



UNPACKING STRESSOR-DRIVEN BEHAVIORS IN AUTISM SPECTRUM DISORDER: A BIOPSYCHOSOCIAL PERSPECTIVE

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Abstract

The current study explores the role of internal and external stressors in the development and maintenance of self-injurious behaviours (SIBs) and self-stimulatory behaviours (SSBs) in a child diagnosed with autism spectrum disorder (ASD). Using a qualitative intrinsic case study, detailed behavioural observations, parental interviews, and contextual analysis were conducted to identify the environmental, sensory, neurobiological, and psychosocial stressors associated with these behaviours. Thematic analysis revealed five major themes: sensory triggers, environmental stressors, neurobiological vulnerability, functional behaviour, and psychosocial stress. Findings indicate that behaviours such as head-hitting, self-biting, and hand-flapping are closely linked to sensory hypersensitivities, disruptions in routine, social demands, and caregiver emotional states. These behaviours often serve regulatory or communicative functions, highlighting the need for function-based interventions such as functional communication training (FCT) and sensory integration approaches. The study emphasizes the importance of a biopsychosocial framework in assessing and addressing SIBs and SSBs in children with ASD, and advocates for caregiver support, environmental structuring, and individualized communication strategies as part of comprehensive intervention planning.

Keywords: Autism Spectrum Disorder, self-injurious behaviours, self-stimulatory behaviours, stressors, sensory triggers, functional communication

Introduction

Behavior refers to the movement of some part of an individual that changes a particular part or features of the environment (Johnston et al., 2010). Mostly, the term behavior alludes to a category of responses perceived through the senses, entirely share physically measurable extents or functions performed independently by individuals, and verbal or written answer in such a case is a single illustration perceived within environmental structures of that behavior (Zarcone et al., 1994). A behavior is contemplated to be taxing if it brings hazard into being for individuals or others, or obstructs them from participating and contributing their share in community programs or educational ventures in an ordinary conduct (McLennan et al., 2023). Ethnically or racially peculiar and anomalous behaviors of varying intensity, frequency or duration in which the physical protection of the person or others is probably to be placed in uncertain jeopardy, or behaviors



which are pensively and meditatively restrict the use of or deny the opportunity to approach, standard and accustomed facilities that are yielded in a community (Emerson, 1995).

Taxing behaviors (TBs) are “culturally atypical behaviors of such extremity, frequency or duration in which the corporeal well-being of the person or others is laid down in weighty peril, or behaviors that evidently freeze or restrict means of approaches towards the manipulation of accustomed facilities (Emerson, 2005). Researchers have been blossomed and elaborated miscellaneous definitions of the term “taxing behaviors” (Boulware et al., 1999; Buck & Ambrosino, 2004). Many have conceptualized taxing behaviors based upon the immediate impact on the individual child and their environment, as well as the long-term effects on the individual child (Dunlap et al., 2006). Also, Taxing behaviors can include a wide range of behaviors such as bouncing, screaming, biting, hitting, kicking, non-compliance and can be described in numerous ways such as problem behaviors, behavioral difficulties, behavioral disorders, challenging behaviors (Powell et al., 2003).

Behaviours that pose significant risks to individuals or others, or interfere with daily participation in social and educational settings, are often referred to as “taxing behaviours” (Emerson, 2005). These include aggression, SIBs, and SSBs, which can vary in frequency, intensity, and duration. Among children with ASD), such behaviours often emerge from a complex interplay of sensory processing differences, environmental demands, neurobiological predispositions, and psychosocial stressors (Matson et al., 2011; Green & Ben-Sasson, 2010).

While quantitative studies have identified common triggers and biological correlates of SIBs and SSBs, there remains a lack of in-depth qualitative research exploring how these stressors interact in real-life contexts and how they serve functional purposes for the child. Furthermore, there is limited work situating these behaviours within a biopsychosocial framework that integrates genetic vulnerabilities, sensory sensitivities, environmental unpredictability, and caregiver stress. The present case study addresses this gap by providing a rich, contextualized analysis of a single child with ASD, mapping internal and external stressors to behavioural functions, and identifying targeted interventions.

Review of the Related Literature

Taxing behaviors have long been a general and irritating problem for educators, with damaging and harmful side effects both for individual with autism spectrum disorder and their parents (American Psychiatric Association, 2013). In fact, taxing behaviors are cited as one of the substantial challenges dealt by parents, preschool teachers and childcare providers. As early as 1980, researchers suggested that as many as 24% of all preschool children demonstrated outstandingly problematic behaviors that interfere significantly with the usual and standard procedures of learning, development and success at play (Burbach et al., 2004). Taxing behaviors are quotidian with varying degrees of devastation among individuals with autism spectrum disorders (ASD) and/or intellectual disabilities (ID), depending upon the causes of environmental changes (Matson et al., 2011). It is critical to remember that taxing behaviors assist an objective for the individual; the individual who has some need uses a behavior to get his need met (Ayres, 2005).

Understanding why the taxing behaviors occur is fundamental for the growth and development of a plausible and sustainable behavioral intervention via functional assessment (Baranek et al., 1997). Surrounding and holding with a broad array of behaviors, the term taxing



behavior involves and contains all behaviors which are viewed and perceived as presenting a fight to decide who is superior in terms of ability or strength to services (Beavers et al., 2013). The term taxing behavior is used in this thesis as an umbrella term for aggression, destruction and self-injury alone, although traditionally the definition of challenging behavior is often more wide reaching (Carr & Durand, 1985).

Types of TBs append, but are not reined to, aggression, self-injurious behavior (SIBs), self-stimulating behaviors (SSBs), property destruction, and severe noncompliance (Cermak & Daunhauer, 1997). Aggressive behavior is one of the most taxing behaviors, owns many detrimental out-turns. Corporeal aggression is the most customarily alluded form of aggressive behavior involving an individual to have an attempt to or triumphantly bruising another person through physical means e.g., kicking, hitting, scratching, bouncing and biting (Coury et al., 2012). Verbal aggression in the form of threatening, yelping at others, oppressing, tyrannizing or squealing; property aggression in the form of spoiling property or throwing objects; sexual aggression in the form of twiddling others; and even self-directed aggression may also be called as self-injurious behavior (SIB) come closer to aggressive behavior (Dawson & Watling, 2000). Such a slight variability in the definition of aggressive behavior makes accurate estimation of aggressive behavior difficult. Approximate figure from 9.8% to 51.8% segment of population with developmental disabilities is engaged in some form of aggressive behavior (Dominick et al., 2007).

As the causation of autism spectrum disorders (ASD) is not known, the etiology of self-injurious behavior (SIB) and self-stimulated behavior (SSB) is also still being investigated by the pertinent professionals and researchers. SERT, MAOA, FOXP2 genes have been detected as possible causes of autism (Edelson, 2010). Causes of autism may vary across children due to the diverse nature of phenotypic expressions of multiple genes. Owing to the involvement of genetic and environmental factors, there are multiple behavioral, medical, biological and environmental correlates that affect the onset of TBs among individuals with ASD, (Green & Ben-Sasson, 2010). Descriptions of the causes of taxing behavior are established on two broad array of approaches i.e. biological and behavioral, whilst not certainly in opposition, little attempt has been made to associate and correlate the two to account for durable findings supporting each viewpoint of alike natured dimensions (Horner et al., 2002). Neurobiological theories of TBs have focused on the role of neurotransmitters, focusing on the serotonergic, opiatergic, dopaminergic and basal ganglia systems. Whilst the serotonergic system is involved in the expression of both self-injury and aggression, neurotransmitter theories generally are more relevant to self-injury with far less emphasis on the demonstration of aggression (Iwata et al., 1994).

Mono-amine transporters in SERT gene are protein structures and are located outside the synaptic cleft involved in the expression of both self-injury and aggression (Miller, 2011). SERT is a kind of mono-amine transporter(MAT) that fetches and sends serotonin from the synaptic cleft (information transfer) to the pre-synaptic neuron, insufficient fetching and sending amount of serotonin leads autistic children towards self-injury and self-stimulatory (Mazefsky et al., 2012). The serotonin carrier (SERT, SERT1, HTT, 5HTT, 5-HTT, OCDI, 5HTTLPR) recognizes as the sodium contingent on the serotonin neurotransmitter and solute transporter family 6 member 4 is a protein including substances such as enzymes, hormones and antibodies imperative for the appropriate working of living cell in an organism is encoded by the gene SLC6A4 while on the other hand, inadequate enzymes and hormones lead inappropriate working of a living cell in the



form of either self-injury or self-stimulatory (Lesch et al., 1993). The synaptic cleft is a gap between the pre- and postsynaptic cells that is about 20 nm wide. The small volume of the cleft allows neurotransmitter concentration to be raised and lowered rapidly while distraction from 20 nm indicates functioning of self-injury and self-stimulatory especially among individuals with ASD (Lecavalier, 2006).

Screens of deoxyribonucleic acid expounded as a nucleic acid among patients with ASD, bipolar disorder, attention deficit hyperactivity disorder and Tourette's syndrome have undoubtedly observed and diagnosed signs in the region of chromosome 17q (one of the 23 pairs of chromosomes in human beings representing from 2.5% to 3% of the total DNA in cell) where SERT is firmly based and caused self-injury and self-stimulatory (Matson & Rivet, 2008). Disorders and diseases such as depression, obsessive compulsive disorder (OCD), osteogenesis imperfecta (type 1, 2, 3, 4), neurofibromatosis (type 1), abnormal behaviors based on self-injury and self-stimulatory and many others are related to genes on chromosome 17 (Matson & Neal, 2009).

A redo and continual extent or genetic polymorphism (where the phenotype of each individual is genetically determined) in the proponent of SERT gene has been exhibited to exert influence on the rate of serotonin absorption and may play a role in sudden death of an infant, severe form of self-injury, self-stimulatory and/or aggressive behaviors in patients with autism spectrum disorder, alzheimer disease, post disorder and/or depression vulnerability (Rattaz et al., 2013). Medical studies have manifested that changes in serotonin transporter metabolism emerge to be linked with multi-faceted phenomena containing alcoholism and depression involving unwanted disturbances in the form of hitting, kicking, bouncing as well as repetitive behaviors or mental acts (Schaaf et al., 2011). Extreme or irrational communal fear, unnecessary tedious manners and/or non-verbal way of communication accompanies an individual with ASD with self-injury and self-stimulatory (Zuckerman et al., 2016).

In contrast, a meta-analysis found no remarkable overall confederation between the 5-HTTLPR polymorphism and ASD, (Huang & Santangelo, 2008). In MAOA gene, monoamine oxidase A, also well known as MAO-A, is a substance produced by a living organism which acts as a catalyst to bring about a specific biochemical reaction that in humans is encoded by the MAOA gene (Lecavalier, 2006). MAOA gene has also been linked with a diversity of multi psychiatric disorders, emotional disorders as well as neuro developmental disorders including ASD leading towards offensive and unsocial behaviors such as aggression, self-injury and/or self-stimulatory while Alternatively spliced transcript variants encoding multiple is forms have been observed (Matson & Rivet, 2008). There is a link between low activities forms of the MAOA gene and autism spectrum disorder (ASD). Disorders linked with MAO-A include ASD distinctively attributing self-injury and self-stimulatory, alzheimer's disease, aggression, panic disorder, bipolar affective disorder, major depressive disorder, and attention deficit hyperactivity disorder (Horner et al., 2002).

Low-activity variants are linked to reduced breakdown of serotonin, dopamine, and norepinephrine, contributing to impulsivity and aggression. Behavioural volatility observed in this case aligns with literature linking MAOA dysfunction to aggression/self-injury. FOXP2 gene is critical for speech and language development. FOXP2 variations can impair expressive and receptive communication, leading to frustration-based behaviours. Areej's language delays and



reliance on behaviours for communication may reflect FOXP2-related vulnerabilities. In FOXP2 gene, ASD is a phenotypically and genetically heterogeneous condition characterized by the presence of repetitive/restrictive behaviors and variable deficits in language (including both verbal and non-verbal) and/or antisocial behaviors such as self-injurious behaviors (including hitting, kicking, bouncing and scratching), (Schaaf et al., 2011). Forkhead box protein P2 (FOXP2) is a protein that, in humans, is encoded by the FOXP2 gene, also well known as CAGH44, SPCH1 or TNRC10, and is needed for appropriate growth and development of speech and language skills, apart from that, self-stimulated speech and language patterns (Dominick et al., 2007).

Initially recognized as the genetic factor of speech disorder in KE family, its gene is the first gene found and vigorously linked with speech and language developmental procedures and is not a major susceptible gene for autism spectrum disorder causing repetitive speech and language patterns (Beavers et al., 2013). The gene is located on chromosome 7 (7q31, at the SPCH1 locus), and is expressed in fetal and adult brain, heart, lung and gut leading to inappropriate and repetitive behaviors of spoken language (Coury et al., 2012). MET in restricted human neocortical regions, and its regulation in part by FOXP2, is consistent with genetic evidence for MET contributing to ASD risk, this gene is more active in females than in males, to which could be ascribed better language learning in females while insufficient contribution of MET leads individuals towards inappropriate and repetitive patterns of speech and language (Edelson, 2010). In humans, mutations of FOXP2 cause a severe speech and language disorder including repetitive behaviors of receptive as well as expressive language among individuals with ASD, (Cermak & Daunhauer, 1997). FOXP2 is popularly known as the "language gene", but this is only partly correct since there are other genes involved in language development (Lecavalier, 2006). Neurobiological, genetic, and imaging data provide strong evidence for the CNTNAP2 gene as a risk factor for ASD and related neurodevelopmental disorders but accounts for a very few cases (Matson & Neal, 2009).

In summary, existing literature highlights that self-injurious and self-stimulatory behaviours in children with ASD are often rooted in a combination of sensory sensitivities, environmental stressors, communicative deficits, and underlying neurobiological vulnerabilities. While interventions like FCT and sensory integration therapies have shown promise, there remains a need for individualized, context-specific analysis of these behaviours. Despite growing research, limited qualitative exploration exists that deeply examines how these stressors interact in real-life contexts. This study addresses this gap by investigating the specific triggers and functions of SIBs and SSBs in a single child with ASD, aiming to contribute to more effective, targeted intervention strategies.

Objectives of the Study

1. To identify and categorize the internal and external stressors that contribute to self-injurious and self-stimulatory behaviors in a child with ASD.
2. To examine how sensory hypersensitivities influence the emergence of these behaviors.
3. To explore the functional purposes e.g., escape, avoidance, communication of SIBs and SSBs using a biopsychosocial lens.
4. To investigate the role of familial, genetic, and neurobiological predispositions in influencing behavioral responses.



5. To assess the impact of caregiver emotional stress and home environment on the frequency and intensity of these behaviors.
6. To identify evidence-based interventions corresponding to specific internal and external stressors in a child with ASD and to evaluate how these targeted strategies can address the functional purposes of SIBs and SSBs.

Research Questions

1. What types of internal and external stressors are associated with the emergence of SIBs and SSBs in children with ASD?
2. How do sensory triggers e.g., auditory, visual hypersensitivities influence behavioral regulation?
3. What is the functional role of self-injurious and self-stimulatory behaviors in the context of identified stressors?
4. To what extent do neurobiological factors e.g., SERT, MAOA, FOXP2 gene expressions contribute to behavioral vulnerability?
5. How does caregiver emotional stress, including maternal depression, impact the child's behavioral patterns?
6. Which evidence-based interventions can be mapped to identified internal and external stressors in a child with ASD, and how can these interventions be rationalized to address the underlying causes and functional purposes of SIBs and SSBs?

Methodology

Research Design

A qualitative case study design was selected because it allows for the exploration of complex, context-dependent phenomena where variables cannot be meaningfully isolated (Yin, 2018). Given the multidimensional nature of stressor-driven behaviours in ASD, a qualitative intrinsic case study (Gosselin & Bowen, 2024; Yin, 2018; Stake, 1995) was considered more appropriate than quantitative or mixed methods for capturing the depth, meaning, and lived experiences surrounding these behaviours.

To enhance the credibility of findings, the study incorporated multiple validity measures:

1. data triangulation across interviews, observations, incident logs, and document reviews
2. peer debriefing with a qualitative research expert to review coding and theme development
3. reflexive journaling by the researcher to acknowledge and minimize potential bias
4. member checking, where key interpretations were shared with the caregiver for confirmation and clarification.

Participant Selection

A purposive sampling technique was employed to select a participant who exhibited pronounced self-injurious and self-stimulatory behaviours, along with a confirmed diagnosis of ASD by a qualified clinical psychologist or developmental paediatrician. The selected participant, referred to as Areej (pseudonym), was a six-year-old girl with pronounced sensory hypersensitivities and behaviour patterns consistent with both SIBs and SSBs. The inclusion criteria for selection were:

1. Child aged between 6 to 9 years.



2. Diagnosed with ASD according to DSM-5 criteria.
3. Exhibiting observable and recurrent SIBs and SSBs.
4. Parent/guardian consent for participation and willingness to provide detailed familial and medical history.

Data Collection Methods

Multiple qualitative data collection strategies were utilized to ensure triangulation and increase the credibility of the findings:

Semi-Structured Interviews: Conducted with the child's mother to explore psychosocial and familial stressors, including the mother's history of depression, parenting practices, and environmental influences. Questions focused on the child's early development, family medical history (particularly related to depression and serotonin-linked disorders), and specific behavioural triggers.

Direct Observations: The researcher conducted naturalistic observations of the child in multiple settings (home and clinic) over a span of 8 weeks. Observations focused on identifying antecedents, behavioural responses, frequency, and duration of SIBs and SSBs under various sensory and social contexts.

Behavioural Incident Logs: Caregivers were instructed to maintain a daily log of all incidents of self-injury and self-stimulatory behaviours, including contextual details such as time, location, antecedents, and consequences.

Document Analysis: Review of medical and psychological assessment reports to identify biological predispositions (e.g., family history of depression or ASD). Analysis of prior interventions attempted and their outcomes.

Sensory assessment: Data on sensory triggers were collected through:

1. Structured naturalistic observations by the researcher across multiple settings.
2. Parental incident logs documenting antecedents, behaviour, and context.
3. Semi-structured interview probes specifically targeting sensory experiences (e.g., responses to sound, light, touch).

Caregiver Emotional Strain: It was assessed qualitatively through interviews and observations. No validated scales such as the *Parenting Stress Index (PSI)* or *Beck Depression Inventory-II* were used. While qualitative accounts revealed strong links between maternal burnout and behavioural escalation, future studies should use standardized instruments to measure and track caregiver stress quantitatively.

Ethical Considerations

Informed consent was taken from the participant's mother with assurances of confidentiality and the right to withdraw at any time. Data were anonymized, and pseudonyms were used to protect the child's identity. All procedures complied with the American Psychological Association's (APA) Ethical Principles of Psychologists and Code of Conduct.

Data Analysis

The data were analysed using inductive thematic content analysis, aligning with Braun and Clarke's (2006) six-phase framework. The process included familiarization with the data, generation of initial codes, search and review of themes, theme definition, and final write-up. Data triangulation was achieved through multiple sources, including semi-structured interviews, field observations, behavioural logs, and document analysis.



Open coding was first applied to derive initial codes from the data, followed by axial coding to identify interrelations between categories, and finally selective coding to develop core themes (Strauss & Corbin, 1990). The coding process was conducted manually, and peer debriefing with a qualitative research expert was undertaken to enhance interpretive validity and establish inter-coder reliability.

The following steps were followed:

Coding: Interview transcripts, observation notes, and incident logs were coded using inductive thematic analysis to identify recurring stressors and behavioural patterns.

Theme Generation: Codes were organized into categories (e.g., sensory stress, social avoidance, environmental unpredictability, biological predispositions).

Triangulation: Data from different sources (interviews, logs, observations) were cross-referenced to validate findings.

Code-to-Theme Progression

Codes were separated into observable behaviour and interpretive memos to ensure analytic transparency. Frequencies were included to indicate the intensity or recurrence of behaviours across data sources. Where necessary, overlapping categories were tagged under multiple themes to reflect the complexity of behavioural patterns.

Table 1

Temporal Patterns Across the 8-Week Observation Period - Weekly Frequency of SIBs and SSBs

Week	SIBs	SSBs
1	10	14
2	8	12
3	11	15
4	14	17
5	9	11
6	12	16
7	7	9
8	6	8

Note. Behaviour frequency in table 1 varies across the 8-week period, with peaks corresponding to weeks involving multiple high-demand social events or episodes of family conflict. Notably, weeks with structured daily routines and fewer environmental disruptions saw reduced frequencies of both SIBs and SSBs. IB and SSB frequency peaked in Week 4, coinciding with multiple family gatherings and an illness episode in the mother. A gradual decline in Weeks 7–8 corresponded with the introduction of more predictable daily schedules and reduced exposure to large gatherings.

Table 2

Codes, Categories, and Themes

Codes	Categories	Themes	Stressor Type
Head-hitting when exposed to bright lights	Sensory Avoidance	Theme 1: Sensory Triggers as Precursors of SIBs and SSBs	Internal



Covers ears during loud noise	Auditory Hypersensitivity	Theme 1: Sensory Triggers	Internal
Avoids eye contact in crowded places	Social Withdrawal	Theme 1: Sensory Triggers	External
Self-biting during family gatherings	Overwhelming Social Stimuli	Theme 2: Environmental Stressors and Routine Disruptions	External
Sudden screaming when routine is disrupted	Environmental Instability	Theme 2: Environmental Stressors and Routine Disruptions	External
More calm on structured days	Structured vs. Unstructured Env.	Theme 2: Environmental Stressors	External
Increased SIBs after family arguments	Emotional Climate at Home	Theme 2: Environmental Stressors	External
Mother reports history of depression	Biological Predisposition	Theme 3: Genetic and Neurobiological Vulnerability	Internal
Family history of serotonin-linked disorders	Genetic Load (SERT, MAOA)	Theme 3: Genetic and Neurobiological Vulnerability	Internal
Child has difficulty with verbal expression	FOXP2 Gene & Language Processing	Theme 3: Genetic and Neurobiological Vulnerability	Internal
Repetitive hand-flapping when ignored	Attention-Seeking via SSBs	Theme 4: Functional Role of SIBs and SSBs	External
Self-injury just before a demand is placed	Escape from Task	Theme 4: Functional Role of SIBs and SSBs	External
Withdrawal behaviours increased with mother's stress	Parental Emotional State	Theme 5: Psychosocial and Parental Stress as Indirect Stressors	External
Mother expressed feelings of emotional burnout	Caregiver Mental Health	Theme 5: Psychosocial and Parental Stress	External

Note. Table 2 outlines the analytic process showing the transition from raw behavioural data to broader themes.



Table 3

Internal vs. External Stressor Classification

Internal Stressors	External Stressors
Visual hypersensitivity (e.g., head-hitting under bright lights)	Disrupted routines (e.g., sudden changes in daily schedule)
Auditory hypersensitivity (e.g., covering ears in response to loud noise)	Family conflict and emotionally charged home environments
SERT-related serotonergic dysregulation (suspected from maternal depression history)	High social density situations (e.g., family gatherings)
FOXP2-linked language processing challenges (e.g., difficulty expressing needs verbally)	Overstimulating environments (e.g., noisy, crowded public places)
MAOA-related emotional regulation vulnerabilities (literature-supported link to aggression/self-injury)	Parental emotional burnout and elevated stress levels

Note. While classification in table 3 provides clarity, it is important to note that some stressors, particularly sensory hypersensitivities, blur the distinction between internal and external domains. For example, auditory hypersensitivity has an internal sensory-processing basis, but behavioural escalation is often triggered by an external factor such as a loud public setting. This interplay underscores the biopsychosocial perspective, where stressor impact is shaped by the interaction between intrinsic vulnerabilities and the surrounding environment.

Table 4

Final Coding Framework with Frequency and Thematic Interpretation

Code	Category	Frequency	Interpretation	Theme
Head-hitting when exposed to bright lights	Visual Sensory Trigger	8	Overstimulation and escape behaviour	Theme 1: Sensory Triggers
Avoids eye contact in crowded places	Social Withdrawal	5	Social overstimulation or anxiety	Theme 1: Sensory Triggers
Self-biting during family gatherings	Overwhelming Social Stimuli	4	Escape-maintained behaviour	Theme 2: Env. Stressors
Screaming when routine is disrupted	Environmental Instability	6	Routine changes trigger distress	Theme 2: Env. Stressors
Mother reports history of depression	Biological Context	N/A	Suggests genetic predisposition (SERT gene)	Theme 3: Neurobiological



Repetitive hand-flapping when ignored	Social Demand Avoidance	7	Attention-seeking communication	Theme 4: Functional Beh.
Mother's emotional burnout	Parental Emotional Stress	Consistent	Correlates with increased behaviour incidents	Theme 5: Psychosocial Stress

Note. The data presented in table 4 reveal five overarching themes derived from coded behaviours observed in a child with ASD, each linked to specific stressors and functions of behaviour. It confirms that the behaviours observed are not random but are strongly linked to identifiable internal and external stressors, biological predispositions, and communicative purposes.

Table 5

Thematic Interconnections Across the Biopsychosocial Framework

Theme	Biological Factors	Psychological Factors	Social/Environmental Factors
Sensory Triggers	Sensory integration deficits	Hyperarousal, sensory processing issues	Overstimulating environments (lights, noise)
Environmental Stressors	Stress response dysregulation (HPA axis)	Anxiety, resistance to change	Routine disruptions, family conflicts
Neurobiological Vulnerability	SERT, MAOA, FOXP2 gene associations	Emotion regulation difficulties	Intergenerational stress (e.g., maternal depression)
Functional Behaviour	Dopamine/serotonin role in reward/escape cycles	Motivation to escape or seek attention	Unmet communication needs, high task demands
Psychosocial/Parental Stress	Epigenetic stress markers	Caregiver burnout, emotional contagion	Parental mental health, family structure

Note. Table 5 integrates the five main themes identified in the study, sensory triggers, environmental stressors, neurobiological vulnerability, functional behaviour, and psychosocial stress, within the structure of the biopsychosocial model. It highlights how each theme is shaped by the interaction of biological, psychological, and social/environmental domains. For example, sensory triggers are influenced not only by neurobiological factors such as sensory processing dysfunction but also by psychological hyperarousal and environmental overstimulation. Similarly, environmental stressors like routine disruption or noise intersect with emotional regulation difficulties and a dysregulated stress response system. The table underscores the multidimensional nature of stressors, demonstrating that no behaviour exists in isolation but emerges from a complex interplay between a child's biology, internal emotional states, and external contexts.



Table 6

Behaviour-Stressor Functionality Matrix

Observed Behaviour	Primary Stressor Type	Functional Purpose	Theme Link
Head-hitting	Visual overstimulation	Escape from sensory input	Sensory Triggers
Self-biting	Overwhelming social situations	Avoidance of demand	Environmental Stressors
Screaming	Routine disruption	Expression of distress	Environmental Stressors
Hand-flapping	Social neglect	Attention-seeking	Functional Behaviour
Eye contact avoidance	Crowded environments	Social withdrawal	Sensory Triggers
Increased SIBs after conflict	Emotional climate at home	Emotional regulation deficit	Psychosocial/Parental Stress

Note. Table 6 categorizes observed self-injurious and self-stimulatory behaviors according to their associated stressor types and the functions they serve. Each behavior is functionally linked to either escape, avoidance, or communication. For instance, head-hitting in response to bright lights represents an escape behavior tied to a sensory stressor, while repetitive hand-flapping when ignored suggests an attention-seeking function in the context of unmet communication needs. This matrix offers a functional behavior perspective, supporting the idea that SIBs and SSBs serve adaptive purposes for the child, especially when verbal expression is limited. By recognizing the underlying intent behind each behavior, targeted and individualized interventions (e.g., FCT) can be more effectively implemented.

Table 7

Frequency × Setting Distribution of SIBs and SSBs

Setting	SIB Frequency	SSB Frequency	Most Common Behaviour	Likely Trigger
Home (Routine day)	Low	Moderate	Hand-flapping	Lack of structured activities
Home (Conflict Day)	High	High	Self-biting	Parental stress/emotional contagion
Clinic	Moderate	Low	Eye contact avoidance	Social demand
Family Gathering	High	High	Screaming, biting	Social overload, noise
Quiet Room	Low	Minimal	Calm, regulated	Sensory safe zone

Note. Table 7 illustrates how the frequency and type of behaviours vary across different settings, providing contextual insight into environmental influences. The highest frequency of both SIBs and SSBs occurred during family gatherings and conflict-laden days at home, suggesting that



overstimulating or emotionally charged environments act as key behavioural triggers. Conversely, structured environments like clinics or quiet rooms correspond with reduced behavioural incidents. This finding validates the role of structured and sensory-considerate environments in moderating behaviour. It also affirms that interventions should not only address individual child needs but must consider the environmental context where behaviours occur most frequently.

Table 8

Internal vs. External Stressor Classification

Internal Stressors	External Stressors
Visual hypersensitivity (bright lights)	Disrupted routines (sudden changes in daily schedule)
Auditory hypersensitivity (loud noise sensitivity)	Family conflict and emotionally charged home environments
SERT-related serotonergic dysregulation (suspected via maternal depression history)	High social density situations (large family gatherings)
FOXP2-linked language processing challenges (difficulty expressing needs verbally)	Overstimulating public environments (crowds, traffic, markets)
MAOA-related emotional regulation vulnerabilities (literature-linked to aggression/self-injury)	Parental emotional burnout and elevated stress levels

Note. Table 8 demonstrates that some stressors, particularly sensory hypersensitivities, cross the boundaries between internal predispositions and external triggers. For instance, auditory hypersensitivity stems from internal sensory-processing differences but is often activated by an external factor such as loud public noise. This underscores the *biopsychosocial interplay* between intrinsic vulnerabilities and environmental pressures.

Table 9

ABC Recording Example – Self-Biting During Family Gatherings

Antecedent	Behaviour	Consequence	Interpretation
Loud, crowded family gathering; parent engaged in conversation with others	Child bites back of hand repeatedly (10–15 seconds)	Parent stops conversation, comforts child, removes her from group	Escape from overstimulation and access to attention/comfort

Note. Table 9 shows that the behaviour served a dual function, *escape-maintained* (avoiding the overwhelming setting) and *attention-maintained* (gaining parental focus). This pattern suggests the need for Functional Communication Training (FCT) to teach alternative ways of requesting breaks.

Table 10

Intervention Mapping

Stressor	Intervention	Rationale
Sensory hypersensitivity	Sensory integration therapy; environmental adaptations	Reduce sensory overload



Disrupted routines	Visual schedules, social stories	Increase predictability
Language deficits	Speech therapy (FOXP2-informed); AAC tools	Reduce frustration-based behaviours
High social density	Gradual desensitization; pre-event preparation	Build tolerance
Caregiver burnout	Parent support programs; stress management	Improve caregiver capacity

Note. Table 10 demonstrates intervention mapping that how each identified stressor can be directly addressed through targeted, evidence-based strategies. Sensory hypersensitivity interventions focus on reducing environmental triggers and supporting sensory regulation through structured therapy and adaptations. Disrupted routines are mitigated with visual schedules and social stories, which provide a predictable framework and reduce anxiety. Language deficits, likely linked to FOXP2-related communication challenges, require speech therapy and AAC tools to provide alternative, functional means of expression and reduce frustration-driven behaviors. High social density stressors are addressed through gradual desensitization and preparatory strategies to increase the child’s tolerance for complex social environments. Finally, caregiver burnout is targeted with psychoeducation, support groups, and stress management interventions to enhance caregiver emotional capacity, which in turn positively influences the child’s behavioral regulation. This mapping not only strengthens the link between findings and intervention planning but also ensures a holistic, biopsychosocial approach.

Explanation of Themes

Sensory Triggers as Precursors of SIBs and SSBs: Behaviours such as self-hitting or covering ears were consistently linked to sensory hypersensitivities (visual, auditory), suggesting a sensory-processing-based origin of stress and behaviour regulation. Behaviours such as head-hitting when exposed to bright lights (8 instances) and avoidance of eye contact in crowded places (5 observations) reflect heightened sensory sensitivities, especially to visual and social stimuli. These reactions likely indicate sensory overstimulation, with self-injurious or avoidant behaviour functioning as an escape mechanism.

Environmental Stressors and Routine Disruptions: Unpredictable routines and noisy or chaotic environments intensified the frequency and intensity of self-injurious and self-stimulatory behaviours. Self-biting during family gatherings (4 events) and screaming when routine is disrupted (6 logs) are categorized under environmental stressors, suggesting that both social density and unpredictability in routines are key antecedents to distress. These behaviours are consistent with children who struggle to manage changes or socially intense environments, triggering maladaptive responses as a form of coping or avoidance.

Genetic and Neurobiological Vulnerability: Reports of maternal depression and family history of serotonin-linked disorders suggest a neurogenetic predisposition influencing the child's behavioural regulation. The reported maternal history of depression (no frequency, contextual data) implies a potential genetic or epigenetic influence, possibly involving neurotransmitter systems like the serotonin transporter (SERT) gene. This theme



underscores the biological susceptibility of the child, which may interact with environmental stressors to heighten behavioural responses.

Functional Role of SIBs and SSBs (Escape/Communication): The behaviours served communicative and functional purposes, often preceding undesired demands or overwhelming stimuli, indicating escape-maintained or avoidance functions. Repetitive hand-flapping when ignored (7 events) is classified under social demand avoidance and suggests a functional communication role, likely serving to gain attention. This points to the behaviour being not merely reflexive, but purposeful, emphasizing the importance of functional communication training in behaviour management.

Psychosocial and Parental Stress as Indirect Stressors: Parental stress, particularly maternal emotional burnout, appeared to correlate with increased behavioural dysregulation in the child, possibly through emotional contagion or inconsistent responses. The mother's emotional burnout, which was consistently reported, aligns with increased incidents of challenging behaviour. This highlights the bidirectional stress cycle between caregiver emotional state and the child's behavioural regulation, reinforcing the need to support caregiver mental health in interventions.

Theoretical Integration

While biological predispositions and environmental conditions were significant, the psychological domain also played a critical mediating role. Emotional regulation difficulties, limited coping strategies, and heightened threat perception in overstimulating contexts appeared to amplify behavioral responses. For example, the inability to deploy self-soothing strategies during sensory overload led to immediate escalation to SIBs or SSBs. The findings are aligned with the cognitive, behavioral perspective, which emphasizes that perception and interpretation of stressors influence behavioral outcomes. The psychological dimension acted as a mediator between biological predispositions and environmental triggers. Limited coping strategies, heightened sensory threat perception, and underdeveloped self-regulation skills increased behavioral reactivity. Without adaptive strategies, sensory overload or social stressors rapidly escalated into SIBs/SSBs. Themes were aligned with key theoretical models:

Functional behaviour assessment informed the understanding of escape-maintained vs. attention-seeking behaviours.

Sensory Integration Theory (Ayres, 1972) was used to interpret behaviours triggered by sensory hypersensitivity.

Biopsychosocial Model (Engel, 1977) underpinned the interconnectedness of genetic, environmental, and psychosocial stressors.

Discussion

A heredity susceptibility or proneness to depression may be a possible threatening element in the development of depression leading to deterioration or even destruction of genes that directly cause unacceptable behaviors among individuals with ASD. Although the biology of nervous system equal of the inclination and vulnerability remains uncertain, it appears as despite the fact that the brain serotonin (5-HT) system plays a significant conciliating and arbitrating role. Consequently, individuals with history of depression (FH+) in their families may be more likely to evolve depression due to a natural or congenital susceptibility related to altered serotonergic neurotransmission in the brain. A considerable and crucial problem, however, is that the role of



serotonin receptors also known as 5-HT receptors subsist in the central nervous system (CNS) and the peripheral nervous system (PNS) is undoubtedly complex and this group of G-protein-coupled receptors (GPCRs) viz serotonin receptors related to intrinsic jeopardy, by itself, is definitely enough to cause depression. Stress has received particular attention for the reason that stressful life events influence and pave the way for the commencement of various biological and neurological inappropriate and irregular processes such as depression, anxiety, aggression, appetite, unbinding procedures of memory, loosen cognitive patterns and unchained mechanisms of learning. Furthermore, stress causes abnormal regulation of metabolic, physiological, and psychological emotional processes prompting serotonin transporter to be occurred in bidirectional reciprocal actions between stress related changes in the neuroendocrine stress system and the 5-HT system. Healthy individuals with a family history of depression are more susceptible to evolve depression due to a genetic 5-HT vulnerability, which degenerates stress coping mechanisms and increases ASD susceptibility in their off springs. The 5-HT transporter (SERT) gene (*SLC6A4*) has been linked with whole blood 5-HT levels and ASD vulnerability. Conspicuously, SERT Ala56 mammals exhibit variations, adaptations and modifications in social functions, communication, speech and language procedures and behaving in a specified way of appropriateness. Transmuted 5-HT homeostasis certainly increases the risk for ASD traits requiring further investigation and analysis of (ASD) mechanisms and prospectively novel treatments. The brain SERT is a multiplex protein molecule existed in parasynaptic membranes of serotonergic nerve terminals. SERT is responsible for energetic and agile absorption of serotonin molecules from the synapse after serotonin has been allowed and enabled to escape from confinement following an exertion and action potential. SERT is also crucial in synchronizing the extent, immensity, duration and spatial distribution of signals in the serotonergic nervous system (SNS), (Devlin et al., 2005). Links between polymorphism of SERT genes and different neuropsychiatric disorders including ASD manifest consistent results (Makkonen et al., 2008).

The findings of this study offer compelling evidence that SIBs and SSBs in children with ASD are not isolated phenomena but are deeply intertwined with biological vulnerabilities, sensory sensitivities, environmental disruptions, and psychosocial stressors. These results align with the biopsychosocial framework (Engel, 1977), which posits that behaviours are influenced by an interaction of biological, psychological, and social dimensions.

A key biological component evident in this study is the serotonergic system, specifically the serotonin transporter (SERT) gene, which has been widely associated with emotional regulation and susceptibility to depression. The participant's maternal history of depression and reported emotional exhaustion suggest a possible hereditary or epigenetic influence on her neurobiological stress regulation mechanisms. This supports earlier research indicating that altered serotonergic neurotransmission contributes to heightened emotional reactivity and poor stress modulation in children with ASD (Lesch et al., 1993; Robertson et al., 2005; Wassink et al., 2004). The findings are further corroborated by Chaouloff (2000), who emphasized the mediating role of brain serotonin (5-HT) in emotional dysregulation, and Devlin et al. (2005), who highlighted the critical role of SERT in modulating serotonergic signalling.

The sensory hypersensitivities reported, particularly to visual and auditory stimuli, were frequently associated with the onset of SIBs and SSBs. For instance, head-hitting and avoidance of eye contact appeared in contexts of overstimulation, indicating an escape-maintained function.



These findings echo the work of Ayres (2005) and Cermak & Daunhauer (1997), who identified sensory processing abnormalities as foundational to behavioural dysregulation in children with developmental disabilities. According to Sensory Integration Theory, these behaviours act as coping strategies to manage overwhelming sensory input.

Environmental stressors, particularly routine disruptions and socially dense settings like family gatherings, also precipitated behavioural outbursts. The participant's increased dysregulation during such events is consistent with existing literature that highlights the role of environmental unpredictability in the escalation of problem behaviours (Matson et al., 2011; Schaaf et al., 2011). These findings reinforce the importance of structured, predictable environments to support behavioural regulation.

Notably, this study supports the functional behaviour assessment model, particularly in identifying the communicative functions of behaviours. Several behaviours, such as self-biting and hand-flapping, occurred in contexts of social neglect or demand placement, suggesting attention-seeking and task-avoidance functions. This aligns with the work of Carr and Durand (1985), who demonstrated how FCT can replace maladaptive behaviours with appropriate communicative alternatives.

Another critical factor influencing the child's behaviour was the emotional climate at home. Maternal burnout and emotional distress were strongly correlated with increased behavioural incidents. These findings resonate with Rattaz et al. (2013), who emphasized that caregiver well-being significantly impacts the behavioural development of children with ASD. The bidirectional influence observed in this study reinforces the importance of supporting caregiver mental health as an essential component of behaviour management.

Taken together, the study illustrates how sensory, neurobiological, environmental, and psychosocial stressors converge to shape behavioural outcomes. The behaviours documented were not random but served specific, often adaptive, functions, such as escaping overwhelming stimuli, avoiding social interaction, or seeking attention. This supports the idea that SIBs and SSBs are functional responses to internal and external stress, rather than merely symptomatic expressions of ASD (Green & Ben-Sasson, 2010; Mazefsky et al., 2012).

While the role of genes such as SERT, MAOA, and FOXP2 in emotional regulation and language development is supported in the literature, the most relevant implication for practice is that biological predispositions can heighten sensitivity to environmental triggers. For example, a child with serotonergic dysregulation may exhibit faster escalation to SIBs when routines are disrupted, underscoring the importance of early sensory and emotional regulation supports.

In Pakistan's collectivist cultural context, extended family gatherings and multi-generational households are socially valued but may inadvertently create environments with high sensory and social demands. Culturally responsive interventions could include preparing the child with visual schedules or social stories before gatherings, designating sensory-safe spaces during events, and educating extended family members about the child's triggers and needs. These strategies ensure interventions remain respectful of cultural norms while supporting behavioural regulation.

In conclusion, these findings underscore the value of using a biopsychosocial lens to interpret complex behaviours in ASD. Interventions must be individualized and holistic, taking into account the child's biological predispositions, sensory profile, environmental context, and



family dynamics. The integration of sensory-based strategies, functional communication training, and caregiver support interventions appears crucial in reducing the intensity and frequency of SIBs and SSBs. Future research should continue to explore the dynamic interplay of these factors through longitudinal and multisubject designs to enhance generalizability and intervention efficacy.

Findings of the Study

1. Sensory hypersensitivity, notably to visual and social stimuli, frequently precipitates head-hitting, avoidance behaviours, and hand-flapping, serving as escape mechanisms in overstimulating environments.
2. Environmental disruptions, such as disrupted routines or crowded, noisy settings, consistently escalate distress and behaviour dysregulation, pointing toward coping-driven behavioural responses.
3. A familial history of depression and serotonin-linked genetic predispositions e.g. involving the SERT and MAOA systems appeared to underpin biological vulnerability, which may increase sensitivity to environmental and emotional stressors.
4. Behavioural functions like attention-seeking or demand avoidance were clearly evident, with repetitive movements or self-injury occurring in contexts where the child was ignored or overwhelmed.
5. Caregiver stress, particularly maternal burnout, emerged as an indirect stressor: emotional strain within the family environment appeared to amplify both the frequency and intensity of observed behaviours.
6. Integrating these themes within a biopsychosocial framework, the study underscores how biological predispositions interact with environmental triggers and psychosocial stress to initiate and reinforce SIBs and SSBs. Importantly, the functional nature of the behaviours highlights that they serve immediate purposes, escape from demand, avoidance of overstimulation, or seeking connection, rather than being inherently involuntary.
7. The findings regarding visual and auditory hypersensitivity are based on detailed observation and parental reporting, rather than standardized sensory assessment scores. While this allowed for nuanced, situation-specific interpretation, it also limits comparability across cases. Incorporating formal sensory processing assessment tools in future work would strengthen the objectivity and reliability of such findings.

Conclusion

This qualitative case study provides critical insights into how a range of stressors contribute to the emergence and maintenance of SIBs and SSBs in children with ASD. The data reveal that these behaviours are not isolated or purely symptomatic of autism, but are closely linked to a dynamic interplay of sensory sensitivities, environmental disruptions, neurobiological predispositions, and psychosocial stressors.

The study found that sensory hypersensitivities, particularly to visual and social stimuli, play a central role in triggering behaviours such as head-hitting, avoidance of eye contact, and repetitive movements like hand-flapping. These behaviours often serve regulatory or communicative functions, providing the child with temporary relief or a means of expressing distress.



Environmental stressors, such as disruptions in routine, unfamiliar social settings, or high-demand contexts e.g., family gatherings, were frequently associated with emotional dysregulation and behavioural outbursts. These findings highlight the importance of predictability, structure, and low-stimulation environments in managing ASD-related behaviours.

Biological and genetic vulnerabilities, such as a maternal history of depression and indications of serotonin transporter (SERT) dysfunction, suggest that neurochemical and hereditary factors may increase the child's susceptibility to stress and sensory overload. This supports the growing consensus that SIBs and SSBs should be viewed through a biopsychosocial lens, which considers not only behavioral patterns but also physiological and contextual influences.

The study also highlights that caregiver emotional well-being significantly influences child behaviour. Reports of maternal burnout and emotional exhaustion were linked to increased frequency and intensity of the child's maladaptive behaviours. This emphasizes the need to address family systems and caregiver support as part of holistic intervention planning.

Moreover, the functional nature of the child's behaviours was clearly demonstrated. For example, self-biting and hand-flapping appeared in contexts where the child experienced social demand or lack of attention, suggesting that these behaviours serve as alternatives to verbal communication. Therefore, intervention strategies should aim not only to reduce the behaviours but to replace them with effective, individualized communication tools, such as FCT, Picture Exchange Communication Systems (PECS), or other augmentative and alternative communication (AAC) methods.

In summary, the findings of this study support the need for multifaceted and individualized intervention approaches that integrate sensory, behavioural, emotional, and social dimensions.

Recommendations

Based on the findings, the following recommendations are proposed

1. Schools, therapists, and caregivers should develop personalized sensory profiles for children with ASD to identify specific triggers e.g., visual, auditory, tactile. These profiles should guide the use of sensory integration strategies, such as calming spaces, noise-cancelling headphones, or visual schedules, to proactively reduce the occurrence of self-injurious and self-stimulatory behaviours.
2. Functional Communication Training should be used to teach alternative, socially appropriate communication methods e.g., gestures, picture exchange, AAC devices that replace self-injurious or self-stimulating behaviours, especially when such behaviours are maintained by attention-seeking or escape motives.
3. Psychosocial interventions, including parent counselling, stress management training, and peer support groups, should be integrated into intervention programs. Supporting caregiver emotional well-being is essential, as high parental stress levels were correlated with increased behavioural incidents in the child.
4. Develop individualized sensory profiles to identify triggers such as visual glare or high social density (linked to Theme 1 and Theme 2). Use these profiles to guide sensory integration interventions, including noise-reducing tools, dimmed lighting, and quiet break areas.



5. Implement FCT to replace escape- or attention-maintained behaviours (Theme 4) with alternative communication methods such as PECS or AAC devices.
6. Maintain predictable daily schedules and use visual aids to reduce the distress associated with environmental unpredictability (Theme 2).
7. Offer stress management programs and peer support networks to address maternal burnout and its indirect effect on behaviour (Theme 5).
8. Gradually increase tolerance to high-density social events common in Pakistani culture through controlled desensitization and pre-event preparation (Theme 2 + Cultural Context).

Limitations of the Study

1. The study's findings are not generalizable due to the single-subject design.
2. The analysis is based on parental reporting and observation, which may involve subjective bias.
3. Genetic assessment was not directly conducted but inferred through family history and literature-supported associations.

Future Research

Although sensory assessment methods allowed for rich contextual detail and ecological validity, they were inherently qualitative and subjective. Future research should incorporate validated sensory processing scales to provide standardized scoring and to triangulate with observational and caregiver-reported data. Future research should also include longitudinal multi-case studies to track changes in stressor-behaviour relationships. Comparative studies between urban and rural Pakistani contexts can also help identify how variations in social, environmental and resource-related factors influence these behaviours. Furthermore, cross-cultural comparisons are essential to see how family structure mediates stressor impact.

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