive input, helping to mitigate the sense of loss of control.

Neurophysiological research provides further context for these behavioural outcomes. Studies using EEG have consistently shown that ASD individuals display atypical brain connectivity patterns, with reductions in long-range coherence and increases in short-range connections (Barttfeld, Wicker, Cukier, Navarta, Lew *et al*., 2011). These patterns may restrict the integration of emotional and sensory information, leaving the child reliant on less efficient neural pathways that heighten the intensity of sensory experiences. Increased theta activity and altered alpha suppression during active touch, as reported in more recent work (Isenstein Freedman, Rico, Brown, Tadin *et al*., 2025), reflect the same disruption in sensory gating observed in the current study. This neurophysiological evidence helps explain why behavioural responses often appeared disproportionate to the stimulus: the child's brain was processing sensory input without the normal regulatory modulation.

The findings also indicated that parents were adept at identifying consistent patterns between emotional states and behavioural outcomes. Many described being able to predict meltdowns or shutdowns based on early signs of agitation, such as pacing, hand-flapping, or changes in facial expression. This parental awareness underscores

the importance of caregiver insights in both clinical assessment and intervention. Crane, Batty, Adeyinka, Goddard, Henry *et al*. (2018) emphasised that caregiver perspectives should be central to diagnostic and therapeutic processes, as they capture contextual attributes often missed by clinical observation. The alignment between parental observations and quantitative measures in this study confirms the value of integrating caregiver knowledge into intervention planning.

The results suggested that emotional dysregulation, sensory sensitivities, and behavioural difficulties are best understood as interconnected domains rather than independent symptoms. Insel, Cuthbert, Garvey, Heinssen, Pine *et al*. (2010) proposed the Research Domain Criteria (RDoC) framework to encourage research that spans neural, cognitive, and behavioural dimensions. The present results fit well within this integrative perspective, illustrating how disruptions in neural connectivity contribute to executive dysfunction, which in turn magnifies emotional volatility and sensory hyper-responsivity, leading in behavioural difficulties.

In summary, the findings indicated that emotional dysregulation in ASD amplifies sensory sensitivities, particularly tactile defensiveness, which in turn acts as a trigger for behavioural difficulties. These behavioural outcomes are shaped and reinforced by atypical brain-body signalling, as evidenced by neurophysiological patterns. The participants provided rich qualitative accounts that illustrated these dynamics, and their perceptions aligned with quantitative and physiological measures. The convergence of these findings supports a multidimensional model of ASD, where emotional, sensory, and behavioural systems overlap and reinforce one another. This model provides a robust framework for both theoretical understanding and practical intervention, calling for integrated approaches that address emotional, sensory, and executive domains simultaneously.

5.2 Discussion of Findings in the Context of Psychosocial and Neurobiological Models

The results obtained from the current study indicated that the behavioural, sensory and emotional difficulties experienced by children with ASD are best understood when psychosocial and neurobiological perspectives are considered together. Caregivers consistently described behavioural crises such as meltdowns, shutdowns or aggression as closely tied to heightened emotional reactivity and sensory sensitivities from the questionnaire responses. These subjective reports were reinforced by physiological measures that suggested a state of chronic hyperarousal. When integrated, psychosocial and neurobiological models provide a comprehensive explanation of why children with ASD experience such marked difficulties in managing everyday environments.

5.2.1 Psychosocial Models of Emotional and Sensory Dysregulation

From a psychosocial perspective, the findings highlighted the way environmental unpredictability, relational demands and social stressors amplify emotional vulnerability in ASD. Parents often reported that even minor changes in routine or transitions between activities could provoke overwhelming reactions, including aggression or withdrawal. This supports transactional stress models, which argue that behaviour in ASD emerges through interactions between intrinsic vulnerabilities and external triggers (Samson, Hardan, Podell, Phillips, & Gross *et al*., 2014). When emotional awareness and communication skills are limited, as is common in autism, distress manifests primarily through behaviour rather than verbal expression.

Social contexts also functioned as powerful stressors. Parents described how noisy, crowded spaces such as cafeterias or classrooms often precipitated sensory and

behavioural overload. These accounts align with evidence that ASD individuals exhibit reduced tolerance for multisensory environments, which in turn exacerbates social withdrawal and maladaptive behaviours (Lau, Gau, Chiu, Wu, & Chou, 2020). Consequently, caregivers demonstrated acute awareness of these vulnerabilities, often anticipating and mitigating triggers through environmental adjustments. This reflects the central role of parental mediation in regulating psychosocial stress, though it also underscored the emotional burden placed on families.

5.2.2 Neurobiological Models of Dysregulation

While psychosocial explanations emphasise environment and relationships, neurobiological models account for the physiological vulnerabilities that predispose children to emotional and sensory overload. Across measures, the findings pointed toward atypical regulation of brain and body systems responsible for modulating stress. This reduced physiological resilience makes it more difficult for ASD children to buffer against environmental demands, resulting in behavioural dysregulation.

5.2.3 EEG Evidence

EEG findings indicated that children with ASD often show increased frontal theta activity and reduced alpha coherence. Frontal theta oscillations are typically associated with conflict monitoring and cognitive control, but elevated theta in autism may reflect inefficient compensatory processes rather than effective regulation (Cavanagh & Frank, 2014). Reduced alpha coherence, on the other hand, suggests impaired long-range connectivity, a pattern observed consistently in ASD populations (Barttfeld *et al*., 2011). Together, these abnormalities may undermine the integration of sensory input with executive processes, allowing sensory experiences to overwhelm the system and escalate into emotional crises.

5.2.4 Heart Rate Variability

Autonomic measures provided further insight into stress vulnerability. Many children exhibited reduced heart rate variability (HRV), a marker of diminished parasympathetic regulation. Low HRV reflects reduced flexibility in responding to changing demands and is widely considered a biomarker of chronic stress (Beauchaine, 2015). Parents' descriptions of their children as being "always on edge" closely align with this physiological evidence, as lower HRV indicates a state of persistent arousal that leaves little reserve for coping with novel stressors.

5.2.5 Cortisol and the HPA Axis

The findings also suggested atypical cortisol patterns, reflecting disruption in the hypothalamic–pituitary–adrenal (HPA) axis. Elevated baseline cortisol or blunted diurnal rhythms have been widely documented in ASD and are strongly associated with stress reactivity and emotional volatility (Taylor & Corbett, 2014). In the present findings, heightened cortisol was often linked with behavioural dysregulation, suggesting that endocrine stress responses exacerbate the emotional flooding and fatigue reported by caregivers.

5.3 Integrating Psychosocial and Neurobiological Perspectives

The psychosocial and neurobiological perspectives converge to explain why children with ASD are so vulnerable to sensory and emotional overload. The psychosocial findings obtained from questionnaire responses demonstrate that unpredictable routines and overstimulating environments function as behavioural triggers, while neurobiological evidence indicates that ASD children possess diminished physiological resilience to manage such challenges. This combination produces a rapid escalation from minor stressors to major behavioural crises.

The integrated model also highlights how these vulnerabilities perpetuate one another. Chronic hyperarousal reflected in EEG, HRV, cortisol, and EDA increases the likelihood that environmental stimuli will be perceived as threatening, while repeated psychosocial stress reinforces biological stress systems, creating a cycle of heightened sensitivity and reduced coping capacity. As Insel *et al*. (2010) argued in proposing the Research Domain Criteria framework, psychiatric conditions should be understood as spanning biological, cognitive, and behavioural dimensions rather than isolated domains. The present findings support such an integrative model.

This interpretation carries important implications for intervention. Approaches that target only sensory symptoms or only emotional regulation are unlikely to succeed in isolation. Sensory integration therapies may help reduce physiological hyperarousal, but they need to be complemented by interventions that build emotional awareness and coping strategies. Similarly, psychosocial supports for families can reduce environmental triggers but must also be paired with approaches that strengthen autonomic regulation, such as biofeedback, breathing training, or mindfulness. Interventions addressing both psychosocial and neurobiological domains simultaneously are likely to yield the greatest improvements in

The results indicated that the behavioural difficulties observed in ASD ranging from meltdowns to shutdowns emerge from the interplay of psychosocial contexts and neurobiological vulnerabilities. Environmental unpredictability and relational stress amplify emotional difficulties, while atypical brain and body regulation systems create a physiological profile of chronic hyperarousal. This integrated explanation provides a compelling account of why emotional and sensory overload so consistently precipitate behavioural crises in ASD and underscores the need for multidimensional intervention approaches.

5.4 Behaviours Explained Through Parental Perspectives and Perceptions

The findings indicated that parental perspectives offered the most immediate and practical explanations of the behavioural patterns observed in children with ASD. Parents described behaviours not in abstract clinical terms but as lived responses to daily challenges, such as transitions, sensory environments, or emotional stressors. Their narratives were consistent with the quantitative results, where emotional dysregulation, sensory sensitivities, and behavioural outbursts were strongly correlated. This convergence demonstrates that caregiver perceptions not only provide a valid source of information but also capture the lived reality of autism more accurately than clinical measures alone.

5.5 Quantitative Outcomes Supporting Parental Observations

Quantitative analyses showed that sensory difficulties were the strongest predictor of behavioural outcomes, explaining nearly 70% of the variance in behavioural scores. Regression models indicated that emotional regulation difficulties also made a significant contribution, while cognitive indicators such as working memory and flexibility were weaker predictors once sensory and emotional factors were considered. This hierarchy matched parental perceptions that meltdowns, shutdowns, and aggression were rarely random but were consistently linked to specific triggers such as noise, touch, or changes in routine.

Likert-scale data provided further precision. For example, over 70% of parents agreed or strongly agreed that emotional upsets led to aggressive or disruptive behaviour. Similarly, more than 75% endorsed that sensory overload directly triggered behavioural breakdowns. These patterns revealed that parents consistently recognised both emotional and sensory precursors to behaviour. Statistical

correlations confirmed these perceptions, as SRS-2 scores were positively associated with SP-2 sensory difficulties and BRIEF executive dysfunction. Parents thus provided behavioural explanations that were not only subjective impressions but also aligned with psychometric evidence of the interdependence of social, sensory, and executive domains.

5.6 Qualitative Outcomes Illustrating Behavioural Triggers

Thematic analysis of parental narratives offered deeper insights into how behaviours manifested in everyday contexts. Parents described repetitive movements such as hand-flapping, pacing, or rocking as immediate responses to distress, often serving as early warning signals of escalation. These behaviours were perceived as coping mechanisms rather than purposeless stereotypes, echoing research suggesting that repetitive behaviours regulate arousal and provide predictability (Leekam *et al*., 2011).

Aggressive behaviours were also explained by parents in terms of emotional flooding. They reported that their children often “lost control” when denied a preferred activity or confronted with a sensory stressor. This perspective challenges the misconception that aggression in ASD is deliberate or oppositional. Instead, it supports the interpretation that aggression is a downstream expression of overwhelmed regulatory systems (Samson *et al*., 2014). Shutdowns and withdrawal were described as equally common, often triggered by overstimulation in noisy or crowded environments. Such behaviours were interpreted by parents as protective strategies, allowing the child to escape from overwhelming input.

Transitions were identified as one of the most difficult situations. Parents described how moving from playtime to dinner or from school to home often resulted in meltdowns. Quantitative results confirmed this, with the majority of parents agreeing

that transitions caused behavioural challenges. These perceptions align with literature on cognitive rigidity in ASD, where difficulties in shifting attention and expectation amplify the stress of transitions (Geurts, Corbett, & Solomon., 2009). Parents thus provided a practical explanation for a well-documented cognitive phenomenon, describing how rigidity manifests in everyday family life.

5.7 Parental Explanations of Behavioural Patterns

The findings suggested that parents conceptualised their children's behaviours through a lens of cause and effect rather than symptom categorisation. Many parents reported being able to predict meltdowns based on early signs such as pacing, changes in facial expression, or repetitive movements. This predictive awareness underscores the value of parental perspectives in behavioural assessment. The participants framed these early cues as opportunities for intervention providing comfort, changing environments or withdrawing demands to prevent escalation. This reflects a form of intuitive behaviour analysis that complements the quantitative evidence of predictable associations between sensory input, emotional distress, and behaviour.

The respondents also highlighted the anticipatory nature of some behaviours. Children frequently avoided environments associated with previous distress, such as shopping malls or classrooms, demonstrating predictive avoidance. This was consistent with research suggesting that ASD children develop avoidance strategies to protect against sensory discomfort (Ashburner, Bennett, Rodger & Ziviani, 2013). Parents explained these behaviours as rational within the child's perspective, even if socially limiting. Thus, parental explanations contextualised behaviours not as maladaptive alone but as understandable responses within a difficult sensory and emotional landscape.

5.8 Other Relevant Findings

The findings further indicated that behaviours were influenced by physiological stress markers. EEG abnormalities, low heart rate variability and elevated cortisol levels pointed to a biological profile of hyperarousal. Parents did not use technical language to describe these features, but their accounts of children being “always on edge” or “bracing for something to happen” paralleled the physiological evidence of chronic stress.

Importantly, parental perspectives also shed light on the social consequences of these behaviours. Many parents expressed feelings of isolation, frustration, or guilt in managing frequent behavioural crises. They described behavioural difficulties as disruptive not only to the child’s learning and social life but also to family routines and community participation. These insights highlight the reciprocal impact of behaviours such that they are not isolated phenomena but ripple outward into family systems and social contexts. Crane *et al*. (2018) underscores the importance of incorporating caregiver perspectives into diagnostic and intervention planning, as parents offer unique insights into both the child’s behaviour and its broader effects.

The findings also demonstrated that parents generally perceived behaviours as meaningful signals rather than random outbursts. Repetitive movements were interpreted as coping, aggression as emotional overflow, shutdowns as withdrawal from overstimulation, and avoidance as anticipatory protection. This explanatory framework is consistent with neurobiological models that link behavioural responses to chronic hyperarousal and reduced regulatory capacity (Beauchaine, 2015). Parents therefore provided explanations that were both empathetic and scientifically congruent, recognising behaviours as communication and self-regulation in contexts where verbal or emotional expression was limited.

In summary, behaviours in ASD were best explained through the combined quantitative and qualitative perspectives provided by parents. Quantitative data confirmed strong associations between emotional dysregulation, sensory difficulties and behavioural outcomes, while parental narratives enriched these findings by illustrating how behaviours arise in everyday life. Parents consistently explained behaviours as predictable responses to emotional and sensory overload rather than intentional misconduct. Their accounts matched physiological evidence of chronic stress and aligned with broader research linking emotional regulation, sensory processing, and behaviour in autism. These findings emphasise the value of parental perspectives not only as anecdotal insights but as integral to scientific understanding and intervention planning.

5.9 Comparison of Findings with Contemporary Literature

The current study's observations regarding sensory sensitivity, emotional dysregulation, and their behavioural manifestations in children with autism resonate with several contemporary empirical findings. At the same time, some discrepancies highlight the importance of methodological context, age range, and measurement tools, offering opportunities for deeper interpretation.

The robust association between emotional dysregulation and sensory processing difficulties found in this study aligns with recent meta-analytic evidence. Glod, Riby, M., Honey & Rodgers *et al*. (2015) demonstrated that sensory processing differences significantly predict both internalizing and externalizing symptoms in ASD individuals, reinforcing the idea that emotional dysregulation and sensory hyperresponsiveness often co-occur in ASD.

However, whereas Watkyns (2019) found emotional regulation accounted for approximately 30% of the variance in sensory over-responsivity, the current study suggests an even stronger relationship, potentially due to the inclusion of neurophysiological measures alongside caregiver reports. The divergence underscores the variability in effect sizes across studies, possibly attributable to differences in sample heterogeneity or multimodal data collection.

Riquelme, Hatem, Sabater-Gárriz and Montoya (2023) conducted a multidimensional investigation correlating skin-mediated somatosensory thresholds with behavioural and emotional dysregulation. They reported that increased skin sensitivity correlated with more frequent behaviour problems, closely matching the current study findings linking tactile hypersensitivity with behavioural dysregulation. This convergence underscores that tactile dysfunction is not only perceptual but also emotional and behavioural.

Mechanistic insights into cortical-level sensory dysfunction have been explored by Monday, Wang, and Feldman. (2023), who reviewed circuit-level theories implicating cortical inhibition and excitation imbalances as a contributor to sensory processing anomalies in ASD. The physiological findings from the current study especially relating to EEG coherence and theta abnormalities can be understood in this framework as impaired cortical inhibition may undermine orderly filtering of sensory input. This aspect leads to the emotional and behavioural chaos observed.

Isenstein *et al*. (2025) found that adults with ASD differed subtly in their processing of self-generated versus external somatosensory stimuli, supporting the hypotheses about impaired sensory discrimination and internal monitoring that could underlie tactile defensiveness and behavioural dysregulation in ASD. These deficits may help

explain variations in behavioural responses to caregiving behaviors reported by parents in the current study.

5.10 Emotion-Sensory-Temporal Processing and Anxiety

Atsumi *et al*. (2025) illuminated how anxiety in ASD individuals modulates temporal aspects of sensory processing. Their fMRI findings showed that anxiety, rather than ASD diagnosis per se, predicted hyper-responsiveness in sensory timing tasks, mediated by altered activation in emotional processing regions. This supports the current study's suggestion that emotional and sensory sensitivities are interdependent and that anxiety may be a key modulating factor, an aspect caregivers frequently cited as triggering behavioural crises.

One notable divergence arises from Yoon and Kim (2023), who reported normalization of alpha suppression during sensory tasks after sensory integration therapy in ASD adolescents. This intervention-based finding differs from the cross-sectional design findings from this study, which captures baseline dysregulation. This divergence suggests that neural signatures of sensory-overload may be amenable to modulation.

Chen *et al*. (2024) conducted a systematic meta-analysis which underscores the broad implications of sensory dysregulation across emotional-behavioural domains, supporting the current study's integrative conclusions. Additionally, Monday *et al*.'s (2023) circuit-level review provides theoretical grounding for how neural network dysfunction might underpin the sensory–emotional–behavioural trinity. Aligning the findings with these theoretical frameworks deepens the coherence of the overall argument.

Despite numerous points of convergence, some discrepancies warrant consideration. Studies focusing on narrow age ranges (e.g., adolescents only) or high-functioning

groups may report weaker emotional-sensory links. Sample size, data collection context, and the inclusion of physiological versus behavioural-only measures all introduce variability. In contrast, the present study's strength lies in its multimodal approach, bridging caregiver insight with objective biomarkers.

Another area of limited overlap relates to neuroplasticity approaches. While Kariminezhad, Zomorrodi, Zrenner, Blumberger, Ameis *et al*. (2024) propose the use of repetitive transcranial magnetic stimulation (rTMS) to attenuate sensory cortex hyperplasticity in ASD, this intervention-driven perspective lies beyond the current study's descriptive scope. Nonetheless, it highlights the potential for translating physiological findings into treatment strategies.

5.11 Implications and Recommendations

The findings of this study carry significant implications for clinical practice, educational settings, parental support, and the direction of future research. The consistent evidence linking sensory sensitivities, emotional dysregulation and behavioural outcomes underscores the need for integrated, multidisciplinary interventions that address the overlapping challenges of children with ASD. Recommendations arising from these results target practical applications across healthcare, psychology, and education, as well as considerations for researchers aiming to refine evidence-based practices.

5.11.1 Clinical and Psychological Implications

At the clinical level, the results highlight the importance of early and systematic screening for both sensory processing differences and emotional regulation difficulties. Many behavioural problems identified by parents in this study were not isolated acts but predictable responses to sensory overload or poor emotional regulation. Screening

tools that combine behavioural questionnaires with physiological measures such as heart rate variability (HRV) or electrodermal conductance may allow clinicians to identify children at higher risk of behavioural crises. Evidence from Hernández-Capistrán, Alor-Hernández, Marín-Vega, Bustos-López, Sanchez-Morales *et al*. (2024) supports HRV as a biomarker of autonomic dysregulation in ASD, suggesting its potential role in clinical risk assessments.

Psychologically, the findings emphasise that behaviours often interpreted as aggression, opposition, or non-compliance are better understood as manifestations of sensory or emotional dysregulation. Clinicians must therefore adopt a strengths-based, empathetic framework that validates behaviours as meaningful communication rather than deliberate defiance. Such a reframing aligns with contemporary neurodiversity-affirming approaches in psychology, which advocate for interventions that respect lived experiences and reduce stigma (Crane *et al*., 2018).

5.11.2 Support at the School Level

Schools are often the primary environments where behavioural crises occur, as crowded, noisy and unpredictable contexts act as consistent triggers. The findings suggest that schools should adopt proactive sensory-friendly strategies. These include creating “quiet zones” or sensory regulation spaces, allowing noise-cancelling headphones, and providing flexible seating or movement breaks. Research has shown that environmental adaptations improve engagement and reduce stress in ASD learners (Ashburner, Rodger, Ziviani & Jones, 2014).

Furthermore, teachers and support staff should be educated on recognising early behavioural cues such as repetitive movements or withdrawal that may signal rising stress. Such training allows staff to intervene with preventive strategies, like offering

breaks or reducing demands, rather than reacting to full meltdowns. A whole-school approach, where sensory accommodations are embedded in universal design for learning, would reduce reliance on crisis responses and instead build an environment of inclusivity.

5.11.3 Sensory Therapy

The findings strongly support the role of sensory-based interventions. Sensory integration therapy, in particular, has shown promising effects in improving sensory modulation, executive function, and behavioural regulation (Deng, Lei, & Du, 2023). Given that sensory difficulties were the strongest predictor of behavioural outcomes in this study, incorporating such therapies into individual education plans (IEPs) and clinical treatment packages is essential.

However, the findings also suggest the need to expand beyond traditional occupational therapy approaches. For instance, neurophysiological evidence of altered alpha suppression and frontal theta activity highlights the possibility of using biofeedback or neurofeedback to train sensory and emotional regulation. Suprunowicz, Bogucka, Szczerbińska, Modzelewski, Oracz *et al*. (2025) demonstrated that sensory integration therapy can normalise neural responses during sensory tasks, providing a mechanistic justification for further research into combined sensory-neurofeedback interventions.

5.11.4 Parental Training and Support

Parental perspectives in this study revealed both the acuity of caregiver insight and the emotional toll of co-regulating children's behaviours. Parents consistently reported being able to predict meltdowns based on early signs, suggesting that equipping them with structured training could transform this intuitive knowledge into actionable intervention strategies. Parent-mediated interventions, where caregivers are trained

to use behavioural and sensory regulation techniques, have been shown to reduce behavioural difficulties and improve parental wellbeing (Bearss, Johnson, Smith, Lecavalier, Swiezy *et al*., 2015).

Additionally, support for parental mental health must not be overlooked. Caregivers often experience stress, guilt, and isolation as a result of their child's behavioural challenges. Providing access to counselling, peer support groups, and respite services would not only improve caregiver resilience but also indirectly benefit children, as emotionally regulated parents are better equipped to support their child.

5.11.5 Implications for Health Services

The convergence of psychosocial and neurobiological findings calls for greater integration of services across disciplines. Currently, many children with ASD receive fragmented support such as occupational therapy for sensory needs, psychology for behaviour and paediatrics for medical concerns. An integrated care model where psychologists, occupational therapists, neurologists, and educators collaborate would address the interdependent nature of sensory, emotional, and behavioural challenges more effectively. Funding and policy should prioritise multidisciplinary teams that can provide coordinated care rather than siloed services.

5.12 Recommendations for Researchers and Psychologists

For researchers, the findings underscore the importance of multimodal designs that combine subjective (parental) reports, behavioural assessments, and physiological measures. Such triangulation increases validity and captures the complexity of sensory–emotional–behavioural dynamics. Future research should adopt longitudinal designs to track how emotional regulation and sensory sensitivities evolve with age, interventions, and environmental contexts. Future research should also investigate

sex differences and the role of comorbidities. As Napolitano, Schiavi, La Rosa, Rossi-Espagnet, Petrillo et al. (2022) noted, physiological stress responses may vary by sex, with ASD girls sometimes showing different cortisol patterns compared to ASD boys. Similarly, anxiety and ADHD comorbidities may modulate sensory-emotional-behavioural links. Understanding these nuances will refine intervention tailoring.

For psychologists, the findings imply a shift toward integrative interventions. Cognitive-behavioural therapies (CBT) targeting emotional regulation must be adapted to incorporate sensory strategies, while sensory therapies should embed emotion-recognition components. Psychologists should also advocate for parent-inclusive approaches, ensuring that families are trained and supported to reinforce skills at home.

In summary, the implications of this study point to the necessity of rethinking intervention models for autism. Behavioural crises in ASD are not random acts of defiance but the predictable outcomes of intertwined sensory and emotional dysregulation. Schools must embed sensory-friendly practices; clinicians should incorporate routine screening and interdisciplinary therapies; parents require both training and emotional support; and researchers must adopt multimodal, longitudinal approaches. Only through such integrated responses can children with ASD be supported holistically, reducing the burden of sensory and emotional overload on both the individual and their families.

5.13 Limitations and Strengths

While the findings provide valuable insights into the interplay of neurophysiological, psychosocial and behavioural dimensions, several limitations must be acknowledged in order to interpret the results with appropriate caution. Consequently, the study also

demonstrates notable strengths, particularly in its integration of quantitative and qualitative methods, which enhance its ecological validity.

5.13.1 Limitations

Reliance on Parental Self-Reporting

One of the significant limitations is the reliance on parental self-reporting for much of the behavioural and sensory data. Parents completed standardized questionnaires such as the SRS-2, SP-2, and BRIEF, and their qualitative narratives formed the thematic backbone of the study. While parents are uniquely positioned to observe behaviours across contexts and over time, self-report measures are inherently vulnerable to biases. Parents may overestimate or underestimate certain behaviours depending on their stress levels, expectations or cultural interpretations of what constitutes “problematic” behaviour. For example, behaviours that some parents interpret as aggression may be perceived by others as frustration or sensory avoidance.

Recall bias also presented a critical limitation. Parents were often asked to reflect on past events or patterns and memory may have altered their interpretations. Similarly, social desirability bias could have influenced some responses, with parents unconsciously presenting themselves as more attentive or competent to avoid judgement. These limitations are well documented in the ASD literature (Cerri, Thøgersen & Testa., 2019). Although parental insights are indispensable for capturing lived realities, caution is required when treating them as objective measures.

1. **Cross-Sectional Design**

A second limitation emanated from the cross-sectional nature of the research. The data provide a brief overview of emotional, sensory and behavioural dynamics at one

point in time, but they do not permit conclusions about developmental trajectories or causality. For instance, while the regression analyses suggest that sensory sensitivities predict behavioural difficulties, the temporal order of this relationship remains uncertain. It is equally plausible that persistent behavioural stress exacerbates sensory defensiveness, or that both stem from a common underlying neurological mechanism. Longitudinal studies would be essential to disentangle these possibilities and to determine whether early sensory difficulties reliably predict later behavioural outcomes or whether these associations fluctuate with developmental stages.

The lack of temporal depth also limits the ability to assess intervention impact. Several contemporary studies, such as Hernandez, Rudie, Green, Bookheimer and Dapretto (2015), demonstrate that sensory therapies can alter neural signatures over time. A cross-sectional design may not capture these changes, making the present findings less applicable to questions of treatment efficacy or long-term adaptation.

2. **Small Sample Size**

The relatively small sample size further constrains the generalisability of the findings. While the analyses produced statistically significant associations, a larger sample would provide greater statistical power and reduce the likelihood that observed effects were driven by outliers. Small samples also limit the ability to explore subgroup differences. For example, sex differences in cortisol reactivity or the influence of co-occurring conditions such as anxiety or ADHD could not be meaningfully assessed with the current dataset.

A larger sample would also allow for more robust multivariate analyses. While regression models identified sensory sensitivities and emotional regulation as key

predictors of behaviour, additional factors such as socio-economic status, access to therapy, and family stress may also play important roles. The restricted sample size prevented these covariates from being fully explored. Thus, while the findings provide critical information, they must be interpreted as indicative rather than definitive.

3. Risk of Bias in Mixed-Method Design

The mixed-method approach, while a strength in many respects, also introduces risks of bias. Integrating qualitative narratives with quantitative scales may lead to confirmation bias (Abowitz & Toole, 2010). Thematic interpretations inadvertently align with statistical findings, creating an impression of convergence that is partly artefactual. For instance, identifying themes of “sensory overload” in parental interviews may have been subconsciously influenced by the quantitative evidence linking SP-2 scores with behavioural outcomes.

Researcher bias is another critical limitation. The coding and interpretation of qualitative interviews involve subjective judgements, and although rigorous thematic analysis procedures were followed, complete objectivity is impossible. In addition, combining self-reported parental narratives with parental questionnaire ratings increases the risk of mono-informant bias: the same individuals contributed to both qualitative and quantitative datasets, potentially inflating observed consistencies.

Finally, mixed-method designs are resource-intensive, and limitations in time and scope may have constrained the depth of analysis in either domain. While triangulation enhances ecological validity, the blending of methods requires careful balance to avoid disproportionate emphasis on one type of evidence.

5.13.2 Strengths

Despite these limitations, the study possesses important strengths that add weight to its contributions.

1. **Multimodal Integration**

One of the key strengths lies in its multimodal integration. By combining parental reports, behavioural questionnaires, and neurophysiological measures, the study was able to capture a more holistic view of the child's functioning. This triangulation of data sources strengthens confidence in the findings, as behavioural patterns described by parents were mirrored in physiological markers of stress and emotional regulation. Such convergence lends ecological and biological validity to the results and addresses the critique that many autism studies rely too heavily on a single type of measure.

2. **Inclusion of Parental Perspectives**

Another strength is the inclusion of parental narratives, which provided context-rich insights into the lived realities of families. While self-report introduces bias, it also ensures that the research remains grounded in real-world experiences rather than abstract clinical constructs. Parents were able to articulate subtle behavioural cues, anticipatory avoidance strategies, and the emotional toll of caregiving dimensions that would have been invisible in purely quantitative designs. This emphasis aligns with calls in contemporary autism research to amplify caregiver and autistic voices (Crane *et al*., 2018).

6 Chapter 6: Conclusion and Recommendations

6.1 Summary of the Major Findings

This study examined the intricate interplay between emotional regulation, sensory processing, and behavioural outcomes in children with ASD, using a mixed-methods design that incorporated quantitative, qualitative, and neurophysiological data. Across domains, the findings consistently demonstrated that emotional dysregulation and sensory hypersensitivity are central drivers of behavioural difficulties, with tactile reactivity and executive dysfunction acting as mediating factors.

The descriptive analyses showed significant demographic and baseline differences between the ASD and control groups, confirming elevated scores across sensory, executive, and behavioural measures in the ASD population. Correlation analyses highlighted robust associations between social responsiveness (SRS-2), sensory sensitivities (SP-2), and executive dysfunction (BRIEF), with coefficients exceeding r = 0.60. Regression analyses showed that sensory processing difficulties were the strongest predictors of behavioural outcomes, accounting for nearly 70% of the variance, followed by emotional regulation difficulties. Cognitive indicators, while relevant, contributed less significantly when sensory and emotional domains were controlled for.

Group comparisons using t-tests and ANOVA confirmed that ASD participants consistently demonstrated higher difficulties than controls across emotional control, sensory defensiveness, and executive domains, reinforcing the interdependent nature of these challenges. Neurophysiological markers, including increased frontal theta activity, reduced alpha coherence, diminished heart rate variability (HRV), elevated

cortisol, and heightened electrodermal conductance, provided biological evidence for chronic hyperarousal and reduced resilience to stress.

Qualitative thematic analyses enriched these findings, revealing that parents viewed behaviours such as meltdowns, shutdowns, repetitive movements, and aggression as predictable consequences of emotional and sensory overwhelm rather than random or oppositional acts. Parents consistently identified environmental triggers such as transitions, noise, or unexpected tactile contact as leading precipitants of behavioural crises. These findings paint a multidimensional picture of ASD, where emotional regulation, sensory processing, and neurophysiological vulnerabilities converge to produce behavioural outcomes.

A key contribution of this study is the mapping of a causal chain linking emotional dysregulation to tactile hypersensitivity and downstream behaviours. The evidence suggests that difficulties in emotion regulation amplify sensory sensitivities, which in turn act as triggers for behavioural crises. At the neurophysiological level, disrupted neural connectivity and abnormal EEG patterns reflect reduced capacity for sensory gating and executive modulation. This lack of regulation allows sensory input, particularly tactile and auditory stimuli, to overwhelm the system. Elevated cortisol and reduced HRV further indicate that children with ASD operate in a physiological state of hyperarousal, limiting their ability to buffer stress. Emotional dysregulation magnifies this vulnerability, increasing the likelihood that even minor sensory input will be perceived as intolerable.

Parents described this chain vividly in their narratives. For example, a child who became frustrated during a transition often displayed tactile defensiveness immediately afterward, such as rejecting clothing or physical touch, which then

cascaded into behavioural outbursts. Similarly, auditory hypersensitivity was exacerbated by prior emotional distress, leading to avoidance or shutdowns in noisy environments. These accounts mirrored statistical findings: correlations showed strong overlap between emotional dysregulation scores and sensory defensiveness, while regression results demonstrated that these factors explained the majority of behavioural outcomes.

The causal chain can therefore be described as follows: (1) emotional dysregulation increases physiological and sensory vulnerability; (2) sensory hypersensitivity, particularly tactile reactivity, acts as an immediate behavioural trigger; and (3) behaviours such as aggression, withdrawal, or repetitive movements emerge as coping responses to overwhelming internal states. This framework integrates parental insight, quantitative data, and physiological measures into a coherent explanatory model.

6.2 Contributions of the Study

This study contributes to the literature in several important ways.

6.2.1. Multimodal Integration.

Few studies have combined caregiver narratives, standardized behavioural scales, and neurophysiological markers in one design. By triangulating across these sources, the research validates parental perspectives with objective physiological evidence, reducing reliance on any single type of data.

6.2.2. Emphasis on Parental Perspectives.

The thematic analyses foregrounded caregiver voices, demonstrating that parents can accurately identify behavioural triggers and anticipate crises. This aligns with

contemporary calls to include lived experience in autism research and provides clinicians with a model for integrating family input into diagnostic and intervention processes.

6.2.3. Causal Modelling of Sensory–Emotional–Behavioural Interactions.

By mapping the chain from emotional dysregulation to sensory hypersensitivity and behavioural outcomes, the study offers a theoretical model that unifies disparate findings in ASD research. This causal chain is supported by statistical associations, physiological patterns, and parental accounts, giving it ecological and biological validity.

6.2.4. Translational Relevance.

The findings have immediate practical implications for clinicians, schools, and families. Recognising behaviours as predictable consequences of sensory and emotional overload, rather than oppositional acts, reframes intervention strategies in more empathetic and effective ways.

6.3 Future Research Directions

Despite these contributions, further research is warranted to expand, refine, and validate the findings. Future studies should adopt longitudinal designs to trace the development of sensory, emotional, and behavioural interactions across time. Such research would clarify whether early sensory hypersensitivity predicts later emotional dysregulation, or whether emotional difficulties exacerbate sensory issues as children grow older. Longitudinal mapping would also reveal developmental transitions, such as puberty, when physiological stress systems and executive functions undergo significant changes. Tracking changes in EEG patterns, HRV, and cortisol across developmental stages would help identify sensitive periods for intervention.

There is a pressing need for intervention research that targets the integrated sensory–emotional–behavioural model. Sensory integration therapies should be systematically evaluated not only for behavioural outcomes but also for physiological markers such as alpha suppression or HRV. Similarly, emotion-regulation therapies, such as adapted CBT, should be tested in combination with sensory-focused interventions. Randomised controlled trials (RCTs) using multimodal outcome measures—including parental perspectives, behavioural ratings, and neurophysiological data—would provide strong evidence for effective interventions.

Future research should expand beyond relatively homogenous samples to include broader diversity in terms of socioeconomic status, ethnicity, and comorbidities. Differences in access to services, cultural interpretations of behaviour, and the presence of co-occurring conditions such as ADHD or anxiety may moderate the sensory–emotional–behavioural chain. Larger, diverse samples would also permit subgroup analyses, such as sex-based differences in stress physiology or age-based developmental changes. Emerging technologies, such as wearable biosensors offer opportunities for ecological monitoring of physiological stress in real-world settings. For example, continuous HRV or EDA tracking could be paired with ecological momentary assessment (EMA) of behaviour to capture real-time links between sensory environments, emotional states, and behavioural outcomes. Such tools would overcome the limitations of parental recall and provide granular data on day-to-day fluctuations.

Theoretical Integration

Finally, further theoretical integration is required to position these findings within broader frameworks such as the RDoC. By conceptualising ASD through dimensional

constructs spanning emotion regulation, sensory processing, and neurobiological systems, future research can contribute to a transdiagnostic understanding of psychiatric and developmental conditions.

This study demonstrated that behavioural crises in ASD are best explained through an integrated model that links emotional dysregulation, sensory hypersensitivity, and neurophysiological vulnerability. Sensory processing difficulties, particularly tactile defensiveness, emerged as the strongest predictors of behavioural outcomes, while emotional regulation amplified these vulnerabilities and reduced the child's resilience to stress. Neurophysiological evidence of hyperarousal corroborated these findings, and parental narratives contextualised them within daily life.

The contributions of this research lie in its multimodal integration, its validation of parental perspectives, its causal modelling of sensory–emotional–behavioural interactions, and its translational relevance for practice. At the same time, limitations such as reliance on parental self-reporting, cross-sectional design, and modest sample size caution against overgeneralisation.

The results of this study strongly aligned with the proposed hypotheses. The first hypothesis anticipated that a higher burden of sensory difficulties would correlate with emotional dysregulation and result in behavioural complications. This was confirmed through the regression and correlation analyses where sensory difficulties consistently emerged as the strongest predictor of behavioural outcomes ($r = .806$, $p < .001$; $\beta = .599$, $p < .001$). Emotional dysregulation was also significantly correlated with behavioural issues ($r = .720$, $p < .001$), and neurophysiological measures such as elevated cortisol and reduced HRV further supported the link with abnormal brain activity.

The second hypothesis proposed that parental reporting would be pivotal in identifying emotional and sensory complications. This was supported by the thematic analyses, where parents described behaviours such as meltdowns, shutdowns, and tactile defensiveness as predictable responses to emotional and sensory overload. Their reports aligned closely with quantitative results, underscoring the validity of parental perspectives as critical sources of clinical information. The third hypothesis predicted that behavioural, communication, and social complications would correlate with atypical sensory responses. This was validated by strong associations between SP2 scores, sensory difficulties and behavioural outcomes ($r = .776–.836$, $p < .001$), confirming that sensory processing vulnerabilities underpin the broader behavioural and social profile in ASD.

Final conclusion

In summary, this study shows that emotional dysregulation and challenges with sensory processing are critical determinants of ASD behaviours. These were attributed to sensory hypersensitivity and ability to regulate emotional triggers. Therefore, incorporating multidisciplinary strategies such as parent-focused methods and longitudinal research can help refine therapeutic interventions to provide holistic evidence-based and personalised care to children with ASD.

7 Chapter 7: References

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8 Chapter 8: Appendices

8.1 Appendix 1. Questionnaire

Participant Information and Invitation to Take Part in the Questionnaire Survey

Dear Parent/Carer,

You are kindly invited to take part in this questionnaire survey as part of a research study titled:

"*Theoretical Multi-level Mapping of the Contribution of Emotional Regulation to Sensory Difficulties in Autism Spectrum Disorder – From Brain to Behaviour*."

Purpose of the Study
The purpose of this survey is to explore emotional dysregulation in children with Autism Spectrum Disorder (ASD), which remains connected to sensory processing difficulties, emotional challenges, behavioural and brain function. Your insights will help us better understand how autistic children respond to emotional and sensory environments.

Why Your Input Matters
As a parent or carer, your observations are vital to improving support strategies for children with ASD and filling key knowledge gaps in autism research.

What to Expect
You'll respond to **30 short statements** about your child's emotions, behaviour, and sensory sensitivities using a rating scale. There are no right or wrong answers—just your honest observations.

Confidentiality & Voluntary Participation
Your responses are confidential and used only for research. Participation is voluntary, and you may withdraw at any time.

If you're happy to proceed, please continue to the questionnaire below.
Thank you for your time and contribution.

Warm regards,
[Your Name]
[Institution/University Name]
[Contact Information]

Questionnaire Survey

Research Title:

Theoretical Multi-Level Mapping of the Contribution of Emotional Regulation to Sensory Difficulties in Autism Spectrum Disorders; from Brain to Behaviour

Participant Instructions

Please answer the following questions to help us determine eligibility and gather essential demographic information for the study.

I. Introduction Questions

1. How old is your child? _____ (in years)
2. **Gender:**
   [ ] Male
   [ ] Female
   [ ] Non-binary
   [ ] Prefer not to say
   [ ] Other: _____
3. **Relationship to Child:**
   [ ] Mother
   [ ] Father
   [ ] Guardian
   [ ] Other: _____
4. **Child's Ethnicity:**
   [ ] Caucasian
   [ ] Hispanic/Latino
   [ ] Black/African American
   [ ] Asian
   [ ] Mixed Race
   [ ] Other: _____
   [ ] Prefer not to say
5. **Parent/Caregiver Age:**
   [ ] 18-25
   [ ] 26-35
   [ ] 36-45
   [ ] 46-55
   [ ] 56+

6. **Household Size:**
   [ ] 1-2
   [ ] 3-4
   [ ] 5-6
   [ ] 7+

7. **Geographical Location:**
   [ ] Urban Area
   [ ] Suburban Area
   [ ] Rural Area

II. Screening Questions

1. Age Range of your child?
   [ ] 4-6
   [ ] 7-9
   [ ] 10-12
   [ ] 13-15
   [ ] 16-18

2. Has your child been diagnosed with Autism Spectrum Disorder (ASD)?
   [ ] Yes
   [ ] No
   [ ] Unsure

3. Has your child experienced any of the following? (Check all that apply)
   [ ] Emotional dysregulation
   [ ] Social communication difficulties
   [ ] Sensory processing difficulties
   [ ] Repetitive behaviours
   [ ] Other (Please specify): _____

4. Does your child have any other diagnoses (e.g., ADHD, Anxiety Disorder)?
   [ ] Yes (Please specify): _____
   [ ] No

5. Is a primary caregiver actively involved in your child's treatment?
   [ ] Yes
   [ ] No

6. What is the highest level of education completed by the child's primary caregiver(s)?
   [ ] High school
   [ ] Associate degree
   [ ] Bachelor's degree
   [ ] Master's degree

[ ] Doctoral degree
[ ] Prefer not to say

7. What is the employment status of the primary caregiver?
   [ ] Employed full-time
   [ ] Employed part-time
   [ ] Stay-at-home parent/guardian
   [ ] Unemployed
   [ ] Retired

8. What is your household's estimated annual income (pre-tax)?
   [ ] Less than £20,000
   [ ] £20,000–£40,000
   [ ] £40,000–£60,000
   [ ] £60,000–£80,000
   [ ] More than £80,000
   [ ] Prefer not to say

9. Does your child currently have access to healthcare services?
   [ ] Yes
   [ ] No

III. Questionnaire Survey Instructions

Please indicate how much you agree or disagree with the following statements based on your child's typical behaviour. Answer each item as accurately as possible.

Response Options (5-point Likert scale):

1 – Strongly Disagree
2 – Disagree
3 – Neither Agree nor Disagree
4 – Agree
5 – Strongly Agree

Section A: Emotional Regulation

No.	Statement	Response
1	My child finds it difficult to manage intense emotions such as anger or frustration.	5 4 3 2 1
2	My child frequently experiences emotional outbursts without clear warning.	5 4 3 2 1
3	My child takes a long time to calm down after becoming upset.	5 4 3 2 1
4	Small changes in daily routine cause strong emotional reactions in my child.	5 4 3 2 1
5	My child can identify and describe how they are feeling. *(Reverse coded)*	5 4 3 2 1
6	Compared to their peers, my child experiences emotions more intensely.	5 4 3 2 1
7	I regularly need to assist my child in managing their emotions.	5 4 3 2 1

Section B: Sensory Difficulties (Tactile & General)

No.	Statement	Response
8	My child is overly sensitive to certain textures, such as clothing labels or seams.	5 4 3 2 1
9	My child reacts negatively to unexpected physical contact.	5 4 3 2 1
10	Everyday sensory experiences (e.g. noise, lights, touch) are distressing to my child.	5 4 3 2 1
11	My child avoids certain places or situations due to sensory overload.	5 4 3 2 1
12	My child actively seeks out specific sensory experiences (e.g. rubbing objects, spinning).	5 4 3 2 1
13	My child shows visible discomfort during routine activities involving touch (e.g. hair brushing).	5 4 3 2 1

Section C: Behavioural Outcomes

No.	Statement	Response
14	When emotionally distressed, my child displays repetitive behaviours such as hand flapping or rocking.	5 4 3 2 1
15	Emotional upsets often lead to aggressive or disruptive behaviour in my child.	5 4 3 2 1
16	Transitions between tasks or places cause behavioural difficulties.	5 4 3 2 1
17	My child withdraws socially when experiencing emotional or sensory distress.	5 4 3 2 1
18	Sensory overload directly triggers challenging behaviours in my child.	5 4 3 2 1
19	I can identify a pattern where emotional difficulty leads to problematic behaviour.	5 4 3 2 1

Section D: Cognitive/Neurological Indicators

No.	Statement	Response
20	My child appears to shut down or become unresponsive when emotionally overwhelmed.	5 4 3 2 1
21	My child's responses to sensory input often seem exaggerated or blunted.	5 4 3 2 1
22	During emotional events, my child struggles with attention or thinking clearly.	5 4 3 2 1

Section E: Parent-Child Interaction

No.	Statement	Response
23	My emotional state often influences my child's behaviour and emotions.	5 4 3 2 1
24	I can predict which sensory experiences will upset my child.	5 4 3 2 1
25	Managing both my child's emotional needs and sensory issues is a challenge.	5 4 3 2 1
26	My child and I are able to talk effectively about emotions. *(Reverse coded)*	5 4 3 2 1

Section F: Emotional Awareness (Alexithymia / Interoception)

No.	Statement	Response
27	My child struggles to recognise their own emotional states.	5 4 3 2 1
28	My child often shows emotions without an obvious reason or trigger.	5 4 3 2 1
29	My child has difficulty describing when they are in pain, tired, or uncomfortable.	5 4 3 2 1
30	My child seems unaware of basic bodily needs (e.g. hunger, sleepiness).	5 4 3 2 1

Invitation to Participate in Expert Interview – Research on Autism Spectrum Disorder (ASD)

Dear [Dr./Professor/Mr./Ms.] [Surname],

I hope you are well.

I'm conducting a research project titled:

> ***"Theoretical Multi-Level Mapping of Emotional Regulation and Sensory Difficulties in Autism Spectrum Disorder – From Brain to Behaviour."***

The study explores how emotional dysregulation in children with ASD relates to sensory challenges, behaviours, and neurophysiological patterns. I'm inviting you to share your expert insights through a **30–45 minute semi-structured interview** (via Zoom/Teams).

Your perspectives on emotional regulation, sensory symptoms, behavioural presentations, or clinical gaps would greatly enrich this work. With your consent, the session will be recorded and anonymised.

Participation is voluntary, and ethics approval has been obtained. If you're available or have questions, please contact me at **[Your Email]** to arrange a suitable time.

Thank you for considering this opportunity.

Warm regards,
[Your Full Name]
[Your Position]

[Institution]
[Email / Phone Number]

Interview Questions

A. Emotional Dysregulation in ASD (Emotion Recognition, Coping, Triggers)

1. Based on your experience, how does emotional dysregulation typically present in children with ASD?
2. What are the most common emotional triggers you have observed in autistic children?
3. How would you describe the relationship between emotion regulation and cognitive control in children with ASD?
4. In your opinion, how effectively do children with ASD recognise and communicate their emotional states?
5. Have you observed differences in emotional coping strategies between children with ASD and typically developing children?

B. Sensory Processing and Emotional Response (Tactile Hypersensitivity, Sensory Reactivity)

6. What patterns of sensory sensitivity, particularly tactile hypersensitivity, are most frequently associated with emotional dysregulation in ASD?
7. How do children with ASD typically react emotionally to overwhelming sensory input?
8. Do you believe that sensory processing difficulties precede emotional challenges in ASD, or vice versa?
9. In your view, how do sensory sensitivities influence daily functioning and behaviour in autistic children?
10. What therapeutic strategies have you found effective in managing sensory-related emotional distress?

C. Behavioural Manifestations (Meltdowns, Withdrawal, Social Behaviour)

11. What kinds of behavioural responses do you most commonly see following emotional or sensory overstimulation?
12. In your opinion, how are repetitive or restrictive behaviours linked to emotional regulation difficulties in ASD?

13. Have you observed a pattern where certain sensory environments consistently provoke behavioural disturbances in ASD children?

14. How do emotional dysregulation and sensory difficulties affect peer relationships and social interactions in this population?

D. Neurological and Cognitive Aspects (Brain–Behaviour Mapping)

15. From a neurological or cognitive standpoint, what brain regions or functions do you believe are most involved in emotional dysregulation in ASD?

16. Have you worked with or observed EEG, fMRI, or similar neurophysiological data that link atypical brain activity to emotional or sensory symptoms in ASD?

17. Do you think interoception and alexithymia play a significant role in the emotional-sensory profile of autistic children?

E. Parent-Child Dynamics and Observations

18. How important do you consider parental observations in diagnosing or managing emotional and sensory symptoms in ASD?

19. What advice or strategies do you typically share with parents to support their child's emotional regulation and sensory coping?

F. Final Reflection

20. What do you believe are the most pressing gaps in our understanding of the link between emotional regulation and sensory difficulties in children with ASD?

8.2 Appendix 2. Dataset for SPSS

ID	Group	Child_Age	Child_Gender	Child_Ethnicity	Caregiver_Role	Caregiver_Age	Caregiver_Education	Caregiver_Employment	Household_Income	Household_Size	Location	Healthcare_Access	ER1	ER2	ER3	ER4	ER5	ER6	SD1	SD2	SD3	SD4	SD5	SD6	BO1	BO2	BO3	BO4	BO5	BO6	CN1	CN2	CN3	CN4	PCI1	PCI2	PCI3	PCI4	EA1	EA2	EA3	EA4	EA5
1	1	12	1	2	2	3	4	1	3	3	1	0	5	5	4	3	2	4	4	4	4	4	4	5	4	3	4	5	4	4	3	4	3	5	3	4	4	4	5	4	5	5	4
2	2	12	1	2	1	3	4	3	4	4	1	0	3	3	2	4	4	3	2	4	2	2	2	3	2	4	5	2	2	3	3	3	4	3	2	5	4	3	3	3	3	3	2
3	2	6	1	1	2	3	4	2	3	3	3	1	3	3	2	2	1	4	3	1	3	3	3	3	3	2	3	2	3	3	3	3	4	3	2	3	3	2	4	4	4	4	3
4	2	9	2	1	1	3	4	1	5	3	1	1	3	5	4	3	3	3	3	3	2	2	2	3	3	4	3	3	4	2	3	3	2	3	3	2	4	4	5	4	1	4	3
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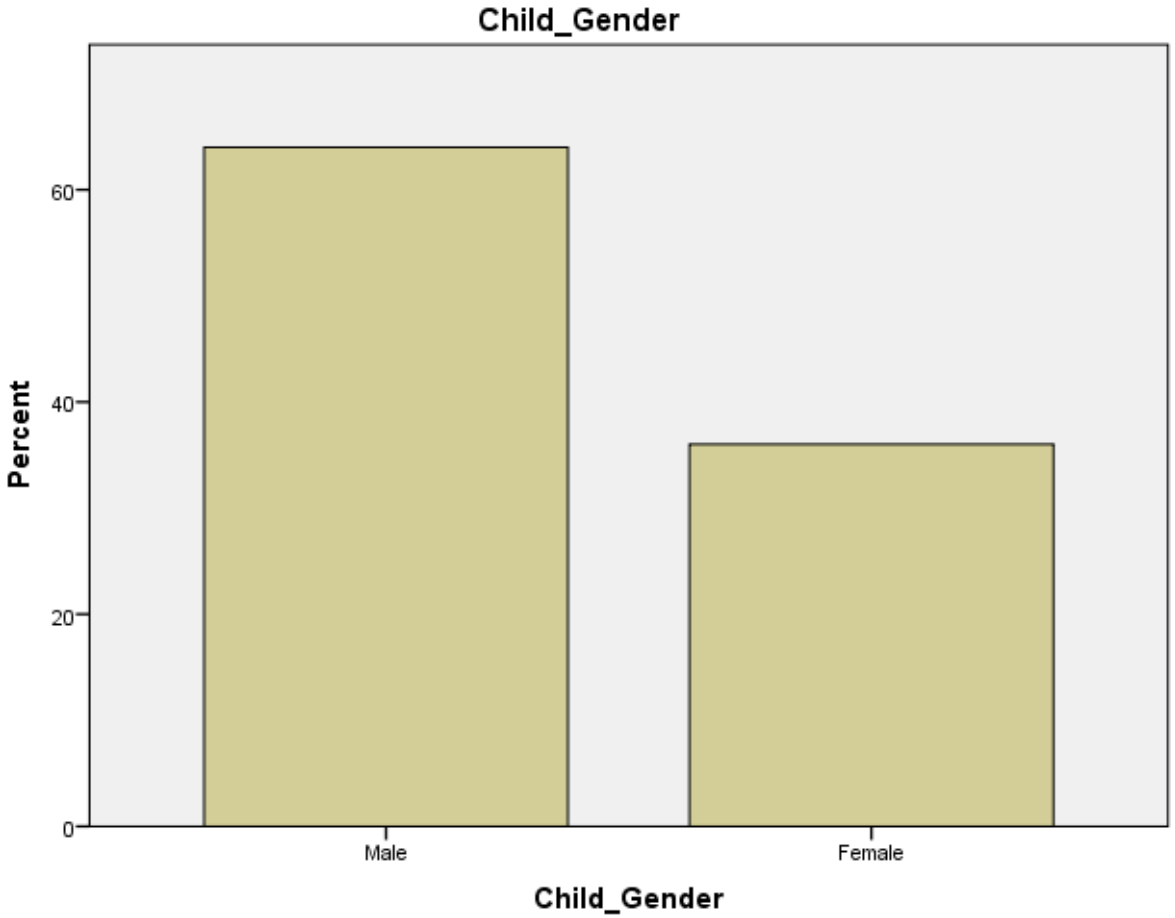
8.3 Appendix 3. SPSS Results

Descriptive Statistics

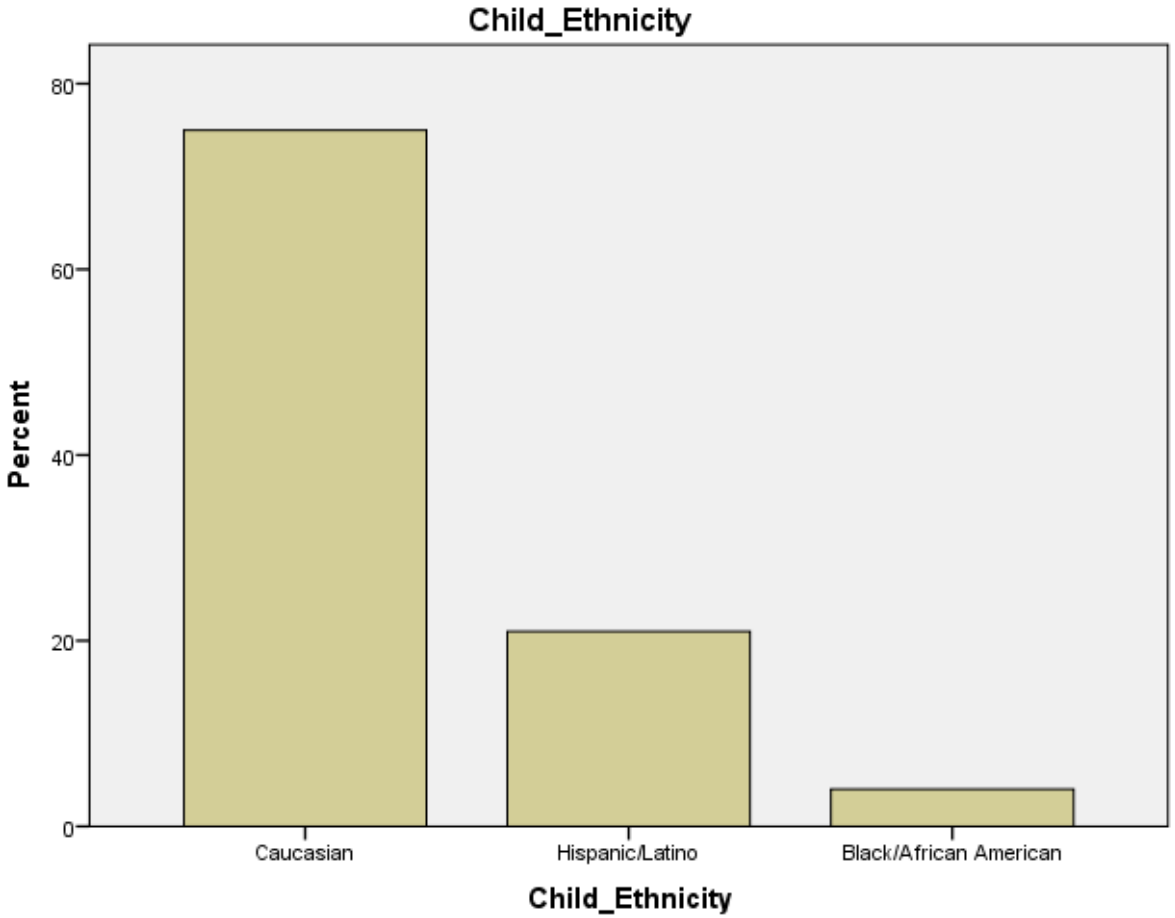
Child_Gender

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Male	64	64.0	64.0	64.0
	Female	36	36.0	36.0	100.0
	Total	100	100.0	100.0	


Child_Gender
Percent
60
40
20
0
Male
Female
Child_Gender

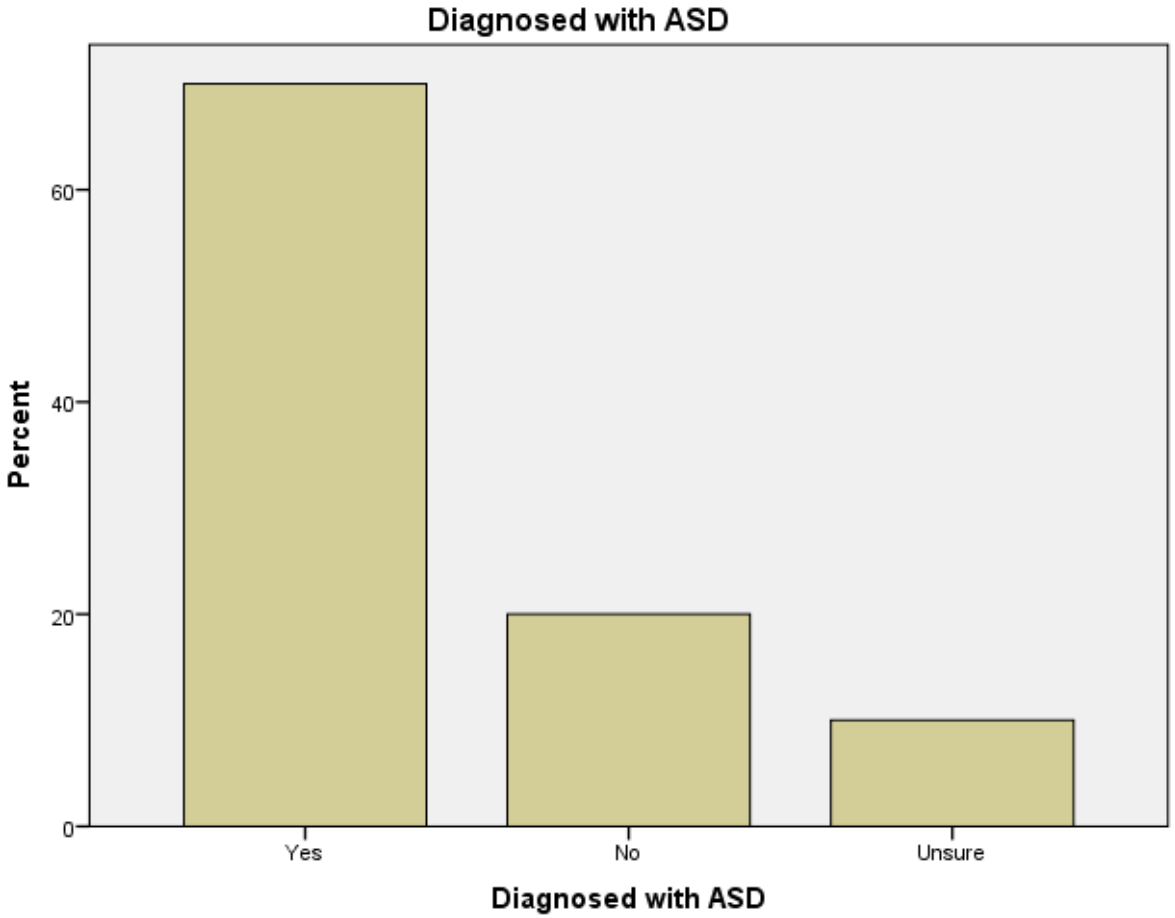
Child_Ethnicity

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Caucasian	75	75.0	75.0	75.0
	Hispanic/Latino	21	21.0	21.0	96.0
	Black/African American	4	4.0	4.0	100.0
	Total	100	100.0	100.0	


Child_Ethnicity
80
60
40
20
0
Percent
Caucasian
Hispanic/Latino
Black/African American
Child_Ethnicity

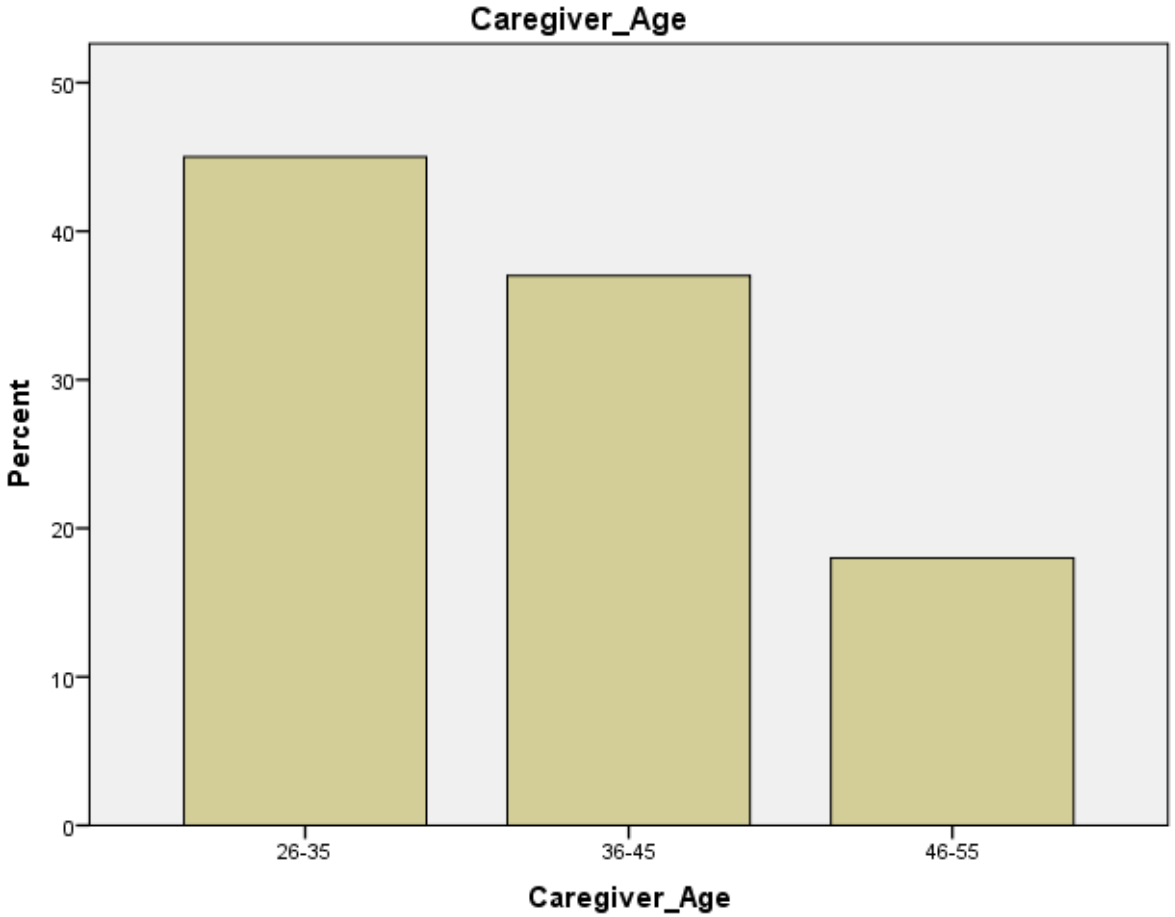
Diagnosed with ASD

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Yes	70	70.0	70.0	70.0
	No	20	20.0	20.0	90.0
	Unsure	10	10.0	10.0	100.0
	Total	100	100.0	100.0	


Diagnosed with ASD
Percent
60
40
20
0
Yes
No
Unsure
Diagnosed with ASD

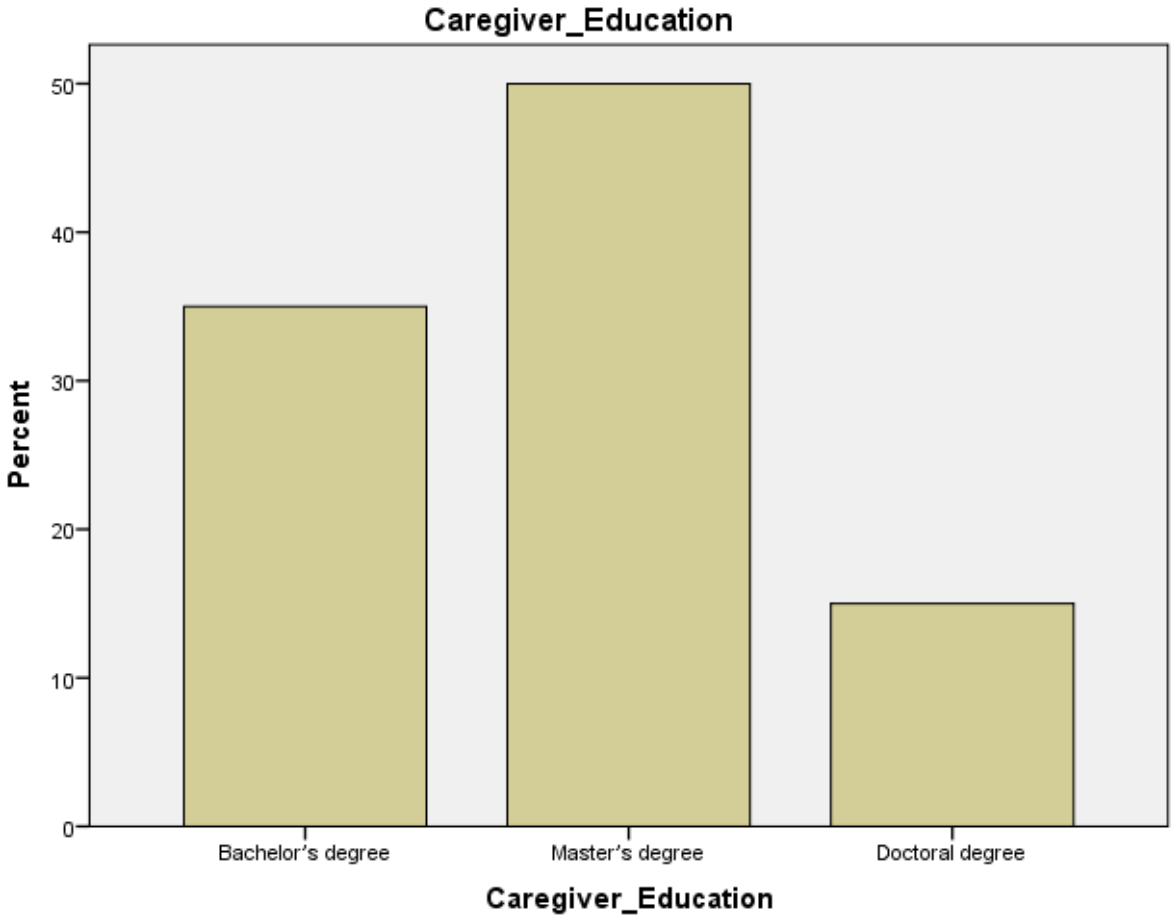
Caregiver_Age

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	26-35	45	45.0	45.0	45.0
	36-45	37	37.0	37.0	82.0
	46-55	18	18.0	18.0	100.0
	Total	100	100.0	100.0	


Caregiver_Age
Percent
50
40
30
20
10
0
26-35
36-45
46-55
Caregiver_Age

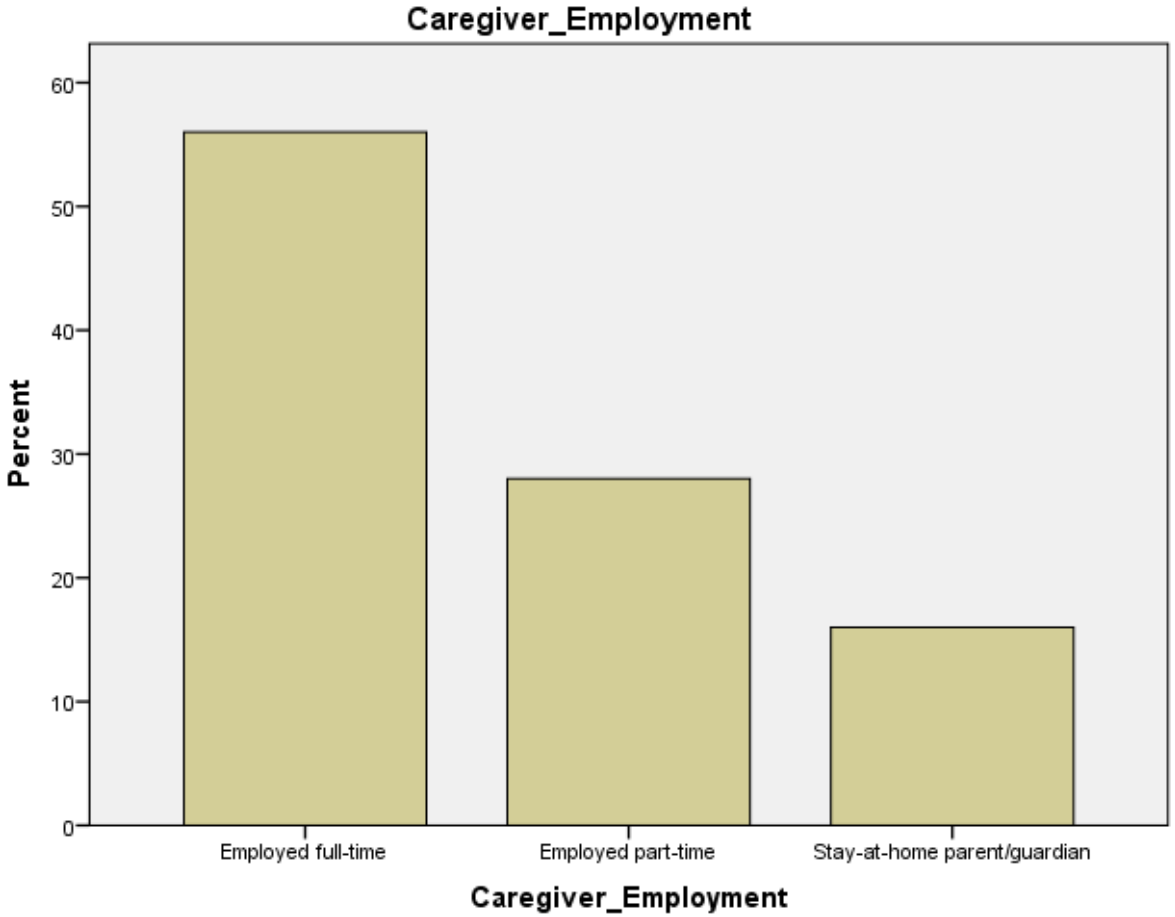
Caregiver_Education

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Bachelor's degree	35	35.0	35.0	35.0
	Master's degree	50	50.0	50.0	85.0
	Doctoral degree	15	15.0	15.0	100.0
	Total	100	100.0	100.0	


Caregiver_Education
Percent
50
40
30
20
10
0
Bachelor's degree
Master's degree
Doctoral degree
Caregiver_Education

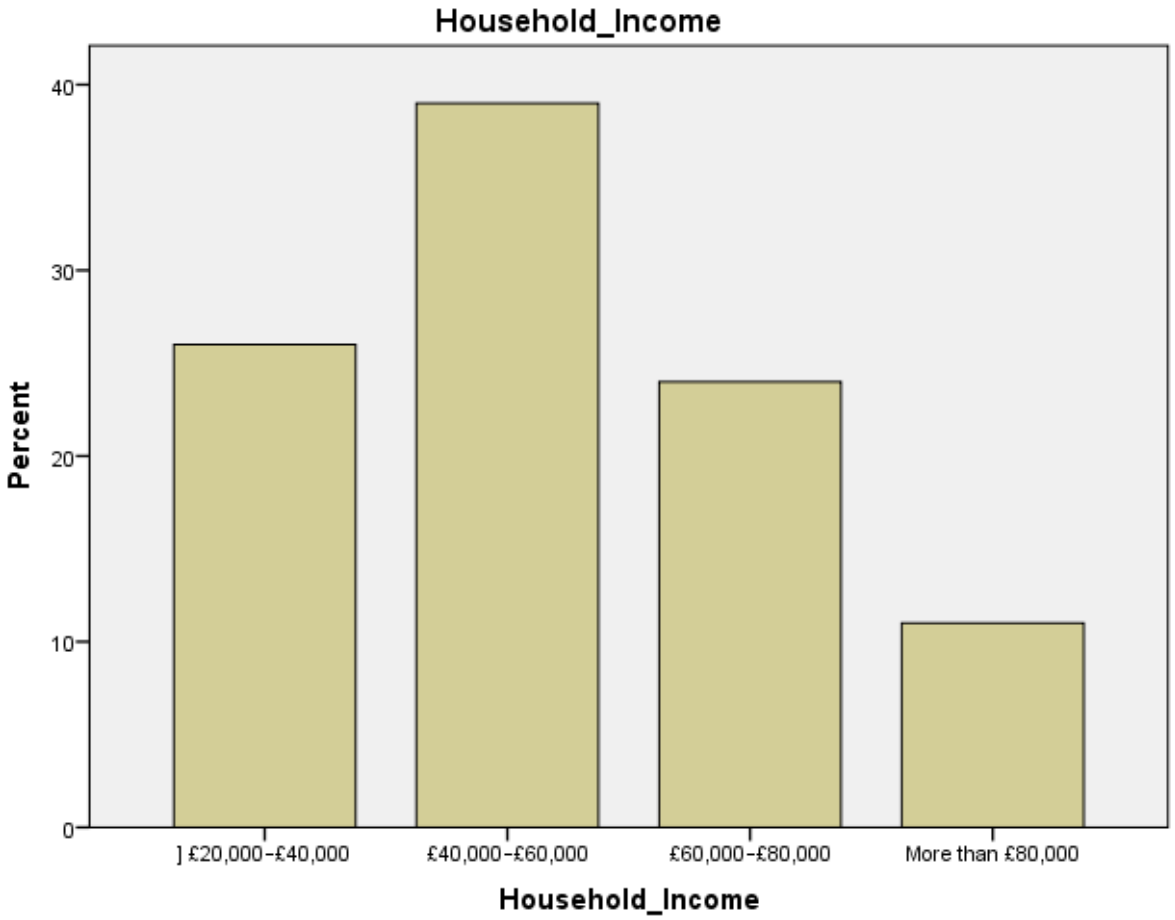
Caregiver_Employment

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Employed full-time	56	56.0	56.0	56.0
	Employed part-time	28	28.0	28.0	84.0
	Stay-at-home parent/guardian	16	16.0	16.0	100.0
	Total	100	100.0	100.0	


Caregiver_Employment
Percent
60
50
40
30
20
10
0
Employed full-time
Employed part-time
Stay-at-home parent/guardian
Caregiver_Employment

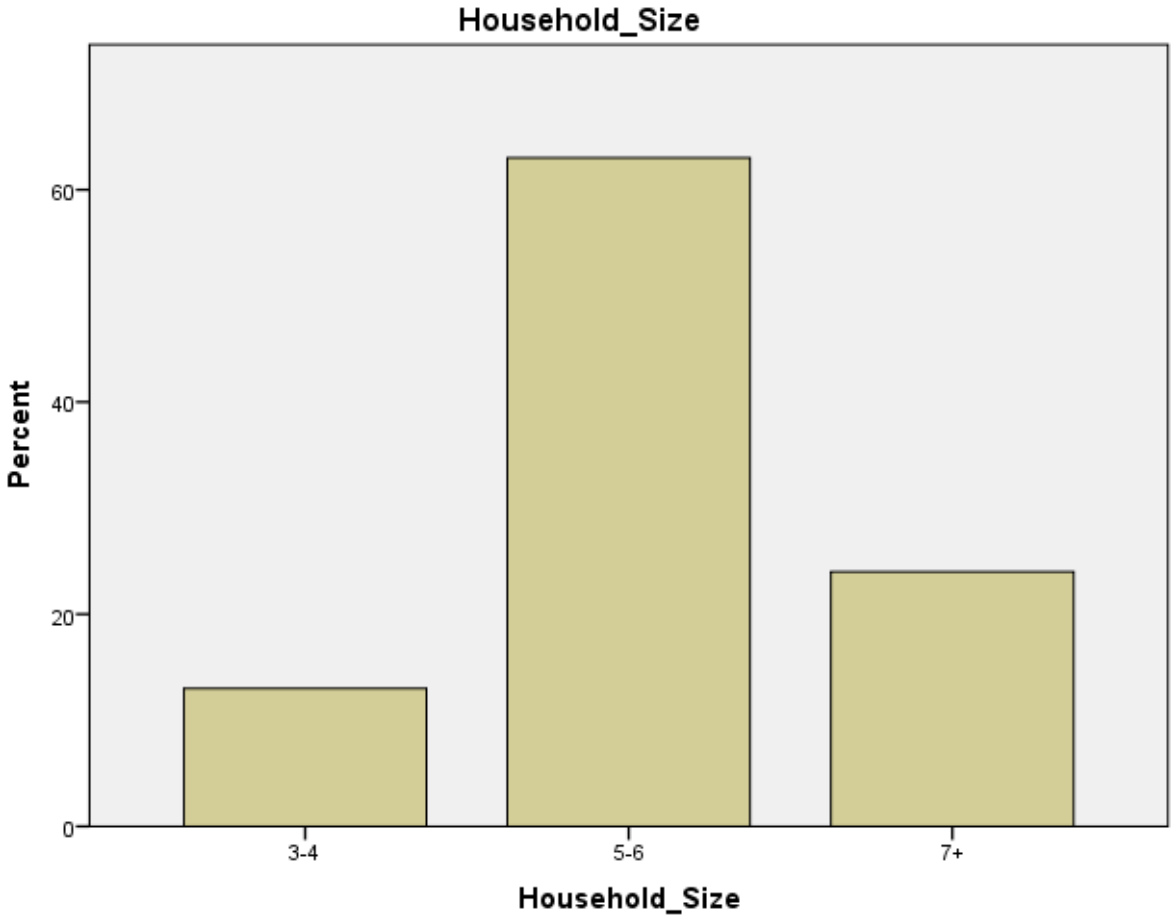
Household_Income

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	] £20,000–£40,000	26	26.0	26.0	26.0
	£40,000–£60,000	39	39.0	39.0	65.0
	£60,000–£80,000	24	24.0	24.0	89.0
	More than £80,000	11	11.0	11.0	100.0
	Total	100	100.0	100.0	


Household_Income
40
30
20
10
0
Percent
] £20,000-£40,000
£40,000-£60,000
£60,000-£80,000
More than £80,000
Household_Income

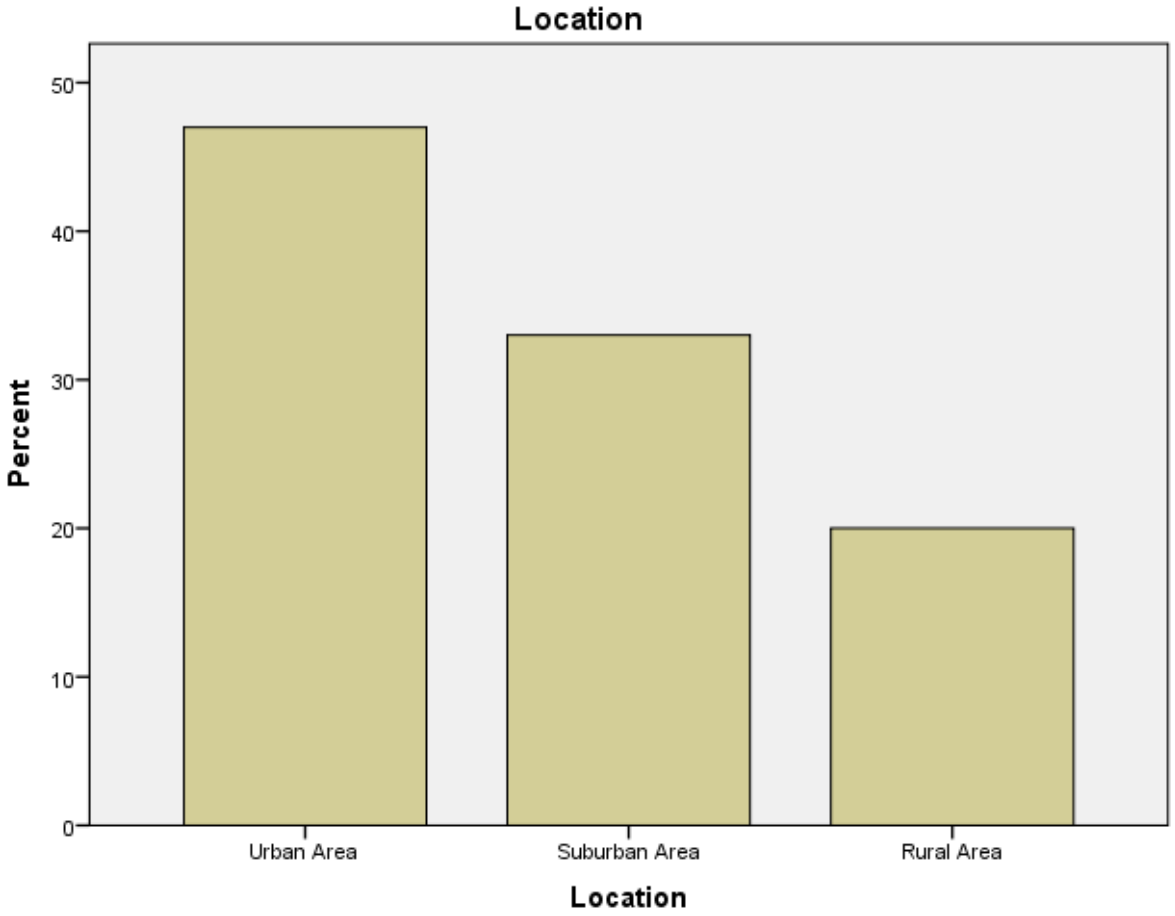
Household_Size

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	3-4	13	13.0	13.0	13.0
	5-6	63	63.0	63.0	76.0
	7+	24	24.0	24.0	100.0
	Total	100	100.0	100.0	


Household_Size
Percent
60
40
20
0
3-4
5-6
7+
Household_Size

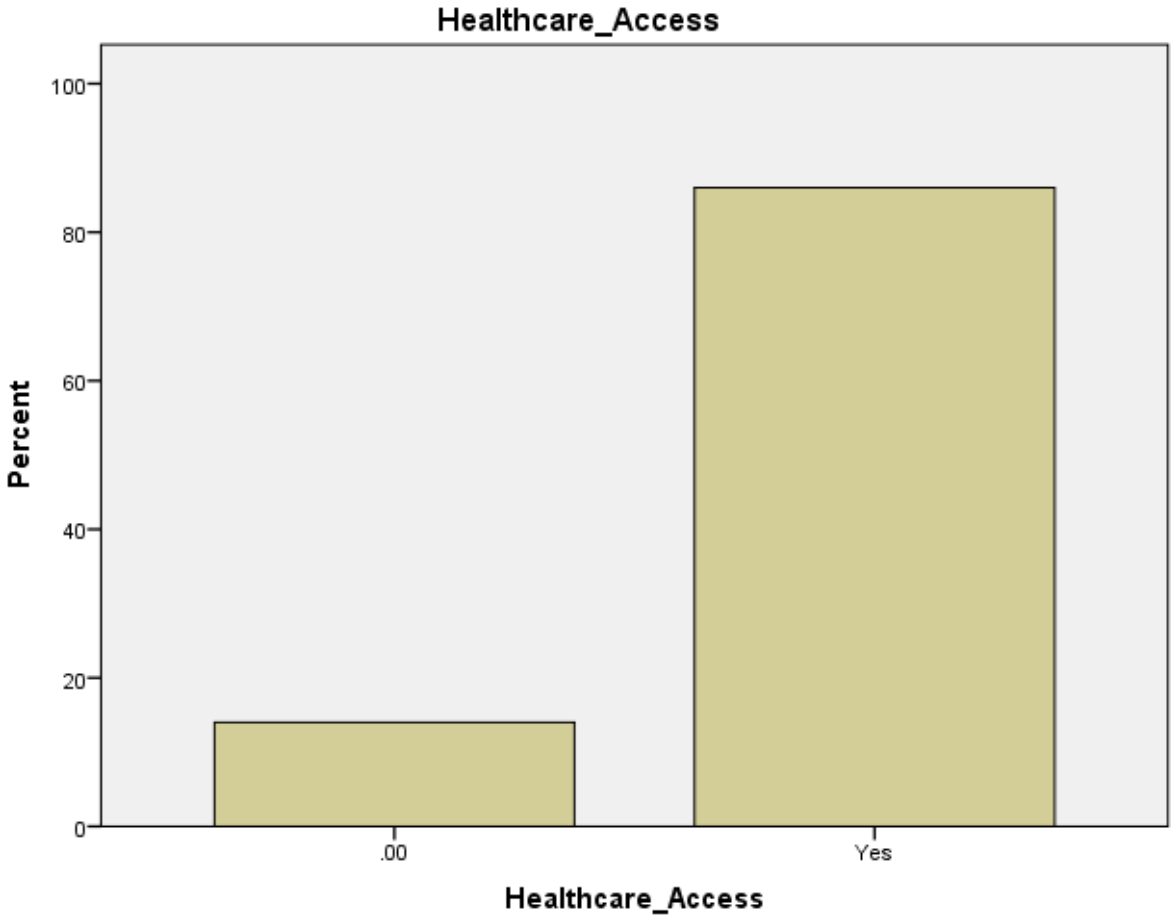
Location

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Urban Area	47	47.0	47.0	47.0
	Suburban Area	33	33.0	33.0	80.0
	Rural Area	20	20.0	20.0	100.0
	Total	100	100.0	100.0	


Location
Percent
50
40
30
20
10
0
Urban Area
Suburban Area
Rural Area
Location

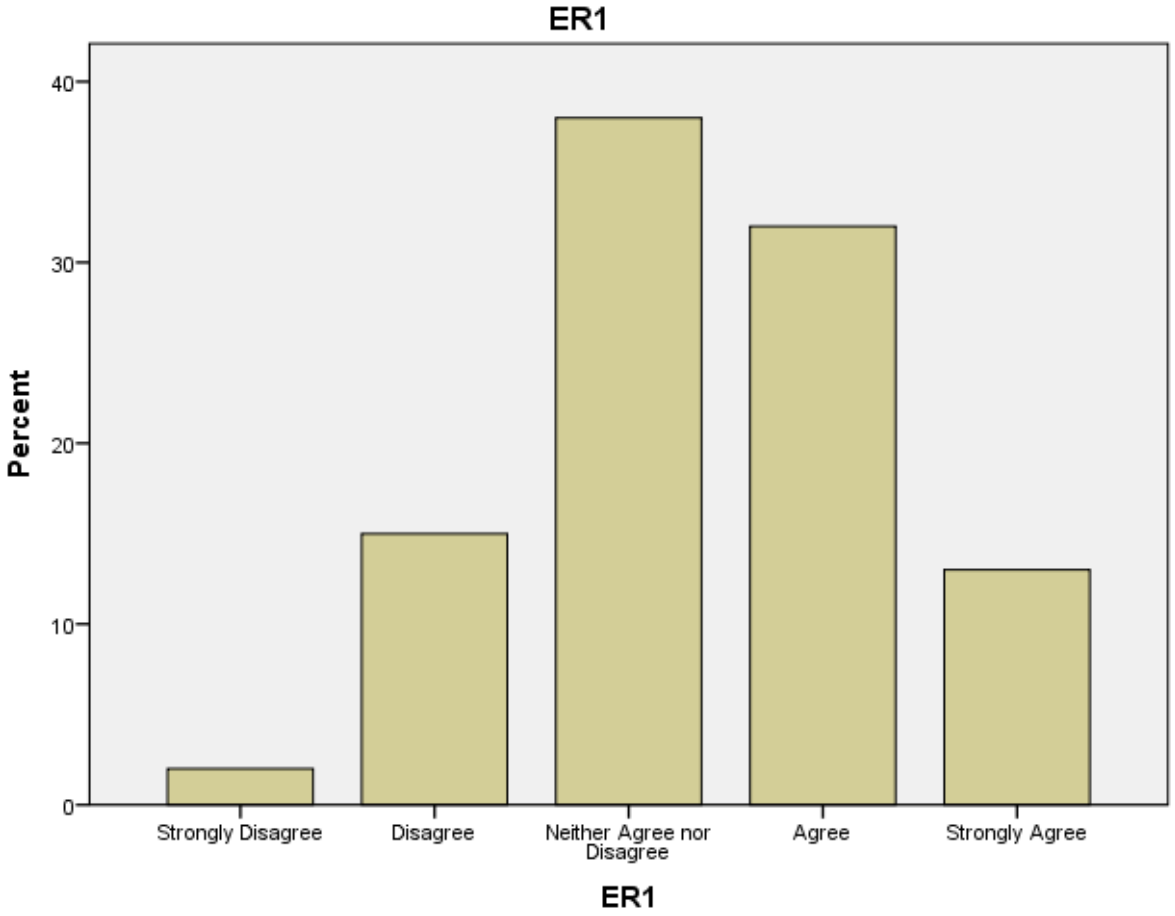
Healthcare_Access

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	.00	14	14.0	14.0	14.0
	Yes	86	86.0	86.0	100.0
	Total	100	100.0	100.0	


Healthcare_Access
Percent
100
80
60
40
20
0
.00
Yes
Healthcare_Access

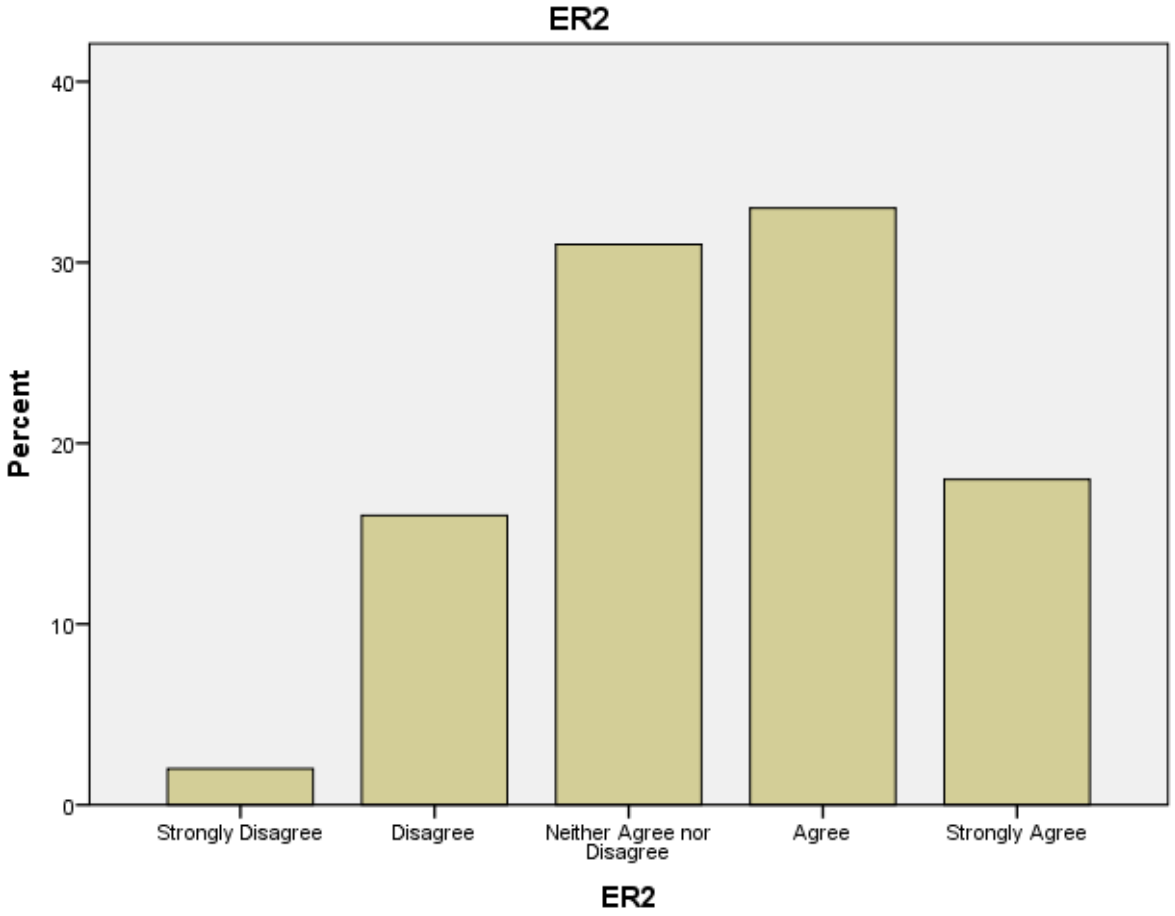
ER1

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	2	2.0	2.0	2.0
	Disagree	15	15.0	15.0	17.0
	Neither Agree nor Disagree	38	38.0	38.0	55.0
	Agree	32	32.0	32.0	87.0
	Strongly Agree	13	13.0	13.0	100.0
	Total	100	100.0	100.0	


ER1
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
ER1

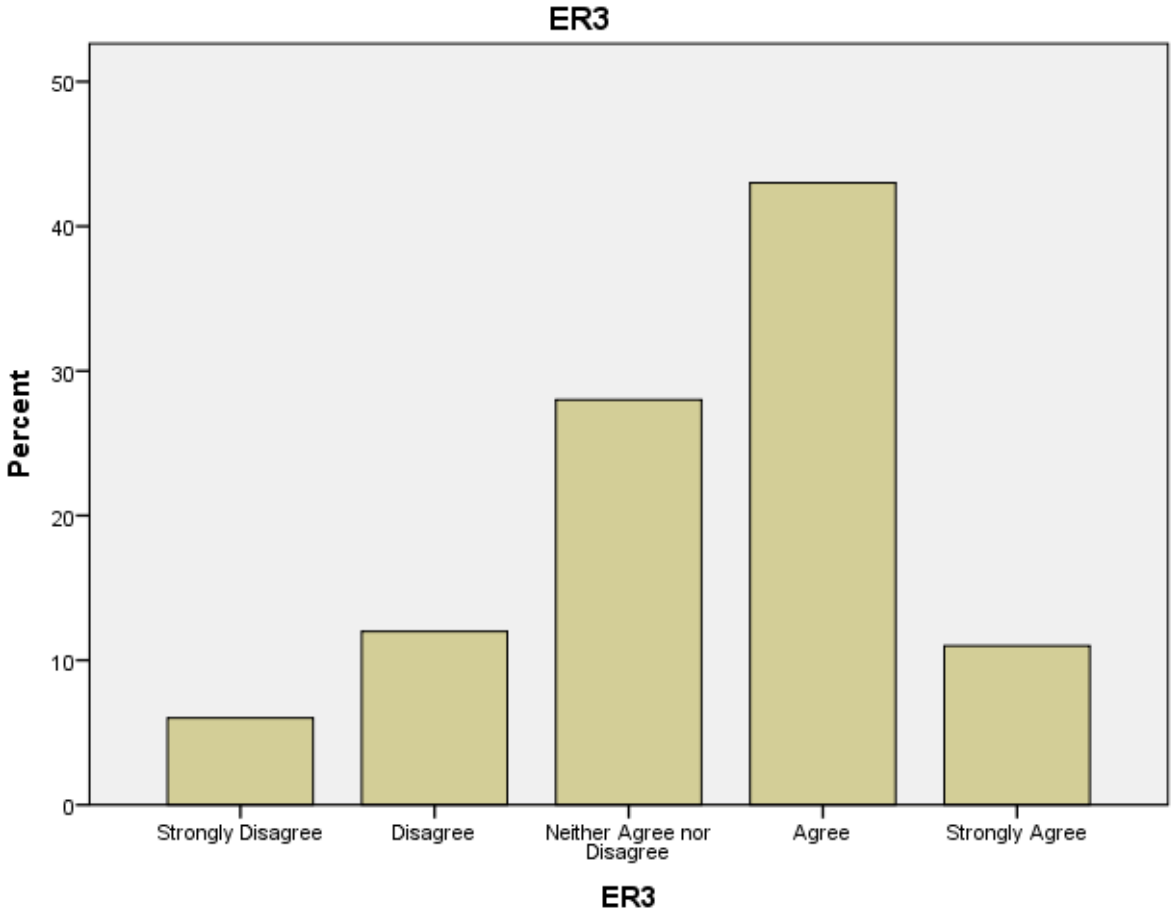
ER2

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	2	2.0	2.0	2.0
	Disagree	16	16.0	16.0	18.0
	Neither Agree nor Disagree	31	31.0	31.0	49.0
	Agree	33	33.0	33.0	82.0
	Strongly Agree	18	18.0	18.0	100.0
	Total	100	100.0	100.0	


ER2
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
ER2

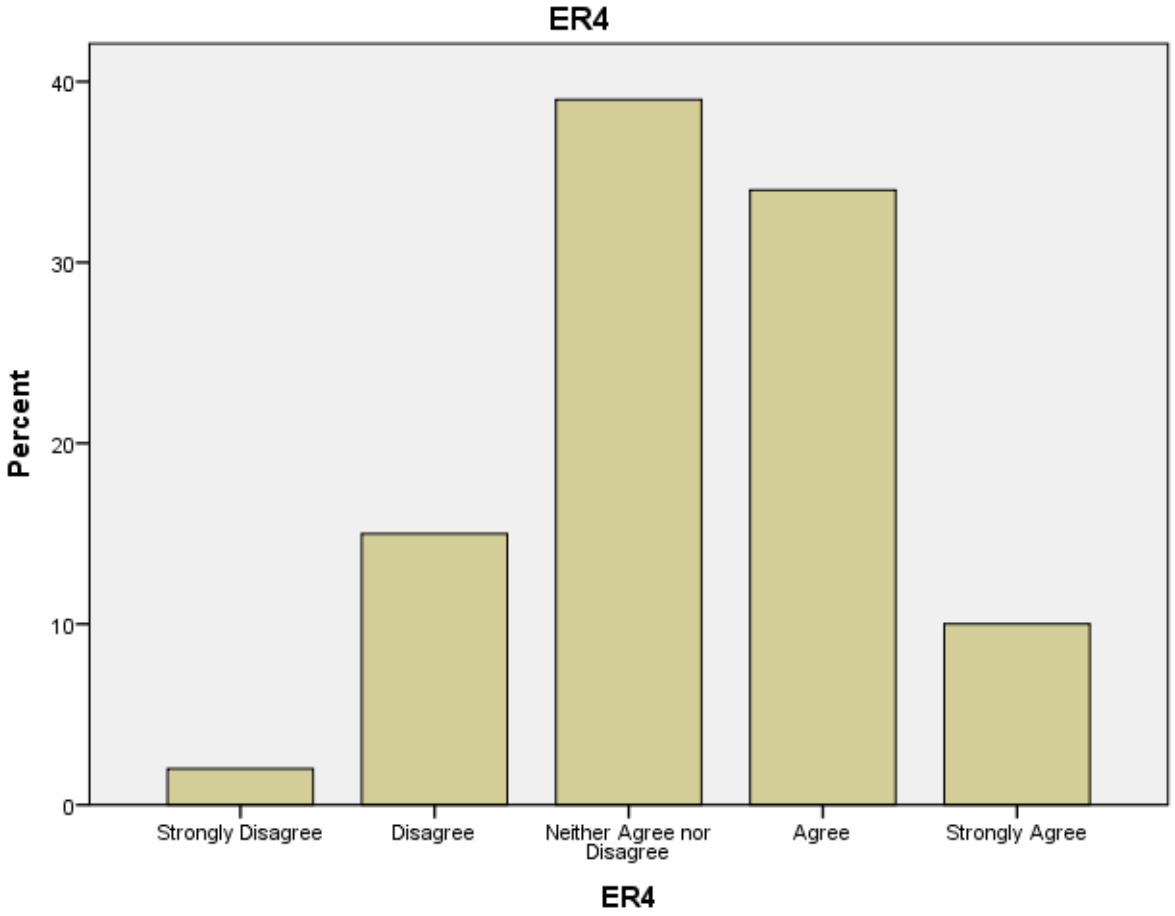
ER3

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	6	6.0	6.0	6.0
	Disagree	12	12.0	12.0	18.0
	Neither Agree nor Disagree	28	28.0	28.0	46.0
	Agree	43	43.0	43.0	89.0
	Strongly Agree	11	11.0	11.0	100.0
	Total	100	100.0	100.0	


ER3
Percent
50
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
ER3

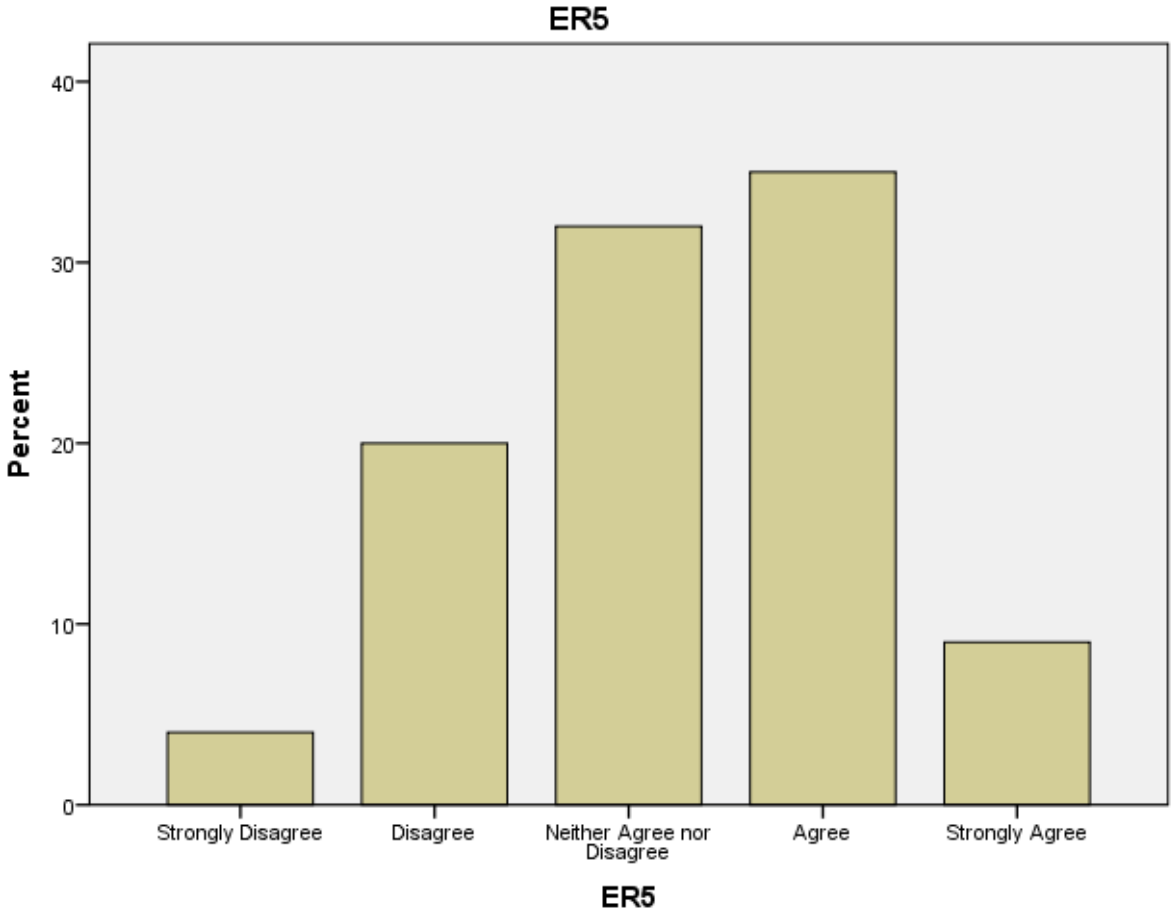
ER4

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	2	2.0	2.0	2.0
	Disagree	15	15.0	15.0	17.0
	Neither Agree nor Disagree	39	39.0	39.0	56.0
	Agree	34	34.0	34.0	90.0
	Strongly Agree	10	10.0	10.0	100.0
	Total	100	100.0	100.0	


ER4
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
ER4

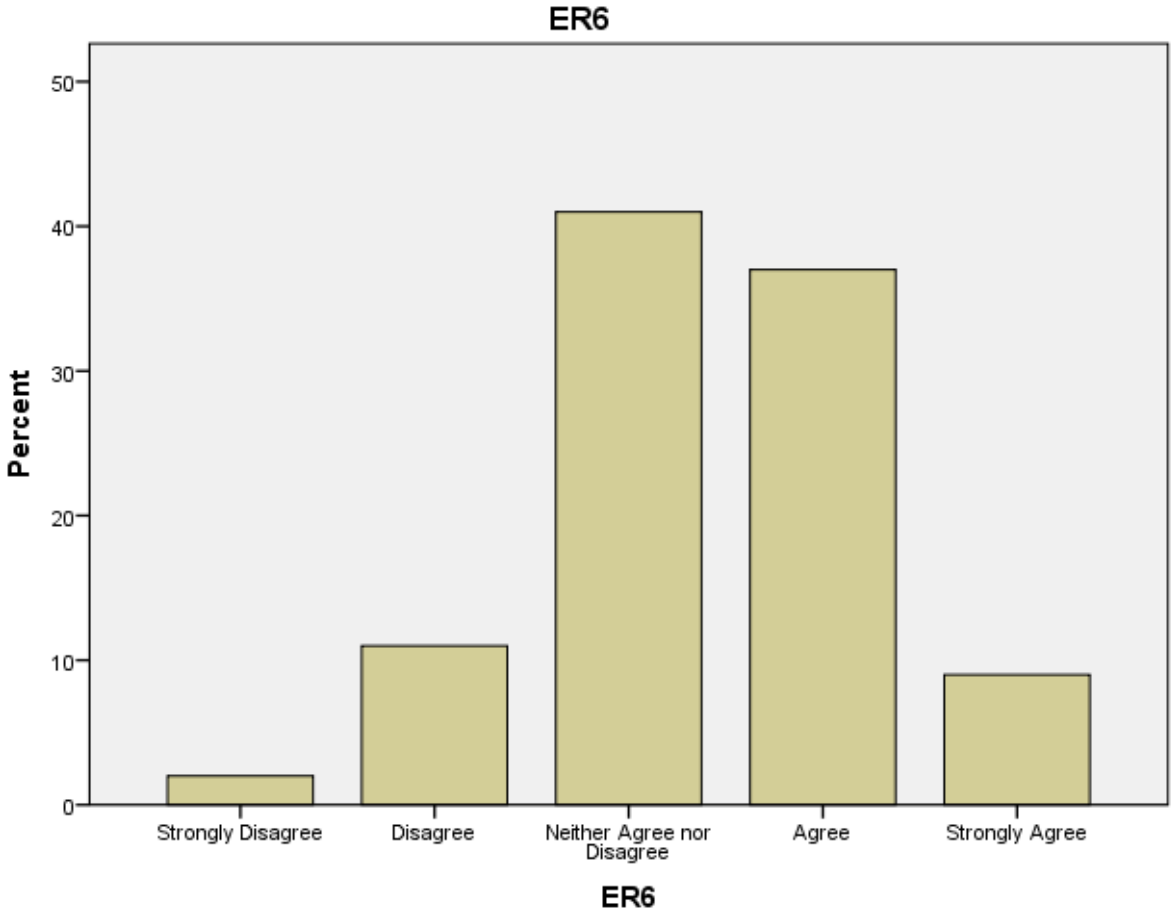
ER5

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	4	4.0	4.0	4.0
	Disagree	20	20.0	20.0	24.0
	Neither Agree nor Disagree	32	32.0	32.0	56.0
	Agree	35	35.0	35.0	91.0
	Strongly Agree	9	9.0	9.0	100.0
	Total	100	100.0	100.0	


ER5
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
ER5

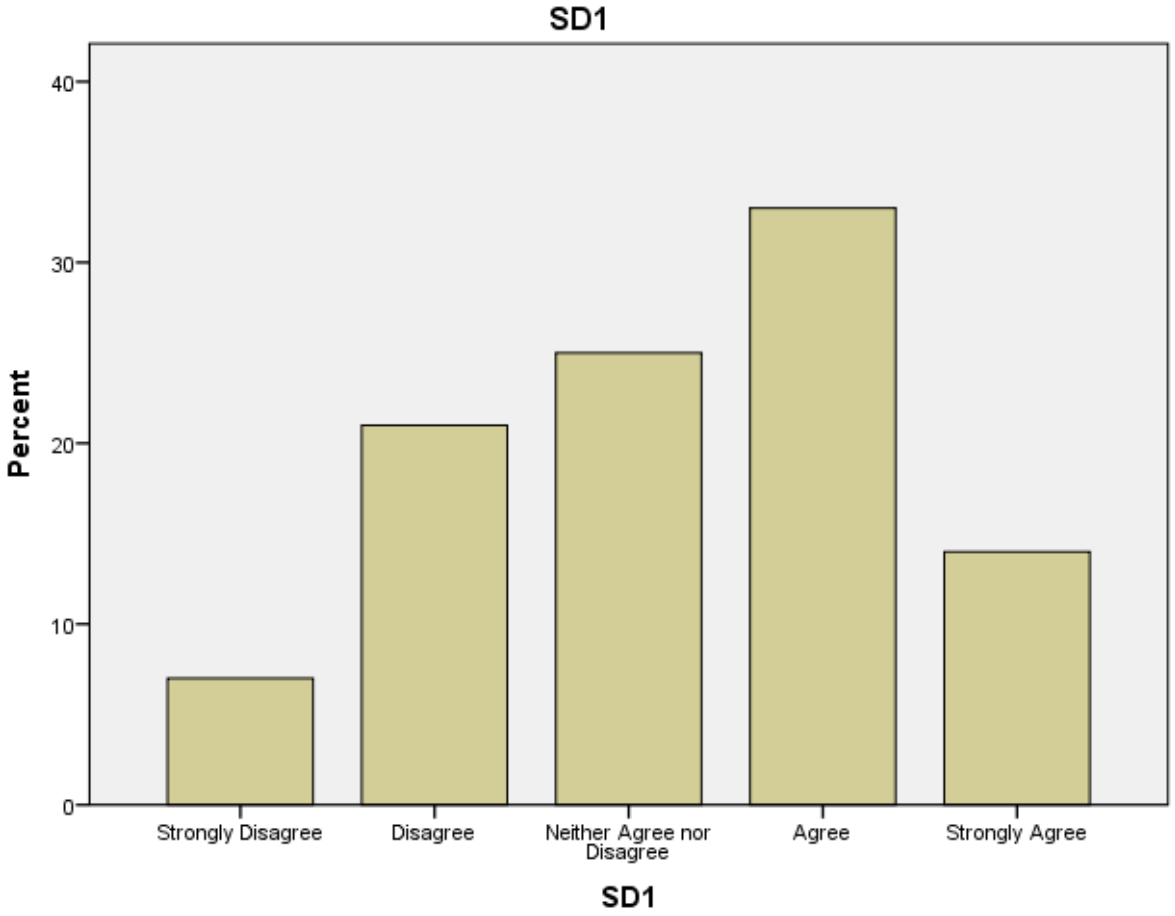
ER6

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	2	2.0	2.0	2.0
	Disagree	11	11.0	11.0	13.0
	Neither Agree nor Disagree	41	41.0	41.0	54.0
	Agree	37	37.0	37.0	91.0
	Strongly Agree	9	9.0	9.0	100.0
	Total	100	100.0	100.0	


ER6
Percent
50
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
ER6

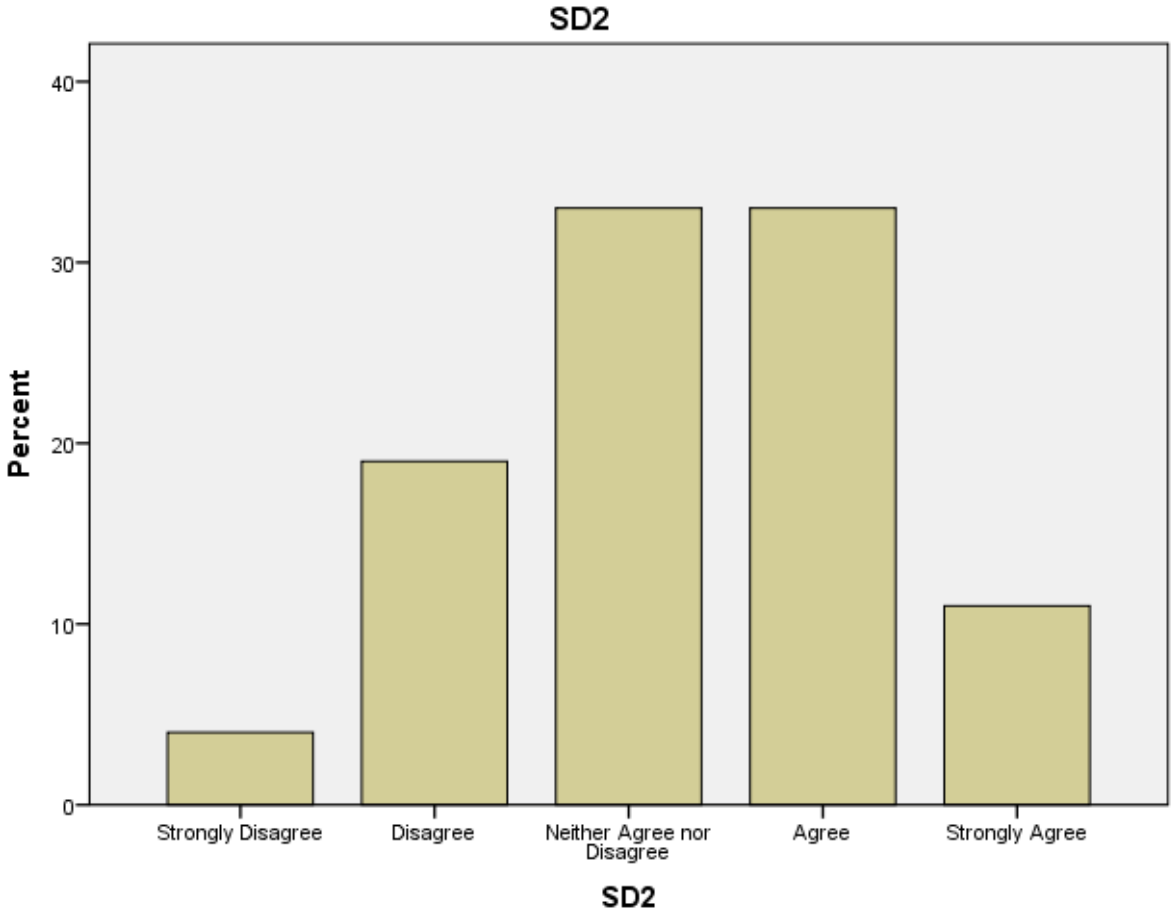
SD1

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	7	7.0	7.0	7.0
	Disagree	21	21.0	21.0	28.0
	Neither Agree nor Disagree	25	25.0	25.0	53.0
	Agree	33	33.0	33.0	86.0
	Strongly Agree	14	14.0	14.0	100.0
	Total	100	100.0	100.0	


SD1
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
SD1

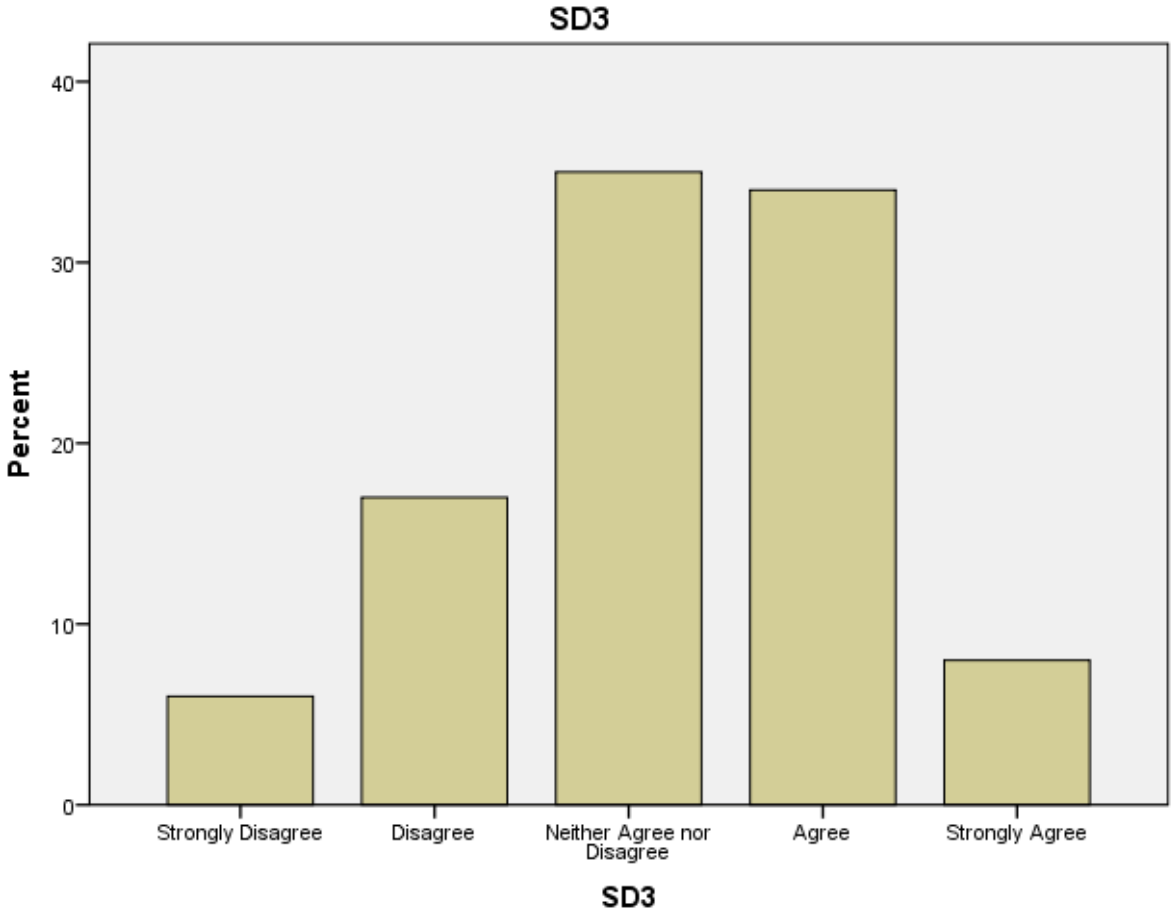
SD2

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	4	4.0	4.0	4.0
	Disagree	19	19.0	19.0	23.0
	Neither Agree nor Disagree	33	33.0	33.0	56.0
	Agree	33	33.0	33.0	89.0
	Strongly Agree	11	11.0	11.0	100.0
	Total	100	100.0	100.0	


SD2
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
SD2

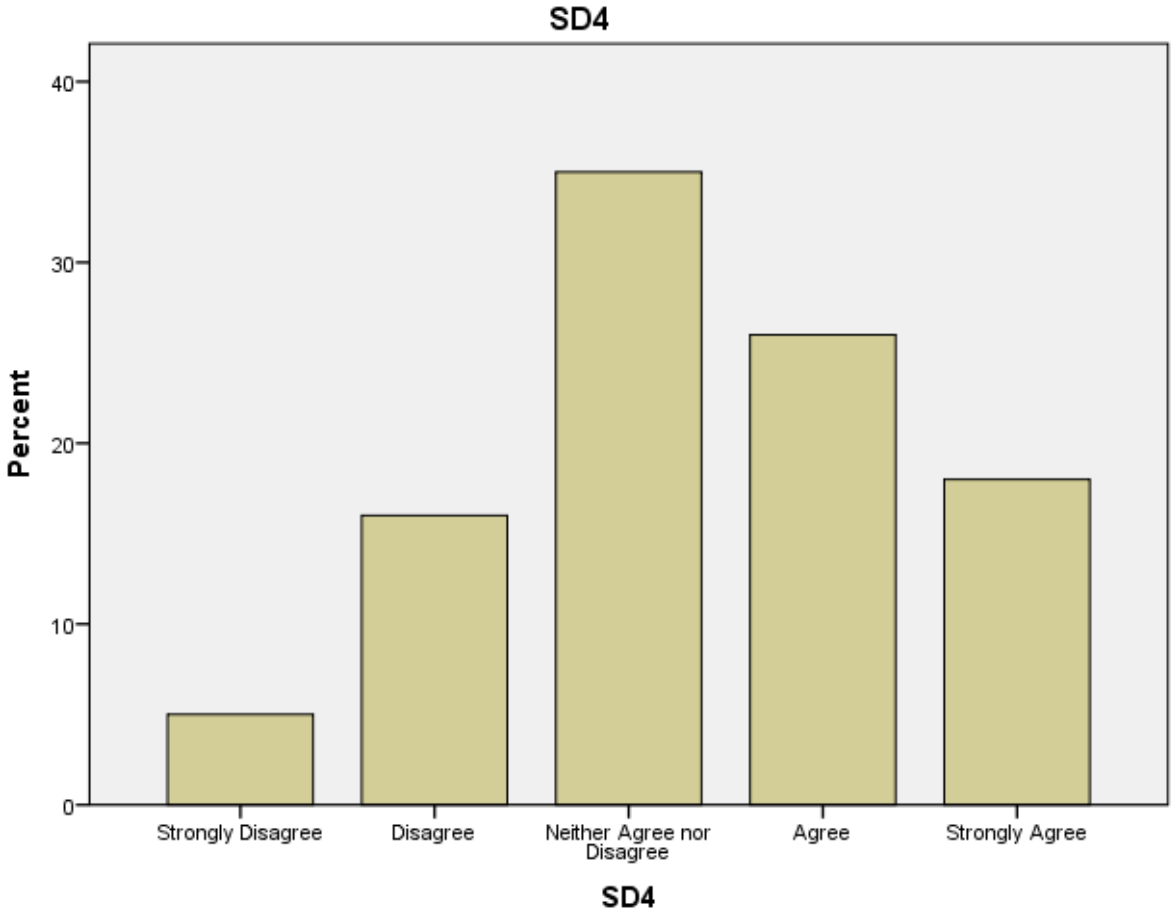
SD3

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	6	6.0	6.0	6.0
	Disagree	17	17.0	17.0	23.0
	Neither Agree nor Disagree	35	35.0	35.0	58.0
	Agree	34	34.0	34.0	92.0
	Strongly Agree	8	8.0	8.0	100.0
	Total	100	100.0	100.0	


SD3
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
SD3

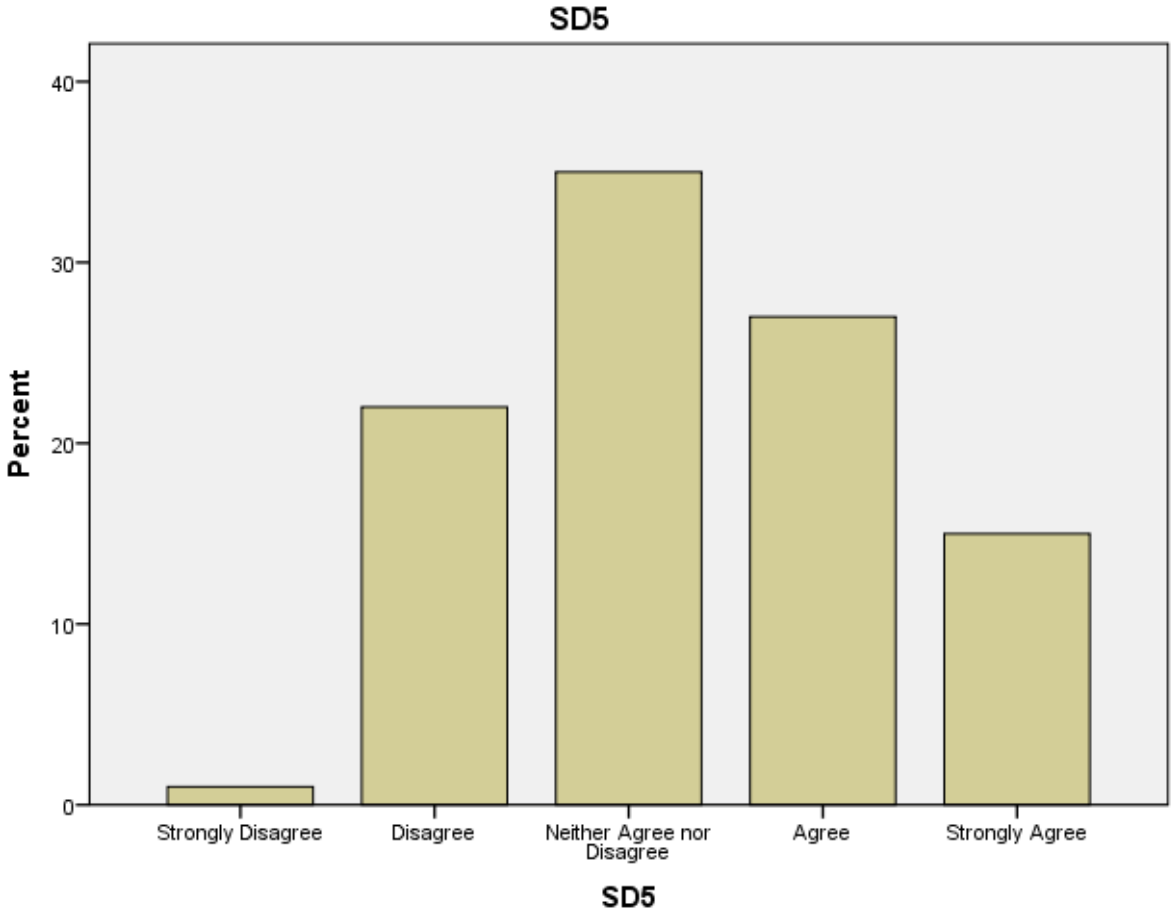
SD4

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	5	5.0	5.0	5.0
	Disagree	16	16.0	16.0	21.0
	Neither Agree nor Disagree	35	35.0	35.0	56.0
	Agree	26	26.0	26.0	82.0
	Strongly Agree	18	18.0	18.0	100.0
	Total	100	100.0	100.0	


SD4
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
SD4

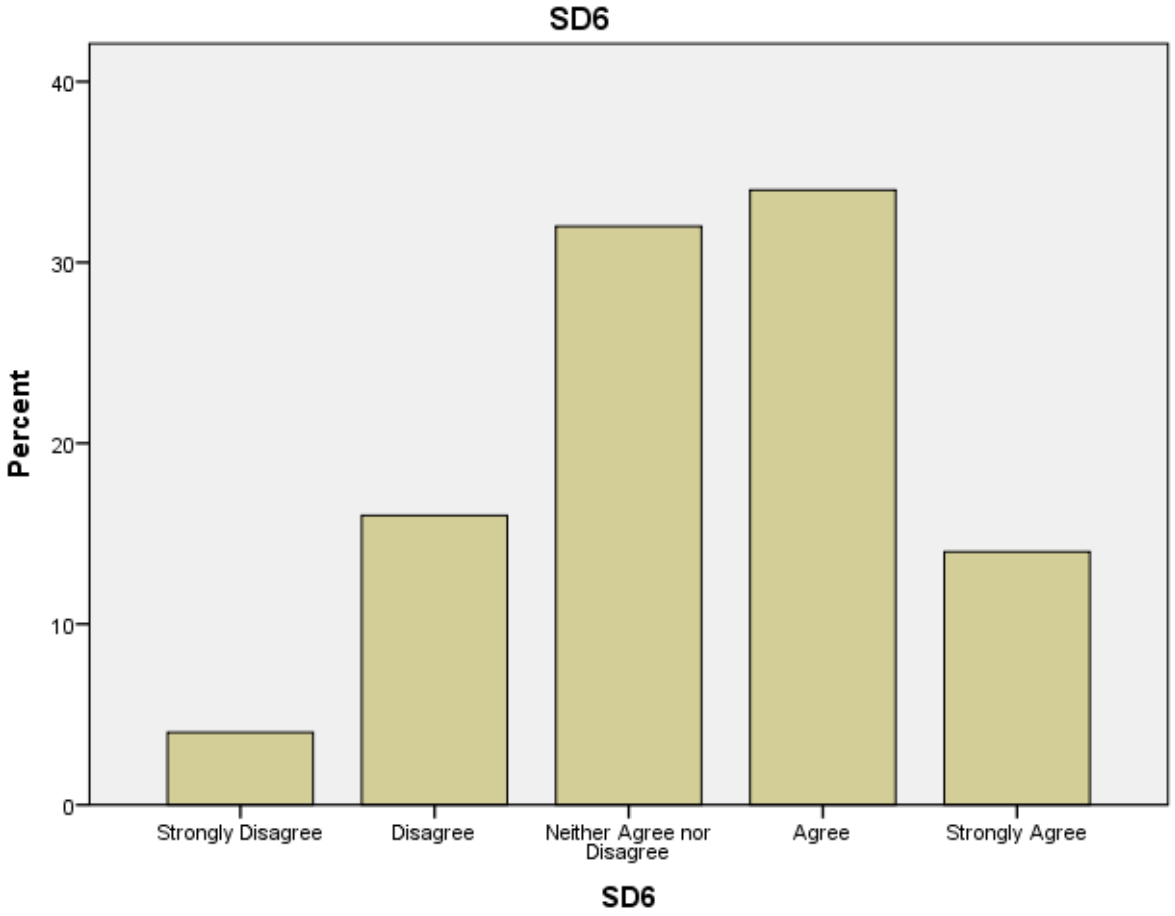
SD5

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	1	1.0	1.0	1.0
	Disagree	22	22.0	22.0	23.0
	Neither Agree nor Disagree	35	35.0	35.0	58.0
	Agree	27	27.0	27.0	85.0
	Strongly Agree	15	15.0	15.0	100.0
	Total	100	100.0	100.0	


SD5
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
SD5

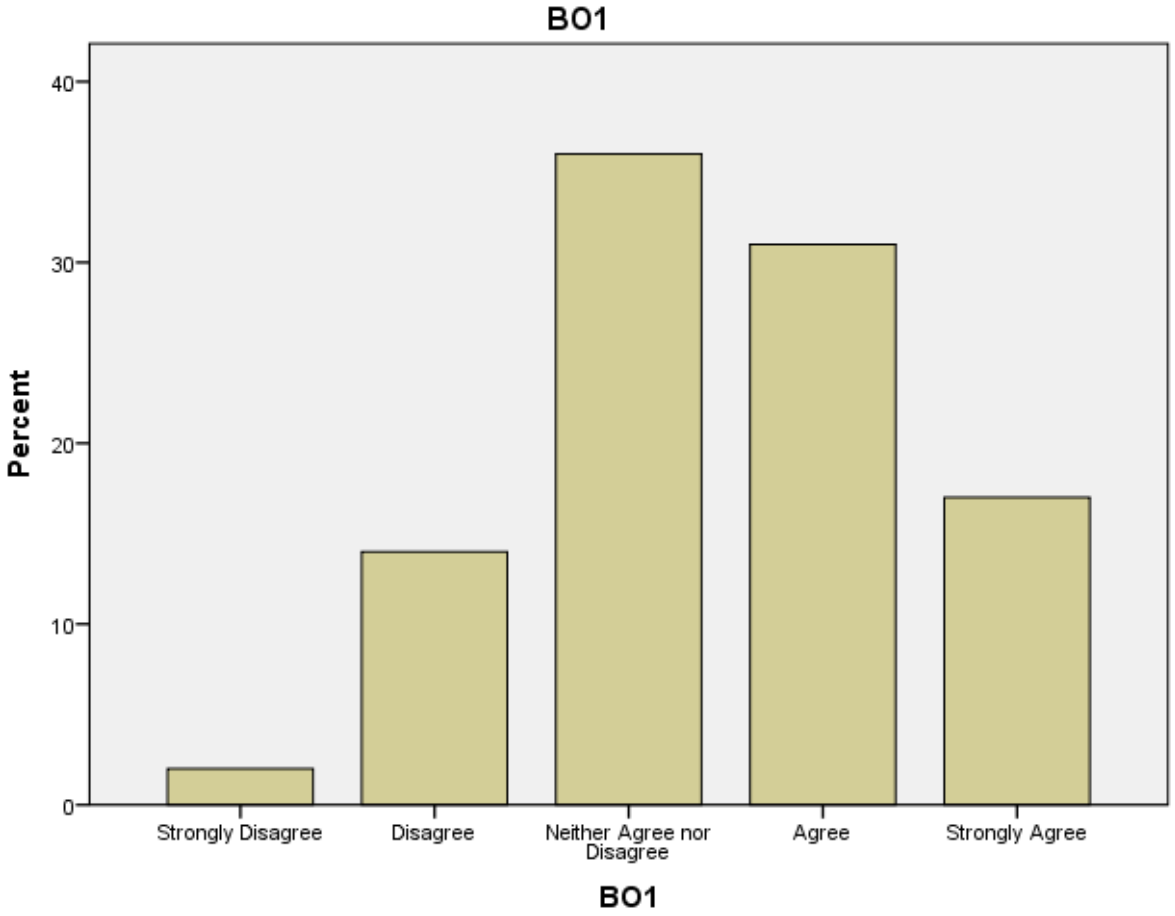
SD6

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	4	4.0	4.0	4.0
	Disagree	16	16.0	16.0	20.0
	Neither Agree nor Disagree	32	32.0	32.0	52.0
	Agree	34	34.0	34.0	86.0
	Strongly Agree	14	14.0	14.0	100.0
	Total	100	100.0	100.0	


SD6
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
SD6

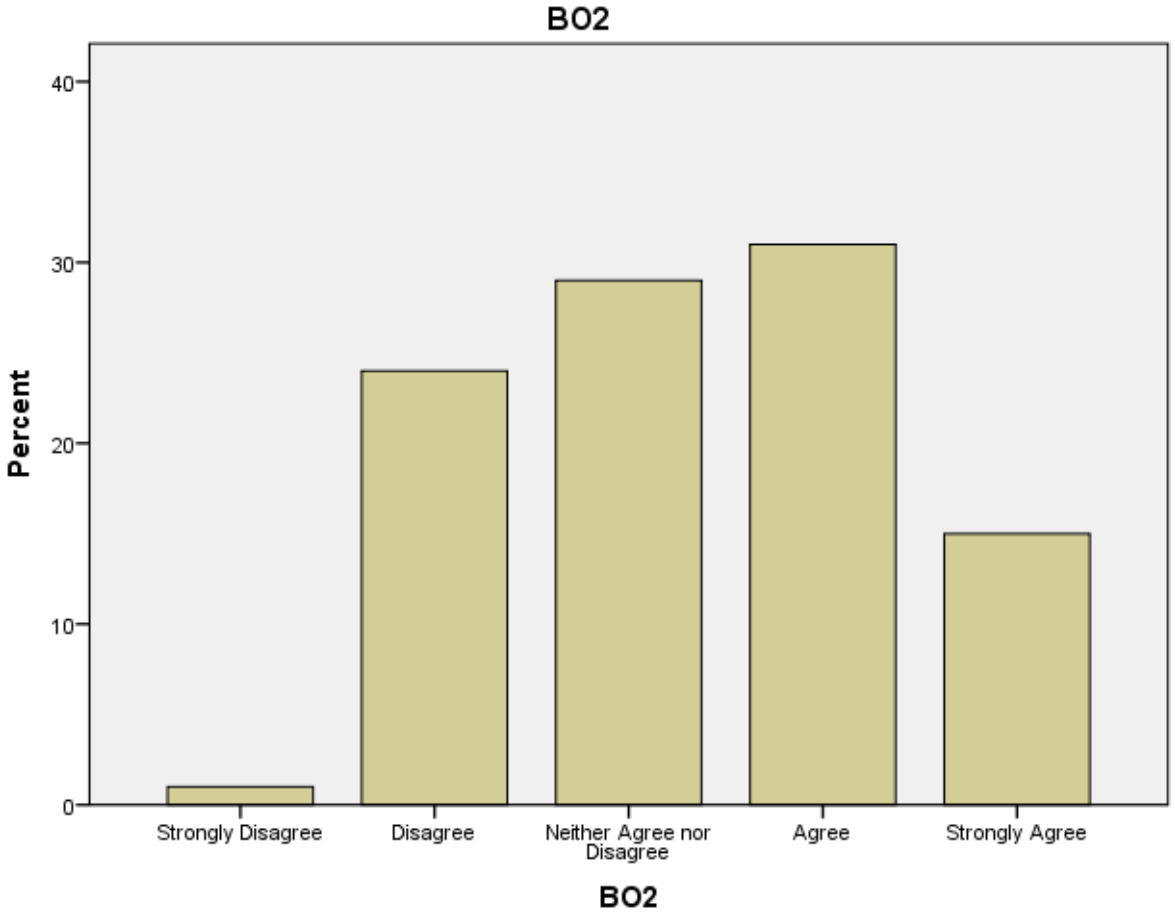
BO1

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	2	2.0	2.0	2.0
	Disagree	14	14.0	14.0	16.0
	Neither Agree nor Disagree	36	36.0	36.0	52.0
	Agree	31	31.0	31.0	83.0
	Strongly Agree	17	17.0	17.0	100.0
	Total	100	100.0	100.0	


BO1
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
BO1

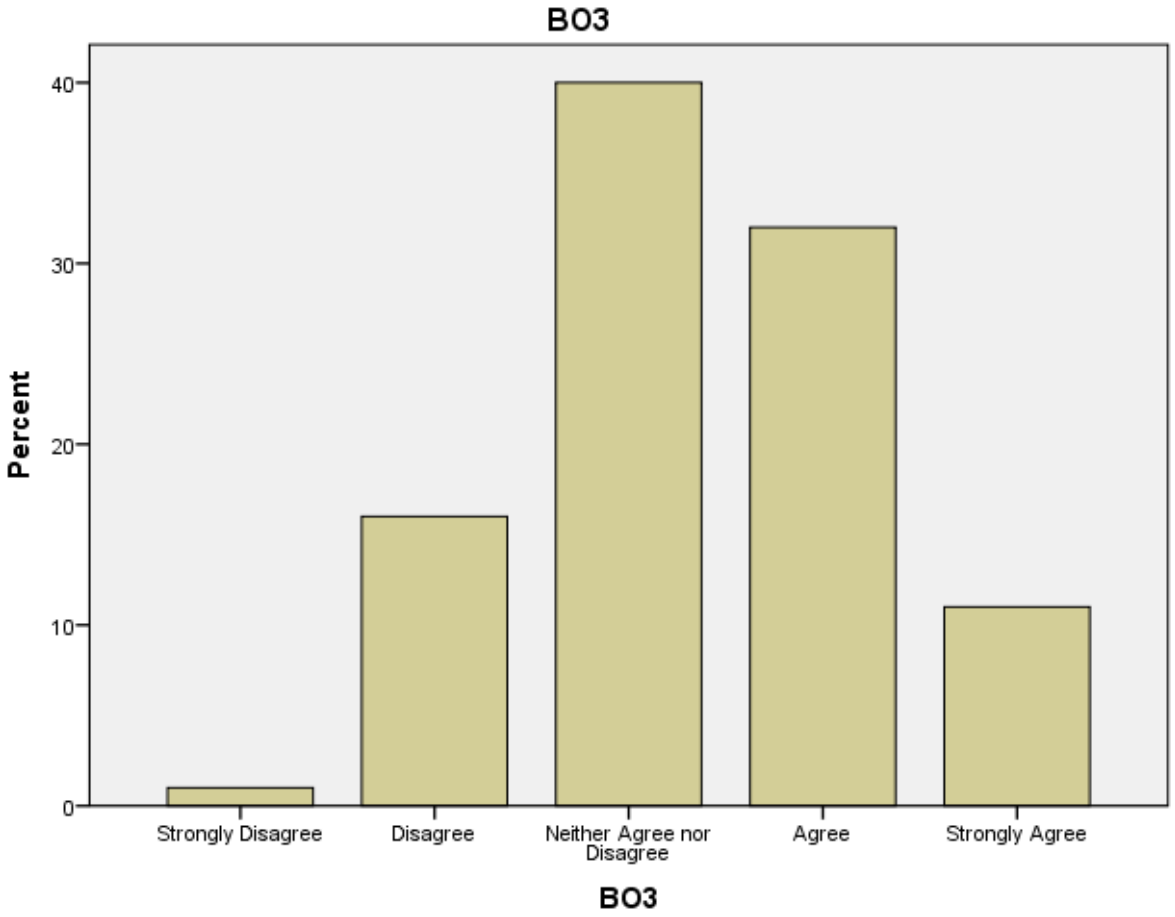
BO2

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	1	1.0	1.0	1.0
	Disagree	24	24.0	24.0	25.0
	Neither Agree nor Disagree	29	29.0	29.0	54.0
	Agree	31	31.0	31.0	85.0
	Strongly Agree	15	15.0	15.0	100.0
	Total	100	100.0	100.0	


BO2
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
BO2

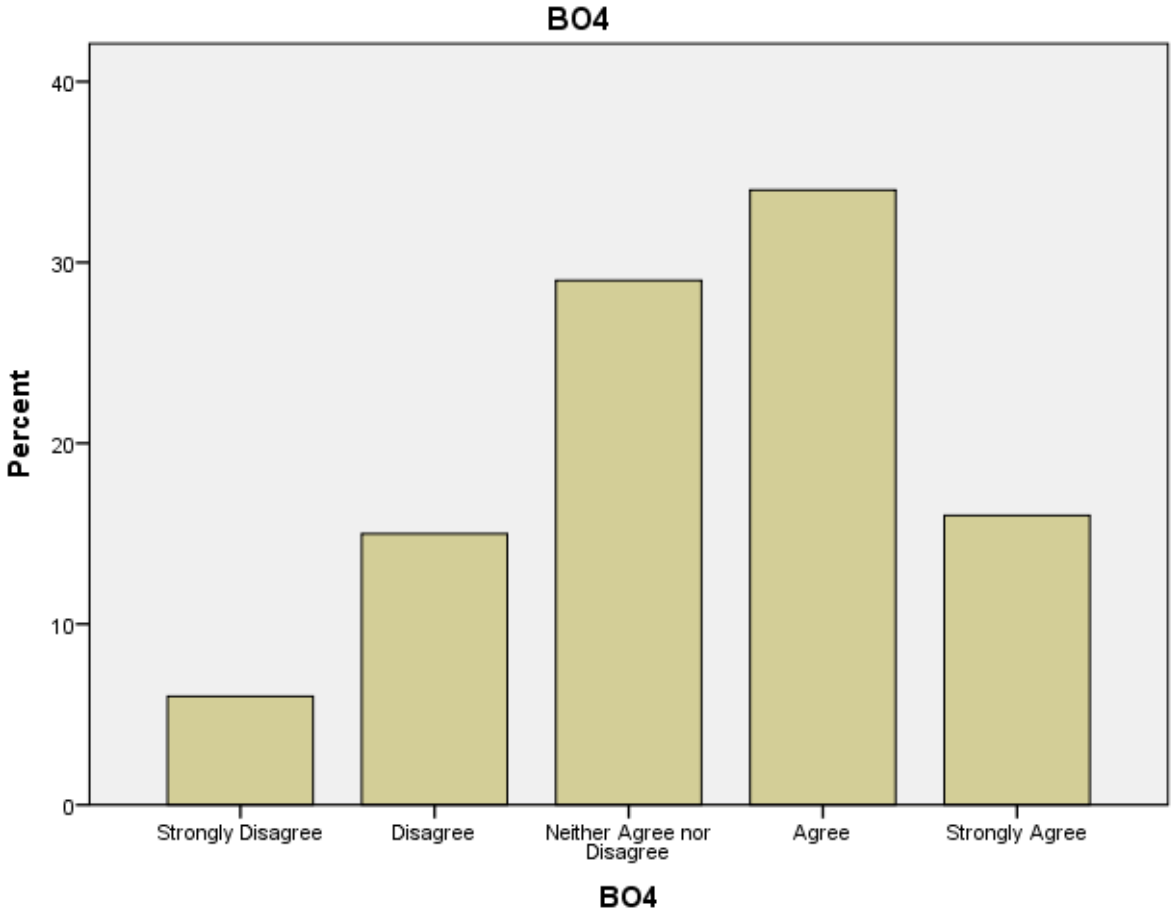
BO3

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	1	1.0	1.0	1.0
	Disagree	16	16.0	16.0	17.0
	Neither Agree nor Disagree	40	40.0	40.0	57.0
	Agree	32	32.0	32.0	89.0
	Strongly Agree	11	11.0	11.0	100.0
	Total	100	100.0	100.0	


BO3
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
BO3

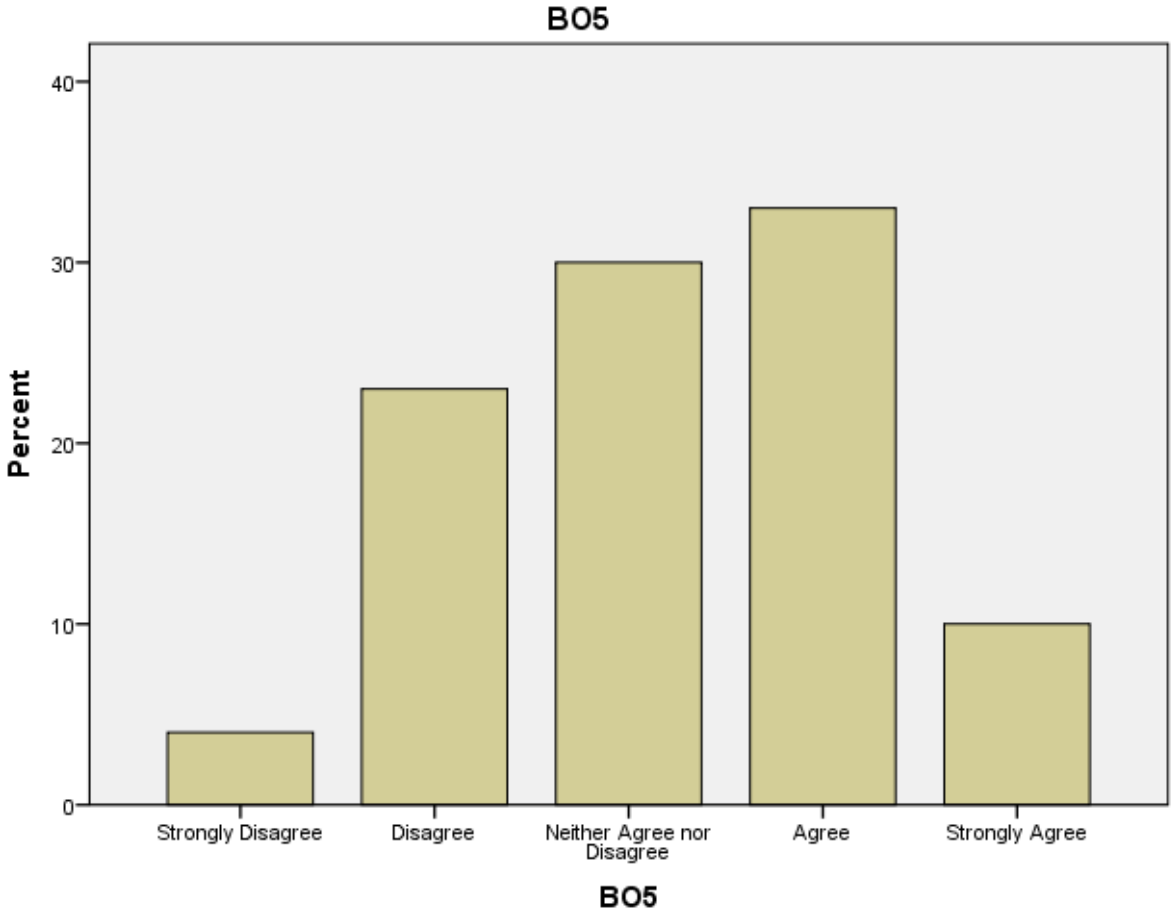
BO4

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	6	6.0	6.0	6.0
	Disagree	15	15.0	15.0	21.0
	Neither Agree nor Disagree	29	29.0	29.0	50.0
	Agree	34	34.0	34.0	84.0
	Strongly Agree	16	16.0	16.0	100.0
	Total	100	100.0	100.0	


BO4
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
BO4

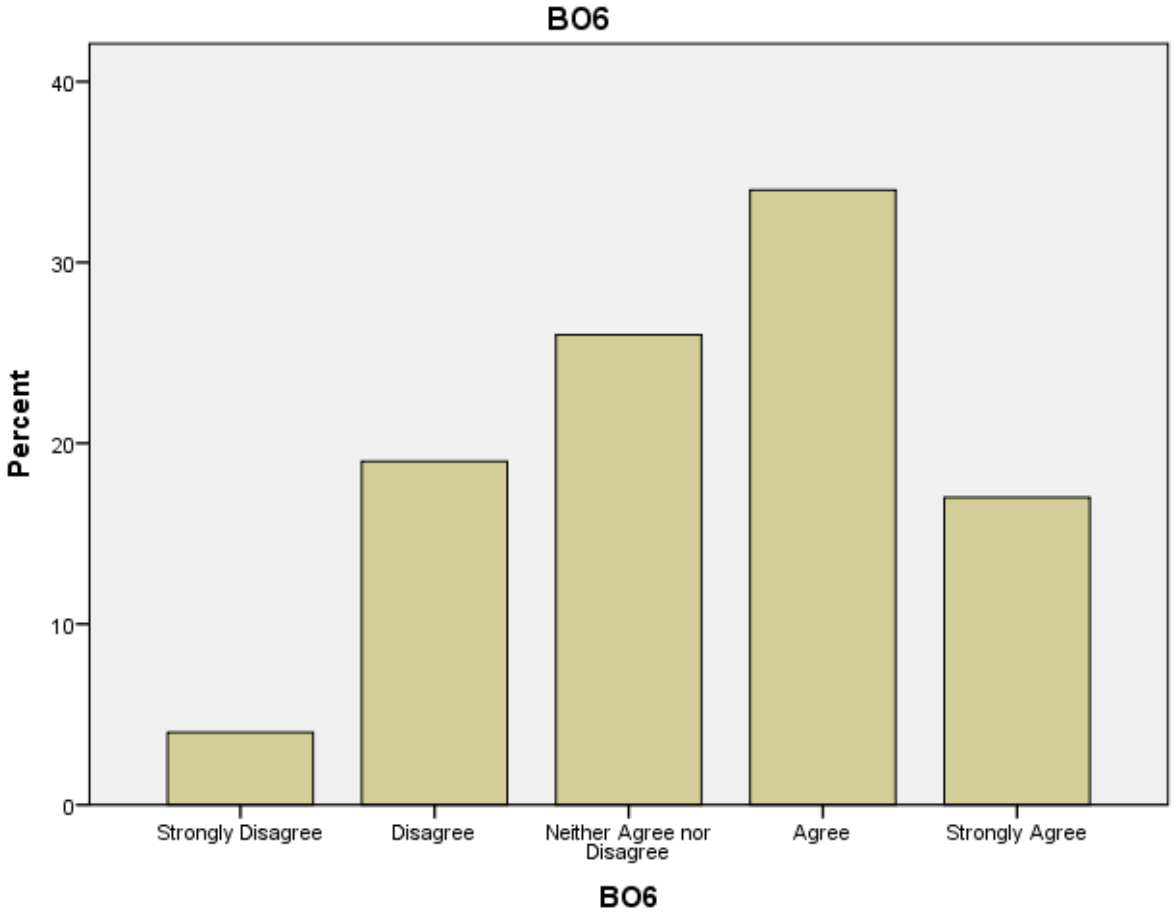
BO5

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	4	4.0	4.0	4.0
	Disagree	23	23.0	23.0	27.0
	Neither Agree nor Disagree	30	30.0	30.0	57.0
	Agree	33	33.0	33.0	90.0
	Strongly Agree	10	10.0	10.0	100.0
	Total	100	100.0	100.0	


BO5
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
BO5

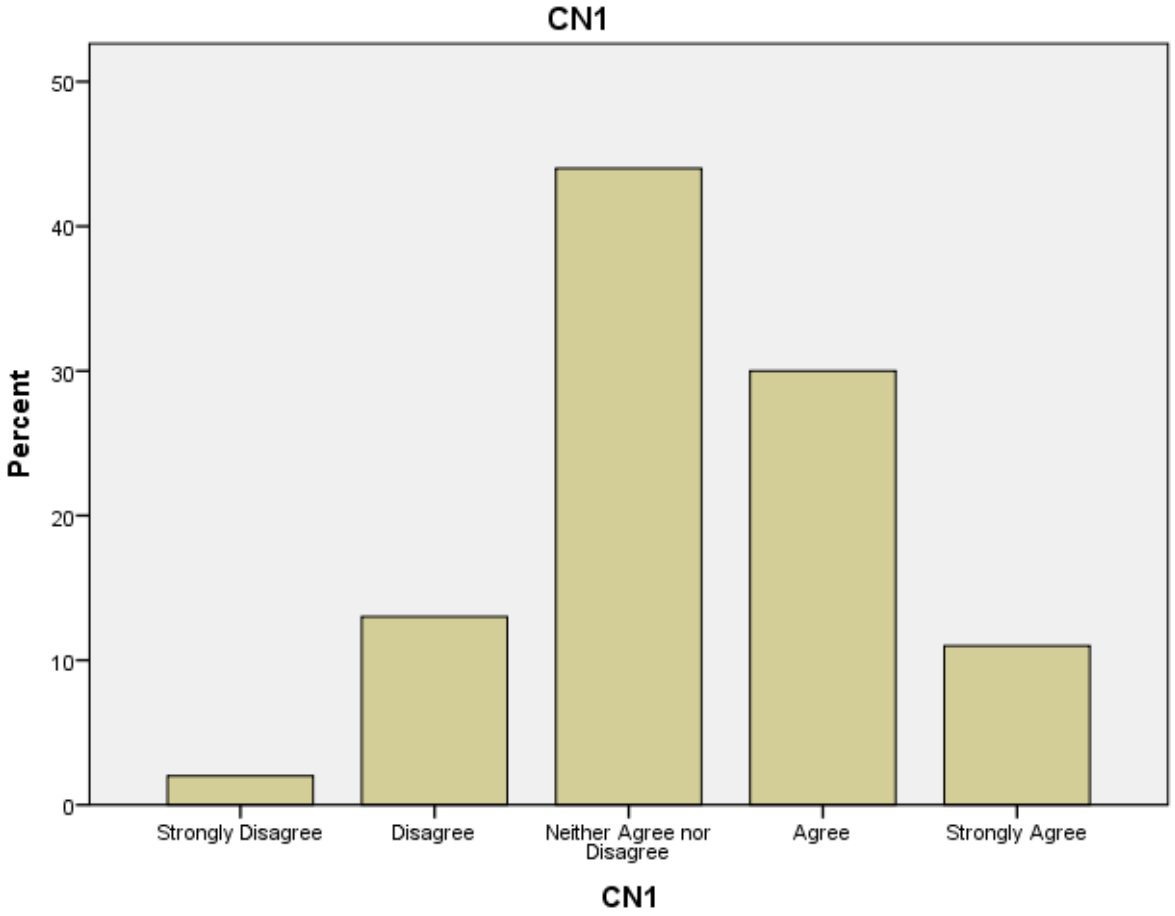
BO6

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	4	4.0	4.0	4.0
	Disagree	19	19.0	19.0	23.0
	Neither Agree nor Disagree	26	26.0	26.0	49.0
	Agree	34	34.0	34.0	83.0
	Strongly Agree	17	17.0	17.0	100.0
	Total	100	100.0	100.0	


BO6
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
BO6

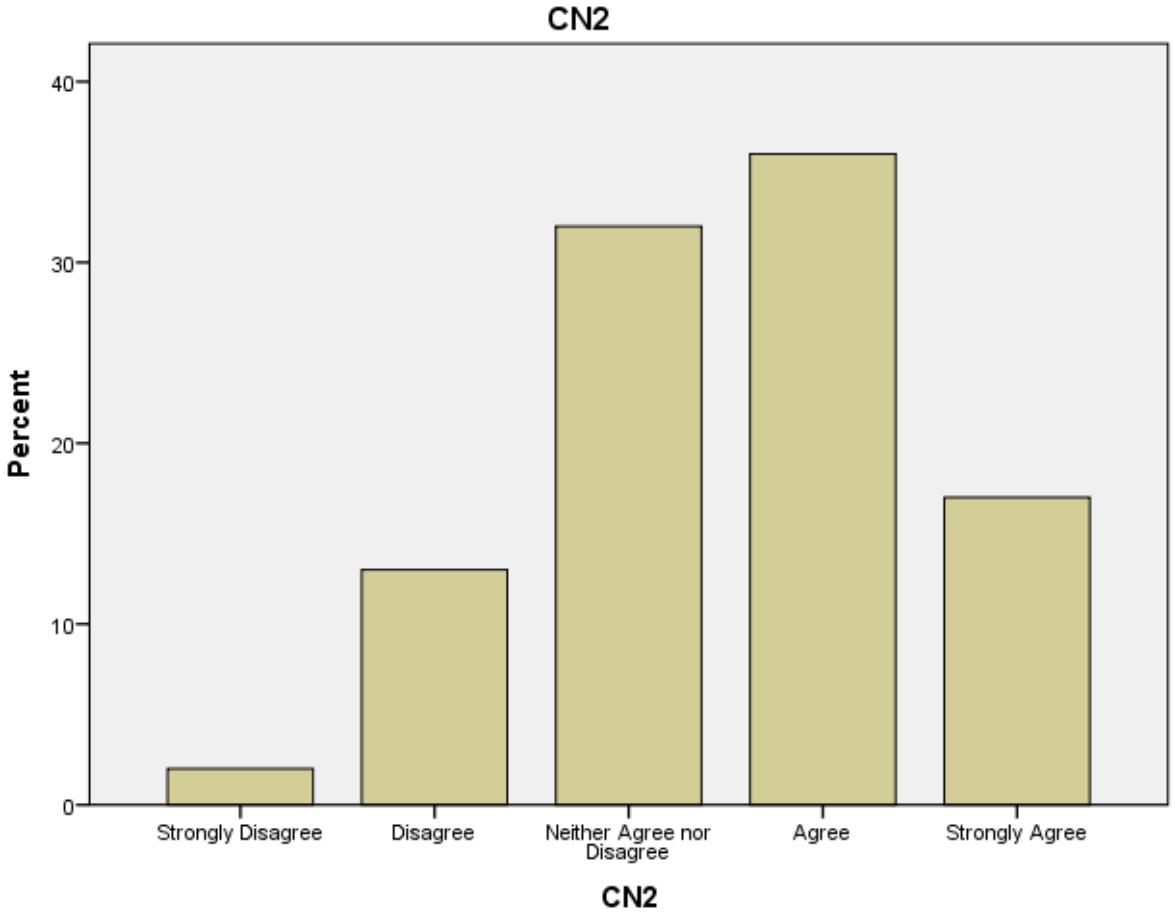
CN1

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	2	2.0	2.0	2.0
	Disagree	13	13.0	13.0	15.0
	Neither Agree nor Disagree	44	44.0	44.0	59.0
	Agree	30	30.0	30.0	89.0
	Strongly Agree	11	11.0	11.0	100.0
	Total	100	100.0	100.0	


CN1
Percent
50
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
CN1

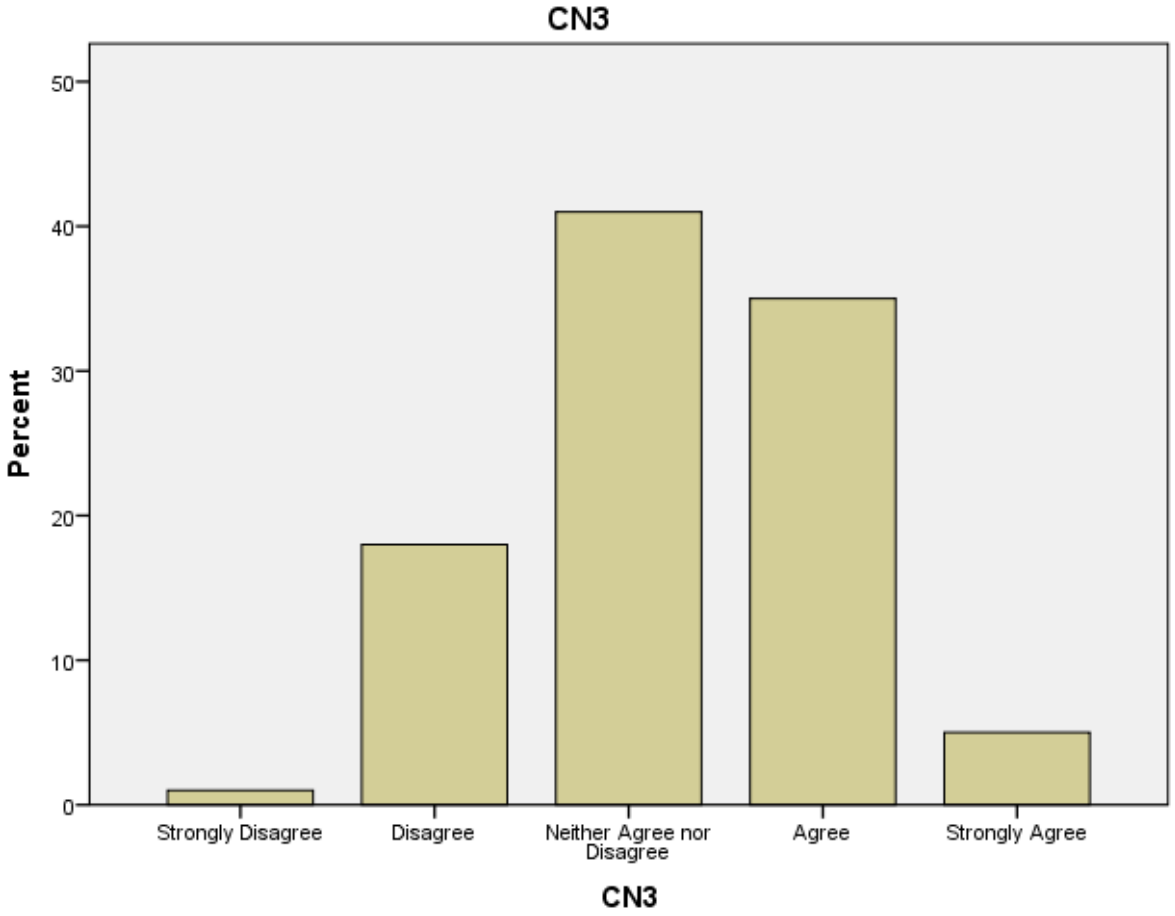
CN2

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	2	2.0	2.0	2.0
	Disagree	13	13.0	13.0	15.0
	Neither Agree nor Disagree	32	32.0	32.0	47.0
	Agree	36	36.0	36.0	83.0
	Strongly Agree	17	17.0	17.0	100.0
	Total	100	100.0	100.0	


CN2
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
CN2

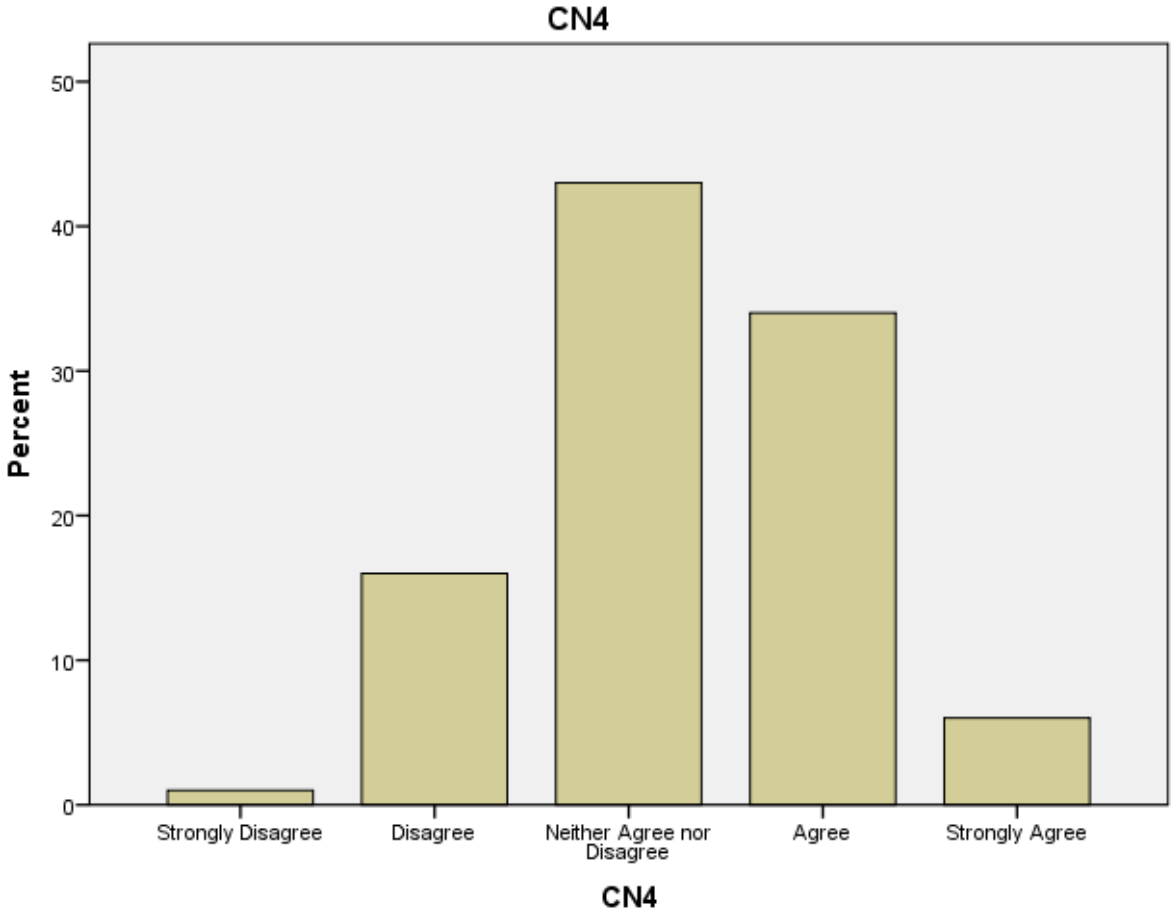
CN3

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	1	1.0	1.0	1.0
	Disagree	18	18.0	18.0	19.0
	Neither Agree nor Disagree	41	41.0	41.0	60.0
	Agree	35	35.0	35.0	95.0
	Strongly Agree	5	5.0	5.0	100.0
	Total	100	100.0	100.0	


CN3
Percent
50
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
CN3

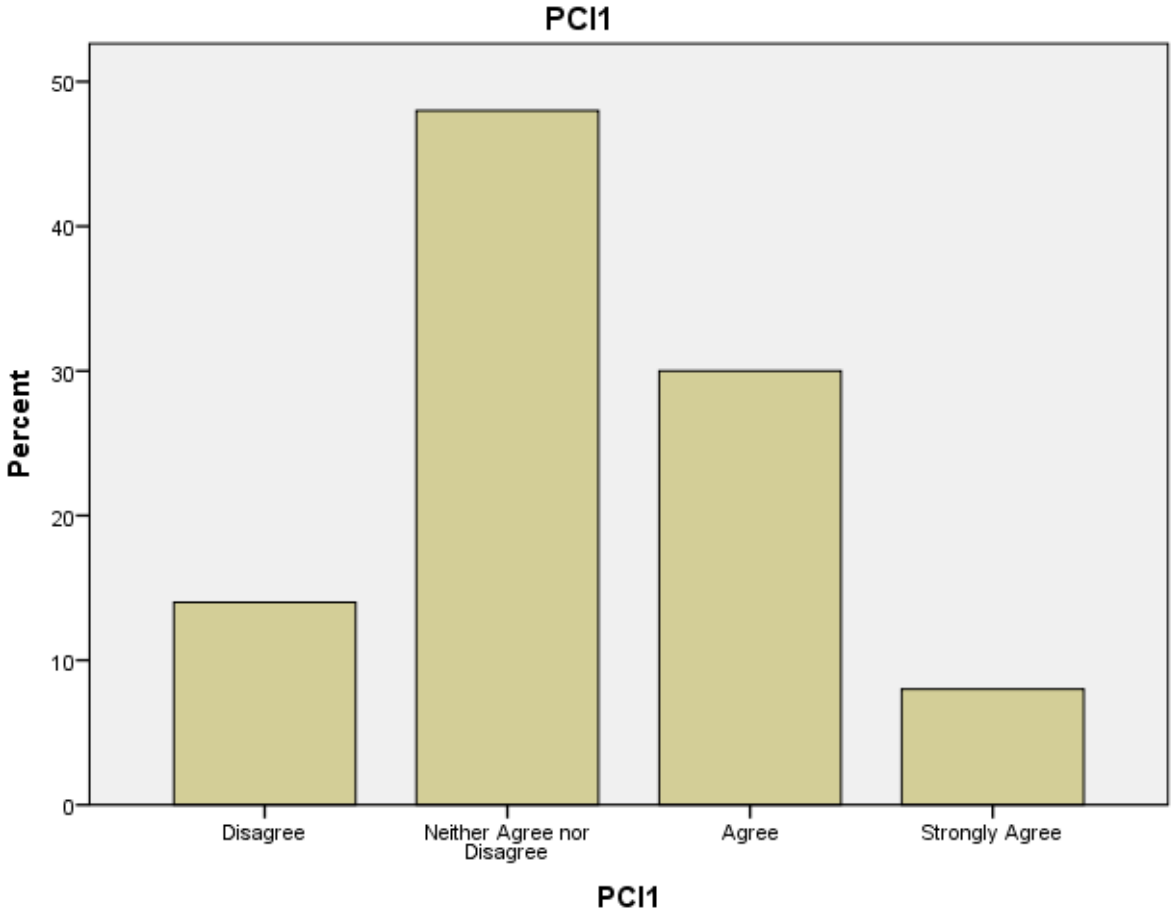
CN4

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	1	1.0	1.0	1.0
	Disagree	16	16.0	16.0	17.0
	Neither Agree nor Disagree	43	43.0	43.0	60.0
	Agree	34	34.0	34.0	94.0
	Strongly Agree	6	6.0	6.0	100.0
	Total	100	100.0	100.0	


CN4
Percent
50
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
CN4

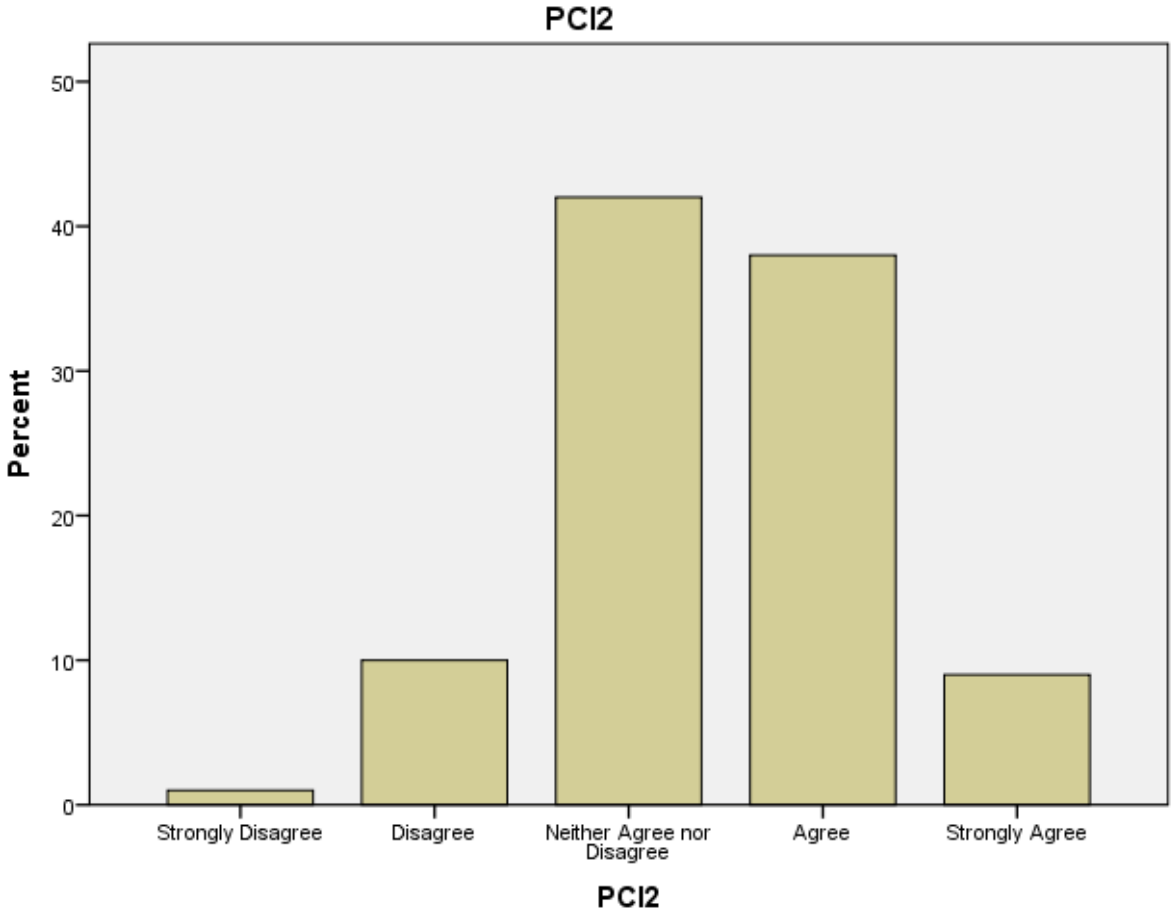
PCI1

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Disagree	14	14.0	14.0	14.0
	Neither Agree nor Disagree	48	48.0	48.0	62.0
	Agree	30	30.0	30.0	92.0
	Strongly Agree	8	8.0	8.0	100.0
	Total	100	100.0	100.0	


PCI1
Percent
50
40
30
20
10
0
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
PCI1

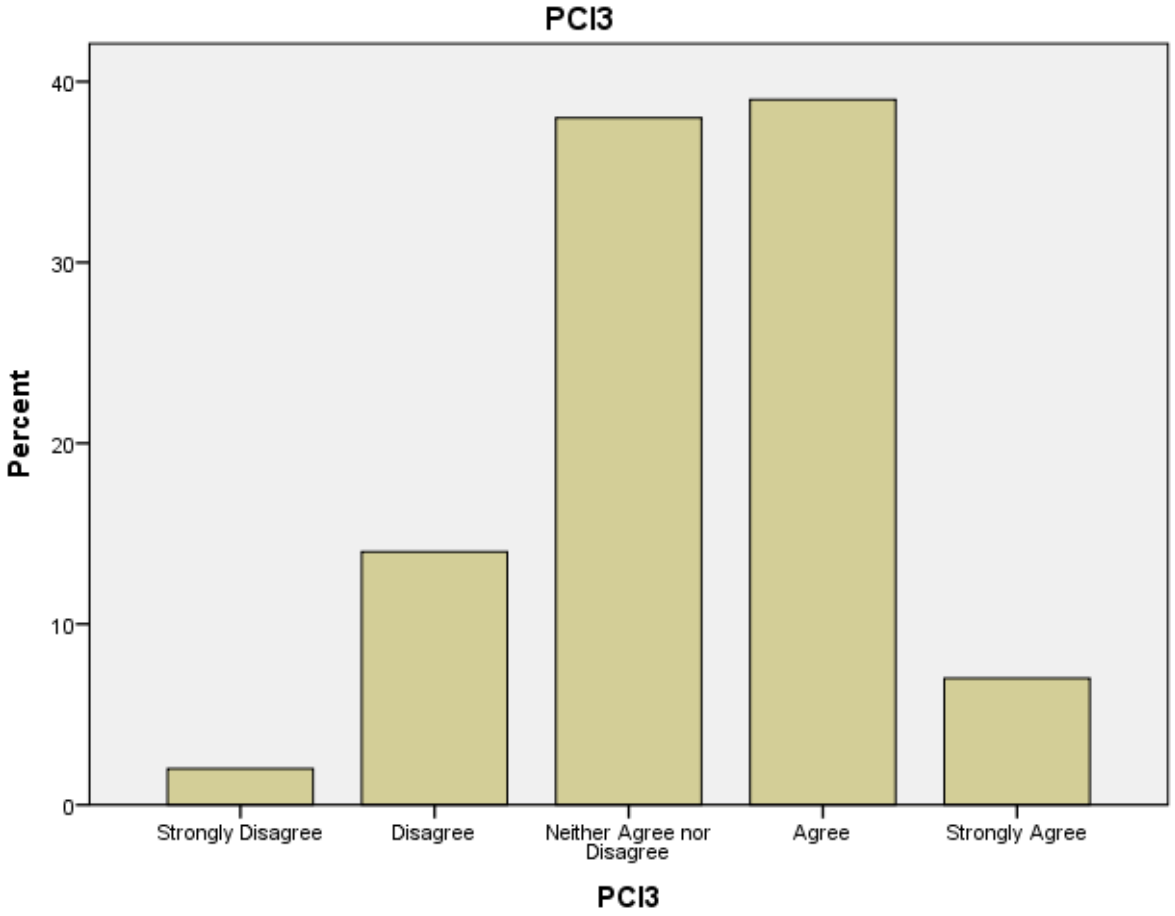
PCI2

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	1	1.0	1.0	1.0
	Disagree	10	10.0	10.0	11.0
	Neither Agree nor Disagree	42	42.0	42.0	53.0
	Agree	38	38.0	38.0	91.0
	Strongly Agree	9	9.0	9.0	100.0
	Total	100	100.0	100.0	


PCI2
Percent
50
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
PCI2

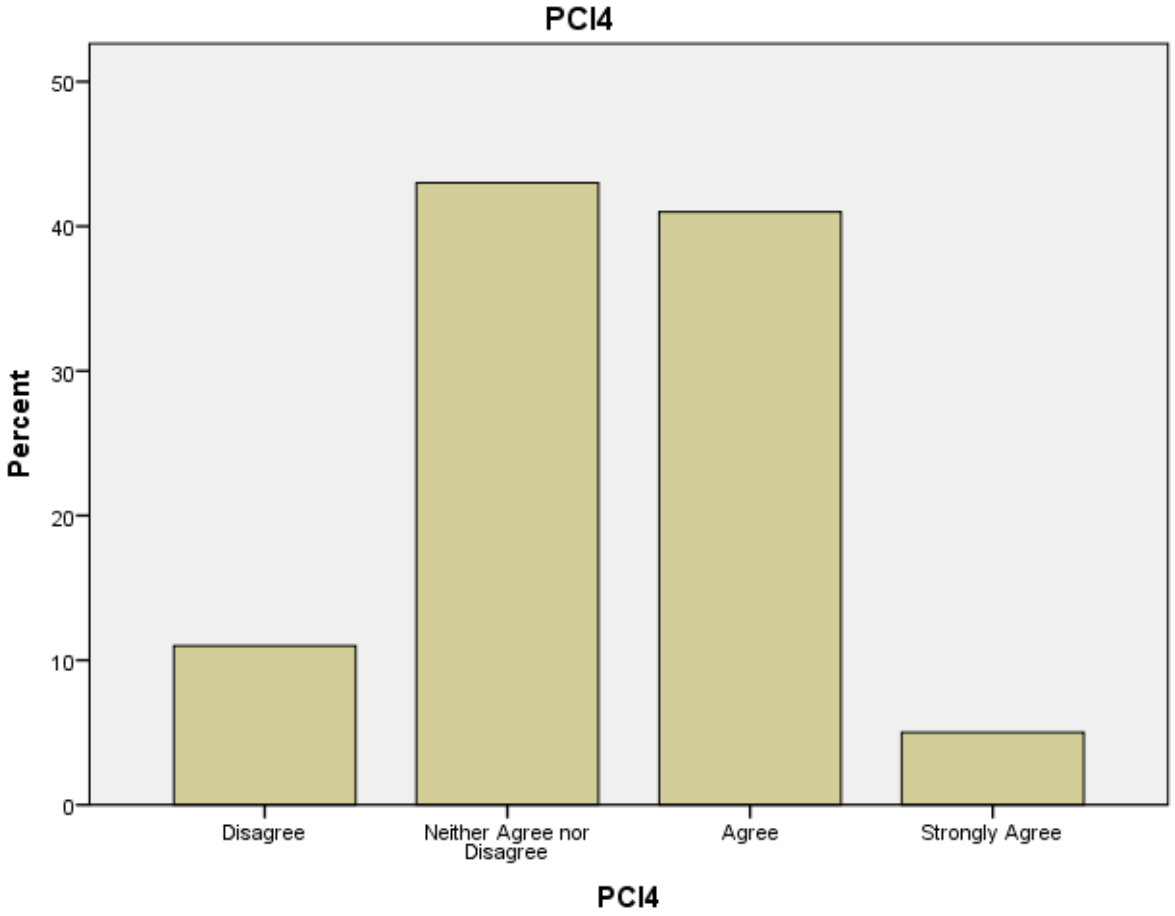
PCI3

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	2	2.0	2.0	2.0
	Disagree	14	14.0	14.0	16.0
	Neither Agree nor Disagree	38	38.0	38.0	54.0
	Agree	39	39.0	39.0	93.0
	Strongly Agree	7	7.0	7.0	100.0
	Total	100	100.0	100.0	


PCI3
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
PCI3

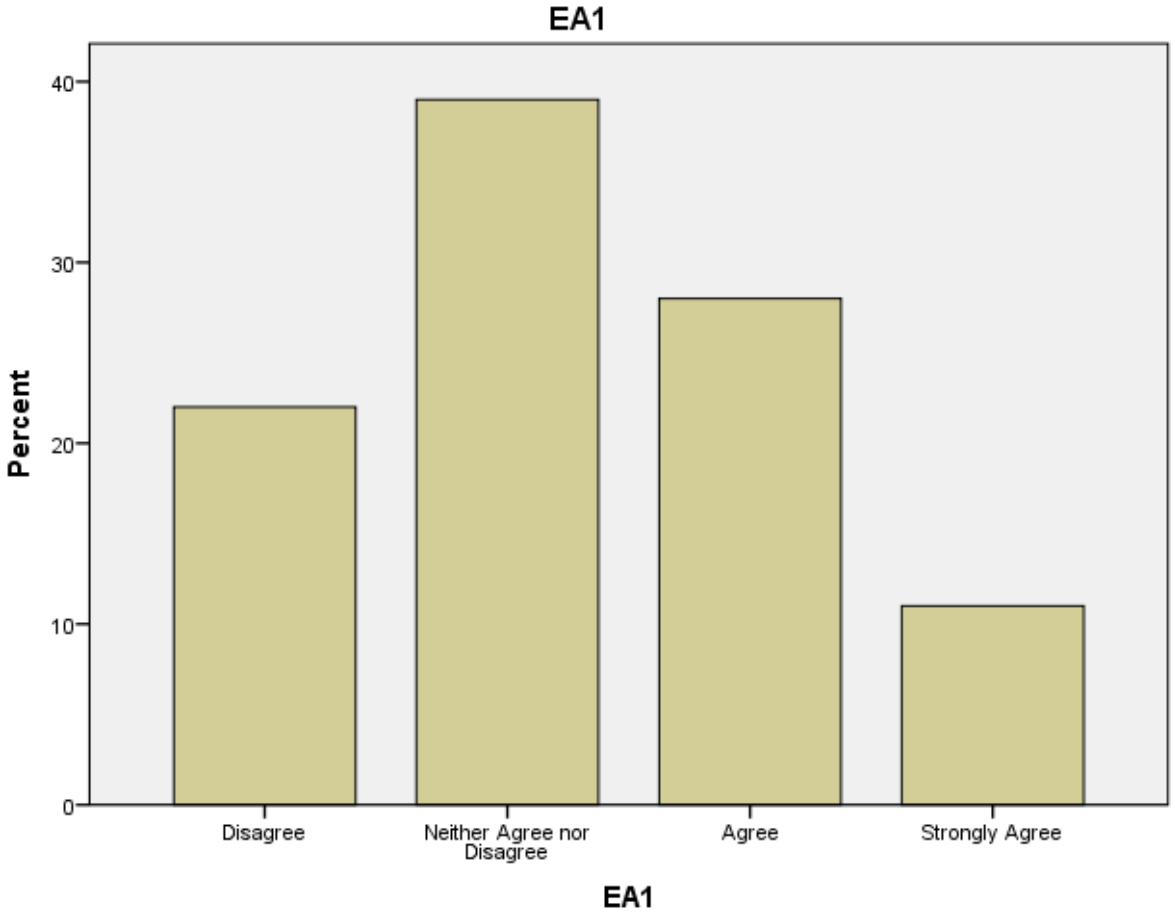
PCI4

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Disagree	11	11.0	11.0	11.0
	Neither Agree nor Disagree	43	43.0	43.0	54.0
	Agree	41	41.0	41.0	95.0
	Strongly Agree	5	5.0	5.0	100.0
	Total	100	100.0	100.0	


PCI4
Percent
50
40
30
20
10
0
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
PCI4

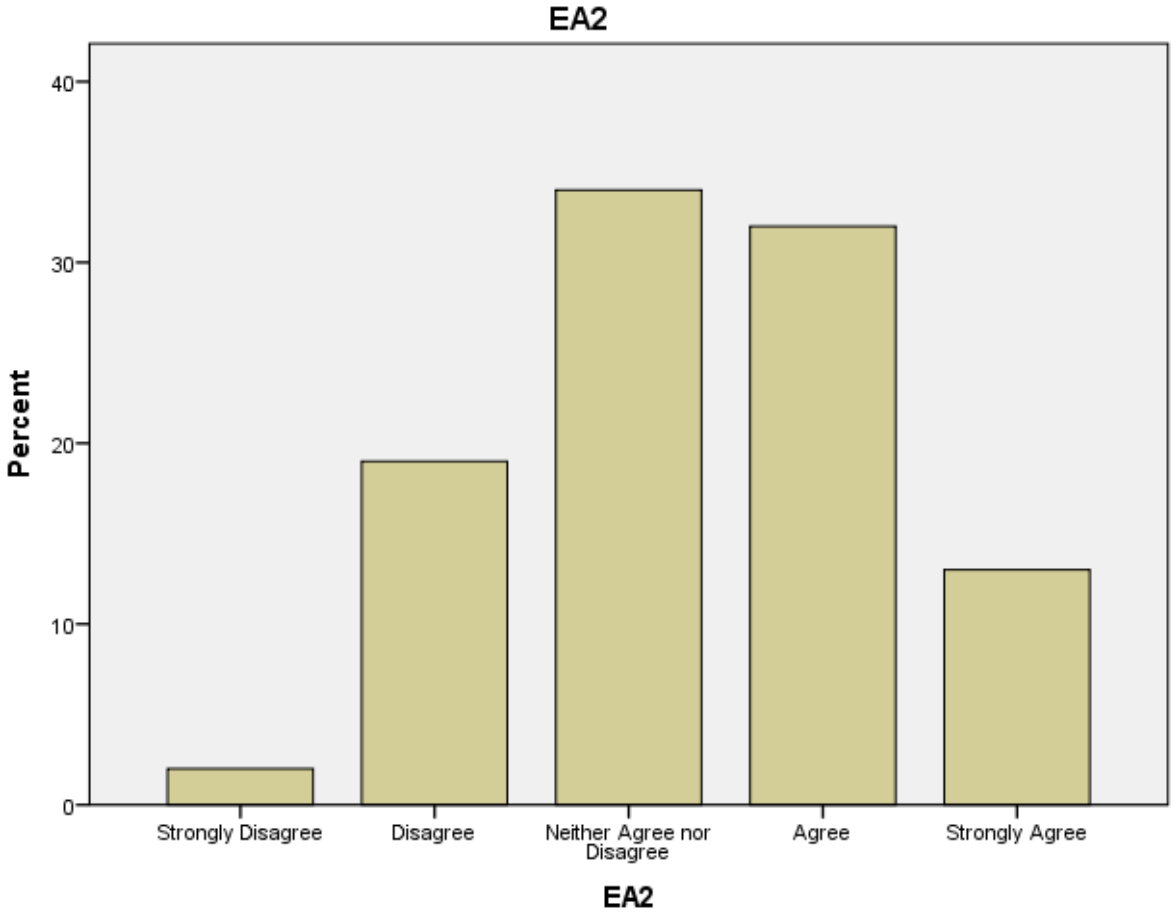
EA1

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Disagree	22	22.0	22.0	22.0
	Neither Agree nor Disagree	39	39.0	39.0	61.0
	Agree	28	28.0	28.0	89.0
	Strongly Agree	11	11.0	11.0	100.0
	Total	100	100.0	100.0	


EA1
Percent
40
30
20
10
0
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
EA1

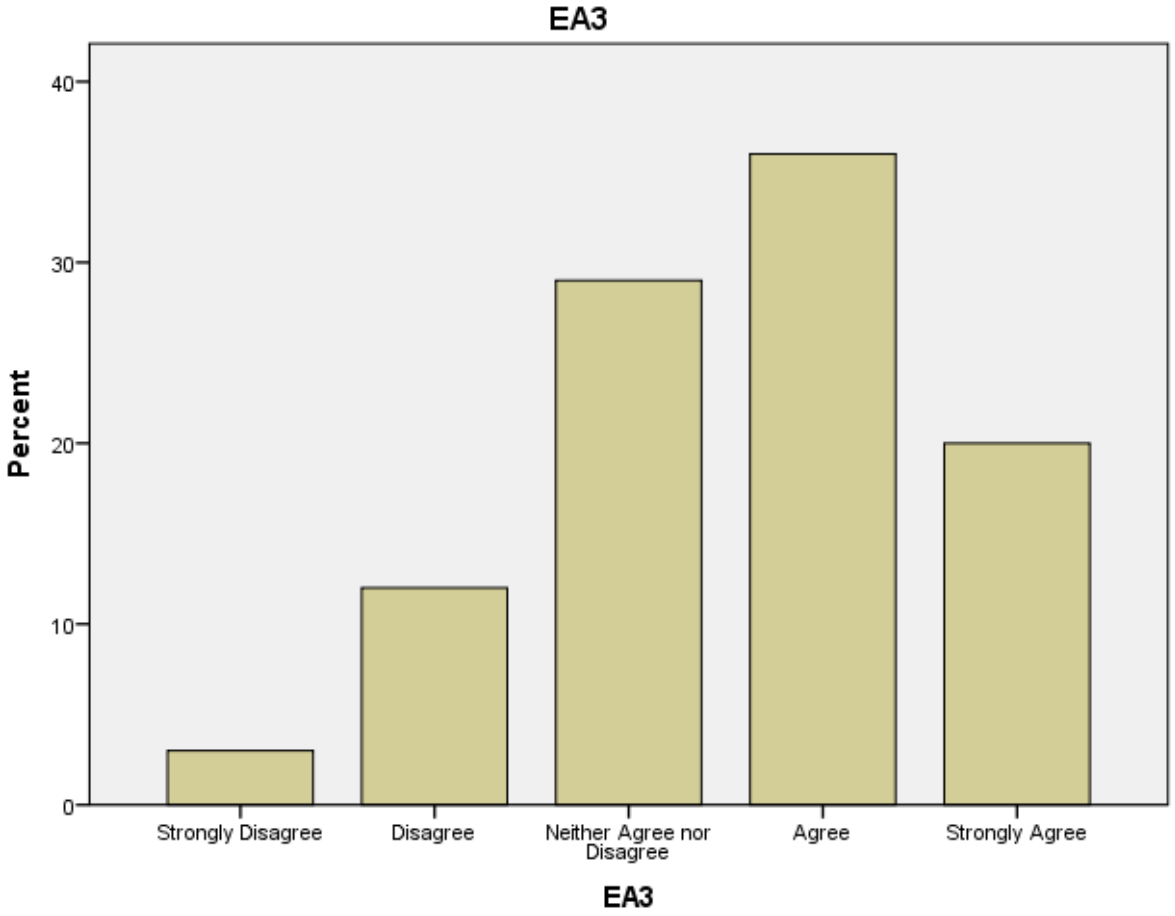
EA2

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	2	2.0	2.0	2.0
	Disagree	19	19.0	19.0	21.0
	Neither Agree nor Disagree	34	34.0	34.0	55.0
	Agree	32	32.0	32.0	87.0
	Strongly Agree	13	13.0	13.0	100.0
	Total	100	100.0	100.0	


EA2
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
EA2

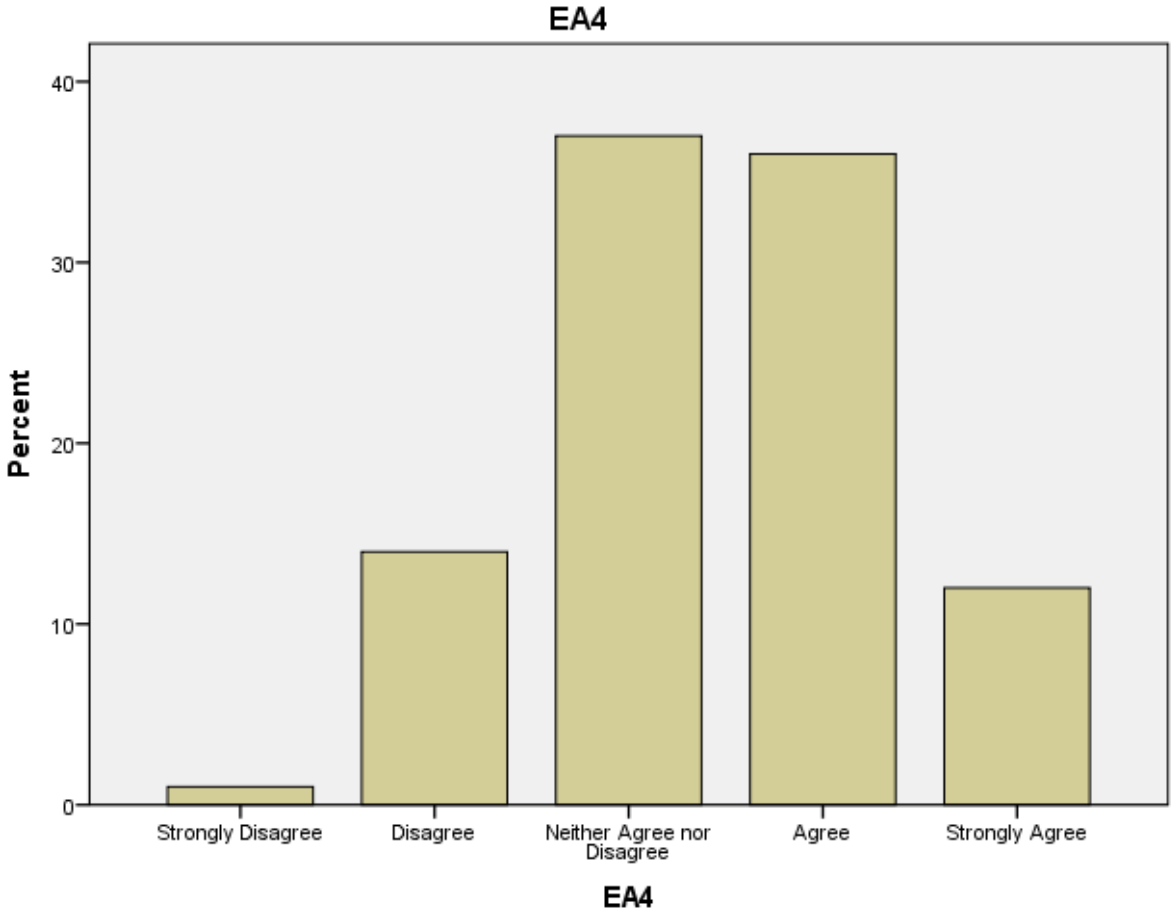
EA3

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	3	3.0	3.0	3.0
	Disagree	12	12.0	12.0	15.0
	Neither Agree nor Disagree	29	29.0	29.0	44.0
	Agree	36	36.0	36.0	80.0
	Strongly Agree	20	20.0	20.0	100.0
	Total	100	100.0	100.0	


EA3
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
EA3

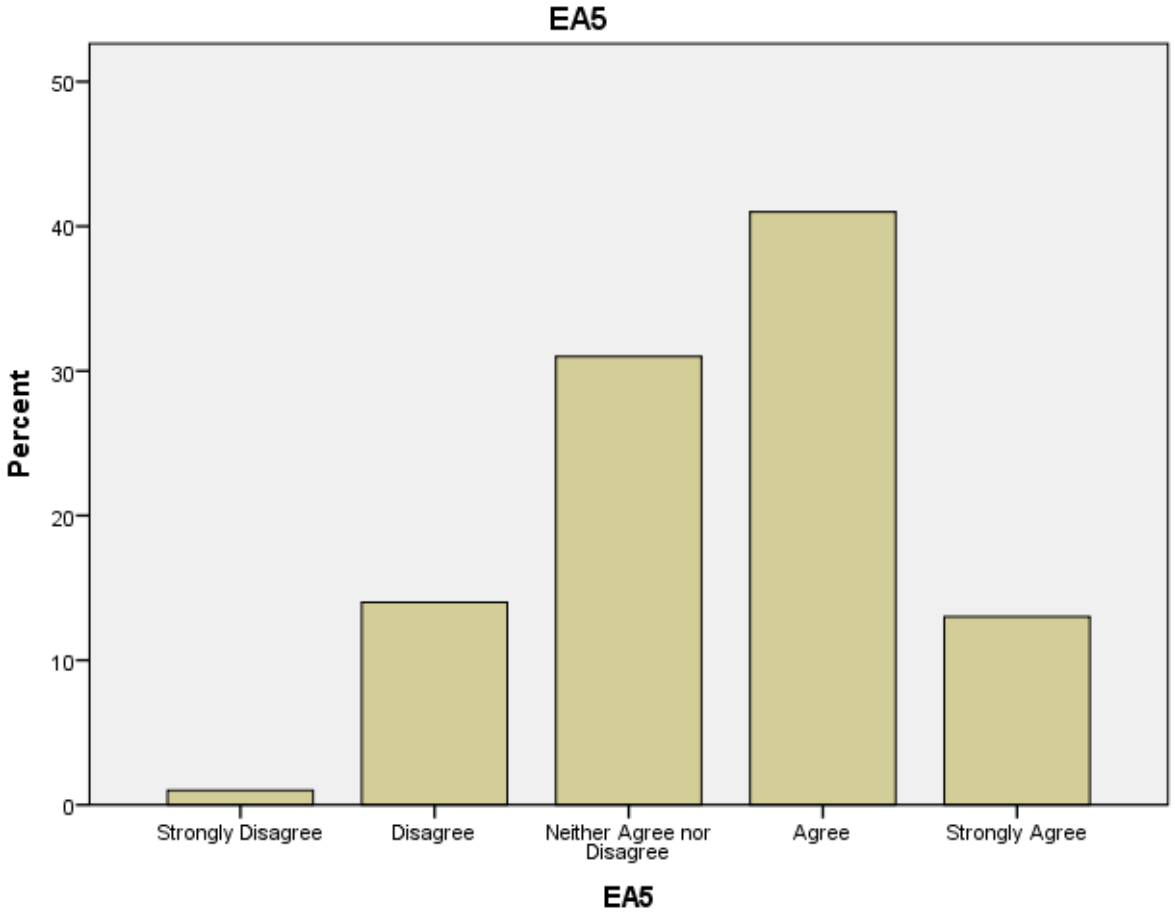
EA4

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	1	1.0	1.0	1.0
	Disagree	14	14.0	14.0	15.0
	Neither Agree nor Disagree	37	37.0	37.0	52.0
	Agree	36	36.0	36.0	88.0
	Strongly Agree	12	12.0	12.0	100.0
	Total	100	100.0	100.0	


EA4
Percent
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
EA4

EA5

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Strongly Disagree	1	1.0	1.0	1.0
	Disagree	14	14.0	14.0	15.0
	Neither Agree nor Disagree	31	31.0	31.0	46.0
	Agree	41	41.0	41.0	87.0
	Strongly Agree	13	13.0	13.0	100.0
	Total	100	100.0	100.0	


EA5
Percent
50
40
30
20
10
0
Strongly Disagree
Disagree
Neither Agree nor Disagree
Agree
Strongly Agree
EA5

Independent Sample Test

ER

Group Statistics

	Group	N	Mean	Std. Deviation	Std. Error Mean
ER1	1.00	53	3.8491	.79412	.10908
	2.00	47	2.8723	.87519	.12766
ER2	1.00	53	3.9811	.84331	.11584
	2.00	47	2.9362	.94188	.13739
ER3	1.00	53	3.9245	.64597	.08873
	2.00	47	2.8298	1.08986	.15897
ER4	1.00	53	3.8113	.70864	.09734

	2.00	47	2.8298	.86776	.12658
ER5	1.00	53	3.7358	.83553	.11477
	2.00	47	2.7021	.90686	.13228
ER6	1.00	53	3.7170	.88529	.12160
	2.00	47	3.0426	.72103	.10517

Independent Samples Test

	Levene's Test for Equality of Variances	t-test for Equality of Means

		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
ER1	Equal variances assumed	.107	.744	5.851	98	.000	.97672	.16693	.64545	1.30799
	Equal variances not assumed			5.817	93.568	.000	.97672	.16792	.64330	1.31014
ER2	Equal variances assumed	.638	.426	5.854	98	.000	1.04496	.17851	.69072	1.39921

	Equal variances not assumed			5.815	93.050	.000	1.04496	.17970	.68811 1.40182
	Equal variances assumed	16.600	.000	6.191	98	.000	1.09474	.17683	.74382 1.44566
ER3	Equal variances not assumed			6.013	72.869	.000	1.09474	.18206	.73189 1.45759
ER4	Equal variances assumed	1.265	.263	6.222	98	.000	.98153	.15775	.66848 1.29459

	Equal variances not assumed			6.147	88.970	.000	.98153	.15968	.66426 1.29881
	Equal variances assumed	.883	.350	5.932	98	.000	1.03372	.17426	.68790 1.37954
E R5	Equal variances not assumed			5.903	94.134	.000	1.03372	.17513	.68601 1.38143
E R6	Equal variances assumed	4.519	.036	4.144	98	.000	.67443	.16276	.35143 .99742

	Equal variances not assumed	4.195	97.328	.000	.67443	.16078	.35534 .99351

SD

Group Statistics

	Group	N	Mean	Std. Deviation	Std. Error Mean
SD1	1.00	53	4.0755	.70299	.09656
	2.00	47	2.3404	.81498	.11888
SD2	1.00	53	3.9434	.71831	.09867

	2.00	47	2.5319	.77603	.11320
SD3	1.00	53	3.8491	.66205	.09094
	2.00	47	2.4894	.85649	.12493
SD4	1.00	53	4.1698	.69989	.09614
	2.00	47	2.4468	.68552	.09999
SD5	1.00	53	3.9057	.88283	.12127
	2.00	47	2.6809	.72551	.10583
SD6	1.00	53	4.0755	.67508	.09273
	2.00	47	2.5957	.79836	.11645

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
SD1	Equal variances assumed	4.611	.034	11.430	98	.000	1.73505	.15180	1.43381	2.03628

	Equal variances not assumed			11.329	91.493	.000	1.73505	.15315	1.43085 2.03925
	Equal variances assumed	3.385	.069	9.444	98	.000	1.41148	.14946	1.11488 1.70808
SD2	Equal variances not assumed			9.400	94.299	.000	1.41148	.15016	1.11335 1.70962
SD3	Equal variances assumed	6.944	.010	8.935	98	.000	1.35969	.15218	1.05769 1.66170

	Equal variances not assumed			8.799	86.243	.000	1.35969	.15453	1.06682
	Equal variances assumed	.448	.505	12.406	98	.000	1.72300	.13889	1.99862
SD4	Equal variances not assumed			12.421	97.016	.000	1.72300	.13871	1.99831
SD5	Equal variances assumed	.605	.438	7.521	98	.000	1.22481	.16285	1.54798

	Equal variances not assumed			7.610	97.462	.000	1.22481	.16095	.90539 1.54423
	Equal variances assumed	4.624	.034	10.041	98	.000	1.47973	.14737	1.18728 1.77218
SD6	Equal variances not assumed			9.940	90.605	.000	1.47973	.14886	1.18401 1.77544

BO

Group Statistics

	Group	N	Mean	Std. Deviation	Std. Error Mean
BO1	1.00	53	4.0377	.75860	.10420
	2.00	47	2.8298	.84233	.12287
BO2	1.00	53	3.8302	.89305	.12267
	2.00	47	2.8085	.92403	.13478
BO3	1.00	53	3.8679	.73479	.10093
	2.00	47	2.7872	.74996	.10939
BO4	1.00	53	4.0377	.78354	.10763
	2.00	47	2.6596	.96181	.14029
BO5	1.00	53	3.8113	.83336	.11447
	2.00	47	2.5532	.82905	.12093
BO6	1.00	53	4.1132	.77609	.10660

2.00	47	2.6170	.84835	.12375

Independent Samples Test

	Levene's Test for Equality of Variances		t-test for Equality of Means					
	F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference
								Lower Upper

	Equal variances assumed	.470	.495	7.546	98	.000	1.20795	.16009	.89026 1.52564
B O1	Equal variances not assumed			7.498	93.280	.000	1.20795	.16110	.88804 1.52786
	Equal variances assumed	.401	.528	5.618	98	.000	1.02168	.18187	.66076 1.38260
B O2	Equal variances not assumed			5.606	95.689	.000	1.02168	.18225	.65990 1.38345

	Equal variances assumed	.012	.913	7.270	98	.000	1.08069	.14866	.78568 1.37570
B									
O3	Equal variances not assumed			7.261	96.068	.000	1.08069	.14884	.78524 1.37614
	Equal variances assumed	5.371	.023	7.890	98	.000	1.37816	.17467	1.03154 1.72478
B									
O4	Equal variances not assumed			7.794	88.853	.000	1.37816	.17682	1.02681 1.72951

	Equal variances assumed	.553	.459	7.553	98	.000	1.25813	.16657	.92758 1.58868
B									
O5	Equal variances not assumed			7.556	96.693	.000	1.25813	.16652	.92763 1.58863
	Equal variances assumed	2.497	.117	9.210	98	.000	1.49619	.16246	1.17380 1.81857
B									
O6	Equal variances not assumed			9.160	93.875	.000	1.49619	.16333	1.17188 1.82049

CN

Group Statistics

	Group	N	Mean	Std. Deviation	Std. Error Mean
CN1	1.00	53	3.6604	.85358	.11725
	2.00	47	3.0000	.85973	.12540
CN2	1.00	53	3.7170	.88529	.12160
	2.00	47	3.3191	1.06539	.15540
CN3	1.00	53	3.4528	.79822	.10964
	2.00	47	3.0213	.84672	.12351
CN4	1.00	53	3.4906	.79958	.10983

2.00	47	3.0426	.83295	.12150

Independent Samples Test

Levene's Test for Equality of Variances		t-test for Equality of Means						
F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
							Lower	Upper

	Equal variances assumed	1.819	.181	3.848	98	.000	.66038	.17160	.31984	1.00092
CN1	Equal variances not assumed			3.847	96.405	.000	.66038	.17168	.31962	1.00114
	Equal variances assumed	1.860	.176	2.039	98	.044	.39783	.19515	.01057	.78510
CN2	Equal variances not assumed			2.016	89.797	.047	.39783	.19733	.00580	.78987

	Equal variances assumed	.417	.520	2.622	98	.010	.43155	.16456	.10498	.75813
CN3	Equal variances not assumed			2.613	94.923	.010	.43155	.16515	.10368	.75943
	Equal variances assumed	.315	.576	2.742	98	.007	.44801	.16338	.12380	.77223
CN4	Equal variances not assumed			2.735	95.490	.007	.44801	.16378	.12289	.77314

PC

Group Statistics

	Group	N	Mean	Std. Deviation	Std. Error Mean
PCI1	1.00	53	3.3208	.80320	.11033
	2.00	47	3.3191	.83683	.12206
PCI2	1.00	53	3.4528	.82196	.11290
	2.00	47	3.4255	.85325	.12446
PCI3	1.00	53	3.4340	.79686	.10946
	2.00	47	3.2553	.96612	.14092
PCI4	1.00	53	3.5472	.63748	.08757
	2.00	47	3.2340	.83958	.12247

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means					
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference
									Lower	Upper
PCI1	Equal variances assumed	.400	.529	.010	98	.992	.00161	.16413	-.32410	.32731

	Equal variances not assumed			.010	95.486	.992	.00161	.16454	-.32502	.32823
	Equal variances assumed	.070	.792	.163	98	.871	.02730	.16766	-.30542	.36001
PCI2	Equal variances not assumed			.162	95.594	.871	.02730	.16804	-.30628	.36087
PCI3	Equal variances assumed	2.046	.156	1.013	98	.314	.17864	.17639	-.17140	.52869

	Equal variances not assumed			1.001	89.46	.319	.17864	.17844	-.17589 .53317
P CI 4	Equal variances assumed	3.229	.075	2.114	98	.037	.31313	.14812	.01919 .60706
	Equal variances not assumed			2.080	85.329	.041	.31313	.15055	.01381 .61245

EA

Group Statistics

	Group	N	Mean	Std. Deviation	Std. Error Mean
EA1	1.00	53	3.6038	.83986	.11536
	2.00	47	2.9149	.90481	.13198
EA2	1.00	53	3.8679	.85570	.11754
	2.00	47	2.7660	.81328	.11863
EA3	1.00	53	4.1321	.80950	.11119
	2.00	47	2.9574	.90787	.13243
EA4	1.00	53	3.8113	.87830	.12064
	2.00	47	3.0213	.76583	.11171
EA5	1.00	53	3.8868	.84718	.11637
	2.00	47	3.0851	.82961	.12101

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means					
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference
									Lower	Upper
EA1	Equal variances assumed	.040	.842	3.948	98	.000	.68888	.17451	.34258	1.03518

	Equal variances not assumed			3.930	94.398	.000	.68888	.17529	.34085 1.03691
	Equal variances assumed	.000	.985	6.578	98	.000	1.10197	.16751	.76954 1.43439
EA2	Equal variances not assumed			6.599	97.514	.000	1.10197	.16700	.77054 1.43339
EA3	Equal variances assumed	.171	.680	6.840	98	.000	1.17463	.17173	.83384 1.51541

	Equal variances not assumed			6.793	92.85	.000	1.17463	.17292	.83124 1.51802
	Equal variances assumed	2.498	.117	4.766	98	.000	.79004	.16578	.46106 1.11903
EA4	Equal variances not assumed			4.805	97.976	.000	.79004	.16442	.46376 1.11633
EA5	Equal variances assumed	.016	.899	4.769	98	.000	.80169	.16810	.46810 1.13527

	Equal variances not assumed	4.775	.030	97.020	.000	.80169	.16788	.46848	1.13489

SP

Group Statistics

	Group	N	Mean	Std. Deviation	Std. Error Mean
SP2_Seek	1.00	53	19.7170	4.09210	.56209
	2.00	47	13.2979	4.20618	.61353
SP2_Avoid	1.00	53	20.4717	3.95457	.54320

	2.00	47	13.2979	4.45711	.65014
SP2_Sensitivit	1.00	53	21.4151	3.58648	.49264
y	2.00	47	13.7234	3.68134	.53698
SP2_Registrati	1.00	53	18.9623	4.73921	.65098
on	2.00	47	13.9362	4.29056	.62584
SP2_Total	1.00	53	80.5660	8.30086	1.14021
	2.00	47	54.2553	6.16606	.89941
SRS_Total	1.00	53	71.6209	10.18841	1.39949
	2.00	47	40.8085	11.17264	1.62970

Independent Samples Test

	Levene's Test for Equality of Variances	t-test for Equality of Means

		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
SP2_Seek	Equal variances assumed	.390	.534	7.727	98	.000	6.41911	.83070	4.77060	8.06761
	Equal variances not assumed			7.714	95.876	.000	6.41911	.83209	4.76740	8.07082
SP2_Avoid	Equal variances assumed	1.829	.179	8.529	98	.000	7.17383	.84111	5.50468	8.84297

	Equal variances not assumed			8.468	92.685	.000	7.17383	.84720	5.49138	8.85627
	Equal variances assumed	.001	.970	10.572	98	.000	7.69169	.72757	6.24784	9.13554
SP2_Sensitivity	Equal variances not assumed			10.555	95.914	.000	7.69169	.72873	6.24516	9.13822
SP2_Registration	Equal variances assumed	.395	.531	5.533	98	.000	5.02609	.90847	3.22327	6.82892

	Equal variances not assumed			5.566	97.953	.000	5.02609	.90302	3.23406	6.81813
	Equal variances assumed	1.896	.172	17.803	98	.000	26.31072	1.47789	23.37789	29.24355
SP2_Total	Equal variances not assumed			18.117	95.185	.000	26.31072	1.45225	23.42771	29.19372
SRS_Total	Equal variances assumed	.135	.714	14.424	98	.000	30.81243	2.13619	26.57323	35.05164

Equal variances not assumed	14.344	93.756	.000	30.81243	2.148 13	26.54712	35.07775

The analysis of the dataset (N = 100) provides insights into the demographic composition and the emotional, sensory, and behavioural attributes of children diagnosed with Autism Spectrum Disorder (ASD) compared to their typically developing (TD) peers. The sample comprised more males (64%) than females (36%), consistent with the higher prevalence of ASD among boys. Ethnically, the majority were Caucasian (75%), followed by Hispanic/Latino (21%) and Black/African American (4%). Notably, 70% of participants were diagnosed with ASD, 20% were not, and 10% were unsure. Caregivers were predominantly aged 26–45 years, with high educational attainment (65% holding master's or doctoral degrees). Employment was also relatively high, with 84% engaged in either full-time or part-time work, and most households reported incomes between £40,000 and £80,000. Healthcare access was widespread, with 86% indicating availability.

On the emotional regulation scale (ER1–ER6), a substantial proportion of caregivers reported agreement or strong agreement with difficulties, highlighting challenges in regulating emotions. Group comparisons revealed that ASD children scored significantly higher on all ER items, with mean differences around one point on the Likert scale, confirming elevated dysregulation. Sensory difficulties (SD1–SD6) also showed clear group distinctions, with ASD children consistently reporting higher levels, particularly in tactile hypersensitivity and sensory sensitivity. Behavioural outcomes (BO1–BO6) followed a similar trend, with ASD children demonstrating markedly higher behavioural and social difficulties. These differences were statistically significant across all items, with large effect sizes, indicating robust group disparities.

Cognitive and neurological indicators (CN1–CN4) were moderately elevated among ASD children, with significant though smaller mean differences compared to TD peers. Parent–child interaction (PCI1–PCI4) did not show consistent group differences, suggesting that caregivers across both groups reported similar relational patterns. However, emotional awareness items (EA1–EA5) revealed significantly higher deficits among ASD children, further supporting the link between alexithymia and social-emotional complications.

Composite measures reinforced these patterns. ASD children scored substantially higher on all SP2 subscales (Seek, Avoid, Sensitivity, Registration) and on SP2_Total, indicating pervasive sensory processing issues. Similarly, Social Responsiveness Scale (SRS_Total) scores were markedly higher in the ASD group, confirming more severe social impairment. Overall, the findings provide strong evidence that ASD is characterised by elevated emotional dysregulation, sensory difficulties, behavioural complications, and reduced emotional awareness, aligning with theoretical expectations and supporting the study's hypotheses.

Hypothesis Testing

Hypothesis 1

Correlations

		BO_average	ER_average	SD_Average	CN_average
BO_average	Pearson Correlation	1	.720**	.806**	.399**
	Sig. (2-tailed)		.000	.000	.000

	N	100	100	100	100
ER_averag e	Pearson Correlation	.720**	1	.744**	.395**
	Sig. (2-tailed)	.000		.000	.000
	N	100	100	100	100
SD_Avera ge	Pearson Correlation	.806**	.744**	1	.458**
	Sig. (2-tailed)	.000	.000		.000
	N	100	100	100	100
CN_avera ge	Pearson Correlation	.399**	.395**	.458**	1
	Sig. (2-tailed)	.000	.000	.000	
	N	100	100	100	100

**. Correlation is significant at the 0.01 level (2-tailed).

Regression

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.826[a]	.683	.673	.40186

a. Predictors: (Constant), CN_average, ER_average, SD_Average

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	33.341	3	11.114	68.819	.000[b]
	Residual	15.503	96	.161		
	Total	48.844	99			

a. Dependent Variable: BO_average

b. Predictors: (Constant), CN_average, ER_average, SD_Average

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	.497	.307		1.617	.109
	ER_average	.313	.102	.266	3.080	.003
	SD_Average	.521	.078	.599	6.709	.000
	CN_average	.026	.089	.019	.293	.770

a. Dependent Variable: BO_average

The regression model for H1 was highly significant, $F(3,96) = 68.82$, $p < .001$, explaining about 68% of the variance in behavioural outcomes ($R^2 = .683$). Sensory difficulties ($\beta = .599$, $p < .001$) emerged as the strongest predictor, followed by emotional regulation ($\beta = .266$, $p = .003$). Cognitive indicators were not significant ($p = .770$). This indicates that higher sensory difficulties and emotional dysregulation are strongly associated with increased behavioural problems in ASD children, with sensory issues showing the greatest impact.

Hypothesis 2

Correlation

Correlations

		PC_average	ER_average	SD_Average
PC_average	Pearson Correlation	1	.079	.080

	Sig. (2-tailed)		.432	.426
	N	100	100	100
ER_avera	Pearson Correlation	.079	1	.744**
ge	Sig. (2-tailed)	.432		.000
	N	100	100	100
SD_Avera	Pearson Correlation	.080	.744**	1
ge	Sig. (2-tailed)	.426	.000	
	N	100	100	100

**. Correlation is significant at the 0.01 level (2-tailed).

One Way Anova

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
ER_average	Between Groups	.264	2	.132	.365	.695
	Within Groups	35.004	97	.361		
	Total	35.268	99			
SD_Average	Between Groups	3.425	2	1.713	2.709	.072
	Within Groups	61.311	97	.632		
	Total	64.736	99			

Multiple Comparisons

Tukey HSD

Dependent Variable	(I) Caregiver_role	(J) Caregiver_role	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
ER_average	Yes	No	.13000	.15231	.671	-.2325	.4925
		Unsure	.02000	.20308	.995	-.4634	.5034
	No	Yes	-.13000	.15231	.671	-.4925	.2325

		Unsure	-.11000	.23266	.884	-.6638	.4438
		Yes	-.02000	.20308	.995	-.5034	.4634
	Unsure						
		No	.11000	.23266	.884	-.4438	.6638
		No	.46357	.20158	.061	-.0162	.9434
	Yes						
		Unsure	.19857	.26877	.741	-.4412	.8383
SD_Average							
		Yes	-.46357	.20158	.061	-.9434	.0162
	No						
		Unsure	-.26500	.30791	.666	-.9979	.4679

		Yes	-.19857	.26877	.741	-.8383	.4412
Unsure							
		No	.26500	.30791	.666	-.4679	.9979

ER_average

Tukey HSD[a,b]

Caregiver_role	N	Subset for alpha = 0.05
		1
No	20	3.2800
Unsure	10	3.3900

Yes	70	3.4100
Sig.		.791

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 18.261.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

SD_Average

Tukey HSD[a,b]

Caregiver_role	N	Subset for alpha = 0.05
		1
No	20	2.9550
Unsure	10	3.2200
Yes	70	3.4186
Sig.		.188

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 18.261.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

The results for H2 show that parent–child interaction (PCI_Average) was not significantly correlated with emotional regulation ($r = .079$, $p = .432$) or sensory difficulties ($r = .080$, $p = .426$). This suggests that in this sample, parental reporting did not directly align with the severity of children's emotional or sensory issues.

The ANOVA results further indicated no significant differences across caregiver roles (mother, father, guardian) in reported emotional regulation ($F = 0.37$, $p = .695$) or sensory difficulties ($F = 2.71$, $p = .072$). Although mothers tended to report slightly higher sensory difficulties, these differences were not statistically significant.

Overall, Hypothesis 2 was not supported: parental observations and role did not significantly influence reported emotional or sensory complications in ASD children.

Hypothesis 3

Correlation

Correlations

		BO_average	SD_Average	SP2_Total
BO_average	Pearson Correlation	1	.806**	.776**
	Sig. (2-tailed)		.000	.000
	N	100	100	100
SD_Average	Pearson Correlation	.806**	1	.836**
	Sig. (2-tailed)	.000		.000
	N	100	100	100

SP2_Total	Pearson Correlation	.776**	.836**	1
	Sig. (2-tailed)	.000	.000	
	N	100	100	100

**. Correlation is significant at the 0.01 level (2-tailed).

Regression

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate

1	.828[a]	.685	.678	.39828

a. Predictors: (Constant), SD_Average, SP2_Total

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	33.458	2	16.729	105.462	.000[b]
	Residual	15.387	97	.159		
	Total	48.844	99			

a. Dependent Variable: BO_average

b. Predictors: (Constant), SD_Average, SP2_Total

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	.788	.187		4.225	.000
	SP2_Total	.016	.005	.340	3.276	.001
	SD_Average	.453	.090	.522	5.027	.000

a. Dependent Variable: BO_average

The results for H3 strongly support the hypothesis. Correlation analysis showed that behavioural outcomes were highly associated with both sensory difficulties ($r = .806$, $p < .001$) and the SP2 total sensory profile score ($r = .776$, $p < .001$). This indicates that children with more severe sensory atypicalities also displayed greater behavioural and social challenges.

The regression model was also highly significant, $F(2,97) = 105.46$, $p < .001$, explaining about 69% of the variance in behavioural outcomes ($R^2 = .685$). Both sensory difficulties ($\beta = .522$, $p < .001$) and SP2_Total ($\beta = .340$, $p = .001$) were significant predictors, with sensory difficulties emerging as the stronger contributor.

Overall, these findings confirm that behavioural issues in ASD are strongly driven by sensory processing complications, highlighting the close link between atypical sensory responses and social-behavioural outcomes.