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Selective Mutism: A Biopsychosocial Perspective on Development and Treatment

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Abstract

Selective mutism (SM) is a rare childhood disorder characterized by a persistent failure to speak in certain social situations, such as school, despite the ability to speak in others, like at home. Although earlier theories explained SM through psychodynamic perspectives focused on trauma and family dynamics, current classifications in the DSM-5-TR place it within anxiety disorders. However, research suggests that SM is a complex and multidimensional condition that cannot be explained by anxiety alone. Using Engel's biopsychosocial model, the essay highlights how biological, psychological, and social factors interact in the development of SM. Biological influences include genetic vulnerabilities, auditory processing differences, temperament traits such as shyness, and familial psychopathology. Psychological mechanisms involve fear of negative evaluation, behavioral inhibition, social withdrawal, and high rates of comorbid anxiety disorders, particularly social anxiety. Social factors include overprotective or controlling parenting styles, parental anxiety, family conflict, stressful life events, and environmental transitions such as starting a new school. Several theoretical frameworks, including the "unsafe world" model, polyvagal theory, and attachment theory, help explain how perceptions of safety and emotion regulation influence speech behavior. Evidence indicates that early intervention is important, with cognitive-behavioral and behavioral therapies—often combined with family and school support—showing the strongest effectiveness in improving speech and reducing anxiety in children with SM.

Keywords

Selective mutism, unsafe world model, behavioral inhibition, social withdrawal

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Introduction

Selective mutism (SM) is a relatively rare disorder, with prevalence estimates ranging from 0.03% to 2% (American Psychiatric Association, 2022; Muris & Ollendick, 2015; Sharkey & McNicholas, 2012; Sharp et al., 2007). The syndrome was first identified by Adolf Kussmaul in 1877, ever since the etiology of the disorder has been debated (Cohan, Price, et al., 2006). Early theories came from the psychodynamic perspective assuming that the condition originates in trauma and functions as a balancing factor of the neurotic family (Hesselman, 1983, as cited in Muris & Ollendick, 2015). Such psychodynamic interpretations are now largely discounted (Muris & Ollendick, 2015), as SM is understood to arise from the interaction of multiple vulnerability factors rather than from a single deterministic cause.

According to the diagnostic criteria outlined in the Diagnostic and Statistical Manual of mental disorders DSM-5-TR (APA, 2022), selective mutism (SM) is characterized by a persistent failure to speak in specific social situations where speaking is expected (e.g., school), despite the ability to speak in other contexts (e.g., at home with close relatives). This disturbance interferes with communication and results in significant impairment in academic achievement or occupational functioning. The duration of the condition exceeds one month, excluding the first month of school. Furthermore, speech failure cannot be attributed to a primary communication disorder, such as childhood-onset fluency disorder, nor does it occur exclusively during the course of autism spectrum disorder, schizophrenia, or other psychotic disorders. Finally, the failure to speak cannot be explained by insufficient knowledge of, or lack of comfort with, the spoken language required in the relevant social context.

While SM was initially categorized under "Disorders of infancy, Childhood, or Adolescence" and "Disorders of social functioning with onset specific to childhood" in DSM-IV and International Classification of Diseases ICD-10 respectively, both DSM-5 and ICD-11 reconceptualized SM as an anxiety disorder (APA, 1998, 2013; World Health Organization, 2010, 2024). Initial classifications reflected a primarily developmental perspective with limited emphasis on anxiety, whereas the current classifications emphasize anxiety and fear as core features. This shift aligns with empirical evidence but may also risk neglecting other relevant aspects of SM, such as oppositional behavior, communication delays, and broader developmental challenges, which are not captured fully by an anxiety-focused framework (Cohan, Price, et al., 2006). Interestingly, in their meta-analysis Driessen and colleagues (2020), concluded that it is not clear whether anxiety plays a key role in SM.

Given this conceptual debate and considering the rarity of the disorder which complicates research, SM appears to be a multidimensional condition. Therefore, an integrative framework, such as Engel's (1977) biopsychosocial model is adopted, which considers the interaction of biological, psychological, and social factors in the onset and maintenance of the disorder.

Biological factors

In a candidate gene study, polymorphisms in the CNTNAP2 gene, a gene that is linked to autism spectrum disorders and language dysfunction, have been associated with a heightened vulnerability to SM and social anxiety traits (Stein et al., 2011). Highlighting, shared genetic influences among SM, related neurodevelopmental conditions and anxiety. For instance, a child with CNTNAP2 polymorphism may inherit a predisposition toward anxiety, making them more sensitive to social evaluation, which can make them reluctant to speak in social situations.

Another interesting finding is the deficiencies observed in the auditory efferent activity (AEA) of selectively mute children, present during vocalization (Arie et al., 2007). These atypical patterns of efferent activity occur despite normal results on pure-tone and speech audiometry, as well as typical brainstem function evidenced by standard auditory brainstem response latencies (Bar-Haim et al., 2004). These auditory processing challenges may heighten anxiety during verbal communication, reinforcing mutism in social contexts such as classrooms.

Other biological factors include familiarity with shyness and preterm birth (Capobianco & Cerniglia, 2018). Notably, a large study in Finland found that parental psychopathology was significantly associated with an increased vulnerability of SM in offspring and that the siblings of children with SM have more neurodevelopmental disorders than control groups (Koskela et al., 2024). Moreover, another family study comparing families having children with SM and families with GAD, found similar associations between these groups, indicating similarities in GAD's and SM's etiologies (Capozzi et al., 2018). Although high rates of disorders within a family are frequently interpreted as indicators of a strong genetic influence, shared environmental factors may also further explain this pattern.

Social factors

Numerous studies examining the family environment in SM have reported elevated rates of marital conflict (Elizur & Perednik, 2003), overprotective (Capobianco & Cerniglia, 2018; Remschmidt et al., 2001), or controlling parenting styles (Edison et al., 2011; Koskela et al., 2020). Notably, parents of children with SM have also been described as frequently shy, anxious, and avoidant, and as exhibiting the personality trait of taciturnity (a habitual tendency toward silence), suggesting broader familial patterns of social inhibition (Capozzi et al., 2018). For example, a child with a naturally anxious temperament facing overprotective or controlling parenting may become increasingly inhibited, as attempts to explore or speak are consistently met with parental restriction or excessive oversight. Additionally, informed by Bandura's (1977) observational learning theory a child might model parental personality exhibitions of shyness, taciturnity and avoidance, hindering its ability to engage in conversations.

Furthermore, single-mother households and lower socioeconomic status (Koskela et al., 2020), as well as exposure to stressful life events or significant childhood trauma (Capozzi et al., 2018; Cunningham et al., 2004; Steinhausen & Juzi, 1996), have been shown to increase the likelihood of SM in offspring. In addition, major environmental transitions, such as relocating to a new area or starting a new school, may precipitate the onset

of the disorder (Koskela et al., 2020). For instance, a child with insecure attachment in a new environment like kindergarden may perceive teacher feedback as threatening, amplifying fear and reinforcing the avoidance of verbal participation.

Interestingly, atypical regulation of physical distance has also been identified as a relevant factor, with children with SM showing a preference for unusually close interpersonal distance, particularly toward caregivers (Schwenck et al., 2022). When considered alongside findings of elevated rates of psychotic, mood, and personality disorders among parents of children with SM (Koskela et al., 2020), and with above mentioned parent's personality characteristics, this may suggest family environments characterized by heightened dependency or emotional dysregulation. From an attachment perspective, such contexts may compromise the development of secure autonomy, increasing vulnerability to anxiety and withdrawal in socially demanding situations, including those requiring speech.

Psychological factors

Conceptualizing SM as an anxiety disorder implies that heightened fear and anxiety are central psychological mechanisms, with fear reflecting an immediate emotional response to actual or perceived threat, and anxiety representing the anticipatory response to potential future threat (APA, 2022). In their analysis of the thoughts and emotions underlying SM, Capobianco and Cerniglia (2018) found that children experience fear of judgment, failure, and mockery, as well as shame, depression, catastrophizing tendencies, and feelings of worthlessness or inadequacy, linking SM to internalizing psychopathology. Supporting this view, children with SM frequently display behavioral inhibition, social withdrawal, and isolation (Capozzi et al., 2018), and some studies report shyness, which can be seen as the social manifestation of behavioral inhibition (Muris & Ollendick, 2015).

Comorbidities are also common in SM, with meta-analytic evidence suggesting that as many as 80% of children with SM meet criteria for at least one additional anxiety disorder, most frequently social phobia (69%) (Driessen et al., 2020). Beyond internalizing symptoms, certain children also exhibit temper tantrums and mild oppositional behavior (Cohan, Chavira, et al., 2006; Diliberto & Kearney, 2016), indicating that SM may involve a broader spectrum of emotional and behavioral difficulties.

The interaction of biological, psychological and social factors

It seems that multiple biological, psychological, and social issues are interwoven in various ways in the life of the patient and are collectively responsible for the development of SM. Biological predispositions, such as genetic vulnerabilities, auditory processing deficits, and familial psychopathology, may lay the foundation for the emergence of the disorder and increase sensitivity to psychological and social stressors. Psychological processes, including fear of judgment, anxiety, behavioral inhibition, and internalizing symptoms, interact with these biological predispositions to reinforce avoidance behaviors and social withdrawal. For example, an inhibited child having a parent with social anxiety may experience heightened anticipatory anxiety during school activities that include verbal communication. This interacts with their biological sensitivity to stress and reinforces SM.

Social and familial factors, such as parenting style, parental shyness, anxiety, and taciturnity, as well as family conflict, trauma, and major life transitions, can further amplify psychological and biological vulnerabilities and shape the child's responses to social situations. From an attachment perspective, these interacting factors may compromise the development of secure autonomy, creating a negative loop between family context and child behavior and increasing vulnerability to social anxiety and withdrawal, ultimately contributing to the onset and maintenance of SM.

Theoretical framework

According to the "unsafe world" model (Melfsen et al., 2021), SM is conceptualized as a stress reaction to situations that are unconsciously misperceived as "unsafe." The model proposes a low tolerance for perceived unsafety, leading to the early activation of dissociative or freeze responses. In line with this view, Cohan, Price, and colleagues (2006) suggest that elevated anxiety levels in SM heighten sensitivity to verbal interactions. Based on these conceptualizations, some researchers argue that SM may not represent a distinct disorder, but rather an extreme or context-specific manifestation of broader anxiety pathology (Bar-Haim et al., 2004).

Nevertheless, the polyvagar theory (Porges, 2011) provides a useful framework for better understanding the mutacic behavior. This theory conceptualizes emotion regulation through the organization of the two efferent branches of the vagus nerve. The vagal system operates in functional opposition to the sympathetic–adrenal system and connects distinct physiological states to specific stress-response strategies, including fight, flight, and freeze behaviors, as well as to adaptive social engagement behaviors. The theory explains how autonomic nervous subsystems correspond to three broad behavioral domains: social communication, mobilization, and immobilization. It proposes that social engagement, including speech, can occur only when an individual experiences sufficient physiological safety.

Regarding safety, another theoretical framework used to understand SM is attachment theory (Bowlby, 1979). Attachment theory posits that the quality of the relationship between a child and their primary caregiver, through the formation of a secure base, plays a central role in the child's perception of safety and capacity for emotion regulation. Research suggests that children with SM are more likely to display insecure attachment patterns compared to typically developing peers, although secure attachment is not absent in all cases (Capobianco & Cerniglia, 2018). Informed by these frameworks, and consistent with cognitive-behavioral principles, treatment of SM primarily targets cognitive and behavioral processes through multimodal interventions and parallel efforts to change environmental dynamics (family and school) that maintain the disorder (Capobianco & Cerniglia, 2018).

Treatment

Researchers agree that an early diagnosis followed by an early treatment yield best outcomes (Capobianco & Cerniglia, 2018; Hipolito et al., 2023; Muris & Ollendick, 2015; Oerbeck et al., 2015). Moreover, the severity of SM and underlying biological and environmental factors influence the extent to which symptoms can improve (Capobianco & Cerniglia, 2018).

Cognitive-behavioral and behavioral interventions are the most systematically supported treatments for SM (Capobianco & Cerniglia, 2018; Cohan, Chavira, et al., 2006; Hipolito et al., 2023; Iimura et al., 2025; Muris & Ollendick, 2015). CBT aims to change maladaptive cognitive biases and emotional responses, while behavioral components target avoidance and reinforce speech (Iimura et al., 2025). Furthermore, Capobianco and Cerniglia (2018) advocate that in order to prevent the maintenance of the symptoms parallel interventions targeting the family and school systems are necessary. For instance, teachers may learn to give neutral prompts and reinforce verbal engagement, while parents could be encouraged to engage with and not overcontrol speech attempts. In addition integrated behavioral therapy (IBT) shows promising results (Muris & Ollendick, 2015).

In a one-year follow-up study, Oerbeck and colleagues (2015) reported that 66.7% of children who received cognitive-behavioral therapy (CBT) demonstrated substantial improvement or no longer met diagnostic criteria for SM, with more favorable outcomes observed among younger children. Consistent with these findings, a recent systematic review and meta-analysis documented promising rates of symptom remission and overall improvements in speech behavior among children aged 3–9 years who received combined systems-based behavioral interventions (Hipolito et al., 2023). Furthermore, a systematic review by Østergaard (2018) found that symptom improvement was observed in 53 out of 60 children treated with CBT, compared to 55 out of 67 children receiving pharmacological interventions and 6 out of 7 children receiving combined treatment approaches.

Although selective serotonin reuptake inhibitors (SSRIs) have demonstrated efficacy, particularly in cases where SM co-occurs with anxiety disorders (Muris & Ollendick, 2015), pharmacological treatment is generally not considered a first-line intervention, especially for younger children. This is largely due to parental concerns about potential side effects, which reduce the acceptability of medication-based approaches (Østergaard, 2018). In contrast, non-pharmacological interventions, such as behavioral and cognitive-behavioral treatments, are typically viewed by parents as more acceptable and appropriate for young children with SM (Hipolito et al., 2023).

On the other hand, a long-term follow-up study indicate that 42–61% of individuals continue to exhibit SM symptoms into adulthood, even after receiving treatment during childhood (average age 8.5 years) (Remschmidt et al., 2001). This highlights the critical importance of early and effective intervention, while also suggesting that SM may be persistent for some individuals, potentially following a chronic course and requiring ongoing support to manage symptoms over time.

Thus, although psychodynamic approaches have received comparatively little attention in the treatment of SM, they may serve as a useful complementary intervention. Fernandez and Sugay (2016) illustrated in a clinical case study that psychodynamic play therapy helped children process emotions and achieve gradual behavioral improvements without being pressured to speak. These methods may be particularly beneficial in cases where emotional or relational factors are central to the child's mutism.

Conclusion

Taken together, SM emerges from biological predispositions amplified by psychological factors and social or environmental influences. Genetic vulnerabilities, neurodevelopmental factors and familial psychopathology interact with fear, anxiety and behavioral inhibitions, while family dynamics and attachment patterns mediate the child's responses to verbal interactions. Evidence based interventions are framed through psychological theories including cognitive-behavioral, polyvagar, attachment and psychodynamic perspectives. Cognitive-behavioral and behavioral therapies, particularly when combined with systemic family and school interventions, show the strongest empirical support, while pharmacological treatments remain adjunctive. Finally, early identification and intervention are deemed critical for optimal results, highlighting the need for an integrated, theory informed approach that addresses the multifaceted nature of SM and tailors' treatment to each child's biological, psychological and social needs.

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